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Vol. 16No. 1 & 2 Feb. – Aug., 2013

EDITORIAL

REVIEW ARTICLES:

ORIGINAL ARTICLES

CASE REPORTS:

EDITORIAL

PERSONALIZED MEDICINE IN PSYCHIATRY

Kumar Anil - Shukla S. R. P.

In medicine knowledge of the disease and an understanding of its mechanisms are required. In Psychiatry we, are dealing with individual human being as a whole the uniqueness of each person makes him / her as an individual in his / her own SELF therefore the course of the disease in response to treatment vary from one subject to another due to differential in; genetic, enzymatic and metabolic factors (Macher Jean Paul 2009). Psychiatry deals with mind which is not an anatomical organ but a treasured perception of an individual where in his / her individuality lies. Precisely, for this reason mental health professionals come across ample opportunities for conscious as well as conscientious learning to understand the inner and the out core of each and every unique psyche (Patkar S V 2013) hence, psychiatry calls of personalized medicines which vary during the course of client's fluctuations in milieu exteriore, passages of life and evolution of defense mechanisms to cope with psychic repercussions to deal with distressed mind i.e. no target organ, it calls for holistic human concern.

In Psychiatry we see a plethora of symptoms manifested as behavioural anomalies, emotional aberration(s), and detoriation in already possessed personal capabilities or lack of maturation congruent with the passages of life on case to case basis. As a consequence of this phenomenon, each case needs to be taken up as a project, unique in itself, which necessitates personalized medicine in psychiatry. This calls for in depth understanding of the pharmacokinetics of each psychotropic agent we use, along with the pharmacogenetics of each subject (patient) which depends upon race, caste, creed, life style, food habits as well as genomorphics of the subject in hand. Haven't we seen patients who fail to respond to tricyclic agents, showing brilliant recovery with tetracyclics or showing brilliant response to piperazine, not with quinolinone derivatives, this bespeaks of psychotropic species impacting differentially on case to case basis. We have seen many cases of classical Manic Depressive Psychosis not adequately stabilized with adequate Lithium; Lamitrogine, but stabilized with Quietiapin; as also classical Schizophrenics with hallucinations, delusions, paranoid features not responding to Haloperidol, Resperidone, but brilliantly responding to Aripiprazole, iloperidone,Mirtazapine combination, these happening ought to make us aware that “personalized medicine is customized behavioural approach to management of health condition which calls for genetic understanding for difference in psycho pharmacotherapy i.e. pharmacogenomics (Downing Gregory J 2009)”. This enables us to optimized individual factors to optimize or prevent psychiatric diseases. For personalized medication we need to know the Pharmacodynamics of the psychotropic agent we use, as well as the remediation of their effluvial effects.

Personalized medicine in psychiatry e.g. in the form of tailored psychotropic agents has already proved its efficacy, notably in terms of adjusted optimum doses and predictable drug responses or drug induced side effects (Evers Kathinka 2009). In order to understand the pharmacogenetics we need to understand as to how the psychiatric disorders operate at molecular level which entails tailoring choice as well as doses of the mind influencing agents we all use, which happens with psychotherapeutic bonding.

Psychotropic agents remain the cornerstone in treatment of psychiatric disorders. However, more than 20% of patients do not initially respond to treatment of drug therapy (Essali A, Al Haj, HN et al 2002). In addition to lack of response, many patients discontinue their medication due to side effects, which can have serious and devastating consequences (Nose M et al 2003). The pharmacodynamics of drug metabolism depends upon Pharmacogenomics which calls for personalized psychiatric medication (de Leon J. 2009) obtained from holistic intervention. Quality and Quantity of life depends upon junk gene expressing as white matter in brain tissue for which proper understanding of medication for expectations from a Psychiatrist is natural, as also possible, provided we amalgamate genetic adiposity versus life style adiposity, the former manifesting as HDL and latter LDL phenomenon which is in our domain of psychotropic intervention. Similarly, the electrical disturbance in heart are nerve conduction aberration phenomenon and need to be managed by us thereby preventing implantation of pace makers, as also is the case with essential hypertension managed at an early age for preventing Coronary Ischemic Disease and other systemic damage. Drugs affecting the hippocampus enhance grey matter. “The ultimate remedy lies in the development of social phenomenology infrastructure to handle their distribution, consolidation and prevention en-route personalized psychiatric medication, Cuijpers P 2009 has reviewed and found a growing number of randomized controlled trials

have shown that it is possible at least in some cases to actually prevent or at lease delay the onset of mental disorder, including depressive and anxiety disorders and some studies indicate that it may be possible to prevent the onset of psychotic disorders in high risk group for which selecting target group by using indices other than odds ratios, relative risks or incidence rate ratios alone is insufficient". For studying the cumulative effect of joint exposures to several risk indicators rather than the effect of a single risk indicator for which shared decision making in mental health management is a necessity. Shared decision making, consists of a Psychiatrist with consultation liaison experience, a Clinical Psychologist, the patient himself and one of the care givers / family member (Drake R. E. et al 2009), would assure personalized Psychiatric intervention impacting the Quality Of Life and enabling harnessing of full potential of an individual for meeting his realistic aspirations in life, which would not be otherwise possible.

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IN THIS ISSUE

Shukla SRP & Kumar Anil

This issue deals with the organic aspects of psychotropic agents as well as dilates the psycho-social ramifications of the dialogue between mental health professionals and patients. In this issue the main focus is pediatric psychiatry and individualized patient care.

REVIEW ARTICLES

- Singh G. P. Loona N. and Singla R. K. provide us with an update on "Aripiprazole, a new generation antipsychotic: current research and clinical practice".

  The coverage is a comprehensive review highlighting the uniqueness of this molecule as regards it being both an agonist of D2 and 5HT2A receptors as also an antagonist at 5 HT2A thus an antipsychotic of use in acting today while preparing for preventing relapse tomorrow, consequent upon neuronogenesis.

- Second review deals with endocrinological repercussions of psychotropic agents under the caption "Antipsychotic Induced Hyperprolactinaemia by Zainab L. D. and De.Souza A.

This paper highlights that atypical antipsychotics often cause rise in prolactin concentration up to 10 times or above the normal levels, culminating in amenorrhoea, perceived as suspected pregnancy, when it is not so, as well as breakthrough bleeding in females. Psychiatrists, using non-classical antipsychotics need to be aware in this regards; refer to a gynaecologist for management of hyperprolactinaemia while apprising him/her in this regards, as well as switching over to antipsychotics with lesser propensity to cause hyperprolactinaemia or choosing an appropriate antipsychotic right in the beginning

- The third review by Mohapatra S and Rath NM. Captioned "Treatment of Depression in Pregnancy" Current trends deals with reproductive systems correlates with depressive disorders.

Pregnancy causes or precipitates psycho-social aberrations consequent upon endocrinological disequilibrium, hence often need's hypothalamo – hypophysiological axis intervention, which we do zealously, but inadvertently forget to ask about pregnancy status, mostly in case of young primigravida and prescribe medication which may cross the placental barrier and thus may impact the foetus. Hence, enquiry about pregnancy is vital which entails taking history about menstrual status. After, all we also see patients with psychiatric disorders at menarche or menopause, after hysterectomy or may be cervical erosion.

- The fourth review article captioned "Expressed Emotion in Psychiatric Disorders – A Review by Mohapatra S and Rath NM. details the dialogue between patient and mental health professionals, highlighting the family's attitude towards the patient as a function of major psychiatric disorder, a pointer towards family being the aetiological agent.

- The last but potent review article is captioned " post traumatic stress disorder : An Illness of recovery by Sengar K. S. & Singh Archna. This article encompasses an update review of PTSD found in 70% of general population a sort of endemic in masses perhaps, due to hazardous life and nature calamities; hence gains significance for mental health specialists to acquire ability to the carefully intervene, more so because PTSD is fully recoverable. therefore this review needs to be carefully read by all of us.

ORIGINAL ARTICLES

- The first original article "Rorschach culture and popular responses by Shweta, Sengar K. S., Bajpai R.C. and Singh A.R. where it has been significant ly demonstrated that the responses to projected test are culture bound, hence need interpretation by culture aware clinical experts.

- The second being "Executive Function Deficits in Patients with Schizophrenia by Kumar Neelam and Prakash Jai. This research's originality lies in finding the information processing faults in Schizophrenia which impact activities of daily life.

- Next comes "Memory Dysfunctions in the cases with Schizophrenia by Bhengra Hitakar Pushpa and Prakash Jai which enables us to information processes, storage and retrieval deficits in brain mal functioning in Schizophrenia Patients.
- The fourth original article is by Mohapatra S and Agarwal V "Anxiety Disorders in Children and Adolescents", posseses originality in apprising us as regards the phenomenology of childhood anxiety and providing an understanding of prevention of childhood anxiety. A pediatric psychiatry perspective.
- Fifth one being a research by Anjali Kumari, Prakash Jai and Kiran Manisha captioned "Parenting Stress in Parents with mentally Retarded children" the beauty lies in deriving from this paper that parents when stressed due to utopian expectations from their children indulge in defensive response and dysfunctional interaction with their child who cannot meet their expectations for he/she is not capable of, due to aptitude paucity.
- The next original article is "Quality of life in the caregivers of Schizophrenic patients" by Mandal A; Ali MS, Mohta J. and Prakash Jai highlights the quality of life which entails satisfaction of an individual's values, goals and needs through the actualization of their abilities. In the care givers of Schizophremic patients their quality of life is jeopardized, interestingly more in patients who have been abandoned into an hospital and less in patients kept at home, this bespeaks of the fact that the care givers of Schizophrenic patients are better off with outpatient treatment of Schizophrenics and giving care at home.
- At the end of original article senses is "Psychiatric Morbidity among Women Engaged In Commercial Sex Work in Kerala" by Mathew Jins and Sengar K. S highlights the here of sex works and it's natural psychiatric consequences originality, lying in this being a fist systematic study of this population.

CASE REPORTS

- Mohapatra S. and Rath N.M. report a case captioned "Pediatric Autoimmune Neuro Psychiatric Disorders with Streptococcus infection. This case report initiates our thought process on an organic basis of Obsessive Compulsive Disease.
- De-Sousa A gives us a case study on sponge eating PICA. Usually, PICA is understood as on associative phenomenon with iron deficiency anemia consisting of eating non-nutritive substances for at least one month or more culminating in appropriate development of the children in breast feeding mothers. De-Sousa A. gives a case of eating sponge in a three and half years old child which is rather unusual and thus needs differential diagnosis between iron deficiency and other aetiopathology of PICA.

  The crux of our case reports is that they commission our creativity of persosnalized psychiatric practice.

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Review Articles :

"Aripiprazole, a new generation antipsychotic: current research and clinical practice

Singh Gurvinder Pal, Loona Neeraj, Singla Rajinder Kumar

ABSTRACT

Aripiprazole is the new generation class of antipsychotic. Chemically, aripiprazole is a quinolinone derivative. Aripiprazole gives its action through the partial agonist mechanism at dopamine D_2 and 5- HT_{1A} receptors, and is also an antagonist at 5-HT_{2A} receptors. Preclinical and placebo controlled trials of aripiprazole have evaluated that aripiprazole is effective in the treatment of schizophrenia. Comparative trials of aripiprazole with typical and atypical antipsychotics have shown that aripiprazole has similar efficacy as haloperidol and olanzapine. Data also suggest that aripiprazole is safe and its tolerability profile is good. This molecule is also showing promising results in the treatment of bipolar disorders but the evidence is still inadequate. This article critically examines the various clinical trials conducted in various countries regarding the therapeutic profile of aripiprazole.

INTRODUCTION

Aripiprazole is the first next generation atypical antipsychotic with a mechanism of action that differs from conventional and atypical antipsychotics. Aripiprazole belongs to a new class of antipsychotics, called dopamine system stabilizers. Dopamine system stabilizer reduce the hyperactivity of dopamine neurons that mediate psychosis and at same time restore dopamine activity in the cortical regions that mediate negative and cognitive symptoms as well as the brain area that regulate motor movements and prolactin. [1] Collectively, aripiprazole is an important new atypical antipsychotic candidate with a favourable safety profile. Aripiprazole was approved by the Food and Drug Administration on November 15, 2002 for the treatment of schizophrenia. This is also approved for the treatment of acute manic and mixed episodes associated with bipolar disorders.

PHARMACOLOGY

Aripiprazole is a quinolinone derivative. Its Chemical name is 7-[4-{4-(2, 3-dichlorophenyl)-1-piperazinyl}butoxy]-3, 4-dihydrocarbostyril. The empirical formula is $C_{23}H_{27}C_{12}N_3O_2$ and its molecular weight is 448.38. Aripiprazole acts as a dopamine system stabilizer, partial agonist at the D_2 and 5-HT_{1A} receptors and is an antagonist at $5HT_{2A}$ receptors. [2-5] It acts like a D_2 antagonist during hyper-dopaminergic activity and D_2 agonist during hypo-dopaminergic activity. Moreover, aripiprazole preserves normal dopamine activity at nigrostriatal and tuberoinfundibular dopaminergic pathways, thereby causing minimal EPS side effects and hyperprolactinemia. [6] In addition, aripiprazole's partial agonist effect at $5HT_{1A}$ receptors and its antagonist activity at $5HT_{2A}$ receptors may improve negative symptoms and cognitive function in schizophrenia.[1,7]

Aripiprazole is well absorbed with peak plasma concentration occurring within 3 to 5 hours. The oral bioavailability is 87%. The mean elimination half-life is about 75 hours for aripiprazole and 94 hours for its active metabolite (dehydro-aripiprazole). The parent drug represents a greater proportion of the drug exposure (60%) in plasma than its active metabolite, dehydro-aripiprazole. At doses of 5-30 mg/day, aripiprazole shows linear and dose proportional pharmacokinetics.[8] Aripiprazole and dehydro-aripiprazole are more than 99% bound to serum proteins (mainly albumin) at therapeutic concentration.[9] Clinical drug-drug interaction studies have not shown an effect of aripiprazole at dose of 10-30 mg/day on the pharmacokinetics of cytochrome isoenzymes CYP3A4, CYP2D6, CYP2C9, CYP1A2, and CYP2C19 substrates.[10] Drugs that induce CYP3A4 could cause an increase in aripiprazole clearance and lower plasma levels. Inhibitors of CYP3A4 (e.g., ketaconazole) or CYP2D6 (e.g., quinidine, fluoxetine or paroxetine) can inhibit aripiprazole elimination.

PRE-CLINICAL DEVELOPMENT

The discovery and characterization of dopamine in the mammalian brain earned Dr. Arvid Carlsson the Nobel Prize in 2000. Along with his many insights about dopamine pharmacology, came his proposal of the existence and critical role of dopamine autoreceptors in the overall regulation of dopamine-mediated neurotransmission. [11] Aripiprazole doesn't cause an upregulation of D_2 receptors or an increase in expression of the c-fos mRNA in the striatum. The mechanism of action of aripiprazole differentiates it from both typical and atypical antipsychotics and hence, may provide important leads for pharmacotherapy of schizophrenia and other psychotic disorders. [12] Aripiprazole displays properties of an agonist and antagonist in animal models. In a study conducted by Burris et al [2] which examined the interactions of aripiprazole with a single population of human D_2 receptors In membranes prepared from Chinese hamster ovary cells that express recombinant dopaminergic receptors, aripiprazole bound with high affinity to both the G-protein-coupled and uncoupled states of receptors. Results of this study supported the identification of aripiprazole as a dopamine-serotonin system stabilizer.

CLINICAL DEVELOPMENT

i) COMPARISON WITH TYPICAL ANTIPSYCHOTIC

A number of clinical trials were conducted to compare the efficacy of aripiprazole with other typical antipsychotics. The results were comparable on various efficacy parameters. Both short-term and long-term clinical trials have provided evidence that aripiprazole is effective in the treatment of schizophrenia. In a short term trial conducted by Kane et al,[13] aripiprazole and haloperidol produced significant improvement on efficacy parameters. Aripiprazole was found to be safe and effective for positive and negative symptoms of schizophrenia. In patients with treatment resistant schizophrenia, Kane et al [7] conducted another clinical trial comparing aripiprazole with perphenazine. Patients were randomly assigned to aripiprazole (15-30mg/day) or perphenazine (8-64mg/day). In results, 27% of aripiprazole treated patients and 25% of perphenazine treated patients were responder after 6week. Perphenazine treated patients had a higher incidence of extrapyramidal syndrome and a higher rate of elevated prolactin levels than aripiprazole (57.7% vs. 4.4%). The quality of life score revealed better results in patients treated with aripiprazole. Mcquade et al [14] conducted a long term comparative study of aripiprazole (20-30mg/day) with haloperidol (7-10mg/day). Higher number of patients on aripiprazole completed study as compared to haloperidol (40% vs. 27%). Aripiprazole was found to be significantly more effective than haloperidol on negative (PANSS) and depressive symptoms (MADRS) in patients of schizophrenia.

ii) COMPARISON WITH ATYPICAL ANTIPSYCHOTICS

A number of clinical trials were conducted to compare the efficacy of aripiprazole with other atypical antipsychotics. Chrzanowski et al [15] compared the long-term efficacy and safety of aripiprazole with olanzapine in patients with either acute relapsing or chronic, stable schizophrenia. Efficacy improvements were similar between aripiprazole and Olanzapine group. Tandon and jibson [16] compared efficacy of first line atypical antipsychotics (risperidone, olanzapine, quetiapine, ziprasidone and aripiprazole) in the treatment of schizophrenia or schizoaffective disorder. This study showed that all the first line atypical antipsychotics were similarly effective for overall psychotic symptoms and positive & negative symptoms of schizophrenia. In a long-term double-blind study of olanzapine with aripiprazole in schizophrenia, treatment groups didn't differ significantly in time to all-cause discontinuation rate (olanzapine, 42.7% vs. aripiprazole, 50.2%). Olanzapine treated patients had significantly longer time to efficacy-related discontinuation and a significantly lower efficacy-related discontinuation rate (olanzapine, 8.9%, vs. aripiprazole, 16.8%). Olanzapine treated patients had a significantly greater mean decrease in PANSS total score than did aripiprazole treated patients. [17] Kinon BJ et al [18] conducted a 5-day, randomized, double-blind trial of olanzapine versus aripiprazole for the treatment of agitation in acutely ill patients with schizophrenia. Significant improvements from baseline in PANSS-EC and secondary efficacy measures were seen for both olanzapine and aripiprazole. Taylor et al [19] conducted an open label, 26-week, multi-centre, randomized study comparing aripiprazole with olanzapine, quetiapine or risperidone in patients with schizophrenia. The authors reported that more respondents rated the aripiprazole medication as 'much better' compared with their previous medication (Olanzapine, quetiapine or risperidone).

iii) SWITCH STUDIES

A number of switch studies were also conducted to prescribe and shift the patients from multiple doses to monotherapy with aripiprazole. Casey et al [20] conducted a

short-term open-label study to investigate the efficacy, safety and tolerability of three dosing strategies for switching chronic, stable patients with schizophrenia from current oral antipsychotic monotherapy to once-daily oral aripiprazole. In this trial, the efficacy with aripiprazole was revealed with numerical improvements compared with baseline in all treatment groups. In another similar clinical trial, Medori et al [21] documented findings in 311 patients with chronic, stable schizophrenia or schizoaffective disorder who had received monotherapy with a typical (haloperidol or thioridazine) or atypical (risperidone or olanzapine) antipsychotic for 1 month. Patients were shifted to aripiprazole monotherapy. The efficacy results were similar across treatment groups. There were no differences in discontinuations due to adverse events across the treatment groups. Antipsychotic efficacy was maintained in all groups throughout the study and improvement was seen from baseline in PANSS total score, PANSS negative and PANSS positive subscale scores, and CGI-Improvement score. Decreases in EPS rating scores, weight, and serum prolactin levels were also associated with switching to aripiprazole.

iv) SAFETY AND TOLERABILITY

Majority of the clinical trials conducted across globe revealed that aripiprazole is safe and its tolerability profile is documented in many studies. Mcquard et al [22] conducted a study comparing weight change during treatment with olanzapine or aripiprazole in randomized double blind controlled trial. In this clinical trial, at the end of twenty six weeks, 37% of olanzapine treated patients have experienced significant weight gain compared with 14% of aripiprazole treated patients. In another trial, the authors reported that olanzapine treated patients had significant greater mean increases in weight and glucose and a significantly greater worsening on lipid parameters in comparison to aripiprazole. [17] Olanzapine treated patients reported more extrapyramidal symptoms (EPS)-related adverse events (18%) than aripiprazole-treated patients (10%). Changes in fasting glucose and lipid levels at endpoint favoured aripiprazole over olanzapine, with significant differences observed for total cholesterol, low and high density lipoproteins. [15]

In one randomized comparative study of olanzapine and aripiprazole, safety of aripirazole and olanzapine was evaluated. Mean changes from baseline in non-HDL-C levels were significantly different with olanzapine versus aripiprazole at weeks 26 and 52. it was concluded that long term aripiprazole treatment is associated with improvements in lipid profiles of schizophrenia patients versus no improvement or worsening during olanzapine treatment. [23] Newcomer et al [24] compared the metabolic effects of aripiprazole versus olanzapine in overweight persons with schizophrenia or schizoaffective disorder who were previously on olanzapine treatment. In total, 173 subjects were randomly assigned to receive aripiprazole or olanzapine for 16 weeks. At week 16, weight decreased significantly with aripiprazole versus olanzapine (-1.8 vs. +1.41 Kg). Significant differences in percentage change in triglyceride levels were observed with aripiprazole versus olanzapine at all time-points. In addition, significantly more subjects receiving aripiprazole had clinically relevant weight loss versus olanzapine (11.1 % vs. 2.6%), and a lower percentage of subjects receiving aripiprazole had clinically relevant weight gain (2.5% vs. 9.1%). This study concluded that a significant improvement in weights and lipids was observed during discontinuation of olanzapine and switch to aripiprazole treatment.

Sedation, weight gain, and metabolic syndrome may be lass problematic with aripiprazole than with some other second-generation antipsychotics. Akathisia may limit its utility in some patients. Perhaps because of partial agonist effects at dopamine receptors, nausea and vomiting can occur when aripiprazole is started. Tolerability may be enhanced in patients if aripiprazole is initiated at 15mg or lower doses for a few days before being increased to as much as 30mg per day. Somnolence and constipation may be encountered with aripiprazole.

ARIPIPRAZOLE IN BIPOLAR DISORDERS

A number of multicenter, double-blind, placebo controlled trials have established the efficacy of aripiprazole monotherapy in acute mania (Keck et al, 2003; Sachs et al 2006). [25, 26] In the clinical trial conducted by Keck et al, [25], patients in an acute manic or mixed episode were randomly assigned 30mg/day or placebo. Aripiprazole produced statistically significant mean improvements in total score on the Young Mania Rating Scale compared with the placebo (-8.2 versus -3.4, respectively) and produced a significantly higher response rate (40% versus 19%). The completion rate was significantly higher with aripiprazole than with placebo (42% versus 21%). Aripiprazole monotherapy appeared to have a broad spectrum of efficacy. Sachs et al. [26] conducted a 3-week placebo-controlled aripiprazole study in the treatment of acute manic or mixed episodes in patients with bipolar 1 disorder. Aripiprazole-treated patients demonstrated significantly greater improvement from baseline to endpoint in mean YMRS total scores compared with placebo-treated patients as early as Day 4

and sustained through week 3. A significantly higher response rate was observed in aripiprazole-treated patients (53% vs. 32% at endpoint). Aripiprazole produced significantly greater improvement from baseline on other efficacy assessments compared with placebo, including Clinical Global Impression-Bipolar Version Severity and Improvement scores.

CONCLUSION

Aripiprazole is the most balanced drugs with dopamine-serotonin stabilizing properties. Aripiprazole is equally effective in the treatment of schizophrenia as compare to typical and atypical antipsychotics. In majority of clinical trials aripiprazole has been compared with olanzapine. Olanzapine has similar efficacy as aripiprazole but more weight gain and deranged lipid profile is reported from olanzapine receipients. Aripiprazole has the low potential for the drug drug interactions. Aripiprazole has the better tolerability profile as compared to typical antipsychotics. Thus, aripiprazole is the favourable option with improved efficacy and tolerability profile in the patients with schizophrenia.

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Anti Psychotic Induced hyperprolactinaemia

Dawoodi Zainab Lokhandwala, De Sousa Avinash

Abstract

Hyperprolactinaemia has for decades been an inevitable and neglected side-effect of antipsychotic medication. The recent introduction of prolactin-sparing antipsychotic agents makes a re-examination of this problem timely. This article aims to review the literature on antipsychotic induced hyperprolactinaemia and its consequences. A literature search was made for key articles, supplemented by cross referencing. During antipsychotic treatment prolactin concentrations can rise to ten times normal levels or above and existing data indicate that a large number of female patients have amenorrhoea with or without galactorrhoea. Survey data, however, suggest that clinicians underestimate the prevalence of these conditions. Antipsychotic-induced hyperprolactinaemia should become a focus of interest in the drug treatment of psychiatric patients.

Key words : ***Antipsychotics, hyperprolactinemia, prolactin.***

Introduction

Prolactin is a polypeptide hormone that exists as a number of isoforms and is involved in a number of physiological processes. Hyperprolactinemia is elevation of prolactin above the norm and can occur due to various causes including side effect of conventional and some second generation antipsychotics. Hyperprolactinemia has been shown to have many physiological consequences, some of them quite severe including sexual dysfunction, osteoporosis and behavioural effects (hostility, anxiety & depression).[1] Hyperprolactinemia is a disorder of the hypothalamo-pituitary-gonadal axis but may be seen as a side effect of typical and atypical antipsychotics as well.[2] It has a prevalence of 0.4% in the general population and may be as high as 9-17% in the reproductive age group.[3] The present paper reviews the physiology and pathology of hyperprolactinemia and management of antipsychotic induced hyperprolactinemia.

The Neurophysiology of Prolactin

Prolactin is a polypeptide hormone secreted by the lactotorph cells of anterior pituitary gland. Prolactin secretion shows circadian rhythm[4], with highest levels occurring during the night and nadir occurring during the afternoon and evening.[5] Normal basal levels of serum prolactin vary between 5 to 25ng/ml in females and 5 to 15ng/ml in males.[6] Levels vary as per phase of menstrual cycle and can also vary according to age. The regulation of prolactin secretion is controlled by various endogenous agents that are released from or act through the hypothalamus via the hypothalamic pituitary portal vessels in response to various stimuli, including stress, sleep and suckling during breast feeding. Factors such as serotonin, estrogens and thyrotropin releasing hormone stimulate prolactin secretion, whereas gama amino butyric acid and acetylcholine inhibit secretion.

Most importantly, prolactin synthesis and secretion by pituitary lactotorph cells is tonically suppressed by hypothalamic dopamine traversing the portal venous system to impinge on lactotorph D2 receptors.[6] Prolactin itself can cause dopamine release from hypothalamus and thus forms a negative feedback loop. Different isoforms of prolactin have different physiological functions and clinical effects.[7] Apart from its well established function in stimulation and maintenance of lactation, prolactin has been found to be involved in over 300 separate functions including water and electrolyte imbalance, growth and development, reproduction, endocrinology and metabolism and immunoregulation.[5] In terms of affecting brain and behaviour, prolactin is shown to increase brain neurogenesis in pregnant mice. Prolactin stimulates an increase in the number of neural progenitors in the forebrain which then migrate to the olfactory bulb. Here these additional neurons are thought to play a role in maternal behaviour because olfactory behaviour is critical for recognition and rearing of offspring.[8] In humans, prolactin also plays a role in the regulation of sexual activity and behaviour. It has been observed that orgasms

cause large and sustained (60 min) increase in plasma prolactin in both men and women9, which is associated with decreased sexual arousal and function. Furthermore, increased prolactin is thought to promote behaviours that encourage long-term partnership.[10]

General aspects of Hyperprolactinaemia

Hyperprolactinaemia is diagnosed when serum prolactin concentrations are greater than 20-25ng/ml (400-500mU/l) on two separate occasions.[11] The Endocrine Society Clinical Practice guidelines (2011) state that in

order to establish the diagnosis of Hyperprolactinaemia, a single measurement of serum prolactin; at a level above the upper limit of normal confirms the diagnosis as long as the serum sample was obtained without excessive venipuncture stress.[12] Dynamic tests of prolactin secretion using TRH, Ldopa, nomifensine, and domperidone are not superior to measuring a single serum prolactin sample for the diagnosis of Hyperprolactinaemia.[13] A single determination is sufficient to establish the diagnosis, but when in doubt, sampling can be replaced on a different day at 15 to 20 min intervals to account for possible prolactin pulsatility.[14]

Hyperprolactinaemia most commonly results from a disorder of hypothalamic –pituitary axis[15] and its causes can be grouped as physiological (sleep, sexual intercourse, pregnancy, nursing) and pathological (pituitary tumours most commonly prolactinomas, hypothyroidism and pharmacological).[16] Traumatic childhood experiences such as parental separation or living with alcoholic father have been reported to produce increased predisposition to hyperprolactinemia.[8] Medications that elevate prolactin levels include antipsychotics, oral contraceptive pills, oestrogens, tricyclic antidepressants, serotonergic drugs, propranolol, methyldopa and reserpine.[8]

Antipsychotics and hyperprolactinemia

Hyperprolactinema is thought to be caused by antipsychotic agents blocking the D2 receptors on the lactrotroph cells and their effects on the tubero-infundibular dopamine pathway, thus preventing inhibition of prolactin secretion. Furthermore it has been suggested that the degree of elevation of prolactin co relates with the degree of occupancy of D2 receptors in excess of 50%.[17]

Traditional first generation antipsychotic drugs

Most studies have shown that conventional anti psychotics are associated with a 2 to 10 fold increase in prolactin levels.[17] The increase in prolactin that occurs due to use of conventional anti psychotics develops over the 1st week of treatment and remains elevated throughout the period of use. Once the treatment stops, the prolactin levels return to normal within 2-3 weeks.[18] It has been suggested that tolerance can develop in patients treated chronically with anti-psychotics and that prolactin levels gradually decline with extended antipsychotic use.19 Prospective studies with an open or double-blind design have shown that medium-term treatment (3–9 weeks) with therapeutic dosages increases mean baseline prolactin levels up to ten-fold.[20-22] Low daily dosing regimens (e.g. 200mg chlorpromazine) can cause significant prolactin elevations and levels have been reported to increase in a dose-dependent manner up to about 600mg chlorpromazine equivalents.[23]

Second Generation Antipsychotic Drugs

In general second generation anti psychotics produce lower increase in prolactin than conventional agents.[24] Risperidone produces the most elevation in prolactin levels amongst the second generation agents.[24] Olanzapine, Zotepine, Amisulpiride and Quetiapine are also associated with increase in prolactin levels. An analysis of double blind studies of risperidone in schizophrenic patients showed that there is dose dependent increase in prolactin concentrations in both men and women.[25] In a randomised, double-blind, parallel group study that compared treatment with amisulpride (1000mg daily) and oral flupentixol (25mg daily) in 32 men and women with schizophrenia who were free of oral antipsychotic medication for at least 4 weeks and depot neuroleptics for at least 3 months. After 4

weeks of treatment mean baseline prolactin levels were significantly elevated in both groups, in the amisulpride group by a factor of 10 and in the flupentixol group by a factor of 5. The difference between amisulpride and flupentixol treatment was significant in the women patients.[26]

Pooled data from two large, randomised, double-blind, controlled clinical trials comparing 8 weeks of treatment with fixed daily doses of risperidone (1mg, 2mg, 4mg, 6mg, 8mg, 10mg, 12mg and 16mg), haloperidol (10mg and 20mg) and placebo. Prolactin measurements were taken at end-point in 259 women. Levels in the risperidone group were increased above the normal range in proportion to the dose and their mean was significantly higher than in women treated with 10mg (but not 20mg) of haloperidol.27 Whether risperidone has a greater effect on prolactin secretion than equivalent doses of haloperidol, as reported in a small 54-week continuation study, requires further analysis.[28]

In another study, researchers measured prolactin levels in 29 men and women with chronic schizophrenia after a 2-week standardising therapy with oral fluphenazine (20mg daily) and 6 weeks after switching to clozapine (mean dose 400mg daily) or risperidone (mean dose 6mg daily). At the end of fluphenazine treatment prolactin levels were increased by about twice the normal reference range in each group. After switching, levels decreased highly significantly into the normal reference range in the clozapine group, whereas they did not change significantly in the risperidone group.[29] Preliminary evidence indicates that zotepine can also cause prolactin elevation in humans after both acute and chronic treatment.30 Studies of patients who are treatment naive or who have been withdrawn from treatment for a period of time indicate that schizophrenia per se does not affect prolactin concentrations. In such patients, prolactin concentrations are not

different from controls31 although the circadian cycle in schizophrenic patients appears to be advanced by 1 to 1.5 hours, an advance that also occurs in patients on antipsychotics.

Clinical manifestations of hyperprolactinemia

Hyperprolactinemia may remain clinically asymptomatic. Women who suffered from hyperprolactinaemia consisting mainly of the trimeric form of prolactin (microprolactin) neither showed any clinical symptoms nor suffered from reproductive dysregulation, despite elevated prolactin concentrations (700-1600mg/l).32 It is thought that these polymeric forms of prolactin assays can be detected by current prolactin assays33, but they are not necessarily physiologically active. Clinical presentation may include hypogonadism, decreased libido and osteopenia in both sexes; infrequent or absent menstrual cycle, galactorrhoea and infertility in women. Low sperm count and reduced muscle mass in men.34 However, in addition to the effects of hyperprolactinaemia on sexual function and reproductive health, hyperprolactinaemia has been linked to other disorders as well.

While untreated schizophrenia patients exhibit decreased sexual desire35, antipsychotic treatment is associated with restoration of sexual desire, yet it entails erectile, orgasmic and sexual satisfaction problems.36 In women receiving antipsychotics, the incidence of menstrual disturbances is 15 to 50 % There may be infrequent or absent menstrual cycles.37 The feedback loop that links prolactin and dopamine also affects the release of gonadotropin releasing hormone (GnRH), which like prolactin , is inhibited by dopamine. When the inhibitory effect of dopamine on prolactin is lost (as in treatment with dopamine antagonist), hyperprolactinemia occurs. This rise in prolactin levels causes a concomitant rise in

dopamine levels which in turn inhibits the release of GnRH .The lower levels of GnRH lead to the symptoms of hypogonadism that are a primary consequence of hyperprolactinemia.[38] The association between hyperprolactinemia and osteoporosis appears to be mediated by oestrogen deficiency secondary to sustained prolactin elevation, although prolactin itself may have a direct effect on bone formation.[39] Some studies state that bone loss occurs secondary to hyperprolactinemia mediated sex steroid attenuation. Spinal bone density is decreased by approximately 25% in women with hyperprolactinemia[40] and is not necessarily restored with normalization of prolactin levels. Several studies have linked hyperprolactinemia to an increase in risk of breast cancer in women. The possible mechanisms include increase in synthesis and expression of prolactin receptors in malignant breast tissue and a prolactin-induced increase in DNA synthesis in breast cancer cells in vivo.[41]

Managing antipsychotic induced hyperprolactinemia

When treating antipsychotic induced hyperprolactinemia, decisions should be made on an individual basis after a full and frank discussion with the patient. These discussions should include consideration of benefits of antipsychotic therapy as well as potential impact of any adverse effects. The importance of discussing symptom impact is highlighted by data showing that only a minority of patients discontinue their antipsychotic medication because of breast tenderness, galactorrhoea or menstrual irregularities.42

The Endocrine society Clinical practice guidelines, 2011 state that no treatment is necessary in an asymptomatic patient with drug induced hyperprolactinemia.43 Increase in prolactin could be due to formation of macroprolactin which does not have serious consequences for the patient. If there

are doubts about the cause of hyperprolactinemia, other possible causes especially tumours must be excluded.44 Sexual side effects are the commonest cause for non compliance. The decision to change the current antipsychotic to an agent with lower dopamine antagonist property or aripiprazole (an atypical antipsychotic with both Dopamine agonist and antagonist activity that can lower prolactin and reverse hyperprolactinemia side effects) should be made on the basis of risk benefit estimation.45

Adjunctive therapies have also been tested to reduce the symptoms of hyperprolactinemia, but these are associated with their own risks. Oestrogen replacement can prevent the effects of oestrogen deficiency, hypogonadal symptoms, but it carries the risk of thromboembolism.[46] Dopamine agonists such as Cabergoline and Bromocriptine have been suggested for the management of hyperprolactinemia in patients receiving antipsychotics, but these are associated with side effects and may worsen psychosis.47

Conclusions

Endocrine symptoms occur in a large proportion of women treated with prolactin elevating antipsychotic drugs. These symptoms can cause significant distress and may affect compliance with medication. A significant proportion of premenopausal women with psychotic disorders may be at risk of premature bone loss and other consequences of chronic hypooestrogenism due to long-term antipsychotic medication. The presence of menstrual irregularities, breast symptoms and sexual dysfunction should be assessed before and during treatment with prolactin-elevating drugs and management options should be discussed with the patient. Thus management of antipsychotic hyperprolactinemia should exclude all

other causes, involve a regular monitoring of adverse effects and include a regular risk benefit discussion with the patient.

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TREATMENT OF DEPRESSION IN PREGNANCY:CURRENT TREND

Mohapatra Satyakam and Rath NM

ABSTRACT

Pregnancy has traditionally been considered a time of emotional well-being for women conferring protection against psychiatric disorders. But depression during pregnancy affects nearly 20% of women. Depression experienced by obstetric patients frequently remains unrecognized and untreated. Lack of adequate management of depression during pregnancy may result in a potentially devastating consequences that impact upon both mother and baby. So clinicians and patients need up-to-date information to assist with decisions about depression treatment during pregnancy.

Key words- ***Treatment, Depression, Pregnancy.***

INTRODUCTION

Major depressive disorder (MDD), a chronic and recurrent illness, **[1]** is a leading cause of disease burden for women aged 15–44 years in both developed and developing regions of the world**[2].** Each year, a substantial number of women, i.e. between 7 and 13% of the global female population, experience MDD**[3, 4,5 6,7].** Its onset coincides with the reproductive yearsand according to the American congress of obstetricians and gynecologists (ACOG), between (14-23)% of women will experience a depressive disorder while pregnant **[8, 9].** For women of low socioeconomic status (SES), rates as high as 50% have been reported **[10]**. Depression experienced by obstetric patients frequently remains unrecognized and untreated**[11]** .This lack of recognition, coupled with a general unwillingness to use medication throughout gestation **[12]** has resulted in the likelihood that depressed pregnant women will not be treated with antidepressant medication**.** In the absence of adequate treatment the depression can accelerate and episodes may become more frequent and severe, resulting in substantial maternal and infant morbidity **[13, 14].**

Major depression during pregnancy

Pregnancy has traditionally been considered a time of emotional well-being for women conferring protection againstpsychiatric disorders. About one third of depressed pregnant women, represents the first episode of major depression.Clinically significant depressive symptoms during pregnancy, particularly observed in the setting of antidepressant discontinuation or with past history of mood disorder. Women with recurrent major depression who have been maintained on an antidepressant medication before conception appear to be at an especially high risk for relapse during pregnancy.In women who have been diagnosed as recurrent depression prior to conception and in whom antidepressant medications have been discontinued, rates of relapse can approximate 75% and can be seen frequently during the first trimester. Pregnant women may have many clinical signs and symptoms overlapping with those seen in major depression (e.g. sleep and appetite disturbance, diminished libido, and low energy). Some medical disorders commonly seen during pregnancy, such as anemia, gestational diabetes, and thyroid dysfunction, may be associated with depressive symptoms and may complicate the diagnosis of depression during pregnancy. Other risk factors for antenatal depression include marital discord or dissatisfaction, inadequate psychosocial supports, recent adverse life events, lower socio-economic status, and unwanted pregnancy.

Functional impairment, inadequate prenatal care, pre-eclampsia, substance abuse **[15]**, increased risk of postnatal depression and ultimately poor pregnancy outcomes have all been associated with depression during the obstetric period.Their babies borne from depressed mothers are often irritable and lethargic, with irregular sleep habits. Lack of adequate management of depression during pregnancy may result in a potentially devastating consequences that impact upon both mother and baby.On the other hand the use of antidepressant medications during pregnancy have been associated with negative

consequences for the newborn.While weighing the risks and benefitsof treating depression during pregnancy following facts should be taken into consideration: risk of untreated depressive disorder, effects of depressive disorder on the fetus, teratogenicity of antidepressant medications,long term behavioral effects on child and incomplete reproductive safety data for medications.So clinicians and patients need up-to-date information to assist with decisions about depression treatment during pregnancy.

Treating a pregnant woman who is depressed

The therapeutic goal of the treatment of depression during pregnancy is to achieve mental stability of the mother, without causing harm to the fetus **[16]**. Thus, it is necessary to weigh the expected benefits to both the mother and fetus against the potential risks of treatment. Treatment options for the management of depression during pregnancy include pharmacotherapy and psychotherapy. Management should be based upon the physician's clinical judgement, the patient's preference, and the availability of professional and support services.

Recommendations

The American Psychiatric Association and the American College of Obstetricians and Gynecologists **[17]** recommend the following:

1. Women who plan to start a family and have mild depressive symptoms for six months or longer may be able to taper off medication. This may not be appropriate for women with a history of severe anxiety or depression, or who have bipolar disorder or a history of suicide attempts.
2. Women who are pregnant, psychiatrically stable, and prefer to continue taking their medication may be able to do so after consulting with their psychiatrist and Obstetricians and Gynecologists.
3. Women who are pregnant and have severe depression or anxiety should remain on medication, as they are at high risk for relapse.

Antidepressant treatment during pregnancy

There are no antidepressant drug efficacy trials in depressed pregnant women. However, there is little reason to think that response would differ between pregnant and non-pregnant women. It is ideal, but not always possible, to evaluate a woman with a past or current depressive illness prior to conception.

Pre-conceptional patients

For women on medication with mild or no symptoms for six months or longer, it may be appropriate to taper and discontinue medication before becoming pregnant. Medication discontinuation may not be appropriate in women with a history of severe, recurrent depression (or who have psychosis, bipolar disorder, other psychiatric illness requiring medication, or a history of suicide attempts). Women with suicidal or acute psychotic symptoms should be treated aggressively. Some women may also benefit from referral to a therapist who can provide psychotherapy. While CBT or IPT are preferable, other types of counseling may be helpful if empiric-based therapies are not available.

Pregnant patient who is not receiving pharmacotherapy

It is common to diagnose untreated depression during pregnancy and to encounter patients who have discontinued their medications but are symptomatic. Psychotherapy may be beneficial in women who prefer to avoid antidepressant medication and is not gravely disabled or at high risk of relapse . For women who prefer taking medication, risks and benefits of treatment choices should be evaluated and discussed, including factors such as stage of gestation, symptoms, history of depression, and other conditions and circumstances (eg, a smoker, difficulty gaining weight). The dose of agents metabolized primarily by cytochrome P450 2D6 or P450 3A4 may require an increase in the second half of pregnancy **[18].**

Patient with current or recent MDD who is taking antidepressants in pregnancy

Psychiatrically stable women who prefer to stay on medication may be able to do so after consultation between their psychiatrist and obstetrical clinician to discuss risks and benefits. Women who would like to discontinue medication may attempt medication tapering and discontinuation if they are not experiencing symptoms, depending on their psychiatric history. Women with a history of recurrent depression are at a high risk of relapse if medication is discontinued. Women with recurrent depression or who have symptoms despite their medication may benefit from psychotherapy to replace or augment medication. Women with severe depression (with suicide attempts, functional incapacitation, or weight loss) should remain on medication. If a patient refuses medication, alternative treatment and monitoring should be in place, preferably before discontinuation.

The impact of antidepressants on birth outcomes

The use of multiple medications during pregnancy makes it difficult to assess the impact of a single compound, such as an antidepressant, on maternal and fetal outcomes. Increased risk for spontaneous abortion is associated with the use of various antidepressants in early pregnancy**[19].** No differences were observed among the various classes of antidepressants.Reductions in birth weightis associated with SSRI use in pregnancy **[20].**Butnot all studies show this association**[21, 22],** although only a few had adequate power to find a difference. Some studies found that preterm delivery is significantly higher among women who used antidepressants, including SSRIs and TCAs**[23,24].** Other studies do not support this association**[25].**The majority of studies have not shown an association between TCA use in pregnancy and structural malformations**[26].**The current data on SSRI exposure show no consistent information to support specific morphological teratogenic risks.**[27]**

While some linked database reports find that compared to unexposed offspring, those exposed to paroxetine during the first trimester are at higher risk of cardiac malformations **[27]**, these results are disputed by other reports including several large case cohort studies**[28].**Infants exposed in utero to an SSRI in combination with a benzodiazepine but not an SSRI alone, may have a higher a incidence of congenital heart defects compared to no exposure**[29]**. Such results raise the possibility that presumed associations between antidepressants and malformations may be complicated by poly-drug interactions. Other antidepressants including bupropion, venlafaxine, duloxetine ,nefazodone, and mirtazapine known not to be teratogenic. **[22,30,31].**

Neonatal neurobehavioral outcomesof antidepressants

In utero exposure to TCAs are associated with increased perinatal complications including jitteriness, irritability and, rarely, convulsions in neonates**.[16,23].** A cluster of symptoms termed “poor neonatal adaptation” has been reported during the immediate neonatal days among infants exposed to SSRIs which include tachypnea, hypoglycemia, temperature instability, irritability, a weak or absent cry, and seizures**[19].** These symptoms occurred in 15–30% of women who took SSRIs in late pregnancy . Symptoms in neonates were transient and typically resolved by 2 weeks or sooner after delivery. An increased risk of persistent pulmonary hypertension (PPHN) was found among newborns whose mothers were treated SSRIs with a greater risk for infants who were exposed later in pregnancy**[32, 33].**

Electroconvulsive therapy during pregnancy

Electroconvulsive therapy (ECT) is considered safe & effective for depression during pregnancy. It is an option for moderate to severe depression in pregnant patients who are unsuitable for or unresponsive to antidepressants, have psychotic features, &/or are suicidal.There is little evidence that it is harmful to the woman or fetus when both are carefully monitored**[34].**

Non-drug treatments for depression in pregnant

There are a number of non-drug treatments that are effective for even major depression in pregnancy . Non-drug treatments include psychotherapy, Omega-3 fatty acids , exercise, bright light therapy and St. John's wort. Many of these can be combined with each other, and are sometimes used in addition to antidepressants (only St. John's wort cannot be combined with medications)

Behavioral treatments for mood disorders

Many patients with mild-to-moderate depression can be treated by psychosocial approaches including individual and group psychotherapy without use of medication. Patients with residual symptoms, those at high risk of relapse, those with comorbid conditions such as panic disorder and those who prefer to avoid medication may benefit from psychotherapy. This is an especially critical option for women preparing for conception or currently pregnant since a large percentage of women may plan to avoid medication. Cognitive behavioral therapy (CBT) or interpersonal psychotherapy (IPT) have been shown to be effective for depression in pregnant women **[35]**.While evidence for supportive and psychodynamic psychotherapy is limited, these approaches are also reasonable if IPT and CBT are unavailable.

SCREENING OFDEPRESSION DURING PREGNANCY

Females shouldbe screened for peri-pregnancy depression during :

1. Pre-conception: should be ask about personal and family history of mental health disorders and treatment.
2. Pregnancy: during the first routine antenatal visit.
3. Postpartum: during routine postnatal visits at 4-6 weeks and 3-4 months postpartum.

Depression screening tools used in pregnancy & postpartum are :

1. Edinburgh Postnatal Depression Scale – validated for use during both pregnancy and postpartum**[36].**

2. Patient Health Questionnaire 9(PHQ-9)
3. National Institute for Health and Clinical Excellence: Screening for Depression During Pregnancy**[37].**

Screening tools do not confirm a diagnosis of depression, but rather identify patients who require further assessment. Using screening tools which focus on somatic symptoms (e.g. Beck Depression Inventory) should be avoided as it can be difficult to distinguish between symptoms of depression versus pregnancy should be avoided.

CONCLUSION

The treatment of depression during pregnancy can be challenging for patients and providers alike. An increasing attention to perinatal mood disorders has led to an expanding literature that is often difficult for providers to navigate. Women who are depressed during pregnancy have been found to have an elevated risk of poor obstetrical outcomes, although studies of the relationship between depression and outcomes are limited. Women who are treated with antidepressants during pregnancy are also at risk for a host of poor obstetrical and fetal outcomes. Understanding the current data and their limitations will allow providers to guide their patients in choosing treatment options. Consistent and simple strategies should be used when discussing the risk-benefit analysis with the patient.

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EXPRESSED EMOTION IN PSYCHIATRIC DISORDERS

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ABSTRACT

Expressed emotion (EE) is currently among the most thoroughly investigated psychosocial research constructs in psychiatry. Expressed emotion (EE) is the general reflection of the family's attitude towards the patient as a precursor to relapse in major psychiatric disorders. On the basis of recent research on EE many family intervention programmes have been developed to reduce EE of the family and consequent relapse of illness

Key words- ***Expressed, Emotion, Psychiatric, Disorders***

Introduction

There is vast majority of evidence that the quality of family relationships is closely related to the development , maintenance and treatment response of many psychiatric disorders[1]. The roles of families in the care of people with psychiatric disorders and the ensuing caregiver burden have been increasingly acknowledged in the research literature in the last three decades[2,3]. The chronic burden of caregiving to a patient with psychiatric illness is likely to generate negative emotions. With the advent of deinstitutionalization, caregivers have increasingly assumed greater responsibility for the care of their mentally ill relatives, with the consequent negative caregiving experience a likely cause of stress manifested in heightened Expressed emotion(EE). "Expressed emotion" refers to a global index of particular emotions, attitudes, and behaviors expressed by relatives about a family member diagnosed with psychiatric illness. The concept of EE was introduced in studies done by Brown et al[4] , where it was shown to have an effect on relapse of schizophrenic patients. In the last 15 years, the EE construct has been extensively studied [5,6]. More than 20 studies, conducted in many countries, have investigated the EE-relapse relationship in patients with schizophrenia. In addition, there is a growing literature concerning the role of EE in unipolar depression[7,8], bipolar disorder[9],eating disorders[10] and dementia[11]. The results of these investigations make two things clear. First, rather than being a construct of interest solely with respect to schizophrenia, EE is a more general predictor of poor outcome across a range of conditions. Second, EE is a construct that is modifiable. EE is of interest to researchers and clinicians because it predicts symptom relapse in patients and because family based interventions that seek to reduce EE have had success in decreasing patients' relapse rates[12, 13].

Components of expressed emotion

George Brown[14,15]explained five components of EE, which includes critical comments, hostility, emotional overinvolvement , positive remarks (regard), and warmth.

Hostility

The hostile attitudes of expressed emotion are negative toward the person with the disorder. The family members put blame on this person because of the disorder. The family perceives the person as the one who is in control of the course of the illness. The relatives feel that the family member is being selfish by choosing not to get better since the illness is an internal conflict. The patient is held accountable for any kind of negative incident that occurs within the family and is constantly blamed for the problems of the family. They have a hard time problem solving within the family because the answer to most problems is settled with the disorder being the cause[16] .

Emotional over-involvement

Emotional over-involvement reflects a set of feelings and behaviour of a family member towards the patient, indicating evidence of over-protectiveness or self-sacrifice, excessive displays of emotion with the use of praise or blame, preconceptions and statements of attitude. Family members who show high emotional involvement tend to be more intrusive. Therefore,

families with high emotional involvement may believe that patients cannot help themselves and that their problems are due to causes external to them, and thus high involvement will lead to strategies of taking control and doing things for the patients[17]. In addition, patients may feel very anxious and frustrated when interacting with family caregivers with high emotional involvement due to such high intrusiveness and emotional display towards them. On the whole, families with high EE appear to be poorer communicators with their ill relative as they might talk more and listen less effectively[18]. Emotional over-involvement demonstrates a different side compared to hostile and critical attitudes but is still similar with the negative affect that causes a relapse. The relative becomes so overbearing that the patient can no longer live with this kind of stress from pity, and falls back into their illness as a way cope[19].

Critical comments

The critical attitudes of expressed emotion are combinations of hostile and emotional overinvolvement. The family members are more open to view other aspects that contribute to the mental illness and the behavior. These attitudes are more open minded than the previous because they view more than one cause of the disorder. However, there is still negative criticism even though other contributions are viewed and accepted by the relatives. Critical expressed emotion from siblings and parents are the cause of future and increasing problems for the patient. Parents who are critical influence their children to be the same way towards the disorder[20].

Positive remarks (regard)

Positive regard comprises of statements that express appreciation or support for patient's behavior and verbal/nonverbal reinforcement by the caregiver.

Warmth

It is assessed based on kindness, concern, and empathy expressed by the caregiver while talking about the patient. It depends greatly on vocal qualities with smiling being a common accompaniment, which often conveys an empathic attitude by the relative. Warmth is a significant characteristic of the low EE family.

Assessment of expressed emotion

Camberwell Family Interview (CFI)

The gold-standard measure of EE is a semi structured interview known as the Camberwell Family Interview (CFI)[21] . The CFI is conducted with the patient's key relative or relatives (typically parents or a spouse) without the patient being present. The CFI is used to make ratings on five scales. These are criticism, hostility, emotional over involvement (EOI), warmth, and positive remarks. Although ratings on five scales are made, practically speaking, the most important EE scales are criticism, hostility, and EOI. It is widely used in clinical population.

The Five Minute Speech Sample (FMSS)

The Five Minute Speech Sample[22] requires the family members to talk about their thoughts and feelings about the patient for 5 uninterrupted minutes. The speech is recorded and later coded for the overall level of EE, criticism, and EOI. There is no hostility rating on the FMSS.

Level of Expressed Emotion Scale (LEE)

The Level of Expressed Emotion Scale [23] is a 60-item, self report measure that assesses the emotional environment in the patient's most important relationships. Items in the LEE Scale are based on the EE construct, and the four subscales are intrusiveness, emotional response, attitude toward Illness, and tolerance and expectations.

Family Attitude Scale (FAS)

The Family Attitude Scale[24] is a 30-item self-report measure of EE. It is similar to the LEE in that either relatives or patients may complete it.

Perceived Criticism (PC)

Of all the alternative measures of EE, the most simple is the perceived criticism measure. This scale recognizes that the most important element of EE is criticism. It consists of only one question, namely "How critical do you consider your relative to be of you?" It is administered as a 10-point Likert-type scale and anchored with the values "not at all critical" and "very critical indeed." This scale takes very less time (1 minute) to administer it. Interviewer can ask patients to rate how critical they think their relatives are of them using this scale.

Expressed Emotion and Relapse

There is an extensive body of literature that delineates expressed emotion (EE) as a general re? ection of the family's attitude towards the patient as a precursor to relapse[25]. EE probably determines relapse through its effect on emotions and symptom control. A stress-vulnerability model of relapse is advanced that incorporates biological factors as well as cycles of mutual influence between symptomatic behaviour, life events, and EE [6] .

Schizophrenia

Majority of studies have demonstrated that a significantly higher number of patients living with high-EE relatives relapse than patients living with low EE relatives. Reviewing 26 studies investigating outcome in 1,323 patients, Kavanagh et a[16] , found a median first-year relapse rate of 48 percent with high-EE relatives and 21 percent with low; and the Bebbington et al [26] , aggregate analyses of data from 25 studies gives relapse rates of 50 percent in patients with high-EE families and 21 percent with low. Studies have found hostility to be a more sensitive predictor of relapse[27,28]. The Chandigarh study in North India conducted by Leffet al[27] found that the only expressed emotion factor to significantly predict the 2-year outcome of schizophrenia was hostility.

Study by Ivanovic et al[29], showed that critical comment was more frequent in families of patients with paranoid schizophrenia, while emotional over involvement was more frequent in families having a hebephrenic offspring. There was also an inverse relationship between relapse rate and warmth, whether paternal or maternal. This was significant in both subtypes, and indicated that the threshold for the positive effect of warmth was higher for fathers than for mothers.

Recently Rylands et al[30], found that high EE stimuli activated brain regions responsible for processing socially aversive information in schizophrenic patients. The emotional valence of the patient's environment signi?cantly impacts upon their well being and illness outcomes.

Mood disorders

Levels of expressed emotion (EE) in relatives are consistent predictors of relapse among bipolar and other mood disorder patients. Patients with high EE relatives reported higher levels of depression over the 2-year term of follow-up, regardless of treatment condition. High expressed emotion from relatives contributes to the change of state from manic to depression in bipolar disorders[31]. An examination of the dimensions of EE (critical comments and emotional over involvement) indicated that a higher frequency of critical comments predicted higher levels of mania and depression at follow-up. EE is also a predictor of symptom severity among bipolar patients undergoing pharmacological and psychosocial treatments[32].

Alcoholism

Study by O'Farrell et al[33], showed that a relapse is more likely to occur with patients that have family members of high expressed emotion more than those that have low expressed emotion. A cycle forms because of the constant criticism of past experiences of drinking which causes a relapse. Family members of high expressed emotion is likely to complain about the drinking before the rehabilitation which causes the start of drinking again. This creates more criticism toward the patient and in addition causes a set back where the person does not care to get better again. This cycle creates problems between the family members and patient that could easily be avoided with less critical comments. Again, the high expressed emotion causes relapse quicker than those with lower expressed emotion because they are less verbally critical of the patient's drinking problem. The fewer negative comments family members make , the longer time there is before a relapse .

Borderline personality disorder

Research in the area of EE and Axis II disorders is only just beginning. Only one study to date has examined the link between EE and clinical outcome in borderline personality disorder (BPD). Hooley et al[34], showed that contrary to prediction, the EE variables of criticism and hostility were not significantly predictive of overall clinical outcome. However, the relatives' level of EOI was significantly predictive of clinical outcome. EE research with BPD patients is still in its infancy , more research is required in this field.

Learning Disabilities

The environment of high expressed contributes to the progress of the children with a learning disability. They are affected socially because of the stress that they have from their parents about simple abilities that they can not do on their own. The attitude from the parents affect the child and cause more problems. Most parents are emotionally over involved with the child because of the learning disability. The stress to improve becomes a big problem for both the parent and child [35].

Culture and EE

EE has been found to be culturally dependent [36] . Research on EE across different national and ethnic groups has suggested that the sociocultural context may influence the family's emotional climate and levels of EE [37]. For example, low estimates of EE have been found particularly in eastern cultural contexts (Japanese: 37% [38] ; East India: 23%[27]), with rural settings showing particularly low levels (0%–8% in India)[39,40] . The highest estimates have been found in European origin cultural contexts (European Canadian:61%[41]; European American: 67% [42]). Mexican American families have shown rates of high EE at 41% [43.]

World Health Organization's study on assessing expressed emotion in first-onset schizophrenia in three centres (Chandigarh, Aarhus and London) [40], reported the lowest ratings on all of the following components : mean number of critical comments; proportion of relatives showing hostility; positive remarks; mean score on warmth; and level of parental over involvement in the Chandigarh sample. Compared with the 54% of relatives classified as showing high expressed emotion in the two European centres, the Chandigarh sample had only 23% of relatives classed as showing high expressed emotion. More than a quarter (29%) of the Chandigarh sample showed hostility but low criticism. The authors concluded that the Chandigarh relatives commonly express both high criticism and high warmth at the same time. One-year follow-up suggested that the better outcome in cases of schizophrenia in Chandigarh may be related to the high proportion of relatives with low expressed emotion. In a further report, the authors suggest that expression of anger in the form of hostility is relatively unmodified by cultural factors[27].

Intervention

The advances in the research on EE in the caregivers of patients with different psychiatric illness in diverse settings have led to the advances in psychosocial intervention strategies with family caregivers. Many family intervention programmes have been developed on the basis of the results of EE research and have components like psycho-education to the patient and caregiver about illness, crisis management, problem-solving skills, clarifying myths and misconceptions, emotional support , and communication skills. Although not all of these programmes are successful, they can reduce the relatives' high-EE score and thus the patients' relapse rates .[44-46]

The aim of psycho-education is to reduce EE by educating them and also to reduce the direct contact with high EE caregivers to less than 35 hours per week. Barrowclough et al[47] proposed two models of education: Deficit model and interaction model. Deficit model suggests that an inadequate knowledge of information about illness results in negative behavior and disseminating of that knowledge will reduce this behavior and result in more positive attitudes and behaviors toward the patients. Interaction model suggests that people make their own explanations of illness and that information provided by professionals will be understandable, organized, and possibly rejected on the basis of the person's own perceptions and explanations.

Results from several trials of family-based treatment indicate that when family EE levels decrease, patients' relapse rates also fall[48]. From a clinical perspective, these findings are clearly very encouraging.

Conclusion

The family's EE has been shown to be predictive of outcome in many psychiatric disorders in a variety of cultural settings. Expressed emotion (EE) is currently among the most thoroughly investigated psychosocial research constructs in psychiatry. Future research should stress on the feasibility and efficacy of the strength-based interventions with ongoing psychosocial interventions at individual or group level for the persons and families of different psychiatric disorders to deal with the negative emotional atmosphere of the family.

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POST TRAUMATIC STRESS DISORDER: AN ILLNESS OF RECOVERY

Sengar K S & Singh Archana

ABSTRACT

Post traumatic Stress Disorder are the disorders caused in reaction to occurrence of some unacceptable, unpleasant incidence leading to intolerable stress/ anxiety. It may be natural or manmade like war, disaster, rape, torture etc. These disorders are found 1 – 3 percent in general population. All most seventy percent people of the general population experiences PTSD any time in their life time. Persistent avoidance of stimuli causing trauma is main feature of this entity. It can occur any time in life but most common in young adults. Most people do not experience the post traumatic catastrophic symptoms after passing the traumatic phase or some time after trauma the recovery starts taking place. However, significant number of people continuously experiences such symptoms and needs specialized intervention. The personality of the individual also plays a vital role on experiencing trauma and its recovery.

Post traumatic stress disorder (PTSD) is a condition marked by the development of symptoms after exposure to traumatic life events. The person reacts to this experience with fear and helplessness, persistently relives them, and tries to avoid being reminded of it.

HISTORICAL BACKGROUND-

18th Century: During Napoleon's time the Diagnosis of *Nostalgia was* established to describe Combat Stress Reaction. “Soldier's heart” was the name given during the US Civil War to a syndrome similar to PTSD. In 1866 the English Surgeon Erickson in his article he attributed conspicuous psychological abnormalities caused due to railway accidents to micro-traumas of the spinal cord, which later on led to the concept of “R*ailway Spine Syndrome”.* Jacob Da costa's (1871). in his paper, “*On Irritable Heart*,” described soldiers with this syndrome. *“Trauma Etiology”* given by Charcot and Janet, pointed out the importance of traumatic experience for origin of hysterical symptoms. During the U S Civil War and World War I, same symptoms were referred to as the *'Da costa Syndrome'* was noted among soldiers during the time of stress, especially when fear was involved. World War I (1914- 1918).: Charles Myres, British Psychiatrist introduced the term *"Shell Shock"* caused by direct exposure to shelling or bombing. World War-II veterans, survivors of Nazi concentration camps, and survivors of the atomic bombings in Japan had similar symptoms, was called combat neurosis or operational fatigue. The psychiatric morbidity associated with Vietnam War veterans finally brought the concept of PTSD. During World War II (1939-1945). PTSD was known as New Syndrome as Combat Stress Reaction, Combat Neurosis/Battle Fatigue. PTSD as a formal Diagnosis was recognized in the year 1980 in DSM III with the emphasis that trauma could produce chronic symptoms in “normal” individuals too. Prior to that if symptoms were found persistent it was due to preexisting psychological problem. The official recognition of posttraumatic stress disorder (PTSD). in the DSM-III (American Psychiatric Association, 1980). has prompted considerable body of research into the psychology, biology, epidemiology, and also in the area of its treatment.

Epidemiology of PTSD:

The life time “incidence” of PTSD is estimated to be 9 to 15% and life time “prevalence” of PTSD is estimated to be about 8 percent of the general population. Among high risk groups whose members experienced traumatic events, the lifetime prevalence rates ranges from 5 to 75%. The life time prevalence ranges from about 10 to 12 % among women and 5 to 6% among man. Although PTSD may appear at any age but it is most prevalent in young adults, because they tend be more exposed to precipitating situations. (DSM-IV).

Co-morbidities:

Although, the diagnostic features of posttraumatic stress disorder (PTSD) are well defined but the condition is not always easy to recognize. Studies in primary care settings shows, recognition rates as low as 2% have been reported. PTSD is often comorbid with obsessive-compulsive disorder, phobic disorder, panic disorder, generalized anxiety disorder, and depression. Other comorbid conditions found in association with PTSD includes bipolar disorder, somatization disorder, substance abuse or dependence, and eating disorders. PTSD is also associated with a high rate of attempted suicide (Jonathan, 2003). PTSD patients are 6 times more likely to attempt suicide than controls. PTSD results in more suicide attempts than in all other anxiety disorders. (Kessler, et al., 1999).

The Current Diagnostic Criteria for PTSD are Unsatisfactory:

It is difficult to distinguish between the PTSD symptom cluster and what could be considered a normal human response to massive trauma. PTSD, generalized anxiety disorder, depressive disorder, and substance dependence are difficult to separate. Symptoms that define PTSD are not strongly correlated with trauma. The diagnosis of PTSD relies on unverified trauma and subjective reports of symptoms. Financial compensation is an important reinforcing factor suggesting secondary gain. (Wilson & Barglow, 2009).

Clinical Features:

PTSD is characterized by recurrent and intrusive recollections of the stressful event, either in images, thoughts, or perception or in dreams. There is an associated sense of re-experiencing of a stressful event and also marked avoidance of the events or situations that arouse recollections of the stressful event, feeling of numbness, and anhedonia. Reminders of trauma arouse intense distress and physiological reactions and are consequently avoidance, including conversation about the event. Patients try to push memories of the event out of their mind and avoid thinking about event in detail, particularly about it worst moments. On the other hand, many ruminate excessively about questions that prevent them from coming to terms with event, for example about why the event happened to them, how it could have been prevented, or how they can take revenge. The patient's emotional state ranges from intense fear, anger, sadness, guilt, or shame to emotional numbness. They often describe feeling detached from other people and give up previously significant activities. Various symptoms of hyperarousal include hypervigilance, exaggerated startle responses, irritability, difficulty concentrating, and sleep problems.

Tools for Assessing PTSD:

***The PTSD Check*list**

The PTSD checklist has two versions.

1. ***Military Version*** which was developed by Weathers F, Huska J, and Keane T, in year 1991.
2. ***Civilian Version,*** this scale was developed in year 1999 by Smith, M.Y, Redd, Duhamel et.al. Both scales contain 17 items. In military version items are related to the stressful military experiences and in civilian version items are a list of problems and complaints that people sometimes have in response to stressful experiences.

Impact of Event Scale (IES):-

Impact of Event Scale (IES) was developed by Horowitz, M., Wilner, M., and Alvarez,W. in year 1979. The IES is a self administered questionnaire evaluating experiences of avoidance and intrusion which attempts to. IES was developed to measure current subjective distress related to a specific event. The IES scale consist of 15 items, 7 of which measure instructive symptoms, and 8 tap avoidance symptoms. The combined scale provides a total subjective scale score. All items of the IES are anchored to a specific stressor. Both the intrusion and avoidance scales have displayed acceptable reliability (.79 and .82, respectively). The IES has also displayed the ability to discriminate a variety of traumatized groups from non traumatized groups. It is an instrument that can be used for repeated measurement over period of time. Its sensitivity to change situation is useful for monitoring the patient's progress in therapy. The Items can be interpreted according to following dimensions:

0-8 Subclinical Range
9-25 Mild Range
26-43 Moderate Range
44+ Severe Range.

Secondary Traumatic Stress Scale (STSS).

Secondary Traumatic Stress Scale (STSS). is a 17-items self-report instrument designed to measure intrusion, avoidance, and arousal symptoms associated with direct exposure to traumatic events via one's professional relationships with traumatized clients.

The Clinician –Administered PTSD Scale (CAPS).

The CAPS (Blake et al, 1995) is considered as the “gold standard” of structured interviews for post traumatic stress disorder. The CAPS has several helpful features, including a standard prompt questions and explicit

behaviorally anchored rating scale and assess both frequency and intensity of symptoms. It generate both dichotomous and continues scores for current (one month). and life time ("worst ever"). PTSD. In addition to the standard 17 PTSD items, the CAPS also contains items tapping post traumatic impacts on social and occupational functioning, improvement in PTSD symptoms since a previous CAPS assessment, overall response validity and overall PTSD severity, as well as items addressing guilt and dissociation. CAPS may require an hour or longer to complete administration, may sometime provide more information than actually is needed clinically, and focuses only on PTSD

Trauma Symptoms Checklist

The TSC-40 by Elliot & Briere (1992) is a research measure that evaluates symptomatology in adults associated with childhood or adult traumatic experiences. It measures various aspects of posttraumatic stress and other symptoms clusters found in some traumatized individuals. The TSC-40 is a 40-item self-report instrument consisting of six subscales: *Anxiety, Depression, Dissociation, Sexual Abuse Trauma Index (STI)., Sexual Problems, and Sleep Disturbance*, can be measured separately as well as a total. Each symptom item is rated according to its frequency of occurrence over the prior two months, using a four point scale ranging from 0 ("never") to 3 ("0ften"). The TSC-40 requires approximately 10-15 minutes to complete and can be scored in 10-15 minutes.

Theories –old and recent

Early theories can be divided into three groups

- ***Social-cognitive theories*** primarily focus on the way trauma breaches existing mental structures and on innate mechanisms for reconciling incompatible information with previous beliefs. Social-cognitive theories provide good accounts of the range of emotions and beliefs occasioned by trauma and of the process of long-term adjustment, without clearly differentiating between PTSD and other types of reaction such as neither depression, nor do they account for the nature of responses to trauma reminders.

- ***Conditioning theories*** deal with learned associations and avoidance behavior. Conditioning theory provides a good account of how trauma cues acquire the ability to elicit fear and of the critical role played by avoidance, but is limited by the absence of cognitive elements in explaining many of the symptoms and data concerning PTSD, especially those dealing with beliefs and perceived threat.

- ***Information-Processing*** *Theories* focus on the encoding, storage, and recall of fear-inducing events and their associated stimuli and responses. Information-processing theories offer clearer descriptions of the cognitive architecture by which the traumatic event may be represented, of effects on attention, and of how the overturning of assumptions increases the number of potential trauma reminders, but are less able to account for the importance of emotions other than fear and of beliefs extending beyond issues of danger to the wider social context.

Early theories:

Horowitz's Formulation of Stress Response Syndrome:

The Horowitz (1973) formulation of stress response syndrome is the most influential cognitive model of reaction to trauma to date. Although derived from the classical psychodynamic psychology, Horowitz's theory is principally connected with discussing such ideas in terms of cognitive processing of traumatic formation such as ideas, thoughts, images, affects and so on. Horowitz has argued that the main inputs within the cognitive system for the processing of trauma related information comes from a completion tendency, the psychological need to match new information with inner models based on older information, and the revision of both until they agree."This tension causes the individual to oscillate between phases of intrusion and denial –numbing as they gradually integrate with long –term meaning representations. The failure of such processing can remain in active memory ever being fully assimilated, thus leading to chronic post traumatic reactions

Janoff-Bulman's Cognitive Appraisal Theory:

The cognitive-appraisal model of Janoff-Bulman (1985) centers almost exclusively on the content of the preexisting beliefs about the self and the world which the individual carries into a traumatic situation. Janoff-Bulman argues that PTSD is the result of certain basic assumptions about self and the world being "shattered". The assumption which Janoff-Bulman is referring to are the assumption of invulnerability, the perception of the world as a meaningful or comprehensible or and the view of the self in a positive light. The proposal is that these assumptions provide a structure and meaningful in the individuals life. But that they cannot be maintained in the phase of a traumatic experience and consequently they "shattered', plunging the individual into a confusion of intrusion, avoidance and hyper arousal

Foa's Fear Network:

Applying Lang's theory of fear structures foa & Kozak (1986) and her colleagues have outlined an information processing theory of PTSD which centers on the formation of a so-called fear network in long term memory. This fear net work encompasses a stimulus information about the traumatic event, information about cognitive, behavioral and physiological reactions to the event and information which links these stimulus and response elements together. Activation of the trauma related fear network by cue stimuli, according to Foa causes information in the network to inter conscious awareness (the intrusion symptoms of PTSD).

Cognitive Action Theory:

The cognitive action theory of Chemtob et al., (1988) is a product of research with veterans of war in Vietnam. According to this theory individuals with PTSD, the fear net work is permanently activated causing them to function in a 'survival mode" thus a way of functioning that was adaptive during the traumatic incident. This permanent activation of the network, it is suggested, leads to the symptoms of hyper arousal and intrusion. Such symptoms are exacerbated by to further functions of the Chemtob et al. model. First, the increase "gain" of the system in PTSD means that vicious circles of increasing arousal operate more quickly and secondly, it is suggested that PTSD sufferers have higher limits on the magnitude to which the threat – arousal system can be activated.

Information –Processing Theory:

The cognitive processing model of PTSD of Creamer et al, (1992) is a derivative approach presented as a "synthesis and reconceptualization of existing formulation". Creamer et al argue for an initial period of intrusion (due to activation of the fear network) with which the individual copes by the calling upon a range of defense and avoidant strategies. More over Creamer et al. suggest that this initial intrusive experience can be used as an index of the degree of net work resolution processing and is occurring. In this analysis, then, high levels of initial intrusion are a predictor of successful recovery whereas low level of initial intrusion is a predictor of poor outcome and chronic pathology.

Recent Theories:

Brewin's Dual Representation Theory:

Brewin's et al. (1995) endeavors to circumvent the short comings of single level theories by proposing two levels in memory at which trauma related information can be represented. The first level of representation is of the individual's conscious experience of the traumatic event. This form what Brewin called verbally accessible memories (VAMS).VAMS are characterized by their ability to be deliberately retrieved and progressively edited by the traumatized individual. VAMS representations, it is argued, as with foa's network, contains sensory, response and meaning information about the traumatic event. The second level of representation proposed by Brewin consists of situationally accessible memories (SAMS). SAMS contains information which cannot be deliberately accessed by the individual and is not available for progressive editing. Dual representation theory propose that VAM and SAM representations are encoded in parallel at the time of trauma and between then account them account for the range of PTSD phenomenology.

The SPAARS Approach:

Schematic, Propositional, Associative and Anological Representational Systems by Dalgiesh & Power, (1995). The SPAARS approach deal with emotions. It is a multi representational model with four levels/formats of representation in which two routes to the generation of emotions are specified. SPAARS is a functional theory of emotion. Within SPAARS emotions are emotional tools which the cognitive system employs to resolve problems with active, valued goals. So, for example if a goal is threatened, this will be appraised within SPAARS and a fear module will be activated. The fear module is essentially a reconfiguration of the cognitive system to deal with the imminent threat and any possible future threats. Within SPAARS, then, emotions are adaptive processes which recognize the cognitive system in different ways to deal these changes in the internal or external environment. The suggestion is that the adaptiveness of emotions can sometime go awry and this can lead to the development and maintains of so called emotional disorders.

Ehlers and Clark's Cognitive Model provides what is currently the most detailed account of the maintenance and treatment of PTSD. They have significantly expanded understanding of the wide range of relevant negative appraisals and have identified both appraisals and a variety of cognitive coping factors that influence the course of the disorder. These aspects of the model have been strongly and consistently supported by empirical research.

Models of Psychopathology in PTSD

Anxiety disorder:

- Fear, anxiety and avoidance behaviour (as with phobias).
- Intrusive phenomena resemble obsessive–compulsive disorder
- Introversion and neuroticism are common personality traits in both PTSD and anxiety
- Some people improve with exposure therapy

Mood disorder:

- **Sadness and** grief, independent of bereavement
- Co morbidity with depression is very common
- Vegetative symptoms (loss of sleep, appetite, libido). are very similar
- Avoidance, numbing and loss of interest as in mood disorders

Dissociative Disorder:

- Flashbacks and amnesia are common

Personality Disorder:

- Considerable overlap of symptoms with borderline personality disorder
- Some overlap of symptoms with antisocial personality disorder (antisocial behaviour, irritability).
- Some evidence that trauma can induce personality change

Separate Neurophysiological disorder:

- Low monoamine oxidase activity
- Increased excretion of urinary beta-endorphin
- Therapeutic response to serotonergic drugs and drugs that affect the locus ceruleus
- Deregulation of hypothalamic–pituitary–adrenal axis resulting in low cortisol levels

The Role of Persistent Dissociation in PTSD

Briere and colleagues (2005). highlights the importance of persistent dissociation in the etiology of posttraumatic stress disorder (PTSD). On the basis of two cross-sectional studies, the authors conclude that "the primary risk for PTSD is less whether one dissociates during (or soon after). a traumatic event than whether such dissociation persists over time". Murray (2002). reported two *prospective* longitudinal studies of motor vehicle accident survivors and came to a remarkably similar conclusion: "Persistent dissociation and rumination at 4 weeks after trauma are more useful in identifying those patients who are likely to develop chronic PTSD than initial reactions". Participants were assessed very soon after the accident and followed for 6 months. Persistent dissociation at 4 weeks was a better predictor of chronic PTSD at 6 months than peritraumatic dissociation measured in the immediate aftermath of the trauma. Halligan et al (2003) showed that persistent dissociation predicted an additional 8% variance of PTSD severity at 6 months over and above what could be predicted from trauma severity and cognitive processing measures, including peritraumatic dissociation.

Risk Factors for PTSD

- *Women are twice as likely to develop PTSD*
- *Being 40 to 60 years old*
- *Being a member of an ethnic minority group*
- *Low SES*
- *History of emotional problems*
- *Prior history of trauma*
- *Living in a highly disrupted or traumatized community, loss of income or livelihood*
- *Having on-going psychosocial stressors*
- *Higher level of exposure, perceived threat to life*

➢ ***Aspect of Trauma***

- *Duration and magnitude of exposure to stressor*
- *Stressors are sudden and/or occur with no warning*
- *There is multiple loss of life, mutilation or grotesque imagery*
- *Criminal violence, especially sexual*

➢ ***Experience during Trauma***

- *Perceived own life to be at real risk*
- *Perceived lack of control of events, intense fear and helplessness*
- *Perception of grotesque imagery, especially of human man or children*
- *Witnessing or carrying out atrocities, e.g. murder, torture*
- *High levels of dissociative symptoms at the time of the event*

➢ ***Characteristics of the Individual***

- *Previous psychiatric illness or neuroticism*
- *Previous exposure to trauma, especially childhood trauma*

- *Previous coping style*
- *Denial of trauma and/or avoidance*
- *Female gender*
- *Previous acute stress reaction*

➢ ***Post-trauma***

- *Denial of trauma by others or dismissal of experience*
- *Lack of social support*

The Role of Spirituality in Recovery

Trauma often seems to precipitate a spiritual crisis, in which the survivors struggle to establish a greater understanding of transpersonal issues which are often left unexplored within the traditional cognitive and cognitive – behavioral approach which are, by their nature, focused on the alleviation of so-called disorder and the symptoms. For many, the experience of trauma represents a powerful positive and existential change in outlook, the exploration and facilitation of which are not easily amenable to the techniques of cognitive and cognitive-behavioral therapies.

Post-traumatic stress disorder (PTSD). and the family

If a loved one has post-traumatic stress disorder (PTSD)., it's essential that you take care of yourself and get extra support. PTSD can take a heavy toll on the family if you let it. It can be hard to understand why your loved one won't open up to you – why he or she is less affectionate and more volatile. The symptoms of PTSD can also result in job loss, substance abuse, and other stressful problems.

Letting your family member's PTSD dominate your life while ignoring your own needs is a surefire recipe for burnout. In order to take care of your loved one, you first need to take care of yourself. It's also helpful to learn all you can about post-traumatic stress disorder (PTSD). The more you know about the symptoms and treatment options, the better equipped you'll be to help your loved one and keep things in perspective.

Issues To Be Taken Care of in Treatment

- Areas of active and important debate on treatment of PTSD include the management of acute posttraumatic reactions and the prevention itself of PTSD after experience of traumatic events. For managing acute stress disorder, the literature supports the use of brief treatments that employ principles of cognitive restructuring and exposure to details of the event. This may include the prolonged exposure/cognitive restructuring described by others (Bryant, 2003)., or newer treatments such as memory structuring intervention (Gidron, 2001). Both of these treatments are administered in the course of two to four sessions in the immediate post trauma period, and they appear to lower the risk of subsequent PTSD.
- A widely promoted intervention for preventing PTSD is psychological debriefing or critical incident stress debriefing. This technique, originally developed for use with rescue personnel, has been widely adopted but with insufficient critical examination. Recent assessments have demonstrated a lack of benefit for single-session debriefing as a means of preventing PTSD is of greater concern, however, is the possibility that such interventions actually worsen the prognosis. (Mayou et al., 2000).?
- Foa (2000). reviewed empirically validated psychosocial treatments for posttraumatic stress disorder (PTSD). and factors associated with successful therapy outcome. Most of the treatments whose efficacy was studied empirically fall within the broad category of cognitive-behavioral therapy. These include exposure therapy, anxiety management programs and cognitive therapy. These modalities of therapy have been developed to modify conditioned fear and erroneous cognitions that are thought to underlie PTSD. Exposure therapy has the most empirical support because it was found to be effective across different populations of trauma victims with PTSD. Combinations of therapies have also been used, and the value of these is discussed. In addition, recent evidence shows the efficacy of eye movement and desensitization reprocessing. A growing body of evidence supports the use of psychosocial treatments for PTSD, but not all patients benefit. Future research should develop programs that increase the motivation of patients to take advantage of these efficacious treatments.
- Randomized controlled trials have now produced positive results for multiple-session trauma-focused cognitive behavior therapy for survivors with acute stress disorder within a month of the trauma (Bryant et al. ,1999)., those with distressing traumatic stress symptoms 1 month after the trauma (Bisson et al ., 2004) and those with acute PTSD between 1 and 3 months after the trauma (Ehlers et al., 2003). These results led the authors capitulate the guidelines delineated by U. K. National Institute for Health and Clinical Excellence in 2005 to recommend that

trauma-focused cognitive behavior therapy be made available to all individuals with acute PTSD between 1 and 3 months after a traumatic event (National Collaborating Centre for Mental Health,2005).

How community respond to traumatic period

Community responses to traumatic period are in four different ways -

Heroic Phase: Communities Pull Together

During and immediately following a disaster, individuals and communities often respond supportively, altruistically, and heroically.

Honeymoon Phase: In this attention stays focused on the victims. The community adopts the four common modes t combat stressors-

1) Intense community mobilization
2) Increased community consensus
3) People from outside the community come to help
4) Organizations adapt to help the community.

Disillusionment Phase: The survivors of any traumatic events are provided the different type of help from different sources. However, they learn to pass time leisurely. But once helpers leave the once the survievers left alone to face the reality the disillusionment phase occurs. Where persons find them difficult ot face the reality and cope with new situations. This issue deals with following points -

- Longest phase of recovery.
- Immediate response teams leave.
- Assistance and help weakens.
- Losses become a reality.
- Widespread discouragement.
- Scapegoating, resentment, disagreement.
- Unity fades.

Recovery and Reconstruction: Finding a New Normal Life.

- May not occur for a year or more post-disaster.
- Social and economic activities recover.
- Gradual return to normal routines.
- Completion of reconstruction and recovery efforts.
- Community tries to find a new normal life.
- Anniversary events.
- Difficulty in recovery decisions is compounded by poor planning and preparedness.

Certain patterns are required to build a new pattern of starting newer phase of life after trauma which needs to be considered for coming back on track.

Seven Supportive Communications:

1. Empathy: "How are you holding up?"
2. Normalization: "You are having a normal reaction to abnormal events and situations."
3. Recognition of efforts to cope: "Everyone copes in his or her own way."
4. Self-care: "Make sure you are doing things to keep yourself healthy."
5. Tolerance for change: "You will find a new normal after this is over."
6. Instilling hope; "You have made it through some tough times before, and you will make it through this, too."
7. Accepting help: "It is okay to take some help when you need it."

Most often past traumatic problems recover gradually by its own by passing the time but still it require psychosocial intervention for better faster and safer recovery from the trauma and sometimes it also needs to be treated by pharmacological agents.

INDICATED TREATMENTS FOR POST-TRAUMATIC DISORDERS:

1. ***Acute stress responses***
   - Debriefing
   - Social supports
   - Pharmacological supports, e.g. hypnotics
   - Information and advice to families
2. ***Acute PTSD:***
   - Exposure therapy may be first-line treatment if intrusive phenomena prominent
   - Cognitive therapy
   - Brief psychodynamic psychotherapy
   - Antidepressants (especially where avoidance prominent).
3. ***Chronic PTSD:***
   - Exposure therapy if trauma never discussed
   - Cognitive–behavioural approaches may still be effective (group or individual).
   - Long-term psychotherapy (group or individual).
   - Antidepressants, lithium, carbamazepine (National Institute of Clinical Excellence, 2005).

COMMUNITY & CULTURE:

Community has its own relevance and greater significance in cases of traumatic events. Community and culture plays significant role in recovery and following points must be considered during intervention.

- Psychosocial Interventions should be fully integrated with the overall relief and rehabilitation activities right from the beginning following a disaster. Respect for local cultures needed in implementing psycho-social interventions. All programmes must be culturally sensitive and appropriate to the local community. Understanding of local culture helps to determine the appropriateness and feasibility of specific interventions.
- Strong belief in traditional healers may affect the choice of interventions in many ways. Attitudes toward intervention is also very important (e.g., preference for or dislike of medication).;

Why Early Interventions In PTSD?

- There is a dearth of naturalistic, prospective studies of the course of posttraumatic recovery, especially the course of adjustment to mass violence and traumatic loss.
- About 90 % people recover after stress management training, but what happens to those who don't.
- There is much conjecture about what puts people at risk for chronic PTSD, but there are few well defined studies.
- Well trained professional are scare, evaluating efficient methods of delivering the key elements of early interventions is crucial
- There is scant descriptive, epidemiological or clinical research on the unique psychosocial needs and outcomes of individuals who suffer from the dual burden of losing a loved one through trauma while experiencing their own acute trauma.
- But there is a paradigm shift in the area of early intervention well before 9-11
- Early intervention is dominated by non - evidence - based practices, poorly defined and anarchical notions about recovery from trauma and the risk for trauma –linked disorders and an apparent unresponsiveness to scientific inquiry.

Delay in Treatments (Perceived barriers in seeking treatment).

Don't trust,

Too embarrassing,

Harm my career,

Colleagues are less confident in me,

Leaders blame me,

Seen as weak (Hoge et al, NEJM, July 1, 2004).

- Stigma
- Low awareness of PTSD
- Professional help not sought (24.5 %).

PROGNOSIS OF PTSD

- Effects on social systems and support
- As in general psychiatric practice, it is important to consider the influence of the disorder on other areas of a person's life, in relation to its effect on the process of recovery and the prognosis. The symptoms of many of the post-traumatic disorders may be troublesome for families and employers, particularly in the first 6–12 months. Families, friends and employers are usually unfamiliar with the timescale of normal recovery. Most people do not realize that normal recovery may take 6 months, or longer if there are further stressors, and they might become impatient with survivors, thinking them difficult or weak. After experiencing trauma a person may be chronically irritable and withdrawn for weeks, in a way which is alien to them and their families. There is good evidence that marital stress and breakdown are increased after traumatic experiences. Work performance may similarly deteriorate because of the person's hyper vigilance, accompanying loss of concentration and irritability. However, people often find it impossible to discuss the reasons for this with their employers. Employers may not be sympathetic anyway, especially if the trauma occurred at work (and compensation is being claimed)., or where there is a work culture of denial of distress. Many major personal disasters are never reported in the press. During peacetime, and between major disasters, the principal cause of traumatic stress responses is crime, of which the impact on the victim is rarely reported unless it is fatal. (Kilpatrick et al, 1989). This applies to both men and women. A good example of this is the plight of the families of murder victims. The killer is often a member of the family and relatives must cope with multiple losses.

Case: 1

"*Ms A. 45 years married with three children, presented with multiple problems after her husband murdered their daughter to prevent her from telling the mother about his 20-year affair with a family friend. This woman lost not only her daughter and her husband; she also lost experience of her marriage and the support of a trusted friend with whom she was confident that she/he will help at each movement. She was also without funds as the husband had been the principal earner and she did not have access to the bank account. The court trial did not take place for a year, and the funeral of the daughter was delayed several times due to some legal proceedings related to post-mortem reports for both defense and prosecution".*

- These social and legal aspects of post-traumatic dysfunction have a profound influence on the management and prognosis of PTSD, and can cause major setbacks in treatment.
- A man who becomes homeless because of domestic violence related to his post-traumatic irritability may be unable to cooperate with or tolerate a treatment programme
- In-patient treatment might be indicated in such instances. People with PTSD as a result of crime have particular problems: not only they have reminders of the stressor, such as police identification parades or court appearances; they might be at continued risk of further trauma, such as threats from the defendant.

Choosing a treatment

There are particular questions relevant to the selection of treatment.

What is the worst problem at the moment?

If intrusive phenomena are prominent, this may suggest exposure therapy as part of a cognitive–behavioral package. If depression and distress are worst, then regular supportive therapy sessions plus antidepressants may be most effective. The support system must be assessed. What support does this person have and what solutions he is adopting?

What supports does this person have?

Many forms of treatment for PTSD are quite stressful. It is therefore important to ensure that the patient will be well supported, and that the families are informed about the nature and process of therapy.

What solutions to stress are the patient adopting now?

If a patient is misusing alcohol or drugs as a means of managing their PTSD symptoms this needs to be addressed before any specific PTSD treatment can be implemented. Rarely, patients present with acts of self-harm such as overdoses, and these should not be dismissed as 'attention-seeking'.

Helping a loved one with PTSD

- ***Be patient and understanding.*** Getting better takes time, even when a person is committed to treatment for PTSD. Be patient with the pace of recovery and offer a sympathetic ear. A person with PTSD may need to talk about the traumatic event over and over again. This is part of the healing process, so avoid the temptation to tell your loved one to stop rehashing the past and move on. Also **try to anticipate and prepare for PTSD triggers**. Common triggers include anniversary dates; people or places associated with the trauma; and certain sights, sounds, or smells. If you are aware of what triggers may cause an upsetting reaction, you'll be in a better position to offer your support and help your loved one to calm down.
- **Don't take the symptoms of PTSD personally.** Common symptoms of post-traumatic stress disorder (PTSD) include emotional numbness, anger, and withdrawal. If your loved one seems distant, irritable, or closed off, remember that this may not have anything to do with you or your relationship.
- **Don't pressure your loved one into talking.** It is very difficult for people with PTSD to talk about their traumatic experiences. For some, it can even make things worse. Never try to force your loved one to open up. Let the person know, however, that you're there when and if he or she wants to talk.

INTEGRATING PSYCHOSOCIAL TREATMENT FOR PTSD & SEVERE MENTAL ILLNESS:

Patients with severe mental illness (SMI)., such as schizophrenia, bipolar disorder, and major depression, are also more likely to have experienced trauma in childhood, adolescence, and throughout their adult lives than the general population. (Bebbington et al, 2004, Goodman,1997). This high exposure to traumatic events such as physical and sexual abuse and assault takes a heavy toll. In addition to the immediate effects of victimization on an individual's quality of life, a history of trauma exposure in persons with SMI is associated with

more severe symptoms, greater impairment of functioning, and higher levels of distress (Briere J, 1997). During the past decade, research has firmly documented that one of the most common consequences of trauma in patients with SMI is their high vulnerability to posttraumatic stress disorder (PTSD). The lifetime prevalence of PTSD in the general population is 8% to 12% (Breslau et al., 2004; Kessler et al, 2005) in comparison to patients with SMI have much higher rates of PTSD, with most reported estimates ranging from 29% to 47%. (Calhoun et al, 2007; Mc Farlane et al, 2001; Mueser et al., 1998, Switzer et al, 1999). Two approaches have predominated in developing programs for SMI. First, some interventions focus specifically on PTSD by adapting treatment approaches shown to be effective in the general population for patients with SMI, such as cognitive restructuring (Mueser et al , 2004; Mueser et al 2007) and exposure therapy.(Frueh et al, 2004). Second, some interventions are more broad-based and address a wide range of trauma squeal, such as poor self-esteem and body image, dysregulated behavior, and problematic relationships. Pharmacological treatments are often included as important components of treatment. Research on the effects of programs specifically developed to address trauma in patients with SMI is still in its infancy. However, clinical reports from early trials of these programs indicate that patients with SMI can be successfully engaged and treated, and that outcomes often improve (Harris, 1998). (Rosenberg 2004). Only 1 randomized controlled trial of a treatment for trauma in SMI has been completed (K. T. Mueser et al, unpublished data, 2007). The results of the trial indicate that participation in the 12- to 16-week cognitive-behavioral therapy program was associated with improvements in PTSD symptoms, other symptoms such as depression, and trauma-related beliefs about oneself and the world

CASE VIGNETTE

- Elizabeth is a 46-year-old woman with schizophrenia and PTSD. As a child she experienced significant physical abuse from her mother and, in an attempt to escape the abuse, frequently ran away from home. As a result, she spent much of her adolescence in a children's home. At 17, she left the home and moved in with her stepfather, who was separated from her mother. She lived with him for about a year, during her stay with step father, he sexually abused her. At one point she became pregnant by him; she told no one, but suffered a painful and confusing miscarriage. Shortly thereafter, she withdrew from people and stopped taking care of herself, which was followed by signs of psychosis, including paranoia, hallucinations, and conceptual disorganization.
- Elizabeth was hospitalized, treated, and discharged free of psychotic symptoms. She began to work as a store clerk, married, and had 2 children. Although her husband physically abused her, she remained fairly stable until he sought a divorce and took custody of their children. Following this, Elizabeth's condition worsened, and she spent several years cycling in and out of hospitals, interspersed with periods of homelessness.
- During a stay at a halfway house, she met a man and fell in love. Despite his drug abuse, they forged a relationship that lasted several years, during which time Elizabeth's symptoms and functioning stabilized, and she began working part-time. Then one day she found him dead, which was followed by deterioration in her functioning.
- Several years later, an evaluation confirmed that Elizabeth had severe PTSD. She reported that the death of her boyfriend was her most distressing traumatic experience. She said that she felt extremely guilty because she had not been able to prevent his death. She also talked about how her history of abuse made it difficult for her to trust anyone.
- The first session focused on teaching breathing retraining to manage her anxiety. During a homework review the following week, Elizabeth stated that she only felt comfortable practicing breathing retraining with her eyes open, but that she found it very helpful.
- The next 2 sessions focused on discussing the symptoms of PTSD, including how they differ from the symptoms of schizophrenia, and how they are affected by each other.
- Elizabeth expressed relief in learning that her PTSD symptoms were common reactions to traumatic experiences.
- During the fourth session cognitive restructuring was introduced. Elizabeth was able to understand the connection between thoughts and feelings but struggled with the related homework. She noted that she was afraid of becoming too overwhelmed when trying to do her homework alone.
- After more sessions in which the therapist helped her using cognitive restructuring to examine and challenge thoughts related to her upsetting feelings, Elizabeth began to practice using the skill on her own to deal with negative feelings.
- She described the therapy program as helpful and agreed to a joint session with her case manager at the end of treatment so that she and her therapist could teach her case management skills that Elizabeth had developed in therapy.
- A recurring concern that was addressed with cognitive restructuring was Elizabeth's fear that nobody would claim her body at her death.

- She felt abandoned by her children, depressed and isolated, and preoccupied by worry about rejection. She was eventually able to identify several core beliefs, including the thoughts that "you cannot trust anybody" and "anyone that you do trust will abandon or mistreat you." Over the course of treatment she was able to recognize that there were some people who had stood by her (eg, her therapist, her psychiatrist, her case manager, her son).
- She was able to gain a more balanced perspective on her feelings of guilt over her boyfriend's death, realizing she was not to blame. As Elizabeth's symptoms gradually improved, she began to reach out to others. She started having brief conversations with people (e.g, at the library)., reconnected with a neighbor, and renewed her relationship with her son.
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Few important Issue on PTSD -

- Early intervention is a must.
- Help to be seek from specially trained professionals.
- Increasing public awareness about trauma related disorders.
- Localization of the treatment programs.
- Client oriented individual therapies.
- Involvement of clients' social networks.
- Local leaders need to be included.
- IES materials need to be distributed.
- Evidenced based research.
- Outcome studies specific to different group of population.

Yet man is born unto trouble, as the sparks fly upward - Job 5:7

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Original Articles :

Rorschach, Culture and Popular Responses

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ABSTRAC

Researches investigating cross-cultural and ethnic group differences on projective tests are relatively scarce. However, significant differences in relation to acculturation, socialization, and the very different cultural subgroups process cognitively and emotionally, had documented. A number of studies have shown that popular responses are associated with cultural background, which is likely to reflect common contribution of the group. This study provides information on how Indian adult non patients' gives variety of popular responses on the Rorschach test administered and scored by following Beck system. The present research is an effort to generate normative data for proper responses in Indian context.

Key words : - ***Rorschach, Popular response, cultural, cultural free, cross-culture***

Introduction:

Human being can be described as a culture-building species. No human have ever survived and perpetuated themselves except as members of social group. Thus, the study of personality should be done in context of one's culture. In this, regard Benedict (1934) says that no man ever looks at the world with pristine eyes. He sees it edited by a definite set of customs and institutions and ways of thinking. Anthropologist had carried out many cross-cultural studies on Rorschach. About eighty years ago, Benedict (1934) and Hallowell (1934 cited that the cross-cultural researcher must have an intimate knowledge of the culture as a whole; researcher must also be aware of the normal range of individual behaviour within the cultural pattern and likewise understand what the people themselves consider to be extremely deviated deviations from this norm.

Boyer (1988). De Vos & Boye, (1989). and Mattlar and Fried (1993) presented a large-scale normative study in Finland and concluded; at least as concerns Europe and the U.S., it would seem that the principle elements of the structural summary could be described as 'sturdy', little affected by nationality. Normative studies of Finland and other Scandinavian countries describes that “Christmas elves” are most frequent response on card II and it is coded as popular (Mattlar & Fried, 1993). Should there be cultures in which such cultural-specific responses as “Christmas elves” occur frequently.

Although the use of several of these tests with diverse groups may be controversial (Frank, 1994, Hale-Benson, 1982; Worchel, 1997). they have been used cross-culturally, without standardized modification. According to Anastasi (1998). personality projective techniques present a peculiar discrepancy between research and practice. When formally evaluated as psychometric instruments, many of the commonly used projective tests as the Rorschach found technically lacking with people of colour, yet the major differences among ethnic group has been documented on instruments such as the Rorschach (Jones, 1978; Kaplan, 1961).

Many critics in the respect of cultural influence have attacked the test. Garcia (1981). Green and Griffore (1980) commented that the standard test, when it comes to content, modality and structure has certain biasness. That further led to a blind alley in its application and interpretation. When it applied with population, whose culture linguistic, economic or social background is different gives an inappropriate result.

The Rorschach is assumed culture- free or at least culture –fair. This means that the test can applicable to any cultural. Since it is content free test as this is “unstructured” in nature it is assumed that subject's responses represents his or her unique response rather than a response to any common meaning the test may have. However, both of these assumptions have been in contrast by several studies (Jaleel, 1990). Takahashi and

Zax (1966) compared American and Japanese college students to examine the stability of meaning of the Rorschach for the two groups. They found the cultural variables resulted in a greater number of significant differences between the Japanese and American subjects than one's mental health. Rabin and Limuaco (1967) found the evidence that the meaning of Rorschach differed quite significantly between these two cultures.

Greenfield (1997) maintained that cross-cultural misdiagnosis and incorrect assessment often occurs when examiner from a dominant cultural group and respondent from a less powerful group using instruments that developed in the dominant culture. (Banks, 1997; Frank, 1994; Obiakor, Algozzine, & Schwenn, 1995). Multicultural researchers are familiar with diversity of cross-cultural affect. It is also known that the any assessment tool is developed in a specific cultural and generalised on group of peoples who belongs to that particular cultural so any approaches cannot be applied blindly to people of different cultural (Jones, 1978; Lonner, 1985; Russell, Fujino, Sue, Cheung, & Snowden, 1996). Additionally, the validity and reliability of a test used with peoples of different cultures who were not part of the standardization group are questionable. It is also important to recognize that diversity may exist between examiners and examinees even when the difference may not be readily apparent. So far, the study on projective technique as we have, indicates that there are variations in person-to-person responses in any given culture (Lindzey, 1961). He further state that following the factor which contribute in giving the responses as follows:

The social class, ethnic group, cultural background, their past life experience and coordination with given situation.

Another, issue which is repeatedly addressed in literature is psychological normality is an absolute or relative phenomenon. A successful adaption always counts with a particular personality characteristics. It is observed that both absolute and relative perspectives on abnormality contribute to reasonable conclusions. Jonathan Swift (1726/1960) gives a classic example in this regard, “Gulliver was too big for the Lilliputians and too small for the Brobdingnagians”.

Maturity, adjustment and maladjustment are depended upon the certain personality characteristics, which learnt from his own culture, followers of this notion, assumed that Rorschach sets certain standards of maturity and adjustment. This view in its maximum, demonstrates ethnocentricism. A minimum expression of this standpoint would be that the Rorschach could be an indicator of closeness of internal imbalance, which would cause problems in whatever social environment. Thus, the question raised that internal imbalance and immaturity that has been grown up in specific cultural can be casually applicable for another culture.

De Vos, et.al. (1989) studied Japanese "normal" families and families with delinquent teens. They noted higher levels of denial and lower levels of H and M in delinquent families, especially to Card III. Japanese as a group gave fewer responses, put more work into integrating them (W>D and Zd higher). and while they gave as many and most of the same popular, for Card VIII they saw a flower in the centre, an uncommon American response. If Rorschach variables do tap into defensive processes, then the defences of a "normal" and "delinquent" family might both differ from American samples, and so distinctions between A and B (like M and C) on the Rorschach here may not hold up in other countries. They found most of the popular were the same, and found significance for ideas behind M and C, T and Y, as well as trends for significance for V. While delinquent families did not give more aggressive responses, such as those in America do, they gave fewer positive and healthy content responses, more isolated, dysphoric, and evasive (maps and islands) responses, and few positive authority content (badges, helmets, and crests). reports of "playing" between people, and fewer symbols of aesthetic value (vases, chandeliers, etc).

Method:

In present study the sample size consisted of 530 respondents with equally distributed gender from the capital of India. The respondents were selected on the basis of stratified systematic random sampling method. The Age range of respondents included in the study was 20 to 30 years, educated up to high school and not having any physical or psychological illness at the time of interview. The subjects having positive family history of mental illness were excluded from the sample. Descriptive statistics were used to analyse the data.

Data were coded, entered and analyzed using SPSS package (version 16.0) and presented in percentage. Chi-square test was used for evaluating association between gender and socio-demographic characteristics and t test / Mann-Whitney U test was used to establish the relationship between two genders and different variables of Rorschach. 'P' value for the level of significance was calculated and the same less than 0.05 was considered.

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Executive Function Deficits in Patients with Schizophrenia

Kumari Neelam, Prakash Jai

ABSTRACT

Background: *The cognitive dimension most widely studied in schizophrenia is executive function. Some of the specific abilities that fall under this rubric include abstraction, planning, mental flexibility, response inhibition, self monitoring, evaluation and decision making. There is strong evidence that neuropsychological impairment is related to deficits in everyday functioning abilities among persons with schizophrenia.* ***Aim:*** *The present study attempts to measure executive dysfunctions in patients with schizophrenia.* ***Method:*** *By using purposive sampling technique, 30 patients of schizophrenia were chosen according to ICD 10 DCR criteria alongwith a normal control group of 30 subjects. GHQ-12 was used as screening tool for normal control subjects and participants who scored below 2 were included in normal control group. Executive functioning was assessed among all the participants of experimental group as well as normal control group by using CTMT, WCST and Cognitive Symptoms Checklist (Executive Function Scale).* ***Result:*** *The schizophrenic patients demonstrated executive functioning deficits in comparison to normal control subjects on Comprehensive Trail Making Test as they took more time to complete all the trails and on Wisconsin Card Sorting Test as they produced lesser number of correct response, committed more errors, exhibited high number of perseverative responses, perseverative errors and non perseverative errors, low number of conceptual responses, took more trails to complete first category and were not able to maintain set. Impairment was also found on other domains of executive functioning related to activities of daily living like processing speed, initiation, sequencing, planning and reasoning.* ***Conclusion:*** *In comparison to normal participants, schizophrenia patients exhibited significant dysfunction in executive functioning and hence in activities of daily living.*

Key Words : ***Executive Function, Schizophrenia, Activities of daily living***

INTRODUCTION

Cognitive impairment is recognized as core characteristic of schizophrenia. Executive function is an important cognitive domain. Executive functions include the capacity to formulate goals, plan and organize goal directed behavior, carry out goal directed behavior fully and effectively and monitor and self correct one's behavior as needed (Lezak, 1987). Because of inconsistencies in conceptualizing and operationalizing the construct, the nature of executive dysfunction is nebulous. Deficit in executive performance is well established in schizophrenia. Schizophrenia patients have varying grades of impairment in their executive functioning which results in difficulties during extended and multifaceted interpersonal interactions. Executive impairment affects various cognitive processes including initiation, sustained attention, switching and flexibility, disinhibition, attention allocation and planning (Chan et al; 2006a). It has also been demonstrated by various studies that schizophrenic patients tend to perform more poorly than normal controls in neuropsychological tests sensitive to frontal lobe lesions, such as Wisconsin Card Sorting, verbal fluency, and trail making (Chan et al. 2006b). Performance on neuropsychological measures of executive skills seems to be associated with differences in certain types of psychiatric symptoms among schizophrenia patients, i.e; those patients with greater executive impairment tend to have more severe or stable negative symptoms, and may have less independence in at least some aspects of everyday functioning. Frontal dysfunctions such as disturbed planning, lack of inhibition, contextual inappropriate responses, reduced cognitive flexibility, poor problem solving, have been commonly described in schizophrenia (Krabbendam et al.,1999; Mahurin et al., 1998; Pantelis et al., 1997). The functional neuroimaging studies also suggest that schizophrenia patients lack normal patterns of frontal

activation while performing these executive tasks. Schizophrenia patients may lack the ability to organize brain activity efficiently in response to have minimal demands for executive control (Taylor, 1996). However the real ecological impact of these neuropsychologically measured deficits is not known.

Recent empirical research has consistently demonstrated that although psychopathological symptoms disrupt patient's lives, deficits in cognitive functioning have the strongest influence on their overall level of independent functioning. The severity of neuropsychological deficits seems to be a determinant of functional outcome of schizophrenia patients but neuropsychological evaluation does not provide specific information about how cognitive impairment influences activities of daily living, i.e; deficit in which cognitive domain affect which behavior. Patients with schizophrenia who are impaired on measures of executive functions have difficulty adapting to the rapidly changing world around them.

The present study has been undertaken with the aim to study the executive function deficits in patients with schizophrenia in comparison to normal control subjects.

MATERIAL AND METHOD

The sample consisted of 30 patients selected as per ICD 10 DCR criteria for schizophrenia from different in patient department of Ranchi Institute of Neuro-Psychiatry and Allied Sciences, Ranchi and 30 normal control subjects. Inclusion criteria for the former included patients in the age group of 25 to 45 years and having atleast 8 years of schooling. Purposive sampling technique was used for selecting the sample. The General Health Questionnaire 12 (Goldberg & Williams, 1988) has been used as a screening tool for detecting psychiatric disorders among respondents in normal control group. Subjects scoring more than 2 were screened out. Then Comprehensive Trail Making Test (Reynolds, 2002) and Wisconsin Card Sorting Test (Heaton et al. 1981) have been administered to assess the executive functioning in the group of schizophrenia patients and normal control subjects. Executive function subscale of Cognitive Symptom Checklist (O' Hara et al. 1993) was administered to elicit information about difficulties in daily living due to impaired executive functioning. Independent sample t test was used to compare the performance of clinical group and normal controls on CTMT, WCST and Executive Function subscale of Cognitive Symptom Checklist.

RESULT AND DISCUSSION

Table 1: Showing Performance of Schizophrenia Patients and Normal Control Subjects on Comprehensive Trail Making Test

Subjects / Variables	Schizophrenia Patients (N=30)		Normal Controls (N=30)		't' Value
	Mean	SD	Mean	SD	
Time Taken for Trail 1	141.93	24.21	48.17	22.70	15.47**
Time Taken for Trail 2	146.87	30.16	52.20	14.70	15.45**
Time Taken for Trail 3	184.10	32.67	61.37	17.83	18.06**
Time Taken for Trail 4	193.23	34.02	52.63	16.78	20.30**
Time Taken for Trail 5	239.23	64.53	73.30	23.03	13.26**
Total Time Taken	905.40	137.05	286.76	71.38	21.93**

** significant at 0.01 level

Obtained data have been scored using standard procedure for further analysis. It has been found that the mean age of schizophrenia patients and normal control group was 31.77±4.73 years and 31.33±5.65 years respectively. Further mean education was found to be 10.73±1.85 years for schizophrenia patients and 10.53±1.63 years for normal control participants.

Table-1 shows the performance of both the groups on comprehensive trail making test. Significant statistical differences has been found on time taken for trail 1 (Schizophrenia Patients: M=141.93±24.21; Normals: M=48.71±22.70; t=15.47, p>0.01), trail 2 (Schizophrenia Patients: M=146.87±30.16; Normals: M=52.20±14.70; t=15.45, p>0.01), trail 3 (Schizophrenia Patients: M = 184.10 ± 32.67; Normals: M= 61.37 ± 17.83; t = 18.06, p>0.01), trail 4 (Schizophrenia Patients: M= 193.23±34.02; Normals: M=52.63±16.78; t=20.30, p>0.01), trail 5 (Schizophrenia Patients: M=239.23 ±64.53; Normals: M=73.30±23.03; t=13.26, p>0.01) and total time (Schizophrenia Patients: M=905.40±137.05; Normals: M=286.67±71.38; t=21.93, p>0.01) underlying impairment in visual scanning, visual search, sequencing, divided attention and ability to shift cognitive set among schizophrenia patients as compared to normal control subjects. Similar results have been obtained by several researchers. Wolwer and Gaebel (2002) concluded that poor performance on trail making test in schizophrenia patients indicated insufficient sequencing, problems in planning and execution. Wolwer and Gaebel (2003) have also reported impaired planning strategies in patients with acute schizophrenia which mainly accounted for patients poor performance on trail making test, which

Table-2: Showing Performance of Schizophrenia Patients and Normal Control Subjects on Wisconsin Card Sorting Test

Subjects / WCST Variables	Schizophrenia Patients (N=30)		Normal Controls (N=30)		't' Value
	Mean	SD	Mean	SD	
Number of Trials Administered	123.84	13.15	101.70	19.52	12.07**
Correct Responses	58.66	21.48	88.73	13.93	3.47**
Error committed	65.13	6.37	12.56	26.79	10.45**
Percent Errors	51.30	19.94	14.56	4.81	9.80**
Perseverative Responses	43.06	36.05	4.46	2.73	5.84**
Percent Perseverative Responses	37.43	28.53	5.43	2.95	6.10**
Perseverative Errors	38.36	28.71	4.46	2.73	6.43**
Percent Perseverative Errors	32.63	23.93	5.43	2.95	6.17**
Nonperseverative Errors	26.63	20.41	8.43	6.22	4.67**
Percent Non perseverative Errors	20.96	15.66	17.06	23.02	5.76**
Conceptual Level Responses	35.43	26.34	64.93	6.58	5.95**
Percent Conceptual Level Responses	29.96	24.61	79.93	8.31	10.53**
Number of Categories Completed	2.00	2.02	5.60	0.00	10.86**
Trials to Complete First Category	59.03	51.46	12.76	4.05	4.90**
Failure to Maintain Set	1.73	1.79	0.36	0.88	3.72**

** significant at 0.01 level

might be a nosologically specific, trait-like characteristic, probably related to neural dysfunctions involving the prefrontal cortex. Wolwer et al. (2003) have reported impaired visuomotor integration in patients with acute schizophrenia, which mainly accounted for patients poor test performance. In another study Mahurin et al. (2006) found that schizophrenia patients made significantly more errors, particularly tracking errors, suggesting a greater degree of cognitive disorganization during trail making performance.

Performance of both the groups on WCST has been given in table-2. For completing the six sorting principles of WCST, more number of trials were administered on schizophrenia patients (M=123.84±13.15) than normal control subjects (M=101.70±19.52) and the difference was found to be statistically significant (t= 12.07, p>0.01) suggesting impaired executive functioning in the former group. Similar results have been obtained by Raffard et al (2009). Schizophrenia patients produced lesser number of correct responses (M=58.66±21.48) and committed more errors (M=65.13±6.37) than that of normal control subjects (M=88.73±13.93; t=3.47, p>0.01) suggesting faulty planning and impaired cognitive flexibility among the clinical group . Similarly due to impaired abstract thinking, schizophrenia patients have given more perseverative responses (M=43.06±36.05) as compared to normal control group (M=4.46±2.73; t=5.84, p>0.01). Perseverative errors were also high in schizophrenia patients (Schiz: M=38.36±28.71; Normals: M=4.46±2.73; t=6.43, p>0.01) which was due to problem in shifting set and working memory. Similar trend was found for non perseverative errors too (Schiz: M=26.63±20.41; Normals: M=8.43±6.22; t=4.67, p>0.01) which suggest inability to utilize available feedback to correct future response. Similar findings has been observed by Divya et al (2007). Low number of conceptual responses were also exhibited by schizophrenia patients (Schiz: M=35.43±26.34; Normals: M=64.93±6.58; t=5.95, p>0.01). Lesser number of categories were completed by schizophrenia patients (M=2.00±2.02) than normal control subjects (M=5.60±0.00; t=10.86, p>0.01). Similar findings were demonstrated by Jai Prakash et al (2005), Sabhesan & Parthsarathy (2005) and Divya et al (2007). The number of trials taken to complete first category was high in the clinical group of schizophrenia patients (Schiz: M=59.03±51.56; Normals: M=12.76±4.05; t=4.90, p>0.01). Failure to maintain set was also high in schizophrenia group (M=1.73±1.79) than normal control group (M= 0.36±0.88; t=3.72, p>0.01). In another study, compared to normal controls, patients with schizophrenia performed significantly worse on Wisconsin Card Sorting Test (P=0.004 for administered trails; P= 0.025 for perseverative responses) indicating significant deficit in attention, psychomotor performance, working memory and cognitive flexibility among schizophrenic patients (Wobrock et al., 2009). The above findings are also consistent with the results of Divya et al. (2007) and Bhatia et al. (2009).

Performance of schizophrenia patients and normal control subjects on executive function sub scale of cognitive symptom checklist has been reflected in Table -3. Statistically significant difference were found on all the variables of executive function scale namely processing speed (Schiz: M=5.07±1.23; Normals: 1.57±0.94; t=12.41, p>0.01), initiation (Schiz: M=3.00±0.79; Normals: M=0.63±0.90; t=12.54, p>0.01), self correction (Schiz: M=3.53±0.90: Normals: M=0.13±0.35; t=19.32, p>0.01), mental flexibility (Schiz: M=3.27±0.94; Normals: M=0.17±0.38, t=16.69, p>0.01), planning (Schiz: M=5.06±1.55; Normals: M=0.33±0.60, t=15.56, p>0.01), sequencing (Schiz: M=4.50±1.22: Normals: M=0.30±0.59; t=16.89, p>0.01), problem solving (Schiz: M= 5.43±0.43; Normals: M=1.25±0.68, t= 19.24, p>0.01), organization (Schiz: M=5.73±1.66; Normals: M=1.20±0.81, t = 13.46, p>0.01) and reasoning (Schiz: M= 8.00±0.98; normals: M=1.36±0.76; t=29.18,p>0.01)

underlying difficulties in performing activities of daily living among schizophrenia patients due to impairment in executive functioning. Findings of the present study has been supported by Semkovska et al. (2004). They observed that schizophrenia patients showed defective abilities in retaining contextually organized events, poor selective and divided attention capacities, difficulties in making self generated strategies and in organizing sequential thinking which could contribute to an inefficient outcomes in activities of daily living.

Table-3: Showing the Performance of Schizophrenia Patients and Normal Controls on Executive Function Subscale of Cognitive Symptom Checklist

Subjects / Executive Function Variables	Schizophrenia Patients (N=30)		Normal Controls (N=30)		't' Value
	Mean	SD	Mean	SD	
Processing Speed	5.07	1.23	1.57	0.94	12.41**
Initiation	3.00	0.79	0.63	0.90	12.54**
Self Correction	3.53	0.90	0.13	0.35	19.32**
Mental Flexibility	3.27	0.94	0.17	0.38	16.69**
Planning	5.06	1.55	0.33	0.60	15.56**
Sequencing	4.50	1.22	0.30	0.59	16.89**
Problem Solving	5.43	0.43	1.25	0.68	19.24**
Organization	5.73	1.66	1.20	0.81	13.46**
Reasoning	8.00	0.98	1.36	0.76	29.18**

** significant at 0.01 level

Further analysis of activities of daily living through a standardized behavioral scale revealed that executive dysfunctions in schizophrenia may specifically affect activities of daily living like more omissions when choosing the menu, more sequencing and repetition errors during the shopping task and more planning, sequencing, repetition and omission errors during the cooking task. Tyson et al. (2008) also found that abstract tests of cognitive function do not indicate which cognitive function could affect what specific aspect of daily function and here lies the importance of ecologically valid tests of attention and executive function (e.g; Behavioral Assessment of the Dysexecutive Syndrome Test) in defining how cognitive deficits in schizophrenia relate to everyday functioning and quality of life. Iampietro et al. (2012) after comparison of schizophrenia groups based on measures of various executive function abilities on everyday action errors concluded that people with schizophrenia demonstrate variable pattern of executive function deficits, and distinct aspects of these executive function deficit patterns of poor mental control abilities which may be associated with everyday functioning capabilities.

CONCLUSION

Schizophrenia patients showed deficits in executive functioning in comparison to normal controls on all five visual searches and sequencing tasks of CTMT and on all measures of WCST. The impairment in different aspects of executive functioning also have its implication on real world functioning.

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Memory Dysfunctions in the cases with Schizophrenia

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ABSTRACT

Background: Memory refers to the process by which information is stored in the brain. It includes encoding, storage and recall of information. Memory encapsulates sense of personal identity, culture and the meaning of individuals' life. Schizophrenia patients consistently show performance deficits on memory tasks, whether the material in question is verbal or nonverbal, recently learned items or older material. Aim: The study has been designed to assess the memory deficits in schizophrenic patients and it's comparison with the normal control group. Method: The sample consists of 100 subjects, out of which 50 schizophrenia patients diagnosed as per ICD-10 DCR criteria and 50 normal control subjects have been included in the study. Wechsler Memory Scale and GHQ- 12 have been used in the study. Result: Schizophrenia patients performed poor in comparison to normal control subjects on subtests of Wechsler Memory Scale. It has further been found that schizophrenic patients were having problems of immediate, recent and remote memory. They were having impaired logical memory in terms of conceptualizing themes of the story and had difficulty in learning and remembering the information. Conclusion: Patients with schizophrenia exhibited impaired information and orientation, disturbed logical memory and had problems in conceptualizing the themes.

Key words : ***Memory Impairment, Schizophrenia,***

INTRODUCTION

Schizophrenia is the most severe mental illnesses characterized by delusions hallucinations, disorganized speech of frequent derailment or incoherence and grossly disorganized behaviour. The deficient processes involved in this disorder are clinically silent until the onset of prodromal or psychotic symptoms, at which time neuropsychological testing demonstrates the presence of cognitive deficits that are often chronic and apparently irreversible (Goldberg et al.,1993). The clinical picture of schizophrenia has led many authors to speculate that particularly the negative features of schizophrenia reflect deficits in executive functioning (Anderesen et al., 1989).

Memory refers to the process of encoding, storage and recall of information. Encoding allows the perceived item of interest to be converted into a construct, storage referring processes of retaining information in the brain and recall or retrieval is the process of re-accessing of events or information from the past, which have been previously encoded and stored in the brain (Klatzky,1980; Howard,1983).

Memory deficits observed in schizophrenia are not restricted to a single element of memory but strike different systems, such as declarative memory, procedural memory and working memory. McKenna et al.,(1990) and Tamlyn et al., (1992) also reported significant memory deficits in the group of schizophrenia. (Aleman, et. al., 1990) have observed deficiency in memory while examining schizophrenic patients. Schizophrenia patients exhibited impaired memory (Goldberg et al., 1988; Goldberg et al., 1989; Saykin et al., 1994)., deficits in recent and remote memory has also been observed by McKenna et al., (1990). Schwartz et al., (1991) have shown that among schizophrenics there is a disturbance of the temporal ordering of information, suggesting a possible disturbance of episodic memory.

The present study has been undertaken with the aim to assess the memory deficits in schizophrenia patients as compared to normal control subjects by using information orientation and logical memory subtests of Wechsler Memory Scale-III.

MATERIALAND METHOD

The sample consisted of 100 subjects, out of which 50 were schizophrenia patients diagnosed as per ICD-10 DCR criteria from different inpatient and outpatient department of Ranchi Institute of Neuro Psychiatry and Allied Sciences, Ranchi and 50 were normal control

subjects. Both male and female participants were included in each group i.e., schizophrenia and normal control subjects. Positive and Negative Syndrome Scale (PANSS) was administered individually to assess the psychopathology of schizophrenia patients. For normal control group GHQ-12 (Goldberg & Miller, 1979) was used to assess any psychiatric problem and subjects having cut off score more than 2 were excluded. Afterwards Wechsler Memory Scale III (Wechsler, 1997) was administered on both the groups to assess the memory dysfunction.

RESULTS

Obtained data has been analyzed by mean, standard deviation and Mann Whitney U test for the comparison of schizophrenia patients and normal control subjects on WMS-III scale.

Table - 1 showing the Performance of Schizophrenia Patients and Normal Control Subjects on Information and Orientation domain of WMS-III.

Subjects Subtests of WMS-III	Schizophrenia Patients (N=50)		Normal Controls (N=50)		Mann Whitney U Test	
	Mean	SD	Mean	SD	U value	Z score
Information & Orientation-I Total Recall	61.10	11.13	72.88	9.75	547.00	4.84**

**** Significant at 0.01 level.**

Table-1 shows the performance of schizophrenia patients and normal control subjects on information and orientation subtest of WMS-III. It has been found that schizophrenia patients had poor information and orientation in comparison to normal control subjects and difference was significant at 0.01 level (Schiz: M= 61.10 ± 11.13, Normals: M=72.88 ± 9.75, U = 547, P 0.01). Further it has found that majority of schizophrenia patients were unable to give information regarding existing and previous prime ministers and chief ministers, had problems of telling months, day of the month, unable to tell place and city. Some of the schizophrenia patients were unable to even tell the days of the week, not having orientation of time and place in comparison to normal control subjects.

Performance of schizophrenia patients and normal control subjects on logical memory subtests of WMS-III has been given in table-2. It has been observed that schizophrenia patients performed significantly poor on logical memory-I (Schiz: M=9.64 ±4.10. Normal controls: M=13.92± 3.48, U value=524.50, Z score=5.012, p 0.01) indicating inability to recall the stories as well themes of the stories in the group schizophrenic patients. Similar trend of impairment has been observed in this group while assessing the thematic memory (Schiz: M=8.10±3.63, Normal Controls: M=14.04 ±2.94, U value=735.00, Z score=3.57, p 0.01).**Table-2: Showing the Performance of Schizophrenia Patients and Normal Control Subjects on Logical Memory domain of WMS-III.**

Subjects Subtests of WMS-III	Schizophrenia Patients (N=50)		Normal Controls (N=50)		Mann Whitney U Test	
	Mean	SD	Mean	SD	U Value	Z score
Logical Memory-I: recall total Scaled score	9.64	4.10	13.92	3.48	524.50	5.012**
Logical Memory-I thematic total scaled scores	8.10	3.63	14.04	2.94	735.00	3.57**
Logical Memory–II recall total scaled scores	7.40	4.17	13.72	2.79	189.00	7.34**
Logical Memory–II Thematic total scaled scores	6.58	3.13	11.46	2.23	242.00	7.00 **
Logical Memory–II % retention	7.36	2.20	11.70	2.95	630.50	4.18**

** Significant at 0.01 level.

When schizophrenic patients were assessed on logical memory II of Wechsler memory scale, it has been found that schizophrenic patients performed poorly as compared to normal control subjects and difference between these two groups has been found to be significant at 0.01 level (Schiz=7.40±4.17, Normal Controls =13.72±2.79, U values =189.00, Z score=7.34, p 0.01).

It has further been visualized that schizophrenic patients performed poorly on logical memory II and thematic total scores of schizophrenia patient's M=6.58 ±3.13 and normal control subject's M=11.46 ±2.23, U value are=242.00 and difference were at 0.01 level, and logical memory II % retention of schizophrenic patient's M=7.36±2.20 and normal control group's M=11.70±2.95 and its U value are=630.50 and difference were significant at 0.01 level respectively, which indicating that schizophrenia patients performed significantly worse than normal control subjects on logical memory subtest. And they were having problem in conceptualizing theme of the story.

DISCUSSION

In this study an assessment of memory dysfunction of schizophrenia patients have been done along with its comparison with normal control individuals. The

obtained results demonstrate significant deficits in the group of schizophrenia patients as compared to normal controls in memory as assessed by Wechsler Memory Scale- III (Table-I) Similar results were found by Ravindran & Rangaswamy (2004) who conducted a study on schizophrenia patients with normal control group in the domain of memory and found that former have difficulty in learning and remembering new information. In logical memory I and logical memory II schizophrenic patients performed poorly which indicates poor performance by most of the patients (Table-II). Item interpretation shows that most of the schizophrenic patients have impairment in ability to remember information immediately after it is orally presented. Percent retention scores also poorer than normal control subjects of schizophrenic patients which indicated that they are unable to sustain the information after 25-30 minutes. Result is consistent with previous research findings. McDonald, et al, (2006) tested episodic memory using logical memory and visual reproduction tasks of the Wechsler Memory Scale (Revised) and observed marked verbal recall deficits in schizophrenic patients compared to normal controls. Similar findings were obtained by Goldberg et al (2008) observed that schizophrenic patients were having impairment in verbal learning and auditory delayed memory. Michael. et al, (2011) have done a study on 65 schizophrenic patients and 45 healthy normal subjects using verbal paired lists and word list tests, and their results revealed that schizophrenic patients had significant deficits than that of normal subjects on both memory tests..

CONCLUSIONPatients with schizophrenia exhibited impaired information and orientation, disturbed logical memory and had problems in conceptualizing the themes. Recall performances showed impaired in schizophrenic patients on information orientation and logical memory subtest of WMS III as compared to normal control subjects.

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ANXIETY DISORDERS IN CHILDREN AND ADOLESCENTS

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ABSTRACT

Anxiety disorders are the most common group of psychiatric disorders in children and adolescents. Anxiety disorders in children and adolescents can be chronic and disabling, and they can increase the risk of comorbid disorders. Anxiety is associated with substantial negative effects on children's social, emotional and academic success. Identifying and treating children and adolescents with anxiety disorders would reduce the burden of this disorder and may help in better management of the co-morbid conditions in these patients.

Key words- ***anxiety disorders, children and adolescents, phenomenology, comorbidities.***

Introduction

Anxiety disorders are the most common group of psychiatric disorders in children and adolescents[1]. They usually remain undiagnosed in children and adolescents owing to the internalized nature of its symptoms [2] . Anxiety disorders are associated with considerable developmental, psychosocial, and psychopathological complications. They impair emotional, cognitive, physical and behavioral functioning in multiple areas and are usually chronic in nature. They can increase the risk of multiple comorbid psychiatric disorders[3]. Pediatric anxiety disorders predict adult anxiety disorders and depression; and other childhood sequelae, such as substance use problems, suicide attempts, and hospitalization[3, 4, 5]. Therefore, it is important to identify and treat pediatric anxiety disorder to reduce the long-term consequences. Over the last 10 years there have been considerable advances in the understanding of the phenomenology, neurobiology, genetics and treatment of anxiety disorders in children and adolescents. A thorough understanding of anxiety is crucial for professionals who are providing mental health services for children.

Classification and diagnosis

The major anxiety disorders included in the DSM-IV-TR are separation anxiety disorder (SAD), generalized anxiety disorder (GAD), social phobia, specific phobia, panic disorder (with and without agoraphobia), agoraphobia without panic disorder, posttraumatic stress disorder (PTSD), and obsessive-compulsive disorder (OCD).

DSM-IV-TR criteria for anxiety disorders are similar for children and adolescents and adults; however, there are some notable differences that need to be considered when diagnosing anxiety disorders in children and adolescents.

***TABLE NO -1**

DSM-IV-TR criteria differences for anxiety disorders in youth from adults

DSM-IV-TR criteria differences for anxiety disorders in youth	
Anxiety Disorder	Differences in Criteria for children and adolescents
Obsessive-compulsive disorder	Children do not need to recognize that the obsessions and compulsions are excessive or unreasonable.
Social phobia	➢ Children must have the ability to develop age appropriate friendships. ➢ Children must endorse anxiety with adults and peers. ➢ Anxiety may be shown through crying, tantrums, freezing or shrinking from social situations. ➢ Children do not need to recognize that the fear is excessive or unreasonable
Generalized anxiety disorder	➢ Requires only one associated symptom
Social phobia	➢ Children must have the ability to develop age appropriate friendships. ➢ Response to the traumatic event may be expressed through agitated or disorganized behavior rather than extreme fear, helplessness, or horror. ➢ Traumatic event may be re-experienced through the use of repetitive play about the trauma. ➢ Children may have scary dreams without recognizable content that is related to the event. ➢ Children may re-enact trauma-specific details.

Epidemiology

Large-scale community-based epidemiological surveys in US ,UK and Canada show that anxiety disorders are the most common children's mental disorders with an estimated prevalence rate of 6.4 per cent[6]. Prevalence rates for having at least one childhood anxiety disorder vary from 6% to 20% over several other large epidemiological studies[7]. A recent British population study of youth aged 5 to 15 years produced the prevalence of anxiety disorders was 3.8% [8]. Study of anxiety disorders among German adolescents estimated from a survey of 1,035 students aged 12–17 years was 18.6%[9]. In Great Smoky Mountains Study (GSMS), conducted over a representative population sample of 1420 children aged 9 to 13 years at showed prevalence of any anxiety disorder was 1.8% to 3.1%[10]. Other epidemiological reports based on large and/or nationally representative samples estimate the prevalence of anxiety disorders in youth to range between 10% and 20%[11]. The most frequent anxiety disorders among children and adolescents are separation anxiety disorder with estimates of 2.8% and 8% and specific and social phobias, with rates up to around 10% and 7%, respectively. Agoraphobia and panic disorder are low-prevalence conditions in childhood (1% or lower); higher prevalences are found in adolescence (2%–3% for panic and 3%–4% for agoraphobia) [12, 13, 14].

In terms of sex differences, all anxiety disorders more frequently occur among females than among males[15, 16, 17]. Although sex differences may occur as early as childhood they increase with age[18] reaching ratios of 2:1 to 3:1 in adolescence .

The average age at onset of any single anxiety disorder varies widely between studies9. There has been relatively little research interest focused on the age of onset of anxiety disorders in children. The anxiety disorders most commonly seen in children include separation anxiety disorder, generalised anxiety disorder, and Specific Phobias. In adolescence, social phobia and panic disorder can become more prevalent (along with GAD and Specific Phobia). Obsessive Compulsive Disorder and Post Traumatic Stress Disorder can occur across the developmental range.

Indian perspective

Prevalence of anxiety disorders in children and adolescents in India is less compared to western countries . An epidemiological study conducted by Indian Council of Medical Research at two-centres Bangalore and Lucknow[19]. Studies at Banglorecentre and Lucknowcentre were community based study and clinic based study respectively. This studyshowed that anxiety disorders in Banglorecentre was (3.93%) and Lucknowcentre was (2.32%). At Lucknowcentre prevalence of Social phobia 0.19%, Separation anxiety disorder 0.09%, generalised anxiety disorder 0.14%, Simple phobia 1.98%, agoraphobia 0.05%, panic 0.05%. At Bangalore centre prevalence of Specific isolated phobia 2.9%, Social phobia 0.3%, GAD 0.3%, Separation anxiety disorder 0.2%, Agoraphobia 0.1%, Panic disorder 0.1%, Social anxiety disorder 0.1%, OCD 0.1%. Various other epidemiological studies done in children and adolescents in India have reported prevalence of anxiety disorders ranging from 1.3% to 4.2%. [20,21, 22, 23].The first systematic study done in India by Malhotra et al, [24] on incidence in child psychiatry revealed 20 children out of 186 followed up patients had psychiatric disorder giving the annual incidence rate of 18/1000/yr. 2 out of above 20 children had Emotional disorders with onset specific to Childhood (As per ICD-10- F93).

Margoob et al, [20] revealed that anxiety disorders are more common (80%) in adolescent age group. This can be explained by the fact that with increasing scholastic demands and expectations from the child and the consequent stress on studies may be responsible for more anxiety in adolescent age group.

Study done by Chadda et al,[21] showed that anxiety symptoms were common in female population (1.55%) as compared to males (0.77%). Margoob et al ,[20] also showed that anxiety disorders were common (80%) in female patients.

Phenomenology of anxiety disorders

Anxiety is considered to be a universal phenomenon existing across cultures, although its contexts and manifestations are influenced by cultural beliefs and practices[25]. Children and adolescents with anxiety disorders can have a clinical picture that is somewhat different from those seen in adults. The differentiation between normal and pathological anxiety, however, can be particularly difficult in children because children manifest many fears and anxieties as part of typical development [26]. For instance, children may not report any worries or anxieties but may have pronounced physical symptoms. Severe tantrums may be their only manifestation of anxiety problems and thus can be confused with mood disorders or oppositional behavior. Clinicians need to distinguish normal, developmentally appropriate worries, fears, and shyness from anxiety disorders that significantly impair a child's functioning.

Specific phobia

Specific phobias are the most prevalent anxiety disorder in children and adolescents according to nearly all epidemiological studies of the general population[27]. In international community samples, prevalence rates for specific phobias in children and adolescents are (2.6–9.1) % with the average near 5%[28]. Lower risk for specific phobias has been reported among Asians and Hispanics compared to Western countries[29]. Specific phobia is also more prevalent among girls than boys[30].

Phenomenology

The DSM IV-TR categorizes specific phobias into five subtypes: animal type, natural environment type, blood-injection-injury type, situational type and another category for fears that do not fit into one of these specific categories. Some of the more commonly occurring phobias in children include fear of heights, darkness, injections, dogs, loud noises, small animals, and insects[31, 32 , 33, 34]. For animal, environmental, or blood-injection injury type phobias the age of onset is typically 12 years or younger[35]. Environmental phobias tend to have an earlier age of onset in boys. The blood-injury-injection subtype has been shown to be significantly more prevalent in females[36]. Animal phobias are also more common in girls with a 3:1 ratio clearly present by age 10 years. Though not specific to children and adolescents, phobias involving lightning, enclosed spaces, and darkness have all been found to be more prevalent in females[37]. Avoidance behaviors in children often take the form of tantrums, crying, and hiding. When the feared stimuli are present, the severity of the fear response and avoidance behaviors indicate the extent of the child's distress.

Comorbidity

Children with specific phobias frequently have comorbid internalizing or externalizing disorders[38,39]. Last et al[38] found that 75% of the children with specific phobias had a lifetime history of additional anxiety disorders (most commonly separation anxiety disorder), 32.5% had a lifetime history of any depressive disorder, and 22.5% had a lifetime history of any disruptive behavior disorder. Similarly, Silverman et al[39] reported that among 104 children with specific phobias majority (72%) of had at least one comorbid diagnosis: 19% had an additional specific phobia, 16% had separation anxiety disorder, 14% had overanxious disorder, and 6% were diagnosed with attention-deficitl hyperactivity disorder.

Social Phobia

The rate of lifetime social phobia in a community sample of adolescents was found to be 1.6%[40] and substantially higher at 14.9% in a clinical sample of children[38]. Mean age of onset for social phobia in clinical samples ranges from 11 to 12 years of age [38,34] and the rate of SP increases with age[40].

Phenomenology

Before the DSM-IV, social phobia was not diagnosed in children and adolescents. Youth who endorsed anxiety and avoidance of engaging with unfamiliar people were commonly diagnosed with avoidant disorder of childhood or adolescence. This diagnosis was excluded from the DSM-IV; children and adolescents who fear social and performance situations are now diagnosed with social phobia. Children with social phobia often have poor social skills and have difficulty initiating and maintaining interpersonal relationships[41,42]. Essau et al,[40] found that the most commonly feared situations in adolescents with social phobia were performing in front of others, public speaking, and engaging in conversations. The most frequent anxiety endorsed by these adolescents was the fear that something would happen to cause them to be embarrassed. Social phobia also seems to have an impact on children's functioning in the classroom. Muris et al, [43] found that higher social phobia symptoms in a nonclinical sample of children (10–12 years old) was associated with poorer general classroom functioning, increased difficulty with peer relationships, and lower selfesteem.

Comorbidity

Beidel et al, [41] found that 60% of children with social phobia met criteria for another Axis I disorder. Thirty-six percent of the comorbid disorders were other anxiety disorders. The most common comorbid disorders included the following: 10% of children had GAD, ADHD, or specific phobia; 8% had selective mutism; and 6% had an affective disorder and substance use disorders. Because of the high comorbidity rates of depressive and substance dependence disorders, social phobia places youth at risk for longterm problems across domains of education, social relationships, and employment.

Indian study on social phobia

Study for social phobia done on 421 adolescents in one high-school in India by Mehtalia et al, [44]. Social phobia was present in 12.8%of high school adolescents and was equally common in both genders. This study showed that fear of doing things when people might be watching (51.8%), fear of talking to strangers (33.3%) were common complaints among adolescents with social phobia.

Generalized anxiety disorder

There are limited data on the prevalence of GAD in youth, as it was not diagnosed in children and adolescents until the DSM-IV. Prevalence of GAD in USA for children and adolescents ranges from 2.9-4.6%. Whitaker et al[45] reported a prevalence rate of 3.7% for GAD in a sample of 14 to 17-year-old students. Some investigators found that in comparison to childhood samples, middle adolescent samples report a higher prevalence of GAD [46,47]. Results regarding gender differences in GAD are conflicting. Wittchen et al, [48] found no significant gender differences in the prevalence of GAD. Costello et al, [47] found a higher incidence of anxiety disorders in girls between the ages of 9 and 16, and Kashani et al, [46] found prevalence rates in 14 to 16-year-old adolescents of 12% and 23% for males and females, respectively. Abe et al, [49] suggest that during adolescence, symptoms of anxiety may peak at an earlier age in females than in males.

Phenomenology

GAD is a relatively new diagnosis in children and adolescents. In the DSM-III-R [50]a diagnosis of GAD required a minimum age of 18 years. Instead, youth with excessive worry were diagnosed with overanxious disorder (OAD). OAD was omitted from the DSM-IV [51] and the age restriction was removed from the GAD diagnostic criteria. Common domains of worry in children with GAD include health of significant others, personal performance, family matters, and world issues52. Three studies [52, 53, 54] show that restlessness is the most common and muscle tension is the least common associated symptom endorsed by youth with GAD.

Comorbidity

GAD is often comorbid with other psychiatric disorders. Masi et al, [53]found that 93% of clinically referred youth with a diagnosis of GAD had at least one comorbid disorder. Depression (56%), specific phobia (42%), SAD (31.8%), SP (28%), obsessive-compulsive disorder (OCD) (19.7%), and panic disorder (16.6%)are common comorbid disorders. Externalizing disorders (i.e. ADHD, oppositional defiant disorder, conduct disorder) occurred in 21% of the clinical sample diagnosed with GAD. Children with GAD and a comorbid diagnosis of depression often have a poorer prognosis, greater symptom severity, and longer duration of symptoms when compared with children without comorbid depression [56].

Separation anxiety disorder

The prevalence of separation anxiety disorder ranges from 1.3% in individuals aged 14-16 years to (4.1-4.7)% in children aged 7-11 years with an average prevalence rate of 2-4%. [57]. Other studies showed that prevalence rate of separation anxiety disorder (SAD) is between 3% to 5% [58, 59] It is more likely to occur in children compared with adolescents. Onset is typically at 7 to 9 years of age[38].

Clinical presentation

The key feature of separation anxiety disorder (SAD) is excessive anxiety about separation from primary attachment figures (eg, parents, grandparents). Children with SAD fear that harm will come to themselves or their attachment figures when separated. Other symptoms include distress at the time of separation, somatic complaints when separation occurs or is anticipated, nightmares with themes of separation, shadowing parents in the home, and sleeping with family members. Children with SAD commonly refuse to attend school and are reluctant to go other places without their parents. A distinguishing feature of SAD is that the child's anxiety is alleviated when with parents, whereas in other anxiety disorders, the presence of an attachment figure has minimal effect on symptom presentation [60]. SAD can be short-lived or chronic and persistent.

Comorbidity

One third of children with separation anxiety disorder have comorbid depressive disorder, and as many as 27% have another disruptive behavior disorder, such as attention deficit hyperactivity disorder (ADHD), oppositional defiant disorder, or conduct disorder. Other comorbid diagnoses include generalized anxiety disorder (GAD), specific phobia, and social phobia (SP)[61].

Panic disorder

Panic disorder was once thought to be a disorder found only in adults and very rarely in adolescents. This notion was based on the idea that there is a strong cognitive component to panic disorder that children were incapable of experiencing [62] .However, there is now a large body of evidence showing that panic disorder does occur in children [63]. Panic disorder (PD) in children and adolescents is a disabling and chronic condition, which is accompanied by psychosocial and academic difficulties both during adolescence and into adulthood. Although Panic disorder was thought to be rare in children and adolescents, the prevalence of Panic disorder in community samples ranges between 0.5% and 5.0 and in pediatric psychiatric clinics from 0.2% to 10%[64]. Panic attacks are reported to be equally prevalent in males and females. But severe panic attacks are more common in females. Panic disorder patients can be misdiagnosed of having neurologic, cardiovascular, pulmonary, or gastrointestinal illness.

Phenomenology

Panic disorder in children can be difficult to diagnose. This can lead to many visits to physicians and multiple medical tests which are expensive and potentially painful. Panic disorder often looks different in young people than in adults, because children tend to report the physical symptoms accompanying panic attacks rather than the psychological symptoms. Children having a panic attack may appear to be suddenly frightened or upset with no easily identified explanation. This behavior is often confusing to others. Panic disorder is distinguished by the unpredictability of the panic attacks. Recurrent "out of the blue" episodes of fear or physical discomfort that are brief. Typically, panic attacks reach their maximum in 10 minutes. Recurrent episodes that are accompanied by physical symptoms, such as fast heart rate, difficulty breathing, chest discomfort, choking sensation, dizziness or faintness, trembling, sweating, nausea, or hot/cold flashes. Recurrent episodes that may include psychological symptoms or worries such as the fear of losing control, the fear of "going crazy," or the fear of dying.

Comorbidity

90% child and adolescent patients with panic disorder have comorbid other anxiety disorders (Agoraphobia, GAD, social phobia etc.) and depression [65]. 50% have other comorbid illnesses like conduct disorder, ODD, substance abuse disorder and somatoform disorder [66].

Obsessive compulsive disorder

Obsessive-Compulsive Disorder (OCD) is one of the most prevalent psychiatric disorders affecting children and adolescents and is projected to be among the ten leading causes of global disability by the World Health Organization (WHO European Ministerial Conference; 2005) [67] . In the United States, Flament et al, [68] reported a lifetime prevalence rate of 1.9%, and Valleni-Basile et al ,1994 [69] reported a prevalence rate of 3%. Studies from other than USA reported prevalence rates of OCD in juveniles of 2.3% in Israel [70], 3.9% in New Zealand [71] and 4.1% in Denmark [72]. The prevalence rates of late adolescents are in line with available estimates for adult samples of 1 to 4 percent. In the British Child Mental Health Survey 73 of over 10,000 five to fifteen year olds, the point prevalence was 0.25% and almost 90% of cases identified had been undetected and untreated. Jenike et al [74] refers to OCD as a “hidden epidemic”, primarily because the disorder is frequently unrecognized and is therefore under diagnosed[75].

In a review of 11 studies that reported the clinical characteristics of children and adolescents who had OCD, the mean age of onset of OCD ranged from 7.5 to 12.5 years, (mean,10.3 years)[75, 76]. Among children and adolescent samples boys outnumber girls with OCD by at least 3 to 2. Boys are found to have a greater incidence of pre-pubertal onset while girls have a greater post-pubertal onset[75,77] .Hence, younger samples tend to have a greater proportion of boys, which equalizes as sample age approaches adolescence.

Examination of age-of-onset reports from a variety of samples of individuals has been interpreted to show two main peaks of onset: an early-onset subgroup occurring during the prepubertal and early adolescent period and the other during late adolescence or after puberty. Of note is the consistent observation that the prepubertal onset group is more highly familial and is more commonly associated with tic disorder co morbidity.

Indian studies on obsessive compulsive disorder

A study involving 13-16 year old, 1100 high school children in Bangalore showed a point prevalence rate of 1.45% and 5.18% respectively for clinical and subclinical OCD respectively 78. Clinic based prevalence rate at the Child and Adolescent Psychiatric Services of the National Institute of Mental Health and Neurosciences (NIMHANS), Bangalore in the year 2003 was 2.8% (27 cases out of 935).

In an epidemiological study of child & adolescent psychiatric disorders in urban and rural areas of Bangalore, India [79], 2064 children aged 0-16 yrs, were selected by stratified random sampling from urban middle-class, urban slum and rural areas. The results indicated an OCD point prevalence rate of 0.1 per cent among children aged 4-16 years.

In all the studies of OCD in children and adolescents reported from India, males have outnumbered female subjects[80, 81, 82, 83]. The proportion of male subjects has ranged from 63% to 85%.

Phenomenology

The phenomenology of OCD in children, adolescent and adult is strikingly similar and perhaps this similarity facilitated the recognition of the disorder in children and adolescent and encouraged clinical studies in this population.

Studies suggest that OCD in young children is characterised by a range of different types of obsession and compulsions, the most common obsession in young people includes worries about dirt and contamination, thought of something terrible happening and concerns about illness or death (Thomsen, 1999). In younger children concern about contamination, aggression and exactness or symmetry is most common[84, 85, 86]. As children get older the type of obsession may change to include obsessions of a sexual or religious nature.

The most common compulsion in young people includes washing, checking, repeating, ordering/ arranging and counting (Thomson, 1999). However, symptoms generally change over time and it is unusual for young people to only ever carry out one type of compulsion [87, 88] . This phenomenology is very much similar to adult indicating the isomorphism between childhood and adult presentation of OCD.

Children and adolescent had significantly higher rate higher rate of aggressive and catastrophic obsession compared to adults. In addition the rate of sexual obsession increased significantly from childhood to adolescents to level similar to that of adults. Religious obsessions are more frequently in adolescents than either in children and adults. Hoarding compulsions are found significantly more often in the child subjects, compared with adolescents and adults[76].

In addition, Swedo et al, [85] suggest that children with OCD frequently display compulsions without well-defined obsessions and that this may be because young children lack the cognitive ability to be able to articulate their internal cognitive process. Compulsion without obsession are frequently tactile (e.g. touching, tapping and rubbing rituals) and may occur more often in young people with co morbid tic disorder[89].

Children frequently display symptoms other than typical washing or checking rituals (e.g., blinking and breathing rituals) [90].

One review on paediatric OCD [76] indicated that although the majority of children exhibit both multiple obsessions and compulsions (mean number over lifetime was 4.0 and 4.8 respectively. [91] compulsions only without obsessions were more common in children than adolescents.

Co-morbidity

One of the important differences between child and adult OCD is the pattern of co-morbidity. Filament et al, [92] report life time co-morbidity rate of 75% for other psychiatric disorders, In an Indian study 69% of the subjects had a co-morbid disorder[81].

Nearly one third to one half of the children with OCD seems to have a current or past history of another anxiety disorder. In children, overanxious and separation anxiety disorders are the commonest whereas in adolescents, generalized anxiety and panic disorders are the commonest. Prevalence of depression is in the range of 13% to 70%. Tic disorders have been reported in 17% to 60% of juvenile subjects[81]. At least 50% of children with TS develop OC symptoms or OCD by adulthood. Rarely schizophrenia can co-occur with OCD. A high prevalence of attention deficit hyperactivity disorder (ADHD), conduct disorder and oppositional defiant disorder have been reported in children with OCD[86] .In Indian studies rates of ADHD have ranged from9% to 18%.[81, 83] , whereas in other studies rates range from 33% to 57%. [86] .There are also reports suggesting increased prevalence of bipolar disorder in children with OCD.

Post-traumatic stress disorder

PTSD symptoms have been identified in adults for more than a century, recognition of this disorder in children only began to emerge recently. Lifetime prevalence of PTSD is 8%[93] Studies conducted within one year of disasters report prevalence figures for PTSD such as: 4.5% three months after the 1999 earthquake in AnoLiosia, Greece [94] 5% three months following hurricane Hugo [95] and 3% of males and 9% females 6 months after Hurricane Andrew[96] Studies reporting prevalence at one year following natural disaster range from no syndrome after a flood [97] to 3.8– 6.2% after Hurricane Hugo[98]; 26.9% after super-cyclone of Orissa [99] and 28.6% mild to moderate PTSD following Northridge Earthquake [100]. This variation in incidence and course of PTSD depend on various factors, including the type of trauma, the proximity to the stressor, and the reaction of the child's parents.

Phenomenology

Manifestation of post-traumatic stress symptoms and syndrome may depend upon variation in cultural and societal response to stress, coping strategies and available support. It has been emphasized that there are numerous cultural considerations which are to be responded to in understanding and treating PTSD across cultures[101]. The phenomena of re-experiencing, numbering and avoidance and hyper- arousal in children are comparable to that in adults. However there can be major differences in which these manifesting themselves. For example, in young children, repetitive play may occur in which themes or aspects of the trauma are expressed; the dreams may not have any specific trauma related contents and the children may actually re-enact the trauma instead of re-experiencing it. Numbing or avoidance may take the form of restlessness, hyper-alertness, poor concentration and behaviouralproblems[102].

The chronicity and the type of trauma can influence the manifestation of PTSD. Acute PTSD presents more with typical physiological hyper-arousal and re-experiencing and sleep problems, the chronic variety presents with dissociation, restricted affect, sadness and detachments [103]. Terr et al,[104] described that type l trauma results in re-experiencing, avoidance and increased arousal and type ll results in denial, numbing, dissociation and rage. Children may have periods during which they have only re-experiencing, or only avoidance and numbing, which

alternate between each other, rather than exhibiting both groups of symptoms simultaneously [105,106, 107].

The results of follow-up studies of children with PTSD are unclear and contradictory. There are reports of the prevalence/ symptom severity decreasing[108], remaining the same[109, 110] or increasing [111] during the follow-ups. The knowledge regarding the PTSD symptoms in children over time and their associated outcomes are not yet clearly known.

Co-morbidity

ADHD, depression, conduct disorder, oppositional defiant disorder and substance use are highly co-morbid with PTSD [112].

Indian studies on post-traumatic stress disorder

Study was done by Margoob et al , 2006[113] on 100 cases of PTSD in children, in the age range of 03-16 yrs, in Govt. Psychiatric Diseases Hospital, Srinagar. The most common traumatic event experienced was witnessing the killing of a close relative (49%), followed by witnessing the arrest and torture of a close relative (15%), (11%) witnessing night raids, (14%) caught up in cross firing, (4%) beaten up / tortured and (7%) hearing about killing of a close relative. Majority (64%) of cases presented with the complaint of somatic complaints (i.e. Headache, stomachache, breathlessness, palpitations, loss of appetite, and insomnia), followed by (50%) episodes of loss of consciousness/ conversion fits; (32%), irritability/outbursts of anger; (22%) decreased school performance, (18%) loss of interest and pleasure, (4%) with stammering and (3%) with enuresis.

Another study done by Kar et al, [114] on 447 children and adolescents after a super-cyclone in Orissa. Post-traumatic stress disorder (PTSD) was present in 30.6% OF subjects and an additional 13.6% had sub-syndromal PTSD. Most common symptoms are experiencing distress when reminded or exposed to cues(83.4%), actual or preferred avoidance (60.0%), distressing persistent recollection (53%), Difficulty recollecting some aspects of the cyclone (41.6%), hypervigilance (41.2%), difficulty in concentrating (36.9%), exaggerated startle (36.2%), Irritability or outbursts of anger (24.8%), reexperiencing, reliving (24.4 %).

Acute stress disorder

Acute Stress Disorder (ASD), introduced in DSM-IV, has received relatively little attention in younger populations Unlike PTSD, which is diagnosed at least four weeks post-trauma, ASD is diagnosed two days to four weeks post-trauma. ASD also differs from PTSD in being explicitly conceived as a dissociative response to trauma requiring at least three of a possible five dissociation symptoms. An important public health marker of the utility of ASD is its ability to predict later PTSD, thus allowing clinicians to focus resources on susceptible individuals [115].

Treatment of anxiety disorders in children and adolescents

The evidence that childhood anxiety disorders cause suffering and impairment and may entail long term liability highlight the need for effective treatments. A multimodal approach is advisable and psychotherapy should be considered as an integral part of the management of childhood anxiety disorder[116]. Pharmacotherapy should preferably be used as adjunct to behavioral or psychotherapeutic interventions rather than as a sole intervention. This approach is important to prevent symptom return after discontinuation of medications.

Currently, an SSRI is the first line choice medication for children and adolescent with anxiety disorders. Efficacy and safety of fluovoxamine& Paroxetine in children and adolescent with separation anxiety disorder , GAD and/or social phobia, of sertraline for youth with GAD, and of fluoxetine for youth with SAD, GAD and/or social phobia has been documented in well designed trials [116]. For separation anxiety disorder Cognitive and behavioral techniques, including contingency management, modeling, relaxation, and exposure based Treatments are often used. For Social Phobia CBT and SSRIs are the first line treatments. Depending on presentation, treatment may begin with CBT alone or CBT plus an SSRI . Treatment for specific phobias differs from CBT of SAD, GAD and social phobia. It primarily involves graded exposure to the feared stimuli, imaginary or actual, according to hierarchy constructed by the child progressing gradually from mild to most significant fear .

For Panic Disorder CBT again is the first line of treatment. Components include 1. Education about the physical experience associated with panic attacks. 2. Breathing and relaxation exercises. 3. Interceptive exposure (i.e. exposure to cues associated with panic). 4. In vivo exposure. 5. Cognitive modification to reduce catastrophic misinterpretation. In practice an SSRI may be added to CBT.

For Obsessive Compulsive Disorder Choice of first line therapy depends on the symptom pattern, severity, and the patient's and family's preference. The technique of CBT needs to be modified in accordance with the developmental age of the child. CBT for pediatric OCD basically encompasses three techniques 1. Exposure and Response prevention 2. Cognitive therapy and 3. Relaxation training. SRIs used in OCD include clomipramine and the

SSRIs i.e. fluoxetine fluvoxamine, paroxetine and sertraline. Many experts and consensus guidelines recommend CBT as the first line approach for the majority of children and adolescents with OCD.

Conclusion

Much has been discovered about childhood anxiety disorders over the past decade, with increased understanding of the phenomenology and associated risk factors and the development of more effective psychotherapeutic and pharmacologic treatments. Early detection and effective treatment may reduce the impact of anxiety on academic and social functioning in youth and may reduce the persistence of anxiety into adulthood.

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Parenting Stress in the Parents with Mentally Retarded Children

Kumari Anjali, Prakash Jai, Kiran Manish

ABSTRACT

Background: *Parenting refers to the aspects of raising a child in terms of physical, social, emotional as well as over all development of the child aside from the biological relationship.*

Aim: *The present study has been undertaken with the aim to assess parenting stress in the parents of mentally retarded children and parents of normal healthy children.*

Methods: *A total of 100 respondents (50 parents of mentally retarded children and 50 parents of normal children) were selected by using purposive sampling technique. Further socio-demographic data sheet, General Health Questionnaire- 12 and Parenting Stress Index Scale were administered.*

Results: *Parents of mentally retarded children showed parenting stress in comparison to parents of normal children. Parents of mentally retarded children had feeling of inability to handle things very easily. They feel trapped by their responsibilities as a parent. Parents of mentally retarded children showed parental distress and found to be having feeling that they are unable to do new and different things, they almost were unable to do things the way they like to do. They used to perceive that child rarely does things for them which make them feel good. They used to perceive that their child cry more than other children and generally wakes up in a bad mood and easily gets upset over the small things.*

Conclusions: *Parents with mentally retarded children showed stress in the areas related to defensive response, parental distress, parent child dysfunctional interaction and difficult child in comparison to parents of normal children.*

Key words: ***Mentally retarded children, parental stress, defensive response and parent-child dysfunctional interaction.***

INTRODUCTION

Parenting is the process of promoting and supporting the physical, emotional, social and intellectual development of a child from infancy to adulthood. Parenting refers to the aspects of raising a child aside from the biological relationship. Usually, parental figures provide for a child's physical needs, protect them from harm, and impart in them skills and cultural values until they reach legal adulthood. The degree of attention parents invest in their offspring is largely inversely proportional to the number of offspring the average adult in the species produces. Davies, Martin (2000)[5]

Raising a child who is mentally challenged requires emotional strength and flexibility. The child has special needs in addition to the regular needs of all children and parents can find themselves overwhelmed by various medical, caregiving and educational responsibilities whether the special needs of the child are minimal or complex, the parents are inevitably affected support from family , friends , the community or paid caregivers is critical to maintaining balance in the home.

Parents of mentally challenged children commonly experience a gamut of emotions over the years. They often struggle with guilt. Occasionally, parents feel embarrassed or ashamed that their child is mentally disabled. Physical exhaustion can take a toll on the parents of a mentally challenged child. The degree of physical exhaustion is usually related to the amount of care needed. The American Academy of family physician relates that these issues can cause significant caregiver stress. The parent of a child with developmental disabilities may have to deal with complex issues related to education. Raising a child with a mental challenge may be more expensive than raising a typical child. These expenses can arise from medical equipment and supplies, medical care, caregiving expenses, private education, tutoring, adaptive learning equipment or specialized transportation.

Gath(1977)[8] studies the effect of an abnormal child upon the parents on 30 families with a newborn baby with Down syndrome and 30 families with a normal baby. Both groups were followed for 18 months and were interviewed six times. Few differences could be found in mental or physical health of the two groups of parents, but marital disharmony was found in nine families with Down syndrome and in none of the controls. Beckman (1991)[1] compared stress of 27 mothers and fathers of children with disabilities with the parental stress of 27 mothers and fathers of normal children. The children with disabilities were moderately to severely retarded children. Mothers generally reported more stress than fathers which appear to have different perception of the effect of their child on their lives demonstrated by the different levels of stress on specific sub domain. Jarvis and Creasey (1991)[12] in a sample of 32 families, found that for both mothers and fathers, parenting stress was significantly related to the insecure attachment of infants. Importantly, the strongest relationships were found between the father's Child Domain scores and insecure attachment in the child. Bruce et al.(2002)[3] Studied early evidence of behaviour problem in 225 five year old children with or without developmental delays and the relative impact of cognitive delays and problem behaviour on the parents. Result showed parenting stress was higher in delayed condition families. Regression analyses revealed that the extent of child behaviour problems was a much stronger contributer to parenting stress than was the childs cognitive delay. Moran et al. (1992)[17] in a study reported that mother of developmentally delayed children found their children to be more difficult than did mothers of normal children (N=19). Their children's cognitive delay was strongly correlated with stress in the child domain. Orr et al. (1991)[19] employed the PSI to determine the relationship between family stress and coping with the stress on a sample of 86 families with children who were mentally retarded. The researcher found that a greater level of stress was associated with the severity of the child's behaviour problems. In addition, a fewer number of resources were also related to higher levels of stress. Gupta and Kaur (2010)[10] in a study examined stress among parents of children with intellectual disability. 102 parents formed the sample of this study, 30 of whom had children without disability. This study has two parts physical stress and mental stress. Results show that, most parents of children with intellectual disability experience stress, physical and mental stress are significantly correlated, gender differences in stress experience occur only in the mental area and parents have higher mental stress as compared to physical stress. Holt (1958)[11] studied two hundred and seven (207) families with a subnormal child living at home in Sheffield found that nineteen percent (19%) of the mother exhausted by physical work and emotional stress .Father were said to suffer to a lesser degree but marriages were strained by parental quarrelling

The present study has been undertaken with the aim to assess parenting stress in the parents of mentally retarded children.

METHODOLOGY

SAMPLE

A total of 100 respondents (50 parents of mentally retarded children and 50 parents of normal children) were recruited from OPD of RINPAS by using purposive sampling technique. Only those parents were included in the study whose children were in the age range of 5-15 years. Parents either father or mother who were staying with the child were considered for the study. After having informed consent, participants who were able to understand test instruction were taken for further assessment.

TOOLS

Semi-structured personal data sheet specially designed for study have been used. It contains information about mentally retarded child and parents, socio-demographic variables like age, sex, education, martial status, religion and clinical variables related to child. **General Health Questionnaire** developed by David and Williams (1988) was administered for detecting psychiatric disorders among respondents in community setting and non psychiatric clinical setting. Reliability coefficient of the questionnaire ranged from 0.78 to 0.95 in different method. **Parenting Stress Index Scale** developed by Richard in 1983 has been used to assess parenting stress in the parents of mentally retarded children and parents of normal children. This scale is meant for assessing four domains – Defensive Responsiveness, Parental Distress, Parent–Child Dysfunctional Interaction, and Difficult Child.

PROCEDURE

The study conducted at Ranchi Institute of Neuro Psychiatry and Allied Sciences (RINPAS) Ranchi, Jharkhand. After having informed consent all respondents (parents of both mentally retarded children and normal children) has been selected. Subjects were explained about the objectives of the study then GHQ-12, Parenting Stress Index Scale were administered to the individual subjects far the purpose.

STATISTICAL ANALYSIS

The data obtained have been analyzed by using the computer software program, Statistical Package for Social Science Version 16.0 (SPSS-16.0). Parenting Stress Index Scale has been analyzed by using 't' test.

RESULT AND DISCUSSION

Table : Showing Stress between Parents of Mentally Retarded Children and Parents of Normal Children on Parenting Stress Index Scale.

Subjects	Parents of Mentally Retarded Children(N=50)	Parents of Normal Children (N=50)	df	t-value
Variables	**Mean ± SD**	**Mean ± SD**		
Defensive Response	26.16 ± 4.81	19.30 ± 4.25	98	7.55**
Parental Distress	44.80 ± 7.98	30.86 ± 7.07	98	9.24**
Parent- Child Dysfunctional Interaction	43.52 ± 5.63	22.48 ± 5.48	98	18.91**
Difficult Child	46.46 ± 8.11	25.12 ± 7.12	98	13.97**

** Significant at 0.01 level.

It is clear from the table that parents with mentally retarded children showed parenting stress in the area related to Defensive Response in comparison to parents of normal children and difference between these two groups was significant at 0.01 level (PMR: M=26.16± 4.81, PNC: M=19.30±4.25; t=7.55, P>0.01). Further it has been found that parents with mentally retarded children were having feeling that they cannot handle things very easily. They feel trapped by their responsibilities as a parent. There are quite a few things that bother them about their life. They feel that having a child has caused more problems than they expected in maintaining relationship with their spouse. They also found themselves alone and without friends, they were not as interested in people as they used to be earlier. This finding has been supported by Moudgil et al. (1985)[18] who have concluded that parents of mentally retarded children found to be depressed and aloofish, disturbed marital harmony and interpersonal relationship. Similar trend has also been observed by Majumdar et al (2000)[16].

It is also noticed that parents of mentally retarded children showed parenting distress in comparison to parents of normal children and difference between these two groups was significant at 0.01 level (PMR: M=44.80±7.98, PNC:M=30.86± 7.07; t=9.24, P>0.01).Further it has been found that parents of mentally retarded children were having feeling that they are unable to do new and different things, they almost were unable to do things that they like to do. They are unable to enjoy themselves in social gathering usually. They also feel like that they don't enjoy things in comparison to parents with normal children. Beckman (1991)[1], Jarvis and Creasey (1991)[12]and Sloper et al (1991)[24] also witnessed similar finding of high level of parental stress in the parents of mentally retarded children.

It is also observed that parents of mentally retarded children showed parenting stress related to parent child dysfunctional interaction in comparison to parents of normal children and difference between these two groups was significant at 0.01 level (PMR:M=43.52±5.63, PNC:M=22.48±5.48; t=18.91, P>0.01).Further it has been found that parents with mentally retarded children were used to perceive that their child rarely does things for them that make them feel good. Sometimes they feel their child doesn't like them and doesn't want to be close to them. They found that their child smiles much less than they expected. When playing, their child doesn't often giggle or laugh and doesn't seem to learn as quickly as most of the other children. Their child is not able to do as much as they expected and it takes a long time and also it is very hard for their children to get used to new things in comparison to parents with normal children. Finding of the present study is consistent with Jarvis and Creasey (1991)[12]. In their study they have concluded that parents of mentally retarded children show insecure attachment the child. Distractibility, demandingness and unacceptability has been found by Orsmond and Baratt (1999)[20] and Ricci et al. (2003)[22].

It is also seen that parents of mentally retarded children showed parenting stress related to difficult child in comparison to parents of normal children and difference between these two groups was significant at 0.01 level (PMR: M=46.46± 8.11,PNC:M=25.12± 7.12; t=13.97, P>0.01). Further it has been also found that parents with mentally retarded children were used to perceive that their child seems to cry more often than most children. Their child generally wakes up in a bad mood. They also feel that their child is very moody and easily gets upset over the small things. They also found that their child's sleeping or eating schedule was much harder to establish than they expected. There are some things which child does and that really bother them a lot. Their child turned out to be more of a problem than they had expected and child makes more demands on them than most children in comparison to parents of normal children.

Similar trend has been observed by other researchers, incompetence, depression, health problems and role restriction (Orsmond & Baratt,1999)[20], day to day struggles of raising a child with mentally retarded (Kravetz et al,1993) [15], adaptability problem and demandingness (Ricci et al,2003)[22] perceived difficult child in terms of difficulty in feeding, bathing and dressing (Erickson &Upshur,1989)[7].

CONCLUSION: Parents with mentally retarded children showed stress in the areas related to defensive response, parental distress, parent child dysfunctional interaction and difficult child in comparison to parents of normal children.

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QUALITY OF LIFE IN THE CAREGIVERS OF SCHIZOPHRENIC PATIENTS

Mondal Anwesha, Ali Mir Shahid, Mahto J, Prakash Jai

ABSTRACT

Background: *Caregivers play an important role in the support, management, recovery and care of schizophrenia patients throughout life. Caregivers not only manage the patient in home situation but also help in improving the condition and prevent from further relapse. While supporting the patients emotionally, financially and socially it often results in feelings of burden in caregivers. This affects the caregiver's overall life and specifically their quality of life adversely.* ***Objective:*** *The present study is an attempt to explore the quality of life in the caregivers of inpatient and outpatient schizophrenia patients.* ***Method:*** *A total of 40* ***s****chizophrenia patients, 20 inpateint and 20 outpatient, along with their caregivers were selected by using purposive sampling technique from PGIBAMS, Raipur (C.G). Brief Psychiatric Rating Scale (BPRS) was administered on the patient to assess the severity of the symptoms and WHO-Quality of Life-Bref was administered on the caregivers to assess the quality of life.* ***Results:*** *Caregivers of inpatient schizophrenia patient showed disturbances in social functioning in terms of impaired interpersonal relationships and poor social support than caregivers of outpatient schizophrenic patients. Severity of symptoms has been found to be negatively correlated with physical health area of the caregivers indicating lack of energy, fatigability, impaired work performance, disturbed sleep and inability to get relaxed.* ***Conclusion:*** *The study highlights that the caregivers of inpatient schizophrenic patients face problems in the social functioning area, personal relationships and support system in comparison to the caregivers of outpatient schizophrenic patients. Caregivers also exhibited problems in their physical health as the severity of symptoms in schizophrenic patients increase.*

Key words : - ***care-givers, quality of life, schizophrenia***

INTRODUCTION

Quality of life refers to the satisfaction of an individual's values, goals and needs through the actualisation of their abilities or lifestyle" (Emerson, 1985). Quality of life is a broad concept that incorporates different aspects of life such as social recognition, social satisfaction, security, physical well being, philosophy of life. (Oort, et al., 2005). It is affected in a complex way by the person's physical health, psychological state, personal beliefs, social relationships and their relationship to salient features of their environment"(Oort, 2005). The WHO de? nes Quality of Life as 'an individual's perception of their position in life in the context of the culture and value systems in which they live, and in relation to their goals, expectations, standards and concerns'. Quality of life is considered to be a result of physical, mental, social and spiritual well-being (Ferrell, 1995).

Caregivers' quality of life get affected adversely while caring the patients suffering from schizophrenia because it is a disabling and severe psychiatric disorder. It effects the patients suffering with it as well as the functioning and psychosocial well being of caregivers. Main caregiver is the person belonging to the patient's informal support system who takes the responsibility of the patient and who commits most of his or her time to that task without receiving any economic retribution (Dwyer, et al., 1994). A caregiver helps a patient with his or her activities of daily living. This responsibility often creates a burden and effects the quality of life of the caregiver viz. social, personal, economic etc.

A vast amount of studies have proposed that there is a significant decrease in the quality of life of the caregivers of schizophrenia patients. Burden on relatives/caregivers has been found associated with an important reduction in their quality of life, causing damage in caregiver's health condition (Fadden, et

al., 1987). Poor quality of life of caregivers of individuals with schizophrenia has also been observed by Boyer, et al., (2012).

Research on caregivers' quality of life is thus of importance both for the caregivers themselves and indirectly for patients' health. The aim of this present study is to assess the difference in quality of life of the caregivers of inpatient and outpatient schizophrenia patients.

METHODOLGY

Sample

For the purpose of study 40 patients diagnosed as schizophrenia according to ICD-10 (WHO, 1992) with a history of illness of past two years were selected. Twenty subjects were selected from IPD and 20 subjects from OPD of Post-Graduate Institute of Behavioral and Medical Sciences, Raipur using purposive sampling method. For the caregivers, both male, female with no prior history of any kind of physical or mental illness was included in the study.

Tools

Socio-Demographic and Clinical Data Sheet: Self prepared socio-demographic and clinical data sheet was used to collect the information regarding patient's and their caregiver's age, sex, religion, family type, family history, income and occupation. Additional information was also gathered regarding type of admission, diagnosis of patient, persons sharing responsibility of patient, earning members in family, knowledge about patient's illness and attitude towards illness.

Brief Psychiatric Rating Scale (BPRS): The Brief Psychiatric Rating Scale is a 24-item measure that assesses the severity of mental illness (Ventura et al., 1993). The 24 items are rated on a 7-piont scale ranging from 'not present' to 'extreme severe'. A total score ranging from 24 to 168 can be calculated with higher scores reflecting higher severity. Reliability coefficients of 0.56 to 0.87 have been reported by the authors.

The World Health Organization Quality of Life (WHOQOL-BREF):

It is 26-item measure to assess the quality of life (WHO, 2004). Items are rated on 5-point scale and measures the quality of life in four domains namely physical health, psychological, social relationships and environment along with the overall quality of life. The alpha score of all domain ranges from 0.59 to 0.87, Cronbach alpha of the all domains are 0.87, the factor loadings of the item ranges from 0.52 to 0.84.

PROCEDURE

Caregivers of 40 diagnosed schizophrenia patients (as per ICD 10) were selected through purposive sampling technique, 20 each from inpatient and outpatient of Post-Graduate Institute of Behavioral and Medical Sciences, Raipur. Both the groups were matched with respect to age and sex. After having informed consent for the study from the care givers, socio-demographic details of both patients and caregivers were collected. Brief Psychiatric Rating Scale was administered on the patients to find out the severity of the symptoms. Finally, quality of life of care givers of the patients was assessed by using World Health Organization Quality of Life- BREF.

STATISTICAL ANALYSIS

Scores thus obtained were analyzed with Statistical Package for Social Sciences (SPSS). Descriptive statistics (percentages and mean), Chi-Square & t-test were used to see the significant differences, if any.

RESULTS AND DISCUSSION

Table 1 shows the socio demographic profile of caregivers of outpatient and inpatient schizophrenia patients. It is clear from the table that no significant difference has been found among the caregivers of outpatient and inpatient schizophrenia patients. It was observed that most of the caregivers of outpatients were males, literates, by occupation farmers, married, were spouse in relation to the patients who mostly spent more than 12 hours, had a family income of less than 5,000, had knowledge about mental illness and had a positive attitude towards patient's mental illness. It is also reflected in the table that majority of the the caregivers of inpatients were females, literates, housewife by occupation, married, illiterates, were farmers by occupation, married, were parents in relation to the patients who mostly spent more than 12 hours, had a family income of less than Rs. 5, 000, had knowledge about mental illness and had a positive attitude towards patient's mental illness.

Table 1: **Sample Characteristics**

Variables		M	(SD)	t-value
Age	OPD	43.20	(13.87)	0.66
	IPD	46.30	(15.72)	

Variables		OPD		IPD		X^2
		N	%	N	%	value
Sex	Male	13	(65.0%)	7	(35.0%)	3.60
	Female	7	(35.0%)	13	(65.0%)	
Education	Literate	12	(16.0%)	7	(35.0%)	0.10
	Illiterate	8	(40.0%)	13	(65.0%)	
Occupation	House wife	6	(30.0%)	9	(45.0%)	5.82
	Student	1	(5.0%)	1	(5.0%)	
	Service	5	(25.0%)	0	(0.0%)	
	Farmer/labour	8	(40.0%)	10	(50.0%)	
Marital Status	Married	17	(85.0%)	14	(70.0%)	4.49
	Unmarried	3	(15.0%)	2	(10.0%)	
	Widow/Divorced /Separated	0	(0.0%)	4	(20.0%)	
Relation with the patient	Spouse	9	(45.0%)	6	(30.0%)	3.92
	Parents	7	(35.0%)	12	(60.0%)	
	Sibling	2	(10.0%)	2	(10.0%)	
	Other	2	(10.0%)	0	(0.0%)	
Hours spend daily	12 hours	8	(40.0%)	8	(40.0%)	0.00
with the patient	>12 hours	12	(60.0%)	12	(60.0%)	
Family income	5,000	9	(45.0%)	13	(65.0%)	1.84
	5,000 – 10,000	5	(25.0%)	4	(20.0%)	
	>10,000	6	(30.0%)	3	(15%)	
Knowledge about	Mental illness	16	(80.0%)	17	(85.0%)	4.03
patient's illness	Effect of supernatural	1	(1.0%)	3	(15.0%)	
	Any other	3	(15.0%)	0	(0.0%)	
Attitude of family	Positive	20	(100.0%)	18	(90.0%)	2.11
members towards illness	Negative	0	(0.0%)	2	(10.0%)	

Table 2: **Quality of Life among Caregivers of outpatient and inpatient schizophrenia patients**

Subjects / Variables	Schizophrenia Patients				t-value
	OPD Patients		IPD Patients		
	M	(SD)	M	(SD)	
Physical Health	14.20	(2.41)	13.85	(2.03)	0.49
Psychological Well Being	13.75	(2.14)	1.90	(2.59)	1.13
Social Relationships	13.70	(2.29)	11.45	(4.04)	**2.16***
Environmental Conditions	13.15	(1.32)	12.85	(2.15)	0.42

*p 0.05 level (2-tailed)

Table 2 reflects quality of life among the caregivers of outpatient and inpatient schizophrenia patients. It is quite obvious from the table that caregivers of outpatient schizophrenia patients were having less health related problems in comparison to the caregivers of inpatient schizophrenia. However, difference was not found significant statistically (OPD: M= ±13.76, t= 0.49; IPD: M= ± 13.85, t=0.49; p 0.05). It is also noticed that psychological problems were less in the caregivers of outpatient schizophrenic patients than inpatient schizophrenic patients but the difference was not significant statistically (OPD: M= ±13.75, t= 1.13; IPD: M= ± 1.90, t=1.13; p 0.05). Caregivers of outpatient schizophrenic patients were more satisfied in their environmental conditions than the caregivers of inpatient schizophrenia patients but the difference was not significant statistically (OPD: M= ±13.15, t= 0.42; IPD: M= ± 12.85, t=0.42; p 0.05).

It has been noticed that in the area of social relationships, significant difference is there among the caregivers of outpatient and inpatient schizophrenia patients and the difference is significant statistically (OPD: M= ±13.70, t= 2.16; IPD: M= ± 11.45, t=2.16; p 0.05). Quality of life of caregivers of inpatient schizophrenic patients has more problems in social functioning like impaired interpersonal relationships, poor social support from other relatives, problems in sexual activity have also been found in some cases.

Studies have shown that admitted schizophrenic patients' caregivers had lower level of satisfaction in social relations domain (**Galuppi**., et al, 2010). Solanki (2010) has concluded negative effect of illness on social functioning of the family members' of schizophrenic patients in a study in Jaipur, India. Another study on admitted schizophrenic patients by Saffar & Yaseen (2009) reported lower quality of life in the domain of social relationships. The difference in quality of life of care givers of outpatients and inpatients may be because of difference in severity of symptoms as inpatients has relatively more severe symptoms than outpatients. Another reason may be that in India, families and social relationships are given very high importance and play an integral part in everybody's lives. If in any family, one of the members suffers from mental illness, the other family members play a very important role in providing support to the person. Because of that, caregivers often don't get time for other issues. In case when the patient is staying at home, at times the caregivers can discharge their psychological and occupational responsibilities, keeping some other family member to watch and care for the patient. But when the patient is admitted in a hospital, the family members has to be more alert regarding the functioning of the patient in hospital, to visit there, in case of emergency. As a result social relationships gets affected due to the presence of disagreements, conflicts among the other members of the family causing lack of social support

and more damage in caregivers' social life. In addition, some close relatives might go away avoiding the responsibility to take care of such patients. Jungbauer et al., (2002) reported that in a study majority of the caregivers in India felt that the patients' illness prevents them from having a satisfying relationship with the rest of the family members and other people.

Table 3: Correlation among the BPRS and WHOQOL-BREF

Variables	Physical Health	Psychological Well Being	Social Relationships	Environmental Conditions
BPRS	**-0.325***	-0.179	0.132	-0.127

*p 0.05 level

Table 3 shows the correlation between BPRS and WHOQOL-BREF scores in the caregivers of outpatient and inpatient schizophrenia patients. It is reflected in the table that there is a significant negative correlation in the area of physical health and BPRS scores (r=-0.325; p 0.05). No significant correlation was found between BPRS and WHOQOL-BREF scores in other areas among the caregivers of outpatient and inpatient schizophrenia patients. It is also clear from the table that the more severe the illness was, the more negative effect it had on the physical health of the caregivers. Previous studies have shown that physical, emotional, economic distress affect negatively caregiver's physical health as a result of a number of unfulfilled needs such as restoration of patient functioning in family and social roles, economic burden, lack of spare time. Decreased physical health may be associated with caregivers' burden, lack of social support, course of the disease and family relationships problems. (Caqueo-Urizar et al., 2009). If the caregiver is working, then working life is also significantly affected. At time, they had to leave their jobs, modify their working hours or change to another job. Moreover, in some cases, stress seemed to be associated with a triple shift: job, household duties, and care for a patient. As a result of which stress problems, anxiety and depression were commonly observed in caregivers. Lower level of satisfaction in physical health domain was reported by Bobes et al (1996) on the caregivers of a sample of 78 schizophrenic patients.

CONCLUSION

Quality of life of caregivers of inpatient schizophrenic patients has more problems in social functioning like impaired interpersonal relationships, poor social support from other relatives, problems in sexual activity have also been found in some cases. Caregivers face a lot of problem in their physical health also like lack of energy, fatigability, pain, discomfort, disturbed sleep, inability to relax and impaired work performance when the symptoms of the schizophrenic patient increases. However, there are a few limitations of this study like the sample size was small, other variables like burden, social support was not included, gender differences with respect to caregivers was not assessed.

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Psychiatric Morbidity among Women Engaged In Commercial Sex Work in Kerala

Jins Mathew and Sengar K S

ABSTRACT

Sex is basic primary instinct inherited into human being and all other animals too. It is inseparable part of life of living creatures. However, time to time the concept of sex and sexuality was changing. Sex work has always raised a concern all over the world. However if its focus of concern changed over time that is to say, it was about social morality in olden time and it changed into mainly a health issue in current scenario in India, Government funds through NACO is executed through its states. However the main focus is not the prevention of sex work but rather change of sex behavior to prevent AIDS and STD. Organizations have been focusing on the rehabilitation of women involved in commercial sex work for years however the main focus of rehabilitation was on occupational rehabilitation, rare concern was recorded regarding the psychological wellbeing as well as rehabilitation. ***Aim****: The present study aim to focus on the psychiatric morbidity among those women, involved in commercial street sex work.* ***Method:*** *30 women actively involved in commercial street sex work and 30 women who are not involved in any form of commercial sex work were drawn as the sample from Ernakulam district of Kerala state, India, for the study. They were administered with MCMI-III to identify the psychiatric morbidity.* ***Results****: The study resulted in revealing the different psychiatric morbidity found with those involved in commercial sex workers such as anxiety, dysthymia, alcohol dependence, drug dependence, post traumatic stress disorder and major depression.*

Key words : - ***Commercial Sex Workers (CSW), Sexually Transmitted Diseases (STD), Psychiatric Morbidity***

Introduction

Prostitution has been considered as the oldest profession in the world as the trace of this profession is found in all ancient literature. Sex workers have been an eminent part of all societies. Sexuality has been identified as a basic instinct of human kind by Freud and he was daring to say it in Hippocratic society of that time who were considered as white washed tombs. Sex work has been defined by many authors and one among them is “sexual exploitation of persons for commercial purpose” (Government of India's 'Prevention Of Immoral Traffic Act', 1956). The term prostitution has its origin in Latin called 'prostituere', meaning “to cause to stand in front of”, implicating that one is offering one's body for sale. In the present study we preferred to use the term Commercial Sex Work for prostitution for various reasons. Sex work has its clutches in almost all societies at different levels from top to bottom. Female sex workers commonly called hookers, whores, and escorts are often classified so on the settings in which they work and street prostitutes occupy the bottom level in their hierarchy and Edgley (1989) reports that the latter group often come from poor socio-economical background and have unhappy child hood. They are also differentiated on the basis of attractiveness, age, social class etc. People always wanted an outlet for sexual instinct and sought after sex workers since ages for various reasons, be it frustration with the partner or was curiosity over sex. However, even today, the stigma associated with prostitution is very high. They are often treated like disposables, people like while using it soon after which it is thrown out and don't even like them to be littered around in their premises. Public health concern has mostly been directed to HIV and sexually transmitted diseases but rarely to mental health of those involved in commercial sex work (Weiner, 1996). Sex work is wide spread in India, and occurs on a much larger scale than in many other countries (National AIDS Control Organization, 2006). As the study by I.H.O survey on 1982 at Kamathipura, Bombay, reports that around 100,000 commercial sex workers are approximately estimated to be in Mumbai, 80,000 in Kolkata, 40,000 in Pune, 20.000 in Delhi and 13,000 in Nagpur Prasad, (2004). There are various reasons reported for one to be into the stream of commercial sex work in India, it is reported to be mostly due to poverty, marital breakup or because they are forced into it (NACO, 2006). Physical violence and psychological blackmailing as well as emotional torture are most commonly associated with

commercial sex workers. Gordon & Snyder (1989) say that 80% of the street sex workers are survivors of rape, sexual abuse or incest. Silbert & Pines (1982) report about 'psychological paralysis' among those who prostitute, characterized by hopelessness, immobility, acceptance of victimization which ultimately result in often being victims of violence. Exner et al. (1977) report that street walkers and drug addicted prostitutes showed higher rates of psychological disturbances which is also supported by the similar findings by De Schampheleire (1990), in his cross cultural study of 41 prostitutes in Belgium. The study conducted in Philadelphia by Sovitz & Rosen (1988), explains that commercial sex workers hardly enjoy sex with their partners and however 60% of them report that achieved orgasm only occasionally. Their partners are often intoxicated while having sex and humiliation is commonly associated with. People often hesitate to talk with them or avoid them in public places though over familiarity is shown in darkness which immensely affect their self concept as a segregated group of the society. Edgley (1989) reports that street walkers do not remain in business for very long, some get married and the others die young from drug abuse, disease, suicide, physical abuse from pimps or customers and those who survive become less marketable over time. These adverse situations often lead them to intoxicate themselves prior to sexual contact with their partners, which later often lead to drug addiction, depression, anxiety and various psychiatric morbidities. However, there are also studies reported that there was no way commercial sex work related to psychiatric morbidity (Romans et al. 2001).

Aim

The present study aims to identify the psychiatric morbidity among those women who are involved in commercial sex work.

Tools used

Socio demographic data sheet was used for recording socio demographic variables. Millon Clinical Multiaxial Inventory-III (MCMI-III) developed by Millon et al. (1997) was used to identify the psychiatric morbidity and the areas assessed are Anxiety, Somatoform Disorder, Bipolar: Manic, Dysthymia, Alcohol dependence, Drug dependence, Post Traumatic Stress Disorder, , Major Depression, Thought Disorder and Delusional Disorder.

Sample

Thirty females who are actively involved in commercial street sex work not less than last five years were selected from four different towns of Ernakulam district, Kerala state, India. They were compared with thirty normal controls who were women not involved in commercial sex work.

Methodology

Thirty females who are actively involved in commercial street sex work were selected on the basis of purposive sampling method from different organizations working for the prevention of AIDS, whose age ranged from 20 to 50 years. Duration for the involvement in commercial sex work was kept as minimum for the past five years for the clinical sample. Their active involvement in commercial sex wok was determined on the basis of personal interview with them. The samples involved married, unmarried as well as divorced women. They were then matched with normal controls who were not involved in any form of commercial sex work. Women who are identified with HIV positive, past history of significant head injury and epilepsy or any other major psychiatric or physiological illness were excluded along with those who had family history of psychiatric morbidity in their first degree relatives and those who did not give consent for participating in the study.

Procedure

The sample once drawn by the criteria mentioned above was given socio demographic datasheet. They were then given the Millon Clinical Multiaxial Inventory-III. The scoring and analysis was done only on the clinical syndrome scales as per the instructions given in the Manual and the data was analyzed on SPSS -16 programme for Windows.

Result

The data was collected, coded, analyzed and was executed. The details are given below in the tables.

Table -1: Characteristics of the sample (PSW and Normal Control)

Variables		CSW	Normal control	
Socioeconomic	Lower	27(90.0)	24(80.0)	
status	Middle	3(10.0)	6(20.0)	
Religion	Hindu	21(70.0)	20(66.7)	
	Muslim	2(6.7)	2(6.7)	
	Christian	7(23.3)	8(26.7)	
Domicile	Rural	7(23.3)	7(23.3)	
	Semi-Urban	15(50.0)	18(60.0)	
	Urban	8(26.7)	5(16.7)	
Marital Status	Single	2(6.7)	1(3.3)	
	Married	20(66.7)	25(83.3)	
	Separated	8(26.7)	4(13.3)	
		Mean & SD	Mean & SD	't'
Age		40.60±5.77	39.67±5.17	.66 NS
Education		7.03±2.57	8.07±1.89	1.77 NS

Table -2: The comparison of the groups on different psychiatric morbidities

	Mean rank		z
	Commercial Sex workers(N=30)	Control group (Normal) (N=30)	
Anxiety	35.73	25.27	-2.32*
Somatoform	33.92	27.08	-1.52
Bipolar (Manic)	31.80	29.20	-.58
Dysthymia	35.43	25.57	-2.19*
Alcohol dependence	37.10	23.90	-2.93**
Drug dependence	43.85	17.15	-5.93***
Post Traumatic Stress disorder	34.53	26.47	-5.73***
Thought disorder	37.32	23.68	-1.79
Major depression	37.32	23.68	-3.03**
Delusional disorder	34.60	26.40	-1.83

***=.05, **=.01, ***=.001**

Discussion:

The result shown in the table 1, indicates that the two groups that are commercial sex workers and normal controls were homogenous on their level of education (40.60±5.77; 39.67±5.17; t=0.66) and age (7.03±2.57; 8.07±1.89; t=1.77). The group of CSW for the study were more in number belonged to lower socioeconomic status (90%), Hindu's (70%) hailing from semi-urban area (50%) and 66.7% of them were married living with their husband.

The groups, when compared on different psychiatric morbidities, were found to have significant difference on many of the psychiatric conditions assessed such as anxiety (z=-2.32; p=0.05), dysthymia (z=-2.19; p=0.05), alcohol dependence (z=-2.93; p=0.01), drug dependence (z=-5.93; p=0.001), post traumatic stress disorder (z=-5.73; p=0.001) and major depression (z=-3.03; p=0.01) in comparison to normal controls. The findings of Graham et al. (1994) probably explain its reason that those involved in prostitution undergo various psychological distresses and that the relation they hold with the pimps is of terror and of higher dependency. Violence, the constant humiliation, and social indignity, invariably associated with commercial sex work gradually change their personality itself which is called 'complex post traumatic stress disorder' (Herman, 1992). El-Bassel et al. (1997) say that those who prostitute at the same time having drug use behavior, possibly the former as a means for obtaining the later are of more psychological distress than those who prostitute alone. Burnette et al. (2008) report high prevalence of substance abuse behavior among those involved in prostitution and also by Farley, and Barkan (1998) reporting their immediate need for hospital admission for substance addiction. The present study with high elevations on the scale such as drug dependence (z=-5.93; p=0.001) and alcohol dependence (z=-2.93; p=0.01) also arrives at the similar kind of conclusion. The adolescents involved in commercial sex workers were identified to have suicidal ideation, depression and substance ideation (Yates et al., 1991). High level of depression along with anxiety in commercial sex workers are also reported by Surratte et al. (2005) and depression is very prominent in them regardless of HIV infection status. Women who prostituted were strongest predictors of low self concept and depression. These various studies support the results of the present study that the significant difference found on the variables such as anxiety (z=-2.32; p=0.05), dysthymia (z=-2.19; p=0.05) and major depression (z=-3.03; p=0.01) in comparison to normal group. The study also shows a prevalence of Post Traumatic Stress Disorder among the commercial sex workers (z=-5.73; p=0.001). Ross et al. (2004) have brought about similar results in their study which say the sex workers are often found to have Post Traumatic Stress Disorder and disorder of mood. Pines (1982) also report that people who are into prostitution undergo tremendous violence which might be quite self explaining for the later development of PTSD along with history of childhood physical and sexual abuse (Simons & Whitbeck, 1991; Silbert & Pines, 1981; Meyerding, 1977).

Conclusion

The present study which assessed the psychiatric morbidity among commercial sex workers resulted with similar findings of other researches in the field. The presence of psychiatric morbidity among CSW in comparison to normal control call the attention of mental health professionals for further researches as well as interventions as most of the findings available are from countries outside India.

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COMPARISON OF ARIPIPRAZOLE VERSUS HALOPERIDOL FOR THE TREATMENT OF CHILDHOOD AND ADOLESCENT NON-AFFECTIVE PSYCHOTIC DISORDERS

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ABSTRACT

***Background**: Aripiprazole, a novel antipsychotic, is an effective and safe agent in psychotic disorders of adults but with limited evidence of use in pediatric patients. This study compares efficacy and safety of aripiprazole monotherapy with haloperidol for schizophrenia and related psychotic disorders in children and adolescents.*

***Methods and Materials:** Total 30 patients with ICD-10 DCR diagnosis of schizophrenia and acute psychotic disorders, were assigned to receive either aripiprazole (10-15 mg/day) or haloperidol (10-15mg/day) for four weeks, with each group containing 15 patients respectively. Primary outcome measure was Positive and Negative Syndrome Scale for Schizophrenia (PANSS) while secondary outcome measures were three PANSS subscales and Clinical Global Impressions-Severity of Illness (CGI-S). Assessments were done at baseline and then on weekly basis until endpoint. Extrapyramidal side effects and akathisia were rated weekly by Simpson-Angus Scale (SAS) and Barnes Akathisia Scale (BAS). Body weight and serum prolactin were measured and electrocardiogram recorded at baseline and at end point .*

***Results:** Endpoint improvement for the aripiprazole group was not significantly different from haloperidol group on PANSS total score, and PANSS positive and negative subscale scores. Haloperidol produced significantly greater improvement in PANSS general psychopathology subscale score and CGI-S score at endpoint. Aripiprazole produced significantly less extrapyramidal side effects and weight gain. Haloperidol produced significantly greater elevations in serum prolactin levels while QT (c) changes were nonsignificant in both groups.*

***Conclusions:** This trial shows that aripiprazole therapy was as efficacious as haloperidol in improving both positive and negative symptoms of psychotic disorders in children and adolescents but demonstrated better tolerability.*

Key words- ***aripiprazole, haloperidol, childhood, psychosis***

INTRODUCTION

Childhood and adolescent psychotic disorders are frequently devastating illnesses, characterized by disturbances in language, perception, cognition, affect, volition and socio- occupational functioning. Although estimates of prevalence of psychotic disorders in childhood vary, there is general consensus that they are uncommon[1]. In a study of first admission rates for various psychotic disorders, including schizophrenia, schizophreniform disorder, atypical psychosis and substance induced psychosis, reported prevalence rates were 1.8 per 10,000 in children under age of 13 years and 17.6 per 10,000 at age 18 years[2]. Conventionally, early onset schizophrenia is defined as onset before 18 years of age, and very early onset schizophrenia, defined as onset before age 13. It is estimated that 0.1 to 1.0 percent of all schizophrenic disorders present before age 10, with four percent occurring before age 15.Atypical antipsychotic agents are currently first choice drugs for treating childhood and adolescent onset psychoses. In a randomized double blind controlled trial of clozapine in early onset schizophrenia, the atypical antipsychotic was significantly superior to haloperidol, in 21 children and adolescents with early onset schizophrenia, on all outcome measures[3]. However serious adverse effects occurred in alarmingly high rates in clozapine treated group, including neutropenia and seizures. Another recent double blind controlled trial established olanzapine and risperidone monotherapy as equally efficacious as haloperidol, with shorter time to respond, in total 50 pediatric patients with schizophrenia and other psychotic disorders[4]. However, metabolic adverse effects are problematic for atypical agents, with high rates of hyperprolactinemia with risperidone[5] and weight gain with olanzapine[6] in pediatric patients.

Aripiprazole, a novel antipsychotic having unique action of dopamine system stabilization has been widely investigated in adult psychotic disorders and found to be as effective as haloperidol[7] and risperidone[8], in improving both positive and negative schizophrenic symptoms in randomized double- blind placebo controlled trials. However, it has been sparsely used in pediatric populations, with absence of any controlled trials. It has been found to be efficacious in pediatric patients in treatment of conduct disorder[9] and bipolar mania[10], in small open label trials and retrospective chart reviews.

Aripiprazole has been established to have a superior tolerability profile in adult psychotic patients, compared to conventional antipsychotics. A meta-analysis pooling data from five short term trials, involving total 1549 adult patients, revealed that aripiprazole produced extrapyramidal symptoms comparable to placebo and significantly lesser than haloperidol[11]. Aripiprazole has also not been associated with significant weight gain or QT(c) prolongation in controlled trials[12]. Aripiprazole is thus an attractive candidate for treatment of psychotic disorders in children and adolescents as it appears to have improved tolerability with less propensity for both extrapyramidal and metabolic side effects, which are particularly troublesome in this population and may compromise treatment adherence.

The primary intent of this open label comparison study was to evaluate the efficacy and tolerability of aripiprazole monotherapy compared with haloperidol in non-affective psychotic disorders in children and adolescents.

MATERIALS AND METHODS

This open label, active comparator controlled study of four weeks duration of active therapy was performed at a single site, a tertiary psychiatric referral hospital in India. The study protocol was approved by the institute's ethics committee, and as all patients were minors, legal guardians provided written informed consent before participation in any study related procedures. Each consecutively recruited patient was alternately allocated to receive haloperidol or aripiprazole, in a 1:1 schedule, with the first patient receiving haloperidol. The aripiprazole group received evening dose of 10mg/day for body weight below 50 kg and 15 mg/day for body weight above 50 kg. Similarly, haloperidol group received evening dose of 10 mg/day for body weight below 50 kg and 15mg/day for body weight above 50 kg.

All doses were fixed. Subjects who could not tolerate the minimal dose were discontinued from the study. Concomitant psychotropic medications were not permitted except for limited benzodiazepine use for agitation and insomnia (i.e. up to 6 mg/day lorazepam) and trihexyphenidyl (up to 6 mg/day) for extrapyramidal symptoms.

Subject eligibility included age _ 18 years, an ICD-10 DCR diagnosis of schizophrenia, schizoaffective disorder and acute transient psychotic disorder and drug naïve or drug free status for at least two weeks (for oral antipsychotics and six weeks for depot antipsychotics). Subjects with any comorbid neurological or psychiatric disorder were excluded. A total of 30 subjects participated in the study till the endpoint for four weeks with assignment into any of two treatment groups of 15 patients each.

A screening assessment (standard history, physical examination and laboratory profile) was conducted during the first visit, followed by baseline assessment on the same day. Thereafter, assessments were conducted weekly until week four. Body weight was recorded and electrocardiogram was performed at baseline and at study completion.

The primary efficacy measure was the Positive and Negative Syndrome Scale for Schizophrenia (PANSS)[13] total score at endpoint. Secondary measures included the Clinical Global Impressions -Severity of illness (CGI-S) scale[14] and visitwise PANSS total scores.

Parkinsonism and akathisia were assessed weekly with the Simpson-Angus Scale[15] and the Barnes Akathisia Scale[16], while spontaneous reporting of other adverse events was recorded at each visit.

Statistical methods

Statistical analysis was done with the help of Statistical Package for Social Sciences version 10 (SPSS-10). Pearson chi-square (^{2}) tests were used for analysis of categorical variables. To avoid Type I error due to multiple comparisons, a series of general linear modeling repeat measures multivariate analyses of variance were performed to assess longitudinal group interactions and effects with PANSS total scores, PANSS individual subscale (positive, negative, general psychopathology) scores, CGI-S scores, Simpson Angus total scores, Barnes Akathisia Scale scores, body weight and QT (c) interval. Mauchly's test of sphericity assumptions were performed beforehand and the findings revealed significant deviation from sphericity assumptions. Consequently, Greenhouse-Geisser corrected F values were taken into consideration

only. Additonally, effect size (partial eta square), observed power and confidence interval were calculated for each comparison.

Response was defined as ≥30% decrease from baseline PANSS total score at endpoint.

All hypotheses were tested at a 2 tailed of 0.05.

RESULTS

A total of 30 subjects entered and completed the four week study with no discontinuations.

Table 1 gives the baseline demographic and clinical profile of the study sample by group assignment. Baseline demographics and baseline PANSS total and CGI-S scores were not statistically different among treatment groups.

Table 1 : Patient characteristics at baseline

Variables	Haloperidol (n = 15)	Aripiprazole (n = 15)
Gender, number female (%)	5 (33.3)	6 (40.0)
Age, mean years (SD)	15.33 (1.72)	15.33 (1.35)
Diagnosis number (%)		
Schizophrenia	8 (53.3)	9 (60.0)
Acute psychosis	7 (46.7)	6 (40.0)
Weight, mean kg (SD)	40.80 (8.56)	42.13 (9.93)
QTC interval, mean msec (SD)	405.33 (20.18)	404.13 (23.03)
Serum prolactin, mean ng/ml (SD)	13.71 ± 5.60	12.05 ± 4.28
Psychiatric profile, mean (SD)		
PANSS total	105.93 (19.80)	103.00 (14.91)
PANSS positive	28.47 (8.32)	30.00 (7.65)
PANSS negative	25.67 (10.37)	26.80 (9.07)
PANSS general psychopathology	51.80 (12.12)	46.20 (8.27)
CGI-S	6.27 (0.59)	6.07 (0.59)

At baseline, PANSS total scores were not significantly different between haloperidol group (mean 105.93±19.80) and aripiprazole group (mean 103.00±14.91) (p= 0.65). Also, there was no significant difference on any of the three subscale scores of positive syndrome, negative syndrome and general psychopathology.

PANSS total scores improved significantly from baseline to end point over four weeks in both haloperidol and aripiprazole groups showing significant treatment effect for both the groups. However, no significant treatment interactions could be observed for the PANSS total or subscale scores, except for the general psychopathology cluster. In the haloperidol group, general psychopathology subscale score improved significantly better than that in aripiprazole group (p=0.03; effect size=0.12; observed power=0.61) (Table 2).

	Variables	Pre (mean ± SD	Post (mean ± SD)	F (Greenhouse-Geisser correction)	P	Partial Eta squaared (Effect size)	Observed power
PANSS total	Haloperidol	105.93 ± 19.80	55.67 ± 18.63	1.28	0.28	0.04	0.24
	Aripiprazole	103.00 ± 14.91	60.00 ± 21.29				
PANSS positive scale	Haloperidol	28.47 ± 8.32	11.93 ± 7.20	0.07	0.90	0.00	0.06
	Aripiprazole	30.00 ± 7.65	12.40 ± 9.02				
PANSS negative scale	Haloperidol	25.67 ± 10.36	15.67 ± 7.48	0.47	0.60	0.16	0.12
	Aripiprazole	26.80 ± 9.07	17.33 ± 7.06				
PANSS general score	Haloperidol	51.80 ± 12.12	28.73 ± 7.37	3.83	0.03*	0.12	0.61
	Aripiprazole	46.20 ± 8.27	30.27 ± 9.58				

[* p 0.05(2- tailed)]

Subsequent analysis according to diagnostic groups revealed a differential effect of diagnosis on improvement of PANSS scores. Improvement over time was significantly better for patients with diagnosis of acute transient psychotic disorder than for patients with schizophrenia (p=0.00; effect size=0.23; observed power=0.93)

At baseline, there was no significant difference in CGI-S score between haloperidol group (mean 6.27±0.59) and aripiprazole group (mean 6.07±0.59). CGI-S scores improved significantly from baseline to endpoint over four weeks in both groups showing significant treatment effects for both groups. However, a significant treatment interaction was noted as, in the haloperidol group, CGI-S score improved significantly better than that in aripiprazole group (p=0.02; effect size=0.14; observed power=0.73) .

At week one, only one patient (6.7%) in haloperidol group and none (0%) in aripiprazole group were found to be responder. At week two, seven patients (46.7 %) in haloperidol group and six patients (40.0%) in aripiprazole group were responders. At week three, 12 patients (80.0%) in haloperidol group and 11 patients (73.3%) in aripiprazole group were responders. At endpoint, at week four, there were 12 responders (80.0%) and three nonresponders (20.0%) in both treatment groups. There were no significant group interactions with responder rates throughout the study period.

A significant interaction between extrapyramidal side effects and treatment group was noticed as parkinsonian side effects were more in haloperidol group than in aripiprazole group throughout study period of four weeks (p=0.00; effect size=0.40; observed power=1.00) (Table 3).

Table 3: Comparison of extrapyramidal symptom profile and serum prolactin levels across two groups

	Variables	Pre (mean ± SD	Post (mean ± SD)	F (Green house-Geisser correction)	P	Partial Eta squaared (Effect size)	Observed power
Simpson Angus total score	Haloperido	0.00 ± 0.00	10.20 ± 3.45	19.00	0.00**	0.40	1.00
	Aripiprazole	0.00 ± 0.00	2.20 ± 3.25				
Serum Prolactin level	Haloperidol	13.71 ± 5.60	41.82 ± 19.19	30.22	0.00**	0.52	1.00
	Aripiprazole	12.05 ± 4.28	10.57 ± 4.33				

[** p 0.005 (2-tailed)]

Barnes Akathisia Rating Score (Global assessment) increased from baseline to endpoint in both haloperidol and aripiprazole groups, with no significant interaction.

Haloperidol group also required significantly higher dose of trihexyphenidyl (mean dose 68.40 $\pm$ 19.72), while use of lorazepam was comparable in both groups.

At baseline, there was no significant difference in serum prolactin level between groups. At endpoint, a significant treatment interaction was noted, as serum prolactin level was significantly increased in haloperidol group compared to aripiprazole group (p-0.00; effect size=0.52; observe power=1.00). On the other hand, aripiprazole group showed a slight nonsignificant decrease in serum prolactin level from baseline to end point.

A significant interaction between body weight change and treatment group was noticed as weight gain was significantly more in haloperidol group than in aripiprazole group (p=0.04; effect size=0.14; observed power=0.54).

There was no significant group interaction in QT (c) interval over four weeks from baseline to endpoint.

DISCUSSION

To our knowledge, this is the first prospective controlled trial of aripiprazole in pediatric patients with schizophrenia and other non affective psychosis. In this trial, both aripiprazole and haloperidol demonstrated significant effectiveness for overall symptom improvement as reflected by PANSS total scores as well as positive and negative subscale scores with no significant between group interactions.

However, haloperidol produced significantly better improvement than aripiprazole in CGI-S score and PANSS general psychopathology subscale score at endpoint. On the other hand, aripiprazole produced significantly less extrapyramidal symptoms and weight gain, than haloperidol.

Similar to results in previous trials in adult psychotic patients, aripiprazole produced improvement comparable to haloperidol in both positive and negative symptoms in early onset psychoses, as expected from pharmacodynamic profile of partial D2 receptor agonism[7]. At low dopamine levels, it stimulates dopamine receptors and at high dopamine levels, inhibition of dopamine activity occurs. Since positive symptoms are thought to be caused by hyperdopaminergic activity in the mesolimbic tract and negative symptoms by hypodopaminergic activity in the mesocortical tract, aripiprazole can produce improvement in both syndrome domains.

However, better improvement in general psychopathology scores with haloperidol may be due to the fact that haloperidol is more sedating and less activating due to higher D2 receptor antagonism while aripiprazole as a partial D2 receptor agonist is more activating, at times producing nonspecific de novo side effects like insomnia, agitation, anxiety and tension[12] which may adversely affect general psychopathology score outcomes. There are emerging case reports of worsening psychosis, increased agitation and even emergent suicidality with use of aripiprazole in adults[17,18].

Decreased risk of extrapyramidal symptoms is consistent with pharmacodynamic profile of aripiprazole as D2 receptor partial agonist, while with haloperidol, a high potency D2 antagonist, dopaminergic neurotransmission is blocked in nigrostriatal tract, causing pseudo-parkinsonism. In contrast, aripiprazole acts as dopamine agonist in conditions of low, endogenous dopamine activity, preventing development of hypodopaminergia in nigrostriatal region. This low propensity for parkinsonian side effects is similar to rates reported in adult studies, which were comparable to placebo and significantly less than haloperidol[11].

As a modest H1 receptor antagonist, aripiprazole is associated with minimal weight changes and has even been found to cause mean weight loss from baseline in some trials[19].

In the current study, aripiprazole group demonstrated slight but nonsignificant reduction in serum prolactin levels from baseline to endpoint, while serum prolactin was significantly elevated over baseline with haloperidol treatment. In fact, data from adult studies indicate that aripiprazole treatment frequently produces reductions from baseline prolactin levels compared to placebo (-56.5% versus 0%) while haloperidol is associated with very significant prolactin elevations (120% over baseline)

This study has several limitations. First, sample size is very small, mostly due to rarity of diagnosis of non affective psychotic disorders in pediatric patients. This is borne out by similarly inadequate sample size in all controlled trials, examining atypical antipsychotic agents like clozapine and olanzapine[3]. Secondly, both treatment groups had heterogenous diagnostic composition, namely schizophrenia and acute transient psychotic disorder. This factor may have contributed to overestimation of clinical improvement in treatment groups, as acute psychotic patients improved better and faster than schizophrenic patients. Thirdly, this is a nonrandomized open label study lacking blinding, which may contribute to selection and assessment bias. Thus, results of this study must be considered as purely preliminary and exploratory, making generalizations difficult.

Lastly, there is lack of placebo control, a requisite part of a pharmacotherapy trial, which was not possible due to ethical constraints. However, use of haloperidol as an active comparator, which has already been established as clearly more efficacious than placebo in a previous randomized controlled study in this patient population[20], has alleviated this problem to some extent.

In summary, this preliminary study indicated that aripiprazole was equally efficacious as haloperidol in improving overall symptoms as well as positive and negative syndrome domains of schizophrenia, while offering better tolerability with respect to extrapyramidal symptoms and body weight, and reducing the need for concomitant antiparkinsonian agents.

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Case Report :

PEDIATRIC AUTOIMMUNE NEUROPSYCHIATRIC DISORDERS WITH STREPTOCOCCUS INFECTION

Mohapatra Satyakam and Rath NM

ABSTRACT

Paediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections (PANDAS) is a unique syndrome which is associated with a recent infection caused by the group A b-haemolytic streptococcal bacteria. Although b-haemolytic streptococcal infection is prevalent in India, we are less aware of cases of PANDAS from the Indian subcontinent. We report a case of OCD who presented to us with features of PANDAS.

Key words- ***EXPRESSED, EMOTION, PSYCHIATRIC, DISORDERS***

Introduction

Swedo et al , 1998 [1] first proposed that some cases of childhood-onset obsessive-compulsive disorder (OCD) and tic disorders might be, like Sydenham chorea (SC) , a post-streptococcal disorder of immunological character. They coined the acronym PANDAS to identify the occurrence of pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections. According to the National Institute of Mental Health (NIMH) [2] , children with PANDAS are clinically identified by five criteria. They are:

1. "Presence of obsessive–compulsive disorder and/or a tic disorder(meeting DSM-IV TR criteria).
2. Pediatric onset of symptoms (age 3 years to puberty).
3. Episodic course characterized by acute, severe onset and dramatic symptom exacerbations.
4. Association with neurological abnormalities (motoric hyperactivity, or adventitious movements, such as choreiform movements).
5. Temporal relationship between GABHS (Group A B- hemolytic streptococcus) infections and symptom exacerbations.

Although GABHS infection is prevalent in India [3] surprisingly there is lack of cases of PANDAS being reported from here. This is possibly due to lack of awareness regarding the same amongst the psychiatric fraternity. The few reports and the poor understanding of this condition need to be overcome.

Case history

Mr. A., 15-year-old adolescent Hindu male presented to the Child and Adolescent Psychiatry outpatient department (OPD) with sudden onset of abnormal involuntary movements involving the face and shoulder and repetitive hand washing, spitting and prolonged bathing for the last 2 weeks. 1 month prior to this patient had developed high grade fever, cough and throat pain which lasted for 10 days. After the treatment from physician, his general condition had improved. Few days after the resolution of fever, the parents noticed involuntary movements involving the face and shoulder. Movements were sudden, rapid and non-rhythmic. As per the parents, these movements were present whenever the child was awake and ceased during sleep. Patient also started washing his hands repeatedly and taking prolonged bathing for which the patient expressed distress to the parents but was unable to stop. His condition worsened with time, his sleep and appetite decreased markedly and he became irregular in school.

Child was born after full-term normal delivery at hospital. Regular immunizations were carried out. At birth, her weight and length were normal. Medical records and history suggested normal development. There was no family history of seizures or other abnormal movements/psychiatric complaints. On examination, the child was well oriented and higher mental functions were intact. Vitals were within normal limits. There were tic movements in both shoulders. Movements decreased but persisted when the child was observed in a restful state. Rest of the nervous system and other body systems were normal on examination. On mental status examination obsessions regarding contamination was elicited.

Hemoglobin (11.6 g/dl), total leukocyte count (8800/ mm), differential leukocyte count (P58n L32 M8 E2), and erythrocyte sedimentation rate (11 mm fall in first hour) were within normal limits. Other blood investigations revealed normal sugar, electrolytes levels, and liver function tests. In view of recent past history of sore throat, anti-streptolysin O (ASO) titers were estimated and found to be high (350 Todd units).

Thus, diagnosis of PANDAS syndrome was made, as our case met all the required diagnostic criteria.

Patient was started on Cap fluoxetine 20mg/day which was raised to 40mg/day on an outpatient basis with psycho education of the patient and family members. Currently patient is in our and paediatrician follow up and showing consistent improvement.

Discussion

PANDAS are a recently described subgroup of childhood disorders, and there has been a great deal of public and physician interest in their pathophysiology, diagnosis, and management. The literature search reveals that only few of PANDAS cases have been reported from India . Our case fits the classical description which is laid down by the NIMH [2] .

There is lack of knowledge among the psychiatrists and pediatricians about the diagnosis and management of PANDAS. Diagnostic criteria for PANDAS was proposed by NIMH. As with any newly identified syndrome, the diagnosis of PANDAS is controversial. Though the laid down diagnostic criteria include episodic course of exacerbations (temporally correlated with GABHS infection) and remissions, what time period constitutes "temporal" association has not been defined. Usefulness of this diagnostic criteria is disputed by some scientists who think this sub-set of patients do not differ significantly from the remainder of the patient population, and that infections do not increase the risk of OCD or tics. Consequently, the PANDAS model is a complex and rapidly-moving area of medical research. PANDAS is currently not listed as a diagnosis by the International Statistical Classification of Diseases and Related Health Problems (ICD) or the Diagnostic and Statistical Manual of Mental Disorders (DSM).

Treatment for the PANDAS subgroup of children with OCD is not different from treatment for others with this diagnosis. Murphy et al , [4] [5] recommended the use of combined behavioral therapies and low doses of selective serotonin reuptake inhibitors (SSRIs) with rapid taper to clinically effective levels as reported in the literature]. Children with PANDAS appear to be unusually sensitive to the side-effects of SSRIs and other medications, so it is important to "start low and go slow" when using these medications. The best treatment for acute episodes of PANDAS is to eradicate the streptococcal infection causing the symptoms (if it is still present). A throat culture should be done to document the presence of streptococcal bacteria in the throat. If the throat culture is positive, a single course of antibiotics will usually get rid of the streptococcal infection and allow the PANDAS symptoms to subside. Early studies [6] of prophylactic antibiotics in children with PANDAS have been complicated by poor compliance and the dilemmas of placebo treatment. Swedoet al, [7] mentioned that "too early" to recommend prophylactic antibiotics for PANDAS. IV immunoglobulin therapy is generally recommended only for severe or persistent cases. Although still experimental, it may have potential for the future, especially in those not responding to conventional treatment.[8]

Some recent development in the field of PANDAS includes indentification of a small group of patients with an adolescent-adult "variant" of PANDAS [9] This possible Adolescent-Adult Variation of PANDAS is under review.

The lack of cases of PANDAS in the Indian context can be attributed to an inadequate awareness regarding this disorder and an infrequent liaison among the various specialties. We reported this case since it is important to keep this disorder in mind when treating children. The presence of the significant co- morbidity can lead to a marked disability in terms of the academic performance and the social adjustments in these children and hence a timely intervention can be helpful[10]. A good cross referral between the paediatricians and the psychiatrists can serve in decreasing and eliminating the morbidity and the disability which are associated with this disease.

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PICA

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ABSTRACT

Association of pica and iron deficiency anaemia have been reported in literature. An unusual but completely reversible form of pica in the form of sponge eating is reported as an odd manifestation in a case of iron deficiency anemia. Medical practice is full of challenges and complexity; and clinicians need to be vigilant all the timein their practice to appreciate unusual and rare manifestations of common clinical conditions iron deficiency anaemia.

Key words : iron deficiency anaemia, sponge eating, pica.

INTRODUCTION

Pica has been described as a persistent eating of non-nutritive substances for at least 1 month in a manner that is unsuitable for a child at the developmental level; it is not a part of the culturally sanctioned practice and is rationally severe to warrant independent clinical attention.[1] This is a peculiarneurobehavioural problem that influences inappropriate development of the children older than 18–24 months. The aetiology remains elusive.[2-3]In this report the authors report a case of pica with iron deficiency anaemia (IDA) manifested through sponge eating and recovering completely with irontherapy.

CASE

A paediatrician referred a three and a half year old boy with a habit of eating sponge since the age of 3 years. The habit for the sponge aggravated to the extent that he couldrip the cushions, car seats and mattresses to get the spongeout. The child was noticed to have a strong, irresistible urgeand was seen finishing a large chunk of sponge from a cushion inless than an hour. Occasionally he had been seen eating curtain fibres and newspaper. He was a medically fit boy with a normal intelligence and no behavioural problems. The examination including general physical and systemic resulted to be unremarkable except pallor. The blood investigation revealed iron deficiency anemia with a haemoglobin of 8.3 g/dL, a low MCV (mean corpuscular volume), low MCH (mean corpuscular haemoglobin) and a low MCHC (mean corpuscular hemoglobin concentration) (MCHC). On referral to the haematologist further investigation revealed a very low serum ferritin of less than 2 ng/mL and anisocytosis onthe peripheral smear. Thalassemia screening test was done and has been found to be negative. The liver function tests and renal function

tests were reported normal. The child was diagnosed to bea case of pica with iron deficiency anemiaand was kept on iron replacement orally. The symptoms of eating sponge disappeared fully by correcting the iron deficiency anemia.

DISCUSSION

Several conditions like mental retardation[4], autism[5], psychosocialstress in the form of parental deprivation, parental neglectand abuse[6-7] and a variety of behavioural disorders[7-8] have been speculated to be causatives factors of pica. Thereare also reports suggesting that low zinc and ironlevels may be linked to pica.[9]Inthe case documented by the authors, spongeeating (pica) and iron deficiency anaemia was observed. The case responded well to the iron therapywith complete resolution of symptoms of pica. There is still a speculation whether pica causes anaemia oranaemia leads to pica.[10]

The commonest forms ofpica are geophagia (soil)[11], pagophagia (ice)[12] and trichophagia(hair)[13] but the sponge eating as pica is very rarely reported.[14] The present case had an atypical and extreme form of sponge eating.Natural sponge contains various proteins and mineralsand is often fortified with silica or calcium salts; however,synthetic sponge consists of cellulose alone. It is not yet clear whether a craving of an unidentified salt fuels the eatingof sponge or the texture of sponge acts as a oral stimulant.[14] The aetiology of sponge eating in our case is unknown but it was completely resolved with iron therapy. It is suggested that physical conditions be investigated in all cases of pica and treatment of underlying conditions may help in resolution of the pica.

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All correspondence will be made with the author specified for that purpose. Manuscripts should be accompanied by a covering letter indicating that the paper is intended for publication, stating the number of figures and tables, and the number of words in the manuscript. The preference for the section cited in the *Journal* will be actively considered along with an assessment by the Editor and the editorial team to decide on the most suitable section for each submission, in consultation with the author. Papers not in accordance with the requisite format, or those which do not meet the requirements for one of the types of articles specified, will neither be sent for peer review nor be returned to the author. Suggestions for resubmission may be communicated to the authors. Manuscripts found unsuitable for publication will not be returned.

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2. **Brief Research Communication:** These manuscripts, with not more than I table/figure, should contain short reports of original studies or evaluations, service-oriented research which may not be methodologically sound but points towards a potential area of scientific research or unique first-time reports. The word limit is 1500 words and up to 20 references, and an abstract (structured format) of not more than 150 words.
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The manuscript should be arranged in the following order, with each item beginning on a new page: (i) title page (ii) abstract (iii) text (iv) references and (v) tables and/or figures. All pages must be numbered.

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The title should be brief and relevant. A short running title should be given. The number of words in the manuscript, names(s) of the author(s) and the number of tables and figures should be mentioned on the upper right-hand corner of the title page. The location of work, name, degrees, designations and addresses (including e-mails) of the authors should be given along with acknowledgements in a separate paragraph. One of the authors should be designated as the corresponding author.

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It should contain no more than 250 words for an original research paper and should be no longer than 150 words for brief research communication and case reports. The abstract for original research papers and brief research communication should be organized under the following heads: background, aim, methods, results, conclusion. Up to fife key words should be given.

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The text should include four major sections: introduction, methods, results and discussion. The methods should present a detailed description of the nature of the study group, methods of recruitment, measurement and statistical techniques.

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American Psychiatric Association (APA). Diagnostic and statistical manual of mental disorders. 4th ed. Washington, DC: APA; 1994.

Issue with a supplement

Limgjaerde O. Ahlofors UG, Bech P, et at. The UKU side effect rating scale. Acta Psychiatr Scand 1987; 76 (Suppl. 334): 81-94.

Chapter in a book

Lezak MD. Assessment for rehabilation planning. In: Meier MJ, Benton AL, Diller L (eds). Neuropsychological rehabililation. London: Curchill Livingstone; 1987:41-58.

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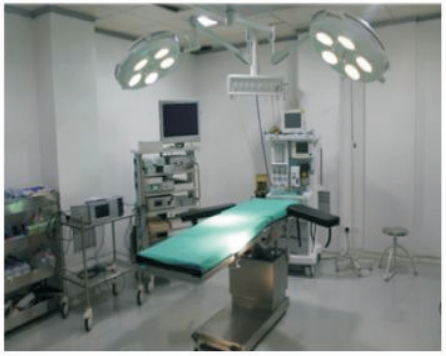

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