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INTERNET & ITS ROLE IN PSYCHOPATHOLOGY

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Of all the revolutions that have influenced mankind in the course of its evolution, mass communication has been the most remarkable one and internet has catalyzed the process to an extent that even Charles Darwin would have been left wonderstruck, for those who make their living forecasting change in social institutions are frequently humbled by the actual flow of events. Developments that seem inevitable (such as “artificial intelligence” or the picture phone) seem to take forever to happen, while seemingly unstoppable institutions or innovations (such as physician practice, management firms) suddenly collapse. Sometimes, however, innovations spring, fully blown and unheralded, seemingly from out of nowhere. The Internet is a global system of interconnected computer networks that use the standard Internet Protocol Suite (TCP/IP) to serve billions of users worldwide. It carries a vast range of information resources and services, such as the inter-linked hypertext documents of the World Wide Web (WWW) and the infrastructure to support electronic mail. The number of internet users in India has reached 354 million and has become the second largest country by the number using the internet. Almost no sphere of life is unaffected and so is mental health, it would not be an overemphasis to say that internet has been instrumental in modernizing psychiatry in more ways than one.

Internet Delusions  
The definition of delusions according to the DSM classification system includes “culture” as a necessary criterion: “delusion” - a false belief … is not one ordinarily accepted by other members of the person's culture or subculture. For example, a few hundred years ago the main content of control delusions, delusions of persecution and reference, involved supernatural entities and witchcraft, while after the invention of modern technologies, electricity, X-rays, laser and modern communication like telegraph, telephone, radio and television, the content of delusions was substantially influenced by these innovations. There is no understanding regarding the time that it takes for cultural innovations to influence delusional beliefs. Delusions about the Internet were not reported until knowledge about the Internet became widely disseminated, suggesting that a level of cultural salience (or perhaps social concern) must to be reached before such concepts become incorporated into paranoid or psychotic experiences. According to Bell et al delusions involving the Internet can vary considerably in presentation. The role of the Internet in such delusional beliefs is largely restricted to two major themes. The first is the use of the Internet as an explanatory tool to account for unusual experiences such as the sense of being under extreme control, hearing voice or having one's thoughts read. A second theme involves the supposed use of the Internet by people who are thought to be conspiring against the affected person. In these delusional concerns, the Internet is not represented as having a direct malign influence, but typically is perceived as a means of hosting chat rooms, photos or recordings about the
person concerned.

**Internet delusions have been conceptualized in the following ways:**

Catalano has proposed that “Internet delusions” may represent a “new subtype of a previously reported psychotic illness” - “delusional disorder with delusional ideas related to the Internet”.

Others state that Internet delusions should not be considered a new diagnostic entity. Rather, these delusions exemplify a new form of delusional content of well-known types of delusions (i.e., delusion of persecution, delusion of control).

A third viewpoint an intermediate position by Eytan and colleagues, who suggest that delusions associated with computer technology, may be a form of a western culture-bound syndrome.

Apart from the content of delusions, technological innovation can also have an impact on the form, etiology or prognosis of the delusions. For example, Schmid & his colleagues labeled a case as 'perception broadcast' which although seeming similar to thought broadcast, it involved visual perceptions and not thoughts, and did not involve the direct participation of others, with the internet mediating technology, thereby not fulfil Schneider's thought broadcast.

**Münchausen by Internet**

With the exponential increase in the number of people with internet access, "virtual" support groups have multiplied. Many of them conducted by e-mail or internet postings and sometimes requiring a free subscription, have been established for persons with particular diseases. Typically, the members of such networks strongly support one another, communicating not only information but strong emotion founded on their personal battles with illness. It is now however seen that these groups simultaneously provide an inexpensive, convenient, and readily accessible forum for people who choose to misrepresent themselves as ill. And so the term "virtual factitious disorder" was coined by Marc Feldman in 2000 to be replaced by “Münchausen by Internet”. Feldman listed the following common behavior patterns in people who exhibited Münchausen by Internet:

- Medical literature from websites or textbooks is often duplicated or discussed in great detail.
- The length and severity of purported physical ailments conflicts with user behavior. For example, someone posting in considerable detail about being in septic shock, when such a possibility is extremely unlikely.
- Symptoms of ailments may be exaggerated as they correspond to a user's misunderstanding of the nature of an illness.
- Grave situations and increasingly critical prognoses are interspersed with "miraculous" recoveries.
- A user's posts eventually reveal contradictory information or claims that are implausible: for example, other users of a forum may find that a user has been divulging contradictory information about occurrence or length of hospital visits.
- When attention and sympathy decreases to focus on other members of the group, a user may announce that other dire events have transpired, including the illness or death of a close family member.
- When faced with insufficient expressions of attention or sympathy, a forum member claims this as a cause that symptoms worsen or do not improve.
- A user resists contact beyond the Internet, by telephone or personal visit, often claiming bizarre reasons for not being able to accept such contact.
- Further emergencies are described with inappropriate happiness, designed to garner immediate reactions.
- Other forum members post on behalf of a user, exhibiting identical writing styles, spelling errors, and language idiosyncrasies, suggesting that the
user has created fictitious identities to move the conversation in their direction.

- Other forum members post on behalf of a user, exhibiting identical writing styles, spelling errors, and language idiosyncrasies, suggesting that the user has created fictitious identities to move the conversation in their direction.

Forum members whose ruses are discovered by their support groups are frequently banned from such communities. At times, when confronted with inaccuracies or inconsistencies, those who are suspected of perpetrating fabrications may compound the deceit by accusing forum members of imposing greater stresses upon them, exacerbating their conditions, or worsening their depression. Users may employ sock puppets - separate online identities controlled by the same person - to accuse other forum members of disloyalty and persecution, or support the user who is under suspicion. Because no money is exchanged and laws are rarely broken, there is little legal recourse to take upon discovery of someone faking illness. Such dramatic situations often polarize online communities, making many members feel ashamed for believing elaborate lies while others remain staunch supporters.

The advent of Internet as a medium of mass communication poses numerous opportunities, challenges and dilemmas for mental health professionals. It is necessary for all mental health professionals, especially those in the developing countries, to remain informed on the latest developments in this area. From its effects on the minds of people using it to its use to treat them as well, the internet is working as a cyber sword.

References


THE INTERFACE BETWEEN OBSESSIVE COMPULSIVE SYMPTOMS AND PSYCHOSIS

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Abstract

The complex relationship between obsessive compulsive symptoms and psychosis has been observed long before. The overlap of the two disorders in terms of course, anatomical abnormality and treatment response has been found quite striking. Obsessive Compulsive symptoms are preceded or succeeded by psychosis is still a matter of debate. However, evidences are enough to suggest that both the disorders have an intertwined etiology and influence each other drastically. It is often difficult for the mental health professionals to dissect the exact symptomatology to differentiate between psychosis and obsession. Insight plays a vital role in determining the psychopathology in both the disorders and current nosology has given a separate specifier for it. Hoarding is emerged as a new entity in DSM V as it is a long standing confusion for the researchers whether to classify it into obsessive symptoms or psychotic symptoms. Treatment modality also varies when one of the disorders is complicated by another. Clinicians have faced tough challenges while dealing with both the disorders simultaneously. As the obsessive compulsive symptoms modify the course of the psychotic disorder, it also has an implication on the prognosis of the illness. The management of the illness is a balance between a pleasurable outcome and risk of exacerbating the symptoms.

Keywords:
Obsessive Compulsive symptoms, Psychosis, DSMV, Insight

History

The concept of Obsessive Compulsive (OC) symptoms and psychosis was first introduced by Jeremy Taylor where he described a case with intrusive thoughts evolving into paranoid delusion in 1660. Later Westphal hypothesized that OC syndrome might actually be a variant schizophrenia. The relationship between Obsessive Compulsive Disorder (OCD) and psychosis may be part of an adaptive defence mechanism during the illness course as stated by Stengel. Some theorists have mentioned the protective role of OCD against the deterioration in personality in schizophrenic illness.

The Overlap

OCD and schizophrenia are clinically similar on many aspects. Both disorders manifest a chronic course, have a similar pattern of age-at-onset, and also affect men and women equally. In both the disorders, similar brain areas have been noted to be involved i.e. prefrontal cortex, anterior cingulate, caudate nucleus and thalamus. Both the disorders respond with almost equal efficacy to antipsychotic agents and serotonin reuptake inhibitors (SRIS),
which may suggest pathophysiological overlap between the two disorders. Although there is an adequate amount of evidence of similarity in both disorders even in its neurobiological and phenomenological aspects, still both disorders remain distinct from each other.

**OC symptoms in Psychosis**

Studies regarding frequency of OC symptoms in schizophrenia and OCD in schizophrenia have a varied result ranging from 3.5% to 46% for OC symptoms and from 7.8% to 59.2% for OCD. Similarly studies on outcome also have a varied picture ranging from poorer outcome in schizophrenia to better outcome in the same. Rosen, observed that the obsessional symptoms either evolved simultaneously with psychotic symptoms or after onset of psychotic symptoms in schizophrenia. He also noted that in most of these patients, the obsessional symptoms persisted unchanged, throughout the course of the schizophrenic illness. Later, he proposed three ways in which obsessions may be transformed into delusions (fig 1):

1. An obsessional idea, first experienced as arising from within oneself, is later believed to be planted in one's mind from outside (and thus becomes a delusion).
2. The obsessional idea is believed to be caused by someone else (and thus develops a persecutory delusional quality).
3. The insight into the obsession is lost, and the idea is believed to be true (and with this loss of insight the obsession becomes a delusion).

He found that 20% of the patients having obsessional symptoms subsequently had the transition, but had a relatively benign course, and a better prognosis. Later some of the studies have contradicted these findings and suggested a poor prognosis in schizophrenia associated with obsessive symptoms. Some authors have tried to explain the obsessive symptoms to be induced or exacerbated by antipsychotic agents in chronically treated schizophrenia patients, while others conceptualise it be part of the natural course of schizophrenia in a subset of patients, in view of the fact that the two disorders apparently involve a dys-regulation of common interacting neurotransmitter systems (e.g., serotonin/dopamine) and neuronal circuits.
There have quite a number of researchers, who have first diagnosed patients with obsessional neurosis or OCD, and then assessed them for schizophrenic or psychotic symptoms. The occurrence of an obsessive personality structure aborts the schizophrenic reactions, makes them mild and/or short lived. This finding was based on the observation that most of the patients displaying psychotic features in the course of OCD were subsequently found symptom free, although some obsessional symptoms were still persistent. Ingram described a subgroup of patients who could not be clearly put under a diagnosis of schizophrenia or obsessional disorder. He concluded that there may be neurotic and psychotic varieties of obsessional illness.

According to some, individuals with schizoid premorbid personalities may have a different clinical presentation of obsessional neurosis, with bizarre symptoms, emotional coldness and social withdrawal. Other authors have suggested that schizophrenia superimposed on OCD is extremely rare, while others believed that there was an entity of non-schizophrenic obsessional psychosis. Simultaneous occurrence of schizophrenia and OC symptoms may point towards a unique subgroup of persons whose condition might be labelled as schizo-obsessive disorder.

**Temporal Relation**

Recently, researchers have developed a growing interest towards OCD–Schizophrenia comorbidity, as the concept of drug-induced OC symptoms in schizophrenia patients, has evolved. So the temporal sequence of the pathology is an important issue, with respect to prognosis, treatment, and neurobiological implications. OCD or even OC symptoms may serve as a precursor for the future development of schizophrenia. Recent evidence strongly suggests the role of OC symptoms as being a part of the prodrome of schizophrenia. If the clinical predictors can be confirmed, it may be possible to intervene the course of schizophrenia at an early stage. If such a temporal sequence in OCD–Schizophrenia does indeed exist, then SSRI use for improving OC symptoms might itself lead to the development of schizophrenia. Finally, the presence of a temporal sequence suggests a shared neurobiology in both disorders. Most of the conceptualisation regarding the mutual temporal relationships and influences between the OCs and Schizophrenia are hypotheses:

a) In case of de novo OCD, delusions can arise in the course of OCD but it may not signify a diagnosis of schizophrenia. Rather, the delusions may represent generally transient reactive, affective, or paranoid psychoses, and may respond to treatment with antidepressants.

b) The stress theory model states that persistent OCD may lead to schizophrenia through exposure to chronic stress, or a continued dysfunction in anatomical regions that are shared by both conditions, like the basal ganglia, orbito frontal cortex, thalamus, anterior cingulum and regions of the temporal cortex.

c) OC symptoms may have a deleterious effect on the course and prognosis of psychosis. Psychological models of OCD might explain this by stating that those persons prone to OCD manifest a cognitive style, characterized by unsuccessful thought suppression and the propensity to make negative interpretations involving the idea that the person's choice may result in harm, consequently necessitating neutralization. Consequent to this cognitive style, occasional intrusive thoughts tend to re-occur, causing distress and thus becoming a symptom.

**The Role of Insight in OCD and Psychosis**

Traditional belief has been that obsessive–compulsive disorder (OCD) is a condition in which patients maintain good insight into their symptoms.
Being shaken by a number of investigators, this belief has now been revised in favour of the possibility that there is a range of insight in OCD. Field trials demonstrated a broad range of insight with 26% having poor insight and 4% were completely lacking insight. This findings prompted the addition of the new OCD specifiers with poor insight and with absent insight in DSM-V, which involves a lack of recognition that the symptoms are unreasonable or excessive, by the patient. Subsequent studies have also reported poor insight in between 15% and 36% of patients with OCD. In severe cases of OCD loss of insight changes obsessions into overvalued ideas and later point of time, patients regard them as justified, and when beliefs become fixed obsessions progresses to delusion. Thus, predisposing to psychosis. Insel and Akiskal described several subjects with OCD who presented the shift from an obsession to a delusion when resistance was abandoned and insight was lost (fig. 2). The shift might take either an affective form or a paranoid form. In addition, some fraction of OCD patients were considered atypical in that they were more severely disturbed, they might entirely lack emotional insight, resist their obsessions in a delirious fashion, show no evidence of anxiety, struggle with very confused family relationships, and show the elements of schizotypal personality.

![Figure 2. SHIFT OF INSIGHT](image)

A study conducted at the Department of Psychiatry at NIMHANS revealed two important findings. Firstly, a substantial portion of the patients had poor insight (25%) and secondly, those with poor insight also had earlier age-at-onset, a longer duration of illness, more number of symptoms, more severe illness with higher comorbidity rate (MDD) and poorer treatment response. These finding has vital treatment implications because OCD had traditionally been recognized to be a disease with insight intact, patients with poor insight could easily get misdiagnosed as psychotic and treated accordingly. There is, however, no evidence to suggest that those with poor insight respond better to antipsychotics. A few studies, on the contrary, have shown that insight improves after treatment with SSRIS. Studies regarding to response to behaviour therapy inpatients with poor insight show mixed results. Some studies found significant relation between obsessive–compulsive personality disorder and an increased level of insight. The recently authors have described a completely different pattern i.e. poor insight patients had a higher frequency of narcissistic and borderline personality disorders, while avoidant personality disorder was more common in the good insight group.

Hoarding has been associated with poor insight compared to other OCD symptoms. Poor insight into hoarding behaviour could be due to two reasons. Firstly, hoarding behaviour is associated with OCPD where insight is known to be lesser compared to that in OCD. However, it may be difficult to differentiate hoarding due to OCPD from hoarding due to OCD.
Secondly, hoarders show great emotional attachment to their possessions and are sentimental about them. Recent data indicate that in a majority of cases problematic hoarding cannot be better accounted for by OCD or OCPD. Hence, hoarding may be better conceptualized as a separate disorder. The hoarding criterion of OCPD excludes sentimental collecting, and thus does not fully correspond with the construct of compulsive hoarding and it qualifies as a new disorder in DSM-V.

Level of insight correlates with 4 clinical factors severity of compulsive symptoms:

- Chronic course of OCD
- Occurrence in 1st degree relatives
- Concomitant OCPD

OCD might present in a substantial proportion of patients with poor insight, and in them, the disease seems to have:

- Earlier age-at-onset
- More severe form of illness
- Higher rate of MDD
- Poor treatment response to medications

Poor insight was, indeed, the best predictor of poor drug-treatment response. Nevertheless, the treatment of OCD patients with poor insight may eventually lead to the development of a good insight, with concomitant improvement of OCD severity and depressive status.

Management

Studies regarding the treatment of OC symptoms in schizophrenia is limited. Initially, clomipramine was studied in detail but results were inconclusive. Some studies reported improvement of obsessive symptoms when neuroleptics were added to clomipramine, while others showed exacerbation of psychosis with the same. Trial have been done with fluvoxamine and fluoxetine in patients with schizophrenia spectrum disorder have yielded encouraging results. Therefore, addition of anti-obsessive agents seem to be efficacious in controlling OC symptoms in some schizophrenia patients.

Newer Antipsychotics:

Some authors have noted both an exacerbation of pre-existing OC symptoms, as well as the emergence of new ones, in schizophrenia patients treated with clozapine. These reported a reduction in these symptoms with the addition of fluoxetine to the clozapine regime. The exacerbation of OC symptoms has also been described in a psychotic patient treated with risperidone as well, with the symptoms remitting in a few days of discontinuing the medication. Induced OC symptoms in a patient treated with risperidone have also responded well to the addition of fluvoxamine. Glick et al. reported that aripiprazole improved the de novo OCS in schizophrenia. However, later studies failed to corroborate this finding. Although aripiprazole primary action as dopamine partial agonist may be therapeutically useful in the treatment of OC schizophrenia, its potent 5-HT2a antagonism with the weak D2 blockade effect may also be related to potential to induce or exacerbate OCS in schizophrenic patients.

As seen in PET studies, at low doses atypical antipsychotics cause high levels of 5-HT2 antagonism which may exacerbate OCD symptoms, whereas relatively high doses are required to produce significant dopamine D2 antagonism which may augment the anti-obessional effect of SSRI. It is likely that these drugs, because of their dual action on serotonin and dopamine receptors may lead to serotonin- dopamine imbalance, thus leading to treatment emergent OCD. The newer modality of treatment for neuroleptic-induced OCS in schizophrenia points to an antipsychotic treatment with a minimal 5-HT2a receptor affinity and anti-dopaminergic (D2/D3), such as amisulpride and haloperidol.

Psychotherapy:

In absence of reliable evidence, psychotherapy carries a concern of psychotic relapse in patients with comorbid illness due to increased stress in the process. The efficacy of exposure and response prevention for OCD, has been considered in patients with comorbid
schizophrenia and OCD, not only in those who are stabilized on antipsychotics but also in putative prodromal stage reducing development of emerging schizophrenia. To address these issues, longitudinal studies of young people at risk for psychosis or for OCD are needed now.

**Augmentation strategy:**

The addition of lamotrigine to treatment with an atypical antipsychotic (with or without an SRI) in schizo-obsessive patients resulted in reduction in the Y-BOCS score, indicating an overall improvement of OCS but no improvement of schizophrenia symptoms. The positive relationship between lamotrigine-induced improvement of depressive symptoms and OCS suggests that OCS and depressive symptoms in schizophrenia may share an underlying glutaminergic mechanism.

The inferences, which can be drawn from the limited studies in case of patients with comorbid schizophrenia and OC symptoms are that these patients are more resistant to treatment and require a distinct therapeutic approach (fig. 3).

- Conventional antipsychotic agents have no role in these patients, probably due to their limited serotonergic properties.
- Induction or worsening of OCD symptoms with atypical antipsychotic occur in patients with a primary psychotic disorder, rather than OCD.
- The obsessions emerged transiently 3 to 15 months after starting the drug. Both risperidone and olanzapine exacerbate OCD symptoms. With clozapine, the frequency and intensity of OCD symptoms is greater. SSRI diminished obsessions in some patients receiving clozapine.
- Antipsychotics may be used combination therapy (with SSRI)

* OCD with poor insight
* Refractory OCD
* OCD with tics
* When there is co-morbidity (schizophrenia and OCD)
Conclusion

To conclude, a substantial proportion of OCD patients experience psychotic symptomatology during course of illness. OC symptoms are not ego-dystonic, as always and loss of insight may lead to emergence of psychotic features in OCD. It is vital for clinicians to differentiate psychiatric symptoms from OC symptoms as management differs. Initial OCS/OCD seems not to be associated with a more severe course of psychotic symptoms, however, comorbid OCD probably worsen the overall prognosis. Course of schizophrenia with OC symptoms may be varied and associated with several other comorbidities as depression, personality disorders and tics. The links demonstrated in various studies, and replicated a number of times by multiple authors, point towards a relation between OC symptoms, the clinical and neurocognitive features of schizophrenia, and the role of causality. The management of this difficult entity is the balance between the different neurotransmitter systems, and clinicians have to measure between a pleasant outcome and risk of exacerbating the symptoms.

Conflict of Interest: NIL

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TARDIVE DYSKINESIA, A CRITICAL APPRAISAL

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Introduction

Antipsychotic drugs have revolutionised the management of major psychiatric disorders and their outcome. They have a range of adverse effects, the most frequent and distressing of which are those resulting in disturbance of voluntary motor function. The first description of any of the tardive like movement disorders was in 1957 by Schonecker, wherein he described a syndrome consisting of 'automatisms with licking and smacking movements of the lips' in subjects who had all been on chlorpromazine for a maximum of only eight week. The term 'tardive dyskinesia' first appeared in a paper by Faurbye et al in 1964 describing a series of neurological features associated with antipsychotic drug use. The word tardive comes from the French 'tardif', meaning 'late' or 'late in the day'. Dyskinesia, in its most literal sense means 'any abnormal kinesis'. Tardive dyskinesia is a syndrome comprising most, but not all, types of involuntary movements developing more or less anywhere in the body, which have been caused by exposure to many, but not all, types of psychotropic medication from a period of at least 6 to 24 months (Owens, 1999).

Epidemiology

- In a review of 56 studies spanning from 1959 to 1979, Kane and Smith (1982) reported point prevalence rates ranging from 0.5% to 65%, with an average point prevalence of 20%.
- Later, in a review of 76 published studies, Yassa and Jeste (1992) reported an overall prevalence of 24% among a total of 39,187 patients.
- A study by Woerner et al (1998) reported overall prevalence of tardive dyskinesia in neuroleptic-treated individuals to be 23.4%, of whom 3.8% had another neurological/medical illness that might have had an etiological role, thus giving a conservative prevalence rate of 19.6%. The rate varied with the setting, being 13.3% at a voluntary hospital with a young population, 23% in a Veterans Administration hospital, and 36% in a state hospital. In the same study, when a group of patients with no evidence of tardive dyskinesia were withdrawn from neuroleptic drugs and examined weekly for 3 weeks, 34% developed emergent dyskinesia.
Risk Factors

Table 1: Factors predisposing to tardive dyskinesia (retrospective literature) (Owens, 1999)

<table>
<thead>
<tr>
<th>Drug related</th>
<th>Non drug related</th>
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<tbody>
<tr>
<td>Type of antipsychotic (potency)</td>
<td>Age</td>
</tr>
<tr>
<td>Maximum daily exposure</td>
<td>Gender</td>
</tr>
<tr>
<td>Duration of exposure</td>
<td>Past physical treatments (Leukotomy, Insulin coma and</td>
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<tr>
<td>Cumulative exposure</td>
<td>ECT)</td>
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<tr>
<td>Polypharmacy</td>
<td>Organicity</td>
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<td>Antipsychotic blood levels</td>
<td>Antipsychotic blood levels</td>
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<tr>
<td>Antipsychotic-free intervals</td>
<td>Antipsychotic-free intervals</td>
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<tr>
<td>Previous EPS</td>
<td>Previous EPS</td>
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<tr>
<td>Anticholinergics</td>
<td>Anticholinergics</td>
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<td>Alcohol</td>
<td>Alcohol</td>
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Table 2: Factors predisposing to tardive dyskinesia (prospective literature) (Owens, 1999)

<table>
<thead>
<tr>
<th>Drug related</th>
<th>Non drug related</th>
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<tbody>
<tr>
<td>Antipsychotic dose</td>
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<td>Affective disorders (unipolar)</td>
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<td>- subsequent to onset</td>
<td>Alcohol abuse</td>
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<td>Tremor</td>
</tr>
<tr>
<td>Duration of exposure</td>
<td>Race (Afro-Caribbean)</td>
</tr>
<tr>
<td>Drug-free intervals</td>
<td></td>
</tr>
<tr>
<td>Prior EPS (especially akathisia)</td>
<td></td>
</tr>
</tbody>
</table>

Apart from the factors mentioned above, some genetic factors also increase the risk of tardive movement disorders. An article by Ohmori et al in 2003 reviewed the various studies that have been undertaken to find the genes predisposing to tardive dyskinesia, and found a positive correlation with the following genes:

Table 3: Positive genetic association studies of tardive dyskinesia (Ohmori et al, 2003)

<table>
<thead>
<tr>
<th>Gene</th>
<th>Polymorphism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cytochrome P450 2D6 (CYP2D6)</td>
<td>*3, *4, *5</td>
</tr>
<tr>
<td></td>
<td>*3, *4</td>
</tr>
<tr>
<td></td>
<td>*10</td>
</tr>
<tr>
<td></td>
<td>*10</td>
</tr>
<tr>
<td>Cytochrome P450 1A2 (CYP1A2)</td>
<td>C/A (first intron)</td>
</tr>
<tr>
<td>Dopamine D2 receptor ( DRD2 )</td>
<td>TaqI A</td>
</tr>
<tr>
<td>Dopamine D3 receptor (DRD3)</td>
<td>Ser9Gly</td>
</tr>
<tr>
<td>5-HT2C receptor</td>
<td>Cys23Ser</td>
</tr>
<tr>
<td></td>
<td>G-697C</td>
</tr>
<tr>
<td>5-HT2A receptor</td>
<td>T102C, A438G</td>
</tr>
<tr>
<td></td>
<td>T102C</td>
</tr>
<tr>
<td>Human leukocyte antigen (HLA)</td>
<td>B44</td>
</tr>
<tr>
<td>Mn superoxide dismutase</td>
<td>Ala – 9Val</td>
</tr>
<tr>
<td>N opioid receptor</td>
<td>A118G</td>
</tr>
</tbody>
</table>
**Drugs leading to Tardive Dyskinesia**

**Phenothiazines**

<table>
<thead>
<tr>
<th>Class</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aliphatic</td>
<td>Chlorpromazine, triflupromazine</td>
</tr>
<tr>
<td>Piperidine</td>
<td>Thioridazine, mesoridazine</td>
</tr>
<tr>
<td>Piperazine</td>
<td>Trifluoperazine, prochlorperazine, perphenazine, fluphenazine</td>
</tr>
</tbody>
</table>

**Thioxanthenes**

<table>
<thead>
<tr>
<th>Class</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aliphatic</td>
<td>Chlorprothixene</td>
</tr>
<tr>
<td>Piperazine</td>
<td>Thiothixene</td>
</tr>
<tr>
<td>Butyrophenones</td>
<td>Haloperidol, droperidol</td>
</tr>
<tr>
<td>Diphenylbutylpiperidine</td>
<td>Pimozide</td>
</tr>
<tr>
<td>Dibenzazepine</td>
<td>Loxapine</td>
</tr>
<tr>
<td>Dibenzoazepines</td>
<td>Clozapine, quetiapine</td>
</tr>
<tr>
<td>Thienobenzodiazepine</td>
<td>Olanzapine</td>
</tr>
<tr>
<td>Pyrimidinone</td>
<td>Risperidone</td>
</tr>
<tr>
<td>Benzisothiazole</td>
<td>Ziprasidone</td>
</tr>
<tr>
<td>Benzisoxazole</td>
<td>Iloperidone</td>
</tr>
<tr>
<td>Substituted Benzamides</td>
<td>Metoclopramide, tiapride, sulpiride, remoxipride, amisulpride</td>
</tr>
<tr>
<td>Indolones</td>
<td>Molindone</td>
</tr>
<tr>
<td>Quinolinone</td>
<td>Aripiprazole</td>
</tr>
<tr>
<td>Tricyclic</td>
<td>Amoxapine</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>Flunarizine, cinnarizine</td>
</tr>
<tr>
<td>N-acetyl-4-methoxytryptamine</td>
<td>Melatonin</td>
</tr>
</tbody>
</table>

**PATHOGENESIS**

**A) The dopamine hypothesis**

The most popular and prevailing theory for the pathophysiology of tardive dyskinesia is the theory of dopaminereceptor hypersensitivity.

- Initially, it was proposed that the acute neuroleptic blockade of dopamine receptors and a simultaneous increase in acetylcholine turnover in nigrostriatal pathways results in parkinsonism, whereas chronic dopamine blockade results in compensatory dopamine hypersensitivity and cholinergic hyposensitivit, leading to decreasing manifestations of parkinsonism and an increased propensity to tardive dyskinesia (Klawans and Rubovits, 1972).
- Later, it was proposed that blockade of one subset of D2 receptors results in dyskinesia whereas blockade of another subset results in parkinsonism (Gerlach and Casey, 1988).
- Recently, with the development of D1 and D2-selective agonists and antagonists, it was found that D1 and D2 systems play opposing roles in relation...
Apart from this, presynaptic dopamine D2 receptors inhibit the release of glutamate from excitatory cortical striatal projections. Therefore, neuroleptic blockade of these receptors increases the synaptic release of aspartate and glutamate in the striatum. Persistent activation of glutamate ionotropic receptors has been shown to cause neuronal degeneration by the same mechanism as mentioned below.

**D) Noradrenergic Hypothesis**

Norepinephrine is synthesized from dopamine in noradrenergic neurons, and there is evidence of synergy between central dopaminergic and noradrenergic neurons (American Psychiatric Association Task Force on Tardive Dyskinesia, 1992). Jeste et al (1984) reported higher CSF concentration of norepinephrine in patients with tardive dyskinesia compared to patients without it. There is also data suggesting an association of noradrenaline over reactivity and tardive dyskinesia in a portion and not all of the patients with tardive dyskinesia.

**E) Dyskinetic movements as a part of schizophrenic illness**

Bleuler (1911) described patients as 'performing all kinds of manipulations with their teeth … grimaces of all kinds, extraordinary movements of the tongue and lips.' The occurrence of 'peculiar, sprawling, irregular, choreiform, outspreading movements' in schizophrenic patients were noted by Kraepelin. These were both described long before the advent of antipsychotic medication. According to a study there was no significant difference in prevalences, severities and patterns of movement disorders in those with a history of antipsychotic exposure compared to those with no such history. On average the drug naive patients were about 10 years older than the drug exposed patients, which was thought to be due to the effect of drugs. A more recent study (Gervin et al, 1998) reported spontaneous dyskinesias in 7.6% of 79 first-episode patients. The movements described in these reports were mainly oro-facial or involving oral dyskinesia: D1 activity promoting, and D2 activity opposing dyskinesia. This lead to the proposal that D1/D2 receptor ratio imbalance in favour of D1 results in dyskinesia. It has also been seen that D1 antagonists can increase both dopaminergic release and firing rate of dopaminergic neurons. It has also been shown that a D1 antagonist can reduce agonist binding to D2 receptor. On the basis of this it has been postulated that in a situation in which small number of D1 receptors are blocked and D2 receptors are less efficient in binding dopamine, the increased stimulation of dopaminergic system would result in dyskinesia (Peacock and Gerlach, 2006)

**B) The GABA Hypothesis**

In the brains of monkeys who had developed dyskinesia during several years of treatment with neuroleptic drugs, there was reduced concentrations of GABA and its synthesizing enzyme glutamic acid decarboxylase in the globus pallidus, substantia nigra, and subthalamic nucleus; these changes were not seen in a control group of monkeys who had received similar neuroleptic treatment without developing dyskinesia. Thaker et al in 1987 studied GABA concentration in the cerebrospinal fluid of schizophrenic patients and found it to be reduced in those with tardive dyskinesia. Although the exact mechanism leading to decreased GABA activity is not known, it is hypothesized that antipsychotic-induced increase in the dendritic release of dopamine (due to chronic blockade of dopamine receptors) mediates the release of GABA at striatonigral terminals and subsequently inhibits activity of the GABA-mediated efferent pathway to thalamus. A reduction in GABA-mediated transmission from SNR to the target nucleus in the thalamus leads to disinhibition of thalamus and is associated with dyskinesias.

**C) Neurodegenerative Hypothesis**

An alternative hypothesis is that tardive dyskinesia is a result of neuro degeneration of striatal efferents from increased oxidative stress. The oxidative stress...
the upper limbs and similar in severity to those seen in tardive dyskinesia patients. These findings have raised the argument that dyskinesias may be intrinsic to the pathophysiology of schizophrenia, and that neuroleptics serve to enhance the process.

F) Maladaptive Synaptic Plasticity

Synaptic plasticity is the ability of the synapse between two neurons to change in strength in response to either use or disuse of transmission over synaptic pathways. In 2012, Teo et al suggested synaptic plasticity as a unifying theory for tardive movement disorders. They postulated that chronic antipsychotic use leads to D2 hypersensitization, which further causes secondary effects on the synaptic plasticity of glutamatergic and other synapses in striatum, resulting in imbalance of direct and indirect pathway, producing abnormal output to the sensorimotor cortex. Also, in the neocortex, chronic antipsychotic use also produces a maladaptive form of synaptic plasticity, which when combined with abnormal basal ganglia output, causes impairments in sensorimotor integration, resulting in miscoding of motor programs and abnormal movements. The persistence of tardive dyskinesia, even when antipsychotics are withdrawn would be explained by maladaptive synaptic plasticity inhibiting the “unlearning” of the miscoded motor programs.

DIAGNOSTIC CRITERIA

The most widely accepted criteria for diagnosing tardive dyskinesia were proposed by Schooler and Kane in 1982. They proposed three prerequisites for making the diagnosis:

1. Exposure to neuroleptic drugs for a minimum of total cumulative exposure of 3 months.

2. The presence of at least “moderate” abnormal involuntary movements in one or more body areas (face, lips, jaw, tongue, upper extremities, lower extremities, trunk) or at least “mild” movements in two or more body areas.

3. Absence of other conditions that might produce abnormal movements.

Later, the American Psychiatric Association Task Force on Tardive dyskinesia (American Psychiatric Association suggested four additional diagnostic criteria. Task Force on Tardive Dyskinesia, 1992) as follows:

1. The abnormal movements are exacerbated or may be provoked by a decrease or withdrawal of and antipsychotic drug. Increasing the dose of antipsychotic will suppress (or dampen) the movements at least temporarily.

2. Anticholinergic medication does not ameliorate and may worsen the movements.

3. Emotional stress may worsen the movements.

4. The movements decrease or disappear during sleep.

Research criteria as proposed by DSM-IV-TR are as follows:

a. Involuntary movements of the tongue, jaw, trunk, or extremities have developed in association with the use of neuroleptic medication.

b. The involuntary movements are present over a period of at least 4 weeks and occur in any of the following patterns: (1) choreiform movements (i.e., rapid, jerky, nonrepetitive) (2) athetoid movements (i.e., slow, sinuous, continual) (3) rhythmic movements (i.e., stereotypes)

c. The signs or symptoms in Criteria A and B develop during exposure to a neuroleptic medication or within 4 weeks of withdrawal from an oral (or within 8 weeks of withdrawal from a depot) neuroleptic medication.

d. There has been exposure to neuroleptic medication for at least 3 months (1 month if age 60 years or older).

e. The symptoms are not due to a neurological or general medical condition (e.g., Huntington's disease,
Sydenham's chorea, spontaneous dyskinesia, hyperthyroidism, and Wilson's disease), ill-fitting dentures, or exposure to other medications that cause acute reversible dyskinesia (e.g., L-dopa, bromocriptine). Evidence that the symptoms are due to one of these etiologies might include the following: the symptoms precede the exposure to the neuroleptic medication or unexplained focal neurological signs are present.

f. The symptoms are not better accounted for by a neuroleptic-induced acute movement disorder (e.g., Neuroleptic-Induced Acute Dystonia, Neuroleptic-Induced Acute Akathisia)

Table 4: Summary of the major clinical features of tardive dyskinesia (Owens, 1999)

<table>
<thead>
<tr>
<th>Tongue</th>
<th>‘Vermicular’ movements (no displacement) Displacement on one or more axes (rotation, lateral movement, ‘tromboning’) Extension beyond dental margin Irregular sweeping of buccal surface (‘bon bon’ sign) Irregular non-recurrent protrusion (‘fly catcher’ sign)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jaw</td>
<td>Mouth opening Lateral deviation Anterior protrusion Chewing Grinding</td>
</tr>
<tr>
<td>Lips</td>
<td>Pursing Puckering Sucking Smacking Retraction of lateral angles (‘bridling’)</td>
</tr>
<tr>
<td>Expression</td>
<td>Elevation/depression of eyebrows Furrowing of forehead Grimacing</td>
</tr>
<tr>
<td>Head, neck and trunk</td>
<td>Shoulder elevation/shrugging Axial hyperkinesis (‘copulatory’ movements)</td>
</tr>
<tr>
<td>Upper limbs</td>
<td>Hyperpronation Wrist/elbow flexion/extension Metacarpopharyngeal flexion/extension (‘piano playing’) Finger filliping Lateral outsplaying Exaggerated arm swing</td>
</tr>
<tr>
<td>Lower limbs</td>
<td>Adduction/abduction Flexion/extension hips/knees/ankles Ankle rotation Inversion/eversion Lateral ‘outsplaying’ of toes Flexion/extension of toes</td>
</tr>
<tr>
<td>Internal musculature</td>
<td>Dysphagia Dyspnoea Spontaneous vocalisations: Grunting, moaning Dysarthria ‘Nasal’ speech ‘Stacatto’ speech (abductor spasm) ‘Breathless whisper’ (abductor spasm)</td>
</tr>
</tbody>
</table>

CLINICAL FEATURES

The movements of tardive dyskinesia may be symmetrical bilaterally, predominantly unilateral or exclusively unilateral in their distribution. These movements usually decrease or disappear when the patient is at rest or sleeping. According to DSM-IV-TR, over three-fourths of the individuals with tardive dyskinesia have abnormal oro-facial movements, approximately one-half have limb involvement, and up to one-quarter have axial dyskinesia of the trunk.
TOOLS FOR ASSESSMENT OF DYSKINESIA

- Abnormal Involuntary Movement Scale (AIMS) (Guy, 1976): The AIMS contains seven items, rated on a severity scale of 0–4, which assess abnormal movements in various anatomical locations including facial and oral, extremity, and truncal movements. It also includes a global judgment of three items (severity of abnormal movements overall, incapacitation due to abnormal movements, and patient's awareness of abnormal movements), also rated on a scale of 0–4.

- Extrapyramidal Symptom Rating Scale (ESRS) (Chouinard et al, 1980): The ESRS is a more comprehensive movement-disorder rating scale and rates symptoms based on severity and frequency, on a scale of 0–6. It includes subjective and objective measures of parkinsonism, dystonia, and akathisia as well as dyskinesia.

- Simpson Tardive Dyskinesia Rating Scale (Simpson et al, 1979): is a 34-item scale, each item being rated on a six-point scale. It offers the opportunity for raters to add additional individualized items.

- The Dyskinesia Identification System-Coldwater (DIS-Co) (Sprague, 1984): A scale consisting of 34 items grouped in 10 body areas and scored on a five-point scale, has been thoroughly evaluated on psychometric properties. This scale has also been abbreviated to 15 items, the Dyskinesia Identification System: Condensed User Scale (DIS-CUS).

- The Smith Tardive Dyskinesia Scale (Smith et al, 1983): Assesses both tardive dyskinesia and parkinsonism in a single 24-item scale. Parkinsonism and tardive dyskinesia sub-scores can be computed separately.

- Accelerometry: Accelerometers are widely used to quantify hyperkinesias such as tremor and dyskinesia. An accelerometer is a miniature piezoelectric device or strain gauge that is responsive to acceleration in a single plane. The device can be attached to the finger, hand, foot, or leg without obstructing normal movements.

- Force Procedures: Force gauges, or load cells, can be used to evaluate dyskinesia. These work on the assumption that dyskinetic movements are direct consequences of random muscle contractions and that these muscle contractions produce changes in force measurable over time (df/dt).

COURSE AND OUTCOME

As per DSM-IV-TR, onset may occur at any age and is almost always insidious. The signs are typically minimal to mild at onset. When individuals receiving neuroleptic medication are assessed periodically, tardive dyskinesia is found to be stable over time in about one-half; to worsen in one-quarter, and to improve in the rest. Younger individuals generally tend to improve more readily; in older individuals there is a greater likelihood that tardive dyskinesia may become more severe or more generalized with continued neuroleptic use. When neuroleptic medications are discontinued, it is estimated that 5%-40% of all cases remit and between 50% and 90% of mild cases remit. The dyskinesia remits within 3 months in one-third of the cases and remits by 12-18 months in more than 50% of cases, although these percentages are lower in older persons. This data does not match with a study in which approximately half the sample showed a 50% improvement in ratings over one year, but signs resolved completely in only 2 per cent of subjects (Glazer and Morgenstern, 1988).

TREATMENT

A) Cessation or Reduction Of Antipsychotic:

Jeste et al (1988) found that discontinuation or reduction was favourable over long term in 30-50% of patients, as was reciprocated by few other studies. But reduction or cessation of antipsychotic may cause worsening of movements in some people as mentioned earlier. In addition, discontinuation of antipsychotics...
should always be weighed against the risk of psychotic relapse.

B) Dopamine Depletors:

These agents effectively reduce dopaminergic synaptic activity, thereby reducing the tardive dyskinesia symptoms without exposing the brain to an offending dopamine antagonist. These include reserpine and TBZ (a synthetic benzoquinolizine). Both reserpine and TBZ inhibit the vesicular monoamine transporter. By preventing monoamines to be sequestered in the nerve terminal's vesicles, they allow the monoamines to be exposed to monoamine oxidase and catabolized, thus being markedly depleted in the nerve terminals. Reserpine is used in dose of 1-8mg/day and tetrabenazine in 25-150mg/day (Ondo, 1999).

The U.S. Food and Drug Administration today approved Ingrezza (valbenazine) capsules to treat adults with tardive dyskinesia. It acts as a vesicular monoamine transporter 2 (VMAT2) inhibitor. This is the first drug approved by the FDA for this condition. Ingrezza may cause serious side effects including sleepiness and heart rhythm problems (QT prolongation). Its use should be avoided in patients with congenital long QT syndrome or with abnormal heartbeats associated with a prolonged QT interval. Those taking Ingrezza should not drive or operate heavy machinery or do other dangerous activities until it is known how the drug affects them.

C) Antipsychotics:

Selective D2 Antagonists: The substituted benzamides are selective D2 antagonists; these include sulpiride, tiapride and remoxipride. Metoclopramide also belongs to this group. Their use has shown to improve the abnormal movements in tardive dyskinesia (Chouinard, 1995).

The most studied antipsychotic for treatment of tardive dyskinesia is clozapine. There are many reports of clozapine successfully reducing the abnormal movements of tardive dyskinesia (Bassitt and Neto, 1998).

Other atypical antipsychotics being studied with mixed results include quetiapine, olanzapine, sertindole and ziprasidone.

D) Antioxidants:

Antioxidants, such as tocopherol (vitamin E), work on the basis of the theory of free-radical toxicity in the basal ganglia leading to tardive dyskinesia. Vitamin E protects against deterioration of tardive dyskinesia but there is no evidence that vitamin E improves symptoms of tardive dyskinesia. Ascorbic acid in particular may be a promising adjunct to vitamin E therapy since it recycles the tocopheroxyl radical of vitamin E.

E) Benzodiazepines:

Apart from having anxiolytic and muscle relaxant properties, they also facilitate GABAergic transmission by opening chloride channel on the GABAA receptor. One recent trial found end point abnormal movement scores to be better for those receiving adjunct benzodiazepines (Bhoopathi and Soares-Weiser, 2006).

F) GABA-ergic Strategies:

These include GABA receptor agonists like muscimol and progabide, GABA transaminase inhibitors like vigabatrin, GABAB agonist like baclofen, GABA mimetics like sodium valproate, but none of these have any proven efficacy for tardive dyskinesia. Gabapentin could be of significant clinical utility in the management of tardive dyskinesia psychotic patients with affective features or those with bipolar spectrum disorders (Hardoy et al, 1999).

G) Cholinergic Drugs:

Use of cholinergic drugs was based on the reciprocal dopamine-acetylcholine balance in the basal ganglia. In a systematic review and meta-analysis including eleven trials (Tammenmaa et al, 2004), cholinergic drugs (choline, lecithin, physostigmine, tacrine, galantamine, donepezil, rivastigmine, deanol etc.) showed a minor trend for improvement of tardive dyskinesia symptoms, but results were not statistically significant.

H) Other Strategies:

In severe cases, where tardive dyskinesia is
different neuromedical conditions. Many of these neuromedical conditions are found more commonly in older people than in younger individuals. When these adventitious movements develop as a consequence of neuroleptic treatment, they are known as tardive dyskinesia. The proprioceptive changes that are associated with edentulousness have not been well characterized and may be etiologically related to the increased rates of dyskinesia seen in older adults, either with or without neuroleptic treatment.

GENDER AS A FACTOR IN THE DEVELOPMENT OF TARDIVE DYSKINESIA

Gender has been suggested to be an important factor in the development of tardive dyskinesia, and some authors have concluded that women have a higher prevalence of tardive dyskinesia than men, and some other have found no gender difference. The prevalence of TD increases fivefold when women over 70 are compared with those less than 50 years of age. Among men, that increase is only twofold. Women were reported to have more severe tardive dyskinesia than men (3.1% vs. 1.3%). Certain investigators have reported that women tend to have more chronic illnesses, larger doses of neuroleptics and longer hospitalizations tend to receive or to experience longer periods of neuroleptic treatment than men. Thus, it is difficult to attribute the higher prevalence of tardive dyskinesia among women. Another possible explanation is that, in some studies, the women were older than the men. Women have longer life spans than men and therefore tend to be overrepresented in surveys of older patients (Yassa et al, 1986). Age does not seem to increase the prevalence of tardive dyskinesia among men to the same degree as among women. Another possibility is that psychosis may tend to develop later in life among women than among men. They showed tardive dyskinesia to have developed after shorter periods of neuroleptic treatment, and in more severe forms, when neuroleptic treatment was initiated later rather than during the acute phase of the illness.

aging and tardive dyskinesia

It has been estimated, for instance, that 1% of elderly people living outside of the institution have spontaneous dyskinesia. Yet patients in institutions have an average dyskinesia prevalence of 5%, with some groups exhibiting much higher rates. In the geriatric-hospital sample, 85% of patients had orofacial movements. Neuroleptic users not wearing dentures were found to be more likely to be rated as having tardive dyskinesia. Intellectual dysfunction, organic mental disorder and negative symptoms are vulnerability factors in the development of tardive dyskinesia in older population. It was hypothesized that impairments of peripheral and central glucose and insulin metabolism may be significant in the pathophysiology of tardive dyskinesia as the high prevalence of diabetes among the elderly population (Karson et al 1990). Abnormal involuntary body movements can develop in many different internal and external body regions, and in association with many painful or causing muscle damage, and all other strategies have failed, the dose of antipsychotic drug can be increased. But this strategy is limited by the increase in other side effects.

Propranolol, fusaric acid and clonidine decrease the noradrenergic activity and have been reported to be useful but require further study to clarify their role.

Other therapies tried without any proven efficacy are lithium, insulin, phenylalanine, piracetam, pyridoxine, tryptophan, cyproheptadine, vasopressin, naloxone, morphine, estrogen, calcium channel blockers, botulinum toxin (for oro-facial dyskinesia) and electroconvulsive therapy (Fahn and Jankovic, 2007).

Deep brain stimulation (DBS) to the globus pallidus interna has been found useful in cases of orofacial tardive dyskinesia though the best results have been for tardive dystonia.
than earlier in life. Perhaps, too, women are simply protected by estrogens earlier in life. Estrogens have an antidopaminergic activity thus possibly protecting premenopausal women from developing tardive dyskinesia. Premenopausal women, therefore, may need lower neuroleptic dosages than do men of the same age (Yassa et al, 1991).

TARDIVE DYSKINESIA AND AFFECTIVE DISORDER

The most compelling evidence for affective disorder as a risk factor for tardive dyskinesia comes from a prospective longitudinal study of tardive dyskinesia development in a large cohort (Kane et al, 1984). In that study, preliminary analysis of 5-year dyskinesia rates showed 23.3% for schizophrenics, 26.9% for schizoaffectives, and 37.9% for patients with affective disorder (including both unipolar and bipolar patients). The relationship between affective illness and tardive dyskinesia remains statistically significant even after controlling for other risk factors such as age, gender, lithium treatment, and ECT. Unipolar patients showed vulnerability comparable to that for bipolar patients, both sets of patients developed tardive dyskinesia after significantly shorter neuroleptic exposures than schizophrenic patients or schizoaffective patients. Observations of mood-dependent dyskinesia have suggested that the state of depression may induce and/or aggravate dyskinesia. Indeed, patients who tend to be rapid-cycling bipolars, responding poorly to conventional pharmacotherapy, have shown increased dyskinesia during depression and decreased dyskinesia during mania. The validity of those observations is not withstanding, mood-dependent dyskinesia has not been observed in the vast majority of bipolar patients.

DIABETES MELLITUS AND TARDIVE DYSKINESIA

On the basis of incidental observations and findings from pilot studies it was found that impaired glucose metabolism is associated with increased risk for tardive dyskinesia. That view was based on the following observations:

1. The proportion of tardive dyskinesia patients also diagnosed as having non-insulin-dependent diabetes mellitus (NIDDM) was considerably higher than would have been expected on the basis of the known rates of NIDDM in the general population.

2. On average, fasting blood glucose concentrations were found to be significantly higher in patients with tardive dyskinesia than in patients without tardive dyskinesia, an effect that remained significant after controlling for variance due to age and gender.

3. A disproportionately large number of nondiabetic tardive dyskinesia patients had a positive family history of NIDDM. The presence of insulin receptors in the brain is well established. Although there are structural differences between the brain's insulin receptors and peripheral insulin receptors both are coupled to tyrosine kinase activity, which is integrally involved in the action of insulin. The cellular actions of insulin involve many discrete cascades only some of which are involved in glucose utilization, and that tyrosine kinase activity is not involved in all of those cascades. This is a critical consideration, as it raises the possibility that the cascade of events following the binding of insulin to its receptor, so critical for the pathogenesis of tardive dyskinesia, may not be the same as that involved in glucose utilization.

ROLE OF ETHNICITY IN THE DEVELOPMENT OF TARDIVE DYSKINESIA

There is limited evidence of differences in the prevalence of tardive dyskinesia among ethnic groups. Yassa and Jeste (1992) reported that patients from Asia seemed to have a lower prevalence of tardive dyskinesia (16.6%) than those from North America (27.6%), Europe (21.5%), and Africa (25.5%). Several reasons were suggested for the differences in the reported rates of prevalence, including ethnicity and variations in practices of prescribing neuroleptics.
Some dramatic ethnic differences in responses to drugs have been reported in the literature.

**SMOKING AND TARDIVE DYSKINESIA**

Numerous epidemiological studies show that cigarette smoking is associated with a lower risk of idiopathic Parkinson’s disease (PD). This has led to the hypothesis that nicotine exposure may protect against the development of PD. The pathophysiological basis of PD (idiopathic or neuroleptic induced) is believed to be the reverse of TD. Thus, smoking might be expected to aggravate the development of TD. However, the underlying mechanism for the association between TD and smoking remains unclear. Suggested neurobiological mechanisms for this association of smoking and TD include increased dopaminergic activity from nicotine leading to dopamine receptor hypersensitivity. Epidemiological studies have shown mixed results on smoking's association with TD with some finding an increased incidence of TD in smokers, while others find no relationship between smoking and TD. Repeated measurements revealed a positive correlation between changes in cigarette consumption and changes of the severity of TD, suggesting a possible cause–effect-relationship between smoking and TD (Diehl et al, 2009).

**VULNERABILITY TO TARDIVE DYSKINESIA IN SCHIZOPHRENIA:**

An exploration of individual patient factors Many studies suggest that patients having family history of schizophrenia, formal thought disorder, cognitive impairment, frontal-lobe dysfunction, abnormalities of structure and function in fronto-temporal and corticostriatopallidothalamic networks, ventricular enlargement or cortical atrophy and reduced caudate volume are the individual risk factor in schizophrenic patients. The extent and form of the structural and functional consequences of any such neuronal "maldevelopment" might be elements in one's vulnerability to tardive dyskinesia. Obstetric complications, perhaps uncommonly frequent in the early history of patients with schizophrenia, suggest one potential source of early cerebral insult, and a family history of schizophrenia might reflect the operation of genetic factors as well. Although both aspects have been considered as potential risk factors for tardive dyskinesia, a consistent picture is yet to emerge. An additional factor might be that patients with schizophrenia have an excess of peripheral markers of neurodevelopmental perturbation reflected in minor physical anomalies (O'Callaghan et al, 1999)

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STRESS AND PSYCHOLOGICAL FACTORS IN ACUTE CORONARY SYNDROMES

**Introduction**

An increasing body of evidence supports a link with acute and chronic stress as risk factors for acute myocardial infarction. The potential role of psychosocial factors in acute disease onset and prognosis after MI has been studied. Acute coronary syndromes are due to coronary thrombosis associated with plaque rupture, which in turn is related to stressful factors.

**Circadian Variation**

Increased frequency of MI, sudden cardiac death, and myocardial ischemia occurs during sunrise. A meta-analysis of 66,635 patients with an acute MI and 19,390 patients with sudden death reported an excess of MIs (relative risk 1.38) and sudden deaths (relative risk 1.29) between the hours of 6 am and noon compared to the rest of the day.

The acute cardiac events are triggered by external activities, particularly those activating the sympathetic nervous system. In a study of 1225 patients an absence of a circadian variation was noted in diabetics, particularly those with evidence of cardiac autonomic neuropathy who have absent heart rate variability, and those taking beta blockers or aspirin at the time of admission for an MI. Trauma, acute stroke or bereavement were the external factors.

Fibrinolytic capacity has a daily variability and contributes to increase in myocardial infarction during sunrise. Plasminogen activator inhibitor-1 activity rises in the early morning while tissue plasminogen activator activity is at its nadir; betablockers can reduce this relatively prothrombotic state.

**Triggering Of Acute MI**

In a study of 849 patients with acute MI, 48 percent described one or more possible triggers, emotional upset was the commonest (14 %). Studies identified possible triggers in up to 10 percent of patients.

Life events and other crises — In a prospective study of middle-aged widowers, for example, a 40 percent increase in the mortality rate in the first 6 months was observed following bereavement; more than half of which was attributed to cardiovascular causes.

The INTERHEART study compared over 11,000 patients with a first MI with over 13,000 matched controls from 52 countries on six continents. Stressful life events occurred more frequently within the prior year among patients than among controls (16.1 versus 13.0 percent; odds ratio 1.48; 95% CI 1.33-1.64). Stressful life events specified included marital separation or divorce, loss of job or retirement, loss of crop or business failure, violence, or other major stress.

An increase in cardiovascular events has also been associated with disasters. This are illustrated by the following observations:

- After the 1981 Athens earthquake, the incidence of cardiac deaths rose from the normal average of 2.6
deaths per day to an average of 5.4, with a peak of 8 deaths per day.

- During the initial week after six blizzards in Massachusetts from 1974 to 1978, a 22 percent increase in ischemic deaths per day was reported in comparison to preceding and subsequent control weeks.

- During the six weeks following the Hanshin-Awaji, Japan earthquake there were 10 cardiovascular or sudden deaths compared to three during the same period in the previous year. A study of 42 elderly patients with well controlled hypertension who lived near the epicenter reported that one to two weeks after the earthquake (when aftershocks were frequent), there were transient increases in blood pressure, blood viscosity determinants, and fibrin turnover.

- The 1991 Iraq war also provided an opportunity to study the cardiovascular effects of psychological stress. During the first week of missile attacks on Israel, 20 civilians developed an acute infarction in the area served by one hospital, compared to only eight during a control period.

- There was a statistically significant 49 percent increase in patients admitted with MI through 16 emergency departments within a 50-mile radius of the World Trade Center in the 60 days after September 11, 2001, compared with the 60 days beforehand (118 MIs after versus 79 before).

The role of other factors such as unaccustomed physical activity, altered sleep patterns, and diet needs to be considered.

Personality and mood — Studies have shown increasing evidence that hostility, cynicism, and anger form a critical "toxic" component of type A behavior associated with enhanced cardiovascular risk.

Anxiety

Anxiety as a cardiovascular risk factor has not been studied well but, two representative studies suggest an association:

- During a two-year follow-up of 33,999 male health professionals in the United States who were aged 42 to 77 years and initially free of diagnosed disease, the age-adjusted relative risk of fatal cardiovascular disease was three-fold greater for those having the highest levels of phobic anxiety compared to those with the lowest levels.

- In a study of 735 men (mean age 60 years) without cardiovascular disease or diabetes, the presence of overall anxiety, as assessed by four scales which included phobia, independently and significantly predicted the development of myocardial infarction during a mean follow-up of 12.4 years.

Depression

A wide range of evidence supports depression as a risk factor for cardiovascular disease, both in otherwise healthy subjects and in those with known cardiovascular disease. The potential mechanisms include hypothalamic-pituitary-adrenal axis dysfunction, inflammatory and prothrombotic changes, dietary factors, low omega-3 fatty acid levels, reduced heart rate variability and adverse behavior including medication non-adherence, smoking and physical inactivity. The following studies support the relationship between depression and cardiovascular disease:

- The largest analysis of this issue comes from data on over 93,000 postmenopausal women aged 50 to 79 years participating in the Women's Health Initiative Observational Study. At four years, patients with current or previous depression had significantly higher rates of cardiovascular death (0.79 versus 0.52 percent) and all-cause mortality (2.87 versus 2.18 percent) than those without depression.

- In the worldwide INTER HEART study, depression was significantly more common among patients with a first MI than among controls (24.0 versus 17.6 percent).

- In the Heart and Soul study of patients with stable coronary artery disease, baseline depressive symptoms assessed using the Patient Health Questionnaire (PHQ) were predictive of subsequent age-adjusted annualized cardiovascular events (10% for PHQ score >10 versus 6.7% without depressive symptoms, HR 1.5, p=0.002). Adjustment for biological factors attenuated the association, which was no longer significant after further adjustment for potential behavioral mediators, in
particular physical inactivity. These findings suggest the need for further investigation of exercise as a possible intervention to ameliorate the link between depression and cardiovascular risk.

The severity of depression has also been correlated with cardiovascular risk. This was illustrated in a prospective cohort of 4493 elderly subjects (≥65 years of age) in the Cardiovascular Health Study who were initially free of cardiovascular disease at baseline and were followed for six years, found that each 5-unit increase in depression score was associated with an adjusted hazard ratio of 1.15 and 1.16 for the development of coronary heart disease and all-cause mortality, respectively. Those with the highest depression scores had a 40 and 60 percent increased risk of coronary disease and death compared to those with the lowest scores. A similar graded relationship was noted for the occurrence of MI in the Baltimore Epidemiologic Catchment Area study; the relationship was independent of coronary risk factors.

A more complex relationship was noted in the Systolic Hypertension in older adults Program (SHEP). In this trial, 4736 subjects aged 60 years or more with isolated systolic hypertension were followed for an average of 4.5 years, with depressive symptoms being assessed by six month questionnaires. Baseline levels of depressive symptoms did not predict future cardiovascular events; however there was an increase in depression score prior to MI, stroke or death. One study found that in men, but not women, over 70 years the recent onset of depression, but not chronic depression, was associated with an increased risk of cardiovascular mortality (relative risk 1.75), cardiovascular and coronary heart disease events (relative risk 2.07 and 2.03, respectively), and all cause mortality (relative risk 1.4) (figure 2).

**Anger**

Anger appears to have a deleterious effect on the cardiovascular system. Patients with trait anger have a relatively stable personality, but manifest rage and fury more often, more intensely, and have longer-lasting episodes. The relationship between anger and coronary heart disease was evaluated in the Atherosclerosis Risk in Communities (ARIC) trial of 12,986 men and women. Compared to normotensive subjects with low trait anger, normotensive subjects with high trait anger were at higher risk for all coronary heart disease events, including acute MI, cardiac mortality, silent MI, or coronary revascularization (hazard ratio 2.2), and for hard cardiac events of acute MI or cardiac mortality (hazard ratio 2.7). In hypertensive individuals, there was no relationship between trait anger and risk of coronary heart disease. Episodes of anger may also trigger an acute MI:

- The Determinants of Myocardial Infarction Onset Study interviewed 1623 patients within one week of MI: 2.4 percent reported episodes of anger within the two hours prior to MI onset. The level of anger corresponded on a scale to the subjects feeling "very angry, body tense, clenching fists or teeth." The most frequent causes of anger were arguments with family members (25 percent), conflicts at work (22 percent), and legal problems (8 percent). The relative risk of MI following episodes of anger was 2.3, especially during the first two hours after an outburst of anger.

- A meta-analysis of four observational studies (n = 4546 cases of MI and 462 cases of acute coronary syndrome) found that the risk of acute cardiovascular events was five times greater in the two hours following an outburst of anger, compared with other times. Anger in response to stress may be related to premature MI in young men. As an example, one longitudinal study of 1055 young medical students established anger reactions to stress by self reporting on a questionnaire administered in medical school. After a median follow-up of 36 years, those with the highest level of anger, compared to those with lower levels, had an increased risk of premature cardiovascular disease developing before the age of 55 (adjusted relative risk 3.1), coronary heart disease (adjusted relative risk 3.5), and MI (relative risk 6.4).

Seasonal pattern — Several series have demonstrated a seasonal pattern of deaths from MI, with more fatal events (20 to 30 percent variation) occurring in the winter than the summer. As an example, a recent report from the National Registry of Myocardial Infarction evaluated 259,891 cases of acute MI from 1474 hospitals during a 25 month period; approximately 53 percent more MIS occurred in the winter or spring compared to the summer (p<0.05) (figure 3). The trends were independent of gender, geographic location, age, and the type of MI (ST elevation or non-ST elevation). In-hospital fatality rates for MI also followed a seasonal pattern, with a peak of 9 percent in winter and a nadir in the spring (8.4 percent). However, a seasonal pattern is absent in diabetics or those taking beta blockers or aspirin, suggesting an important role
for the sympathetic nervous system.

A similar association between the season and death from MI was also noted in another study of 300,000 deaths from MI or stroke in the Canadian Mortality database; however, in this study there was a relationship between mortality and age. Deaths from MI were highest in January and lowest in September, with a relative risk difference of 18.6 percent. The seasonal mortality variation (winter versus summer) increased with increasing age; for those <65, 65 to 74, 75 to 84 and >85, mortality was 5.8, 8.3, 13.4, and 15.8 percent, respectively. An interesting explanation for the association of coronary disease with the winter months was noted by the Eurowinter Group. Among various European regions, the increase in coronary mortality was greater in warmer than in colder regions. The increase in risk was associated with low living-room temperatures and the wearing of fewer clothes when people went outdoors. Other social factors — In addition to psychosocial factors, a number of other social factors have been identified as triggers of an acute MI:

- An acute coronary ischemic syndrome is the most common cardiac pathology associated with cocaine abuse and can occur with all routes of cocaine intake. In a survey of 10,085 adults between the ages of 18 and 45, cocaine use accounted for 25 percent of nonfatal MIS.

- Smoking may be a rare trigger of MI. In one report of 3882 patients with an acute MI, 124 (3.2 percent) reported smoking marijuana in the prior year, 37 within 24 hours and nine within one hour. The risk of MI was increased 4.8-fold over baseline in the 60 minutes after marijuana use and then rapidly declined with time.

- Exposure to particulate air pollution also may be associated with MI. This may be due, in part to a sympathetic stress response, as detected by changes in heart rate variability, the production of cytokines, and an increased vulnerability to plaque rupture. In a study of 772 patients with an acute MI, the risk of an MI was increased in the two hours after exposure to elevated levels of fine particles in the air (odds ratio 1.48 compared to low levels of fine particles); this effect lasted for up to 24 hours after exposure.

- Exposure to vehicular traffic has been implicated as an MI trigger. In one analysis, 691 patients with an acute MI were interviewed about activities during the four days preceding the event. Traffic exposure (defined as time spent in a vehicle) occurred during the hour before MI in 75 (12 percent) of 625 patients. Compared to the frequency of traffic exposure during the preceding three days, the likelihood of traffic exposure during the hour preceding MI was significantly increased (adjusted odds ratio 2.73).

- There was a four-fold increase in relative risk of MI over baseline in the first hour after the stress of traffic.

- A daily variation in MI and stroke has also been described with a peak incidence on Mondays. Holidays such as Christmas and New Year's day are also associated with increased cardiac mortality.

**PATHOPHYSIOLOGY**

There are several mechanisms by which emotional stress might trigger an acute MI. The physiologic changes that have been described in the morning period of enhanced cardiovascular risk — increases in blood pressure, heart rate, vascular tone, and platelet agreeability — also may result from mental stress. These factors may all be related to abnormalities in autonomic tone and activation of sympathetic nervous system activity, which may enhance platelet aggregation and increase the susceptibility to serious ventricular arrhythmia. Support for the role of autonomic dysfunction comes from a study of 804 post MI patients with and without depression who underwent 24 hour ambulatory monitoring. Compared to patients without depression, those with minor or major depression had significantly decreased heart rate variability, indicating excessive sympathetic and/or reduced parasympathetic tone. Myocardial ischemia and plaque rupture — Mental stress produces significant increases in heart rate and blood pressure that may lead to increased myocardial oxygen demand and plaque disruption. In addition to a rise in the rate pressure product, there is also evidence that mental stress may lead to a primary reduction in myocardial oxygen supply. Whereas coronary arteries of normal patients dilate during mental stress, impaired dilation and even constriction have been demonstrated in atherosclerotic arteries. The vaso constriction induced by stress may not be immediate. In a dog model, profound coronary vaso constriction could be
demonstrated two to three minutes following elicitation of anger. The vaso constriction persisted well after heart rate and arterial blood pressure recovered. Myocardial ischemia, as evidenced by ST depression and more sensitive means such as radionuclide ventriculography and positron emission tomography, has been shown to be precipitated by psychologically stressful circumstances (eg, public speaking). One study, for example, found that among 29 patients with coronary artery disease with exercise induced wall motion abnormalities, 21 (72 percent) also exhibited wall motion abnormalities following mental stress. The ischemia was usually silent, often without ECG abnormalities. Altered platelet activity — Although studies have not all shown consistent findings, mental stress enhances platelet aggregation secondary to sympathetic nervous system activation, and may promote mitogenic activity in plasma due to platelet-derived growth factors. There is a compensatory increase in fibrinolytic activity following acute stress, but a diminished fibrinolytic response due to endothelial dysfunction may lead to a prothrombotic imbalance. Altered cardiovascular risk factors — Although the effects of chronic stress on cardiovascular risk factors are less clear, depressed mood scores and increased sympathetic reactivity have been associated with elevated cholesterol levels. The interaction of altered sympathetic and cortisol levels with oxidized LDL and macrophage activation is receiving increasing attention.

The new information on triggering can be incorporated into a hypothesis of the progression of coronary artery disease. The onset of MI may occur when a vulnerable atherosclerotic plaque disrupts in response to mental stress or anger that produces transient pressure surges or vasoconstriction. If the plaque disruption is major with extensive exposure of collagen and atheromatous core contents to the lumen, this may lead immediately to occlusive thrombosis, with MI or sudden cardiac death. If the disruption is minor, it may lead to non occlusive thrombosis. In this setting, the patient may be asymptomatic or develop unstable angina or non-ST elevation MI. The lesion may gradually heal with smooth muscle cell proliferation and a greater degree of stenosis. Alternatively, a further increase in coagulability or vaso constriction may precipitate occlusive thrombosis, MI, and sudden cardiac death.

CENTRAL NERVOUS SYSTEM DYSFUNCTION

The association between psychosocial stress and myocardial infarction may involve dysfunction of neural circuits that include the amygdala, which participates in sympathetic responses to stress. A retrospective study identified patients without known cardiovascular disease (n = 293) who underwent positron emission tomography/computerized tomography and were followed for a median of four years. After controlling for potential confounding factors (eg, diabetes, hypertension, and smoking), the analyses found that increased resting metabolic activity in the amygdala was associated with an increased risk of cardiovascular events such as myocardial infarction, stroke, and unstable angina (hazard ratio 1.4, 95% CI 1.1-1.8). In addition, the study suggested that the link between amygdalar activity and cardiovascular disease may be mediated by arterial inflammation. A separate cross-sectional study of individuals with chronic stress (ie, posttraumatic stress disorder; n = 13) suggested that perceived stress was positively associated with amygdalar activity and arterial inflammation.

DEPRESSION AND PSYCHOSOCIAL STRESS POST MI

Major depression develops in almost 20 percent of patients after MI, while over 33 percent have significant symptoms of depression soon after an MI. Depression is associated with poor compliance with recommendations for post MI therapy.

In addition, depression (even if minimal) and other psychosocial stress factors, including hostility, social isolation, anxiety, anger, and marital stress, are related to a poor prognosis among patients recovering from an MI and to a reduced quality of life:

- A review of 53 studies and 4 meta-analyses by the American Heart Association found that depression after acute coronary syndrome is a risk factor for adverse outcomes, including increased all-cause and cardiac mortality. Despite the lack of compelling evidence that treating depression improves survival following acute coronary syndromes, the association nevertheless concluded that clinicians should consider comprehensive evaluation and treatment of severe or persistent depression.
A substudy from the GISSI-2 trial involved 2449 patients who completed a questionnaire dealing with psychological variables and were followed for six months after MI. The presence of extroversion and neurotic behavior had a protective effect, reducing risk by 40 percent. In contrast, the presence of vital exhaustion (excess fatigue, lack of energy, and sleep disturbances), depression, or lack of anxiety about the MI increased risk of death by two to three fold. The impact of these factors was comparable to that of well established clinical predictors such as left ventricular function, arrhythmia, and eligibility for exercise testing.

High levels of social support may influence mortality among depressed post-MI patients. A study of 887 patients who completed questionnaires seven days after an MI found that evidence of mild to moderate depression, present in 32 percent, was associated with increased cardiac mortality during a one year follow-up. High levels of social support alone did not influence cardiac mortality, but were associated with improvements in depressive symptoms. In addition, high levels of social support appeared to buffer the impact of depression on mortality.

A population-based case-control study found an association between MI and increased risk of suicide. The study identified 19,857 people aged 40 to 89 years who died by suicide, and randomly selected 190,058 controls matched by sex, date of birth, and calendar time (control was alive on the day that the case committed suicide). Patients who suffered an MI and had no history of psychiatric illness were more than three times as likely to commit suicide within one month of the MI, compared with individuals who had no history of MI or psychiatric illness (incidence rate ratio 3.3, 95% CI 1.6-6.6). The risk of suicide remained high for at least five years after an MI.

Additional information about risk factors for adverse outcomes after acute coronary syndromes is discussed separately.

**Treatment**

Spontaneous remission of depressive symptoms occurs in approximately one half of cases of post-MI depression; the other cases either persist or remit and relapse within one year. This suggests that a substantial number of patients may benefit from treatment of depression following an MI. Standard treatment of depression can include psychotherapy, drug therapy, or both; the efficacy of these therapies in non-post-MI patients is similar. A number of patients with increased stress (without depression) post MI may also benefit from psychosocial interventions.

**Psychosocial interventions**

Psychosocial interventions have been evaluated for efficacy with regard to both depressive and cardiovascular outcomes. Observational studies suggest that stress reduction post-MI may have cardiovascular benefits. In one report, stress assessments were made before discharge and by telephone on a monthly basis in 461 men with a recent MI; those determined to have high stress levels received outpatient visits from nurse. This intervention was associated with a 41 percent reduction in cardiac mortality and a 46 percent reduction in nonfatal cardiac events during a five year follow-up; there were also reductions in psychologic distress, systolic blood pressure, heart rate, and the serum cholesterol concentration.

One high quality study is the ENRICHD trial, which evaluated 2481 patients post-MI who had either depression or a low level of perceived social support or both. All patients were randomly assigned to six months of usual medical care with or without psychosocial intervention with individual and group cognitive therapy and social support counseling; antidepressant drugs were added for up to 12 months if there was severe depression or no response to cognitive therapy. At a mean follow-up of 29 months, patients receiving psychosocial intervention had less depression and better levels of perceived social support, but there was no reduction in mortality.

In the CREATE trial, which evaluated 284 coronary heart disease patients (69 percent postacute coronary syndrome) with moderate to severe depression. Patients were randomly assigned to 20 to 25 minute weekly sessions of clinical management from a trained psychotherapist with or without an additional 45 minutes of interpersonal psychotherapy. No added benefit on depression score was noted with the interpersonal psychotherapy. Two factors that may have contributed to the lack of benefit noted in the CREATE trial were, compared to ENRICHD, a greater degree of underlying depression and a higher level of psychological support in the control group. In addition, a cognitive behavioral approach was used in ENRICHD.
Collaborative care interventions may also be useful. A 24 week, randomized trial (Management of Sadness and Anxiety in Cardiology [MOSAIC]) compared collaborative care with usual care in 183 patients with depression and anxiety, who were hospitalized for acute cardiac disease (including acute coronary syndrome) and then discharged. Collaborative care was administered over the telephone by a social worker who coordinated treatment recommendations made by psychiatrists and general medical clinicians; the trial was open label, except for outcome assessors. Improvement of depression, health related quality of life (eg, pain, energy, and psychological distress), and functioning were greater in patients who received active treatment. In addition, time to cardiac readmission was longer with collaborative care than usual care (92 versus 63 days). Additional information about collaborative care is discussed separately. (See "Unipolar depression in adult primary care patients and general medical illness: Evidence for the efficacy of initial treatments", section on 'Collaborative care'.)

Drug therapy
Most patients who experience depression after an MI do not receive antidepressants. This is probably related to several factors, including the high rate of spontaneous remission of depressive symptoms and fears regarding the safety of antidepressants in post-MI patients.

Tricyclic antidepressants have many properties that may adversely affect outcomes after MI, including orthostatic hypotension and similarities to class IA antiarrhythmic drugs, which increase arrhythmic deaths and can prolong the QT interval.

Cohort studies have reported conflicting results with regard to the risk of MI in patients taking tricyclic antidepressants, but the results are difficult to interpret since it is not clear if the cardiovascular effects are related to the underlying depression or the treatment. Furthermore, these studies did not look specifically at post-MI patients.

There are more data documenting cardiac safety with selective serotonin uptake inhibitors (SSRIS) post-MI and, at least one SSRI sertraline has been reported to be safe in both a case-control study and randomized, controlled trial. In the SADHART trial (Sertraline Antidepressant Heart Attack Randomized Trial), 369 patients with depression who had been hospitalized for an MI or unstable angina were randomly assigned to 24 weeks of treatment with sertraline (50 to 200 mg/day) or placebo. Sertraline was no different from placebo in its effect on any measure of cardiovascular safety including the left ventricular ejection fraction (the primary end point), blood pressure, heart rate, arrhythmias, or QTc prolongation. The incidence of severe cardiovascular adverse events tended to be lower in the sertraline group (14.5 versus 22.4 percent), although this difference was not statistically significant.

The findings from SADHART are encouraging, although several limitations of this study should be noted:

- The sample size was too small to identify rare adverse events or drug-drug interactions.
- Antidepressant treatment was not initiated until an average of 34 days following an MI; thus, the safety of sertraline in the immediate post-MI period is not clear.
- Patients who had other medical conditions were excluded from the study, so that the results may not be generalized to the typical population of post-MI patients.

The efficacy of SSRIS in the treatment of depression in patients with MI has been evaluated in the SADHART and CREATE trials:

- In SADHART, the depression scale ratings did not improve significantly in the total study population with sertraline therapy compared with placebo. However, the subset of patients who had a history of major depression prior to their MI did have significant improvements in depressive symptoms with sertraline.
- In CREATE, which had a 2 x 2 study design to evaluate both psychotherapy (as noted in the preceding section) and drug therapy, 284 patients with moderate to severe depression were randomly assigned to citalopram or placebo. After twelve weeks of therapy, the citalopram group had a statistically significant greater decline in a standard measurement of depression compared with placebo. However, the United States Food and Drug Administration issued warnings that citalopram causes dose-dependent QT interval prolongation that can lead to arrhythmias, and thus recommends avoiding citalopram in patients with recent acute myocardial infarction. Additional information about the citalopram warnings and cardiac effects of SSRIS.
is discussed separately. (See "Selective serotonin reuptake inhibitors: Pharmacology, administration, and side effects", section on 'Cardiac'.)

Neither trial was powered to evaluate hard cardiovascular outcomes.

Summary
The benefits of treating depression are unclear. Both psychotherapy and drug therapy improve depressive symptoms, but may not affect cardiovascular outcomes. Several nonrandomized studies have reported a cardiovascular benefit with stress reduction and enhanced social support post-MI.

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FDA Drug Safety Communication: Revised recommendations for Celexa (citalopram hydrobromide) related to a

Social Factors in Mental Illness

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Abstract:
Social factor is an important role in the development of psychiatry which influenced by many factor such as gender, social class, race and ethnicity, household patterns and some other social situation, such as disability, social security systems, labor markets, and health care organizations. The individual capacity to cope effectively persons with mental illness depends significantly on the social arrangements affecting family, work, income support and medical care. This paper provides a review of the emerging international literature to find out a linked to a wider conceptual review undertaken as part of a major project. Research have been explored in detail in relation to three areas that had been identified by the wider review as central to recovery: empowerment and control over one's life; connectedness (including both inter-personal relationships and social inclusion); and rebuilding positive identities (often within the context of stigma and discrimination). The epidemiology of mental illness in later life is complicated by the interaction among symptom occurrence, drug use, and physical illness and the varying ways individuals and families define illness, evaluate its significance and take remedial action. With aging of the population, the cumulative burdens of mental illness are increasing in most societies. Such burdens depend on not only the magnitude of dementia, depression, schizophrenia and other mental illnesses but the types of social supports and institutional arrangements that help to insulate individuals, families and communities from the most disruptive stresses and facilitate functioning.

Introduction:
“Socialization is the process by which the child acquires a cultural content, along with selfhood and Personality” (Green 1981). The personality development and the personality of different individuals have been closely related to the process of socialization and how one accepts the socially and culturally accepted behaviors. The maladaptive behavior patterns, personality traits and finally the different mental disorders may be a result of problems in socialization or the influence of different cultural and social factors one belongs to. The prevalence of different types of mental illness, crime and addiction to alcohol or drugs has been shown to be higher in areas of city lives, as compared to areas where socially the more affluent and socially better-integrated people live.

Mental Illness:
Mental illness can be seen in purely sociological terms, as a deviation from socially approved standards of interpersonal behavior, or as an inability to perform one's sanctioned social roles. Mental illnesses have direct relationship with the elements of society the basic reasons for considering mental illness as social issues are as follows;

- Development of mental illness in one person often resulting in the negative consequences such as labeling, stigma, shame and guilt.
- The cost of mental illness is high both in terms of social and monetary terms.
- A wide variety of social forces and factors like, modernization, unbridled urbanization leading to crisis and inadequacies in every aspects of life.
The widespread occurrences of mental disorder may cause trouble for the existing social system. In modern changing societies, occurrences of mental disorder can be attributed to the lack of having a supportive environment and palpable scarcity of resources for people's positive growth and development.

Social Theories:
Major Social Theories: Explaining Relationship between Society & Mental Illness

There are many theoretical propositions that explain the mutually interactive relationship between society and mental illness. Some of them give the aetiological role of social factors in mental illness or some others explain the societal reactions to mentally ill people or explain society's interpretation of mental illness or explain how social factors play an important role in the long term course, outcome and prognosis of mental illness.

A. Aetiological theories on role of Social Factors in Mental Illness

1. The Bio - Psychosocial Model: The biopsychosocial model is an approach that delivers that biological, psychological and social factors, all play a significant role in human functioning in the context of disease or illness. As per this approach health can best be understood in terms of a combination of biological, psychological, and social factors rather than purely in biological terms. The biopsychosocial approach supports the famous mind–body connection of various disorders, which addresses more philosophical arguments between the biopsychosocial and biomedical models. The model was theorized by psychiatrist George L. Engel in 1970s (Engel, 1977; McLaren, 2002).

2. The social Disorganization and Urbanization Hypothesis: Robert Farris and Warren Dunhan (1939), hypothesized that under extreme social disorganization high rates of severe mental illness like schizophrenia could become rampant among the people.

3. Social Selection and Causation Hypothesis: “the social selection” perspective suggests that mental disorders are over represented in the lower socioeconomic strata as a consequence of impaired social mobility. Selection processes would likely to operate both within and across generations. Within a generation mental disorders may cause downward mobility among adults and cause the downward “drift” of these people into the lower socioeconomic strata (Jarvis 1971).

4. The Cultural Disintegration Hypothesis: This theory promulgates that social changes occurred by the events like disaster, extensive and wide spread poverty, extensive migration or widespread ill health, existing culture of the society leads to changes in the social formation which results in the broken homes, high rates of crime and delinquency, fragmentation of communication networks and wide spread delinquencies.

5. The Breeder and Attraction Hypothesis: Some pockets of a large geographical location would provoke the severe mental illness to the people because in those 'pockets' social isolation becomes a way of life and noticeable thing is 'lack of social responsibility' among the dwellers (The Attraction Hypothesis) (Gregory, 1960).

6. The Social Class Hypothesis: Developed by Hollinshead and Redlich in 1958. They found prevalence of mental disorder increased as social class decreased. They also found that certain types of psychopathology were associated with different social classes

7. Vulnerability-Diathesis-Stress-Model: This approach posits that a person is more likely to suffer an illness if he or she has a particular diathesis (i.e., vulnerability or susceptibility) and is under a high level of stress. Diathesis factors can be family history of substance abuse or mental illness; individual psychological characteristics such as hostility or impulsivity; biological characteristics (e.g., cardiovascular reactivity, hypothalamic-pituitary-
adrenal responsibility); and environmental characteristics such as childhood maltreatment or low socioeconomic status. The term stress refers to events and experiences that may cause psychological distress. Stress can influence mechanisms that help to maintain the stability of an individual's cognition, physiology, and emotion (Hankin & Abela, 2005).

2. Bio-Psychosocial-Spiritual Model: This model primarily explains the schema or the complex web of substance addiction. This model explains the complex pathway of 'alcoholism' and 'drug addiction' and how individual factors and other factors are tied and work together in substance addiction.

Firstly, Biological or genetic factors related to heritability (Clonginger et al., 1981) which are the basic premises of the disease model.

Secondly, psychological factors which incorporate individual factors and characteristics that can be assessed, many of which have been described as “risk” and “protective” factors (Hawkins et al., 1992).

Thirdly, social factors which include environmental, cultural, familial, and peer factors that have been related to social learning (Bandura, 1977).

Fourthly, spirituality and religiosity have some implications in substance addiction pathway. Positive spirituality and religiosity is a protective factor against substance addiction.

3. Poverty, Economic Crisis and Mental Disorders: Poverty indeed is a multidimensional phenomenon, which includes elements like inability to satisfy basic needs, lack of control over resources, lack of education and poor health. Poverty is associated with many long-term problems, such as poor health and increased mortality, school failure, crime and substance misuse (Murali & Oyebode, 2004).

4. Migration and Mental Disorders: Migration is the process of social change whereby an individual moves from one cultural setting to another for the purposes of settling down either permanently or for a prolonged period. Migration to another country and culture is a stressful thing and several studies in the 1980s and 1990s showed that rates of schizophrenia were higher among migrant groups (Bhugra, 2000; Cochrane & Bal, 1987; King et al., 1994).

B. Theories on Societal Attitude, Reaction on Mental Illness:

1. Stereotyping and stigma

Stereotyping and stigma refers to the tendency of human beings to attribute fixed and common characteristics to whole social groups. The shift from stereotyping to stigmatization involves an enlargement of prejudicial social typing (an error of reasoning). Globally two most common prejudicial attitudes about mental illness are: a) authoritarianism (the belief that persons with mental illness as a class are inferior to normal persons and therefore require coercive handling) and b) benevolence (kindness to unfortunates, leading to behaviour akin to how parents treat children). Both these two attitudes are negative in nature (Corrigan et al., 2001).

C. Labeling Theory:

Labeling theory emphasizes the people's reaction and putting a particular tag on the sick person of being 'sick', 'incapacitated' so on. The labeling or societal reaction approach observed that once an individual is identified as mentally ill a number of forces work to reinforce and solidify that person's mentally ill image. The labeling or societal reaction approach observed that once an individual is identified as mentally ill a number of forces work to reinforce and solidify that person's mentally ill image (Rosenhan, 1973).

D. The Modified Labeling Theory:

Developed by Link et al (1987, 1989) to show that stigma is not only an internal process (perceptions of stigmatizing behaviors exhibited by others) but a
process that inherently involves the negative responses of persons in the environment, defined as the 'labeling' behavior of others.

**Relationship between Various Social Agencies & Mental Health and Illness**

**Culture and Mental Illness:**

Mental illness is the result of a complicated chain of events that implicate flawed biological, psychological, social, and cultural processes. Culture influences mental illness in many ways. The content of people's delusions, auditory hallucinations, obsessional thoughts, and phobias often reflects what is significant in their culture.

The incidence of mental disorders, particularly of behavioral disturbances, such as attempted suicide and alcoholism is closely associated with social situation. Severe mental disorders, such as dementia and schizophrenia, are far less influenced by socio-cultural factors than are minor mental disturbances.

**Religion and Mental Health:**

The positive impact of spirituality on adherence to treatment is explained by an improved quality of life, a better social support, and more positive representations of the illness by believers. The close relationship between religion and mental abnormality explained in various psychiatric literatures, which, have focused on 'religiosity' of mentally ill people or tried to distinguish between healthy and pathological religious commitment (Tseng 2003).

**Gender and Mental Illness:**

Feminist criticized the general outlook of the society to female patients in the lines of labeling theory and constructivist frameworks. These feminists are critical to the societal view of portraying women as vulnerable to being labeled mentally disordered when they fail to conform to stereotypical gender roles as mothers, housewives, and so on or if they are too submissive, too aggressive or hostile to men.

**Race /ethnicity and Mental Illness:**

Various minority or smaller ethnic groups within a large multicultural umbrella conceptualize, treat and cope with mental disorders much differently than the 'people of dominant' culture. Very often ethnic minority people have to face problems like 'discrimination', 'fear of being swallowed by dominant culture' (Kleinman, 1988), 'economic and political disparities', etc. Those factors would make them susceptible for developing mental disorders. Those factors would make them susceptible for developing mental disorders (Karlsen & Nazroo, 2002).

**Social Capital and Mental Illness:**

The most famous definition of social capital was given by political scientist Robert Putnam (Putnam, 1993). This definition includes five parameters, thus, social capital attempts to describe some very innate and unique features of populations such as levels of civic participation, social networks and trust. The most famous definition of social capital was given by political scientist Robert Putnam (Putnam, 1993). This definition includes five parameters:

- Community networks, voluntary, state, personal networks and density
- Civic engagement, participation and use of civic networks
- Local civic identity, sense of belonging, solidarity and equality with local community members
- Reciprocity and norms of cooperation, a sense of obligation to help others and confidence in return of assistance
- Trust in the community

**Three Dimensions of Social Capital:**

1. **Structural/Cognitive**

   Structural social capital describes the relationships, networks, associations and institutions that link people and groups together (McKenzie, 2008).
2. Bonding/Bridging

It can be thought of as the type of social capital that a family unit has or that which is found in small, close-knit migrant groups that rely on mutual support. Bridging social capital is outward-focused and links various minority or smaller ethnic groups within a large multicultural umbrella. Conceptualizing, treating, and coping with mental disorders much differently than the 'people of dominant' culture. Very often ethnic minority people have to face problems like 'discrimination', 'fear of being swallowed by dominant culture' (Kleinman, 1988), 'economic and political disparities', etc. Those factors would make them susceptible for developing mental disorders. Those factors would make them susceptible for developing mental disorders (Karlsen & Nazroo, 2002).

References:


MARITAL ADJUSTMENT, LIFE SATISFACTION AND QUALITY OF MARITAL LIFE-A COMPARATIVE STUDY BETWEEN WORKING AND NON-WORKING MARRIED WOMEN

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Abstract:

Objective:
The motive of the study is to compare the marital adjustment, life satisfaction and quality of marital life between working and non-working married women.

Methodology:
A cross sectional study with sample of 100 married women [50 working and 50 non-working women] selected from various areas of Khammam town. The marital adjustment, life satisfaction and quality of marital life were assessed using Marital-satisfaction scale developed by Brunda, Amrithraj and Indira Jai Prakash, Marital Quality Scale developed by Shah, Anisha and Dyadic adjustment scale developed by Spanier. Statistical analysis was done by Independent t test. Results: There is a significant difference in marital satisfaction and quality of marital life among working and non-working women. Conclusion: The findings suggest that non-working women has more satisfied marital life than working women. In addition, working women has better quality of marital life than working women.

Keywords:
Quality of marital life, marital adjustment, Life satisfaction.

Introduction:
Marital adjustment is the state in which there is an overall feeling in husband and wife of happiness and satisfaction with their marriage and with each other. It is a social phenomenon. Marital satisfaction is a mental state that reflects the perceived benefits and costs of marriage to a particular person. Marital Quality is the subjective evaluation of a married couple's relationship on a number of dimensions and evaluations. Status of women in the society has been changing fast due to multiple factors such as increased level of education, urbanization, awareness of right,
Industrialization. The occupation is one of the most important factors which brings many changes in the life of women; it brings along with it many expectations, pressures, time demands and commitments that may affect the mental health of women. Working women at present are more prone to marital adjustment problems because they have to work in two environments, one is the work environment and the other is home environment. Her employment not only affects her personality but also her family relationships and is also liable to face crisis of adjustment. The success of marital life much depends upon the success in marital adjustment by the husband and wife. Marital maladjustment results in conflicts, tensions, and many a time divorce. Marital adjustment and psychological well-being is related to each other. Psychological well-being is particularly viewed as a positive functioning of individuals and is described as the quality of life of a person. Hashmi et al demonstrated that Marital adjustment has been related to personality, job & home stresses, mental illness, depression, education, sex role attitude, happiness and success in life. Sheema Aleem et al demonstrated that there is no significant difference in marital satisfaction among the dual career women and single career women. Marital maladjustment results in conflicts and tensions and many a time divorce. Jamabr et al demonstrated no significant marital difference in working and non-working women. Tiwari et al and Gupta et al demonstrated that non-working women were better at marital adjustment than working women. Hashani et al demonstrated that working married women have to face more problems in their married life as compare to non-working married women. Ramesh et al demonstrated that non-working women are more adjusted than working women emotionally, socially and health. Nathawat et al, Bhardwaj et al and Patoliya et al demonstrated that working women reported significantly better marital adjustment. Current study compares the marital adjustment, life satisfaction and quality of marital life between working and non-working married women in local population of Khammam town.

### Materials And Methods:

#### Research Design:
Descriptive research design is selected to assign the knowledge of working and non-working women.

#### Sampling Method:
Random sampling technique.

#### Procedure:
The study is cross-sectional. The sample consists of 100 married women (50 working and 50 non-working women) selected from various areas of Khammam town, conducted from 1st June 2014-1st May 2015 under supervision of Dept of Psychiatry, Mamata Medical College, Khammam.

#### Inclusion Criteria:
- Working and non-working married women who have given verbal consent.
- Working and non-working married women between ages 18-50 years who are living with their husbands.

#### Exclusion Criteria:
- Working and non-working married women who have not given verbal consent.
- Divorced, separated, or widowed women.
- Women suffering from medical or psychiatric illness.

The research ethics committee approved the study. Subjects were briefed in detail about the nature and purpose of the study. Confidentiality was assured and informed consent was taken. Following questionnaires were administered to the subjects as described below.

#### Tools Used:

1. **Marital-satisfaction scale** developed by Brunda Amrithraj and Indira Jai Prakash 19859. The scale consists of 38 items in 3 point Likert type responses and the scores extend from 0-2. Both negative and positive questions are there in the scale. Maximum score possible in this scale was 60, minimum score was 0.
2) **Marital Quality Scale developed** by Shah, Anisha in 1995\textsuperscript{10}. The Marital Quality scale (MQS) is a multidimensional scale developed by Shah, Anisha (1995) which has 50 items. The scale had 28 positively worded items and 22 negatively worded items. Total marital quality score ranges between 50-200. Higher score indicates poor quality of marital life. Overall range of marital quality further divided into three classes viz. Good (Total score ranges between 50-100), average (Total score ranges between 101-150) and poor (Total score ranges between 151-200).

3) **Dyadic adjustment scale** developed by Spanier in 1976\textsuperscript{11}. A 32-item measure of relationship quality. Score less than 97 are considered to be “Distressed.”

There were no conflicts of interest for the present study.

**Statistical Analysis :**

Comparison of the marital adjustment, life satisfaction and quality of marital life between working and non working married women assessed by student's t test using SPSS version 20.

**Results :**

**Table 1: COMPARISON OF SATISFACTION BETWEEN WORKING AND NON WORKING WOMEN**

Non working women has more marital satisfaction compared to working women.

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>MEAN ±SD</th>
<th>p value</th>
<th>t value</th>
</tr>
</thead>
<tbody>
<tr>
<td>WORKING (N=50)</td>
<td>39.58 ±10.74</td>
<td>0.002 (s)</td>
<td>3.149</td>
</tr>
<tr>
<td>NON WORKING (N=50)</td>
<td>45.72 ±8.63</td>
<td>p&lt; 0.05</td>
<td></td>
</tr>
</tbody>
</table>

**Table 2: COMPARISON OF QUALITY OF MARITAL LIFE BETWEEN WORKING AND NON WORKING WOMEN**

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>MEAN ±SD</th>
<th>p value</th>
<th>t value</th>
</tr>
</thead>
<tbody>
<tr>
<td>WORKING (N=50)</td>
<td>103.74 ±26.78</td>
<td>0.065 (ns)</td>
<td>-1.863</td>
</tr>
<tr>
<td>NON WORKING (N=50)</td>
<td>112.38± 18.91</td>
<td>p&lt; 0.05</td>
<td></td>
</tr>
</tbody>
</table>

Non working women has better quality of marital life than working women.

**Discussion :**

There is a significant difference in marital satisfaction and quality of marital life among working and non-working women. SheemaAleem et al\textsuperscript{5} reported no significant difference in marital satisfaction among the working and non-working married women. Adegoke et al\textsuperscript{12} and Rogers et al\textsuperscript{12} reported that working women are more satisfied with their lives than non-working women. The result revealed that working women face more marital problems in comparison to non working women, because of the added responsibilities of job. Working women may suffer from more severe maladjustment at home or in the office, while on the other side they have to take care of the children, husband and in-laws and perform other domestic duties\textsuperscript{4}. No matter how responsible a woman is, her marital life suffers a lot because of social inclinations. Working women cannot pay full attention to their family and are unable to satisfy their members. They face stress because of multiplicity of work performed by them, which in turn affects their well-being too. The non-working married women have more time to their household task and their married life goes smooth. Jamabo et al\textsuperscript{6} reported that there is no significant difference in marital adjustment among working and non-working women of Port Harcourt metropolis in Nigeria. Gupta et al\textsuperscript{4}, Tiwari et al\textsuperscript{4} and Ramesh et al\textsuperscript{48} demonstrated that non working women...
The findings suggest that non-working women has more satisfied marital life than working women. The non-working women has better quality of marital life than working women. There is no significant difference in marital adjustment among working and non-working women, but in comparison to working women, non-working women are more adjusted.

**Conclusion:**
The findings suggest that non-working women has more satisfied marital life than working women. The non-working women has better quality of marital life than working women. There is no significant difference in marital adjustment among working and non-working women, but in comparison to working women, non-working women are more adjusted.

**References:**


SUICIDALITY IN LITHIUM-TREATED PATIENTS WITH BIPOLAR DISORDER

Abstract:
The study was conducted with the aim to find out whether lithium protects all patients from suicidal behavior in bipolar disorder.

Method:
Cases were collected from psychiatric outdoor and indoor on basis of screening for bipolar disorder according to ICD 10 DCR criteria. The patients were further screened for a suicidal attempt in recent episode of bipolar disorder in phases of mania and depression. A total of 100 cases were included. Their data were collected by self-made semi-structured socio-demographic and clinical proforma. Data were analyzed by SSPS 70 version for windows.

Result:
44 (44%) of patient population were on valproate, 32(32%) were on lithium, 14(14%) were on both, 10 (10%) were on other drugs like olanzapine, quetiapine. It implies lithium use does not prevent suicidal behavior in all patients. Conclusion: Even lithium use do not prevent suicidal behavior in all patients.

Introduction:
Suicidal ideation refers to thoughts of harming or killing oneself. Attempted suicide is a non-fatal, self-inflicted destructive act with explicit or inferred intent to die (Simpson 1975). Suicide is a fatal self-inflicted destructive act with explicit or inferred intent to die. Suicidality refers to all suicide-related behaviors and thoughts including completing or attempting suicide, suicidal ideation or communications. Non-suicidal self-harm sometimes referred to as ‘parasuicide,’ is viewed as distinct from suicidal behavior and most commonly involves self-cutting without suicidal intent. Cipriani & colleagues (2005) in their metaanalysis of RCT assessing the efficacy of lithium to reduce suicidality attempts in bipolar disorder showed that patient receiving lithium has a lower rate of suicidality. Fredrick et al (2003) in a retrospective cohort study found that lithium reduces suicidal risk in patients than does Divalproate in bipolar disorder. Gibbons et al (2003) in a pharmaco-epidemiological cohort study found no significant difference in suicidal attempts in patients treated with the antiepileptic drug (13/1000 PY) and patients treated with lithium (13/1000 PY) in bipolar disorder. In our study, we aimed to find out whether lithium protects all patients from suicidal behavior.
Material:
ICD 10 DCR.
Semi-structured proforma for sociodemographic and clinical variables.
Suicide questionnaire a self-designed semi-structured Questionnaire.
Written consent form in patients' language.

Result:

<table>
<thead>
<tr>
<th>TABLE 1: Age Distribution in the Sample</th>
<th>No. of Patient</th>
<th>Age Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>52</td>
<td>18-28</td>
</tr>
<tr>
<td></td>
<td>17 (17%)</td>
<td>40-50</td>
</tr>
<tr>
<td></td>
<td>22 (22%)</td>
<td>51-60</td>
</tr>
<tr>
<td></td>
<td>9 (9%)</td>
<td></td>
</tr>
</tbody>
</table>

Remarks
It implies the majority, more than half of patients who attempted suicide were in young age group.

<table>
<thead>
<tr>
<th>TABLE 2: Age distribution in mania and depression</th>
<th>Total No. of Patient while attempting suicide</th>
<th>Age Group</th>
<th>No. and Percentage of Patient while attempting suicide</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>62 patients</td>
<td>18-28 years old</td>
<td>29 (46.8%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>29-39 years old</td>
<td>18 (29%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>40-50 years old</td>
<td>9 (14.5%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>51-60 years old</td>
<td>6 (9.7%)</td>
</tr>
<tr>
<td>Mania</td>
<td>38 patients</td>
<td>18-28 years old</td>
<td>23 (60.5%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>29-39 years old</td>
<td>4 (10.5%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>40-50 years old</td>
<td>8 (21.1%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>51-60 years old</td>
<td>3 (7.9%)</td>
</tr>
</tbody>
</table>

Remarks
It implies the majority of patients in depressive (more than one thirds) and manic (more than half) phase who attempted suicide were in young age group.
TABLE 3: Gender distribution in the sample

<table>
<thead>
<tr>
<th>Gender</th>
<th>No. and Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>76 (76 %)</td>
</tr>
<tr>
<td>Male</td>
<td>24 (24%)</td>
</tr>
</tbody>
</table>

Remarks: It implies a majority, more than three-fourths of patients who attempted suicide were female.

TABLE 4: Gender distribution in mania and depression

<table>
<thead>
<tr>
<th>Depression</th>
<th>Total No. of Patient while attempting suicide</th>
<th>Gender</th>
<th>No. and Percentage of Patient while attempting suicide</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Female</td>
<td>50 (80.6%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td>12 (19.4 %)</td>
</tr>
<tr>
<td>Mania</td>
<td>38 patients</td>
<td>Female</td>
<td>26 (68.4 %)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td>12 (31.6 %)</td>
</tr>
</tbody>
</table>

Remarks: It implies the majority of suicide attempters in the phase of mania (More than two thirds) and depression (more than three fourth) were female.

TABLE 5: DRUG-ON

<table>
<thead>
<tr>
<th>Medication</th>
<th>No. and Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valproate</td>
<td>44 (44%)</td>
</tr>
<tr>
<td>Lithium</td>
<td>32 (32%)</td>
</tr>
<tr>
<td>Both</td>
<td>14 (14%)</td>
</tr>
</tbody>
</table>

Remarks: It implies less than half of patients attempting suicide was on valproate.

TABLE 6: Drug on vs. Phase Illness

<table>
<thead>
<tr>
<th>Depression</th>
<th>Total No. of Patient while attempting suicide</th>
<th>Medication</th>
<th>No. and Percentage of Patient while attempting suicide</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>62 patients</td>
<td>Valproate</td>
<td>34 (54.8%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lithium</td>
<td>12 (19.4%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Were on both</td>
<td>8 (12.9%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other drugs</td>
<td>8 (12.9%)</td>
</tr>
<tr>
<td>Mania</td>
<td>38 patients</td>
<td>Valproate</td>
<td>10 (26.3%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lithium</td>
<td>20 (52.6%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Were on both</td>
<td>6 (15.8%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other drugs</td>
<td>2 (5.3 %)</td>
</tr>
</tbody>
</table>

Remarks: It implies the majority of patients attempting suicide in the phase of depression more than half was on valproate, in the phase of mania more than half was on lithium.
Discussion:

Goldstien et al (2012) examined 413 youths in a longitudinal study in Los Angeles found in patients with Bipolar Disorder 18% had suicide attempts 41% had multiple attempts. Natacha et al (2013) in a longitudinal study studied more suicide attempt in adolescent and youth than children in bipolar disorder. In a longitudinal study of two years follow up by Galfalvy et al it was found that the only demographic factor that is important is a younger age for an attempt of suicide in Bipolar Disorder (Galfalvy et al, 2006). In a cohort study on bipolar disorder, it was found that adults with an early age of onset (65%) are at increase suicidal risk (Lewhison et al, 2003,). Fernandez et al (2013) in a cross-sectional study found more suicide in early age of onset in bipolar disorder. In our study we found majority 52 (52%) were in age group 18-28 years. It implies young age is a risk factor for suicide attempt in bipolar disorder. Nordentoft et al (2011) in a longitudinal study in Denmark Finland and Sweden reported gender difference in suicidal attempt males (8%) and females (5%) in long-term follow up. Chen et al (1996) in a cross-sectional study found males (19%) and females 34% made a suicidal attempt in bipolar disorder. A high rate of attempted suicides in females and completed suicide in men with bipolar disorder was found (Mann et al, 2002). In our study, more female 76(76%) attempted suicide. Both in the depressive phase and mania more female attempted suicide [50(80.6%) and 26(68.4%) respectively], which supports the previous finding. Cipriani & colleagues (2005) conducted met analysis of RCT assessing the efficacy of lithium to reduce suicidality attempts in bipolar disorder shows patient receiving lithium has a lower rate of suicidality. Fredrick et al (2003) in a retrospective cohort study in San Fransisco found lithium reduces suicidal risk in patients than does Divalproate in bipolar disorder. Gibbons et al (2003) in a pharmaco-epidemio logical cohort study in Massachusetts found no significant difference in suicidal attempts in patients treated with the antiepileptic drug (13/1000 PY)- V/S treated with lithium (13/1000 PY) in bipolar disorder. In our study we found majority 20 (52.6%) of patient in phase of mania who attempted suicide were on lithium, this is in accordance with finding by Gibbons et al (2003) who found no significant difference in suicidal attempts in patients treated with anti epileptic drug (13/1000 PY)- V/S treated with lithium (13/1000 PY) in bipolar disorder. This can be due to poor compliance to lithium, or use of multiple drugs. According to Gazella et al (2006) in a cross-sectional study in Brazil, it was found in bipolar disorder 48.5% of patient presented with suicidal attempts and 84% of them were using more than one medication. In addition, recent starting of lithium may be a cause for a suicide attempt, for this, we need a further study where the duration of lithium institution and intent of suicidality can be assessed.

Limitations

Patients in manic phase who attempted suicide was on new or old instituted lithium therapy, as it is well known according to meta-analysis of RCT by Cipriani And Colleagues, 2005 lithium has an anti-suicidal effect we needed a longitudinal design to see how many days or weeks lithium takes to initialize anti-suicidal effect. The cross-sectional design of the study is another limitation of the study, for which we could not calculate the incidence of suicide attempt in special age group, gender.

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A STUDY ON BURNOUT AMONG DOCTORS AND NURSES IN A TERTIARY CARE HOSPITAL IN EASTERN INDIA

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Abstract:
The present study attempted to understand the level of burnout, social support, general health, of two groups (doctors and nurses) of healthcare professionals working for a Tertiary Care Teaching Hospital in Eastern India. 30 doctors and 30 nurses, working in PBM Hospital, KIMS Bhubaneswar were administered with Mashlach Burnout Inventory (MBI), Berlin Social Support Scale (BSSS), General Health Questionnaire (GHQ), besides a demographic profile containing their personal information, no of working hours, income. The result revealed a low level of emotional exhaustion and moderate level of depersonalization in the health professionals of KIMS. The doctors had less burnout in personal accomplishment dimension and in other dimensions of burnout, which are emotional exhaustion and depersonalization. The perceived emotional support and perceived instrumental support was found to be higher in doctors compared to nurses. Nurses reported higher levels of general health problems. The implications were discussed.

Key Words -- burnout, doctors, nurses, social support

Burnout is increasingly being recognized as a serious problem affecting many people, particularly professionals employed in human services.

Maslach and Pines (1,2,3) defined burnout and summarized a number of symptoms. In addition to physical exhaustion (and sometimes even illness), burnout is characterized by an emotional exhaustion in which the professional no longer has any positive feelings, sympathy, or respect for clients or patients and there is a deterioration in the quality of care or service that they provide.

Professionals those have frequent contact with individuals are more sensitive to develop burnout. Among the different health professions, nursing has been considered a profession highly susceptible to stress. Burnout is associated with a lower effectiveness at work, a decreased job satisfaction and a reduced commitment to the job or the organization.

Kogoj, Travnik and Zaletel Faculty(4) in their study confirmed a strong relationship between stress and burnout in the studied group. Koonce et al(5) conducted a study on mental health professionals and found some predictors of burnout were caseload size, type of cases being treated, gratitude and job satisfaction, age, gender, experience, work environment and perceptions of equity. Moreover, age was negatively linked to burnout. Similar study was conducted by Geurts, Wilmar, Schaufeli and Jonge 1998(6). They found a relatively low level of burnout in the study sample.
Study by Embriaco, Papazian, Barnes, Pochard and Azoulay: 2007(7) found high levels of burnout in critical care physicians and nurses. Orzechowska, Talarowska, Drozda, Mirowska, Florkowski, and Gałecki (2008) (8) in their study on level of the burnout syndrome intensity among nurses and doctors found out that among doctors and nurses all three factors of burnout syndrome were presented: emotional exhaustion (WE), low level of personal service that they provide.

Professionals those have frequent contact with individuals are more sensitive to develop burnout. Among the different health professions, nursing has been considered a profession highly susceptible to stress. Burnout is associated with a lower effectiveness at work, a decreased job satisfaction and a reduced commitment to the job or the organization.

Kogoj, Travnik and Zaletel Faculty (4) in their study confirmed a strong relationship between stress and burnout in the studied group. Koonce et al (5) conducted a study on mental health professionals and found some predictors of burnout were caseload size, type of cases being treated, gratitude and job satisfaction, age, gender, experience, work environment and perceptions of equity. Moreover, age was negatively linked to burnout. Similar study was conducted by Geurts, Wilmar, Schaufeli and Jonge 1998 (6). They found a relatively low level of burnout in the study sample.

Study by Embriaco, Papazian, Barnes, Pochard and Azoulay: 2007(7) found high levels of burnout in critical care physicians and nurses. Orzechowska, Talarowska, Drozda, Mirowska, Florkowski, and Gałecki (2008) (8) in their study on level of the burnout syndrome intensity among nurses and doctors found out that among doctors and nurses all three factors of burnout syndrome were presented: emotional exhaustion (WE), low level of personal satisfaction from work (ZA) and depersonalization (DP). Nurses in comparison to doctors have a higher level of the emotional exhaustion, a lower level of an involvement in work and a lower level of an internal locus of control.

Tipandjan and Sundaram (2012)(9) ; Their study explores both the social support and personality factors on the burnout of 79 hospital nurses. The
results revealed that specific dimensions of personality do significantly and differentially correlate with the experience of the three components of burnout. Findings also indicate that nurses differ significantly on burnout due to their perceived social support. Kar, Roy, Das (10), did a cross cultural study was to explore the main determinants of burnout among nurses working in tertiary care hospitals. Depersonalization, personal accomplishment, professional recognition and professional uncertainty emerged as the main statistically significant job stressors. Burnout remains a significant issue and each organization should have their own set of data to estimate their burnout problem in order formulate their own local strategy to deal with it.

The present study was aimed to assess this problem locally

Aims and Objectives
1. To assess and compare the level of burnout, social support, health status of the doctors and the nurses. 2. To understand the relationship between burnout, perceived social support and general health.

Method of Study
Sample:
This study was conducted in Pradyumna Bal Memorial Hospital, Kalinga Institute of Medical Sciences (KIMS) which is a 1750 bedded tertiary care teaching general hospital located in Bhubaneswar, Odisha. The participants of the study were two groups of healthcare professionals namely the doctors and nurses in the age group of 23-50 years working in OPD and ward setup and purposive sampling was used. A semi structured interview schedule was developed and administered individually. The data was collected over a period of two months. The participants were personally contacted after purposive selections of Doctors and Nurses working in various Departments. The total no. of collected cases are 60, which includes 30 doctors and 30 nurses.

Instruments used for the study Socio-demographic Performa
Socio-demographic information such as age, gender, income, was obtained using a Performa developed for this purpose.

Maslach Burnout Inventory( MBI )
The Maslach Burnout Inventory (MBI) (11) is the most commonly used tool to self-assess whether one might be at risk of burnout. The MBI explores three components: exhaustion, depersonalization and personal achievement. The scoring criteria is explained below.

Emotional Exhaustion:
High scores on which reflect that individuals are experiencing strain approaching beyond their normal coping capabilities; they are "at their rope's end/ emotionally speaking, or are approaching that end. There are 7 items in this section of MBI. Total 17 or less refers to low-level burnout, total between 18 and 29 inclusive refers to moderate burnout, total over 30 refers to high-level burnout. The range of score is 0 to 42.

Depersonalization:
High scores implies that a person distances self from others, considers them as things or objects. There are 7 items in this section of MBI. Total 5 or less refers to low-level burnout, total between 6 and 11 inclusive refers to moderate burnout, total of 12 and greater refers to high-level burnout. The range of score is 0 to 42.

Personal Accomplishment (REVERSED):
High scores indicates that a person is working efficiently on worthwhile organizational jobs. There
are 8 items in this section of MBI. Total greater than 40 refers to low-level burnout, total between 34 and 39 inclusive refers to moderate burnout, total 33 or less refers to high-level burnout. The range of score is 0 to 48.

**Berlin social support scale**
The Berlin Social Support Scales (BSSS, Schwarzer & Schulz, 2000)(12) was developed for measurement of social support.

It has four dimensions which are- perceived emotional support, perceived instrumental support, need for support and support seeking. In perceived emotional support section there are 4 items, the range of score is 0 to 16. There are 4 items in the perceived social support and the range of score is 0 to 16. In the need for support section there are 4 items and the range of score is 0 to 16. There are 5 items in support seeking the range of score is 0 to 20. Besides, the need for support section, increased scores in all the sections refers to increased support. Need for support is reversed scored which means if the person is scoring more then he does not need any support.

**General Health Questionnaire (GHQ)**
GHQ (Goldberg 1979)(13) accessed the health status of the professionals. It includes 28 questions which indicates how the physical and mental health is. It measure somatic symptoms, anxiety/insomnia, social dysfunctioning, and severe depression. Higher the score lower the health status. The range is from 0 to 28.

**Procedure**
The Participants for this study were doctors and nurses of KIMS whose medium of training was English. The participants were approached after a purposive selection of Doctors and Nurses working in various Departments such as General Medicine, Pulmonary Medicine, Dermatology, Medicine, Psychiatry, General Surgery, Orthopaedics, Radio diagnosis, ENT, Anaesthesia The participants were explained on the voluntary participation, confidentiality and anonymity during the data intake and processing. Consent for the study was taken prior to their participation. Total 130 persons were contacted. Among them not all were interested to participate in the study. Some of them accepted the form but did not filled up and there was not positive response about the return of those forms. Only 35 forms from the doctors were collected successfully and 42 forms from the nurses were collected successfully. But 5 forms from the doctors were partially filled and also 13 forms from nurses were partially filled. So, 30 forms were successfully filled up from doctors and from nurses.

The participating individuals were assessed using MBI,BSSS and GHQ. Their socio-demographic characteristic was recorded separately. The cross-sectional Study began from 04/02/2016 and the data intake finished in one month or 4 weeks.

**Results**
The data obtained was directly observed as well as computed using SPSS package 18. The analysis was conducted using t test, Pearson correlation test, and Chi square test. The mean age of the doctors was 42 years while the nurses was 35 years. 80% of the doctors were males while all the nurses were females. Declared Income of the doctors ranged from rs 35000 to 1.5 lakhs while that of nurses was rs 8000 to 25000 which was categorized according to the subjective satisfaction. Average working hours of doctors was 8 hours which was similar to that of nurses.
### TABLE 1

Mean of the variables, standard deviation of the two groups and 't' value

<table>
<thead>
<tr>
<th></th>
<th>Doctors</th>
<th>Nurses</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Emotional (MBIA)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exhuastion</td>
<td>13.33</td>
<td>9.869</td>
</tr>
<tr>
<td>Depersonalisation (MBI B)</td>
<td>7.83</td>
<td>6.983</td>
</tr>
<tr>
<td>Personal (MBIC)</td>
<td>37.93</td>
<td>8.288</td>
</tr>
<tr>
<td>accomplishment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perceived (BSSS1)</td>
<td>14.40</td>
<td>1.831</td>
</tr>
<tr>
<td>emotional support</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perceived (BSSS2)</td>
<td>13.80</td>
<td>2.552</td>
</tr>
<tr>
<td>Instrumental support</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Need for support(BSSS3)</td>
<td>12.03</td>
<td>2.484</td>
</tr>
<tr>
<td>Support seeking(BSSS4)</td>
<td>16.67</td>
<td>3.325</td>
</tr>
<tr>
<td>GHQ</td>
<td>9.20</td>
<td>2.894</td>
</tr>
</tbody>
</table>

**p<.01, *p<.05

Table-1 explains about the mean scores, sd and t-score of doctors and nurses on the measures of MBI A, MBI B, MBI C, BSSS1, BSSS2, BSSS3, BSSS4, GHQ.
The table explains that the difference between the doctors and nurses are significant in these following dimensions: MBIC, BSSS1, BSSS2, BSSS3, BSSS4 and GHQ. There is difference between the doctors and nurses in the MBIC, the t-score is 1.088, df is 58, probability is .037 (p<.05) which is significant. Which refers that in case of personal accomplishment, there is difference in doctors and nurses. Doctors are having less burnout in personal accomplishment compared to nurses. The difference between the two groups in BSSS1 is 2.771 which is highly significant .000(p<.01). So, it states that the perceived emotional support is more in doctors compared to nurses. In the BSSS2 dimension the difference is 2.708, the probability is .044 (p<.05). So, the instrumental support is higher in doctors compared to nurses. In BSSS3 dimension the difference between the two groups is -.219, the probability is .051 (p<.05). So, in need for support doctors need more support compared to nurses. In BSSS4 dimension the difference between the two groups is 1.296, the probability is .046 (p<.05). Here doctors are seeking more support compared to nurses. The GHQ scores reveal that the difference between the doctors and nurses is -1.467 the probability is .056 (p<.05). It suggests that nurses reported more physical and mental symptoms compared to doctors.

**Chart 1. Difference between the Nurses and Doctors with respect to Burnout, Social Support and GHQ Scores**
TABLE 2
Correlation between three subscale of burnout with Perceived social support and General Health amongst Nurses and Doctors

<table>
<thead>
<tr>
<th>MEASURES</th>
<th>Perceived Emotional Support (BSSS1)</th>
<th>Perceived instrumental support (BSSS2)</th>
<th>Need For Support (BSSS3)</th>
<th>Support seeking (BSSS4)</th>
<th>GHQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>MBI A (emotional exhaustion)</td>
<td>.077</td>
<td>-.082</td>
<td>.208</td>
<td>-.123</td>
<td>.210</td>
</tr>
<tr>
<td>MBI B (depersonalisation)</td>
<td>-.042</td>
<td>-.291*</td>
<td>.163</td>
<td>-.346**</td>
<td>.293*</td>
</tr>
<tr>
<td>MBI C (personal accomplishment)</td>
<td>.369**</td>
<td>.322*</td>
<td>-.022</td>
<td>.243</td>
<td>-.324*</td>
</tr>
</tbody>
</table>

*P<.05, **P<.01

The findings as depicted in table 2 suggest that those who are having increased emotional exhaustion they are having increased depersonalization ('r' value of .556, p<.01) Those who are scored high on emotional exhaustion they experienced more general health problems, MBI B had positive significant correlation with GHQ. It showed that those who are having more depersonalization effect they perceiving less instrumental support ('r' value of -.291p<.05).

Professionals who had more depersonalization, they sought less social support. If depersonalization is more, then general health problems are more('r' value of .293p<.05), MBI C had positive significant correlation with BSSS1 and BSSS2. Those who had high personal accomplishment they experienced less general health problems('r' of -.324,p<.05).

In comparison to doctors, significantly more number of nurses reported dissatisfaction with their income. The chi-square statistic is 8.5311. The p-value is .003491. This result is significant at p < .05. however this did not translate to the amount of depersonalization they showed. the chi-square statistic is 1.0227. The p-value is .311873. This result is not significant at p < .05

Discussion and Conclusion

The present empirical investigation is an effort to examine various aspects of burnout in healthcare professionals of PBMH, KIMS. In western countries the socio-cultural set up and work load is different and there were number of studies, which suggest that burnout is very prominent in western countries among healthcare professionals. In India doctors and nurses experience burnout but in mild to moderate degree which was reflected in our study. Another study by Joena et al (14) from Madurai had similar finding.

The socio-demographic profile was similar to other studies in India. Similar to study by Khanna (15) and Langade et al(16), we found higher level of burnout in females.
The present empirical investigation is an effort to examine various aspects of burnout in healthcare professionals of PBMH, KIMS. In western countries the socio-cultural set up and work load is different and there were number of studies, which suggest that burnout is very prominent in western countries among healthcare professionals. In India doctors and nurses experience burnout but in mild to moderate degree which was reflected in our study. Another study by Joena et al (14) from Madurai had similar finding.

The socio-demographic profile was similar to other studies in India. Similar to study by Khanna (15) and Langade et al (16), we found higher level of burnout in females.

The health professionals (doctors and nurses) here are scoring high on personal accomplishment, but lower level burnout in the emotional exhaustion. This is in contrast to finding in another Indian study (16). This can be explained as our sample is from academic setup and the professionals spend a considerable time of working hours in teaching. In our study population we found moderate level of burnout in depersonalization which may translate to detachment from patients in their professional life. In case of personal accomplishment, doctors are having less burnout compared to nurses. That could be due to varying socio-economic status of doctors in comparison with nurses. Compared to nurses, doctors perceive more emotional support.

The professionals who are experiencing higher level of burnout they express more general health problems. In this study, nurses reported more health problems compared to doctors. This finding raises concern about absenteeism and even job change.

The personal accomplishment of the professionals also affects the general health problems and psychological distress. If their personal accomplishment is high the general health problems and psychological distress are low.

In view of the above findings, this study will help the institutional policy makers to facilitate support network services and look at the working pattern of nurses and financial incentives. Limitation of the present study is small sample size. More than 50 percent professionals who were approached did not consent to participate in the study, so the results might not be generalized.

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## COGNITIVE BEHAVIOUR THERAPY FOR THE MANAGEMENT OF SECONDARY ENURESIS

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### Abstract

Enuresis is a highly prevalent (5-10%) elimination disorder underrated and buried under Indian culture because peoples feels embarrassing to disclose or seek help for it and this perpetuate the problem with gradually increasing severity. Behaviour Therapy (BT) and Cognitive Behaviour Therapy (CBT) were found to be significantly effective for the management of secondary enuresis. This paper attempts a single case conceptualization and management of CBT for secondary enuresis.

### Key Word:

Enuresis, Cognitive Behaviour Therapy

Enuresis involves repeated voiding of urine into inappropriate places after the age achieved normal bladder control (5 years), the behaviour does not attributed by the physiological effects of substance or another medical condition and causes significant distress or impairment in personal, social, academic or other significant arrears of life (DSM-5). Enuresis has been described in two types – primary and secondary. In primary enuresis individual has never established urinary continence and in secondary enuresis disturbance develops after a period of established urinary continence (DSM-5). There are several reasons to develop secondary enuresis such as modelling, learned helplessness, emotional distress, sexual abuse, family life transition, stressful life event, co morbid psychiatric problem and deep sleep (Houts, 2003; Walker, 2003; Clayden et al., 2002; Shaffer, 1994; Buchanan, 1992; Sluckin, 1981).
Cognitive behaviour therapy (CBT) is a combination of cognitive therapy and behaviour therapy. CBT focuses on the interconnection between thought, behaviour, and mood. CBT is an evidence-based, practical, and present focused psychotherapeutic technique found to be effective in the management of enuresis by targeting the irrational beliefs that cause and maintain the wetting (Caldwell et al., 2013; Ronen et al., 1992 & 1995). The present case study is a detailed description of the cognitive behaviour therapy case conceptualization and management of enuresis.

**Case Summary:**

Mr. X is a 14 years old Hindu, Hindi speaking Boy, studying in 6th standard, belongs to lower middle socioeconomic status came for consultation by his father with the chief complaints of frequent bedwetting at night, avoids to playing and talking with neighbourhood children, spend maximum time at home, reduced academic performance, irritability, anger outburst, fearful to see others fighting with each other and frequently annoying parents by not listening them from last 6 years. The illness developed insidiously, illness's course was continuous and illness progress was deteriorating.

Index boy was born at hospital after full term normal delivery. No prenatal, perinatal and postnatal complication was found. The physical, cognitive and social developmental milestones were achieved at appropriate age without any delay and complication. He achieved his bowel and bladder control at the age of 3 years. At the age of 7 years his joint family separated following death of his grandfather. This separation leads highly strained family environment due to property issues and he had exposure of verbal and physical fights between his uncles and aunts. That was the first cruel experience of the child having fight between his loving persons. It's unbearable and frightening for him, so he starts weeping and trying to avoid that place, which is not possible because every family member involves there. Following this incidence he had bedwetting that night and this was gradually maintained by exposure of frequent fight between family members. Whenever he had experience of other family members fight or strained family environment, bedwetting was certain that night. Soon the bedwetting behaviour was generalized with the exposure of other peoples fight, strained family environment, academic failures, fight with friends and brother, parental criticism, feeling embarrassment and guilt about bedwetting and social isolation chosen by him. Sometimes the lack of bladder control was appear in day also which embarrassed him more and he was started avoiding to going out of home for play with friends, spends maximum time at home brother. Gradually he was started avoiding going out of home for any work except school and his academic performance is also significantly reduced due less attention in studies. He becomes irritable on minor issues and used to annoy his parents by not listening them. He became homebound and spends maximum time with his younger brother for playing but his irritability and anger outburst leads frequent fight between them, which further increases the strained environment of family and leads more bedwetting. Bedwetting is highly embarrassing in Indian culture therefore parents hides it for longer but when gradually other problems emerging especially poor academic performance, housebound symptom, frequent fight with brother and anger outburst. Then the parents consulted to the medicine specialist and found no significant change in behaviour after treatment, so switch to psychiatric help, where they were referred for psychotherapeutic intervention. There is no history suggestive of structural bladder control problem, neurogenic bladder control problem, urine infection, medication side effect, head injury, epilepsy, brain fever, substance abuse, intellectual disability and other medical conditions. There is no family history of any psychiatric or severe physiological illness in family members. Mental Status Examination reveals normal cognitive functioning, no active psychopathology except sad mood with grade V insight.
Factors affecting the enuresis behaviour of present case

The enuresis problem of present case was precipitated by the exposure of familial verbal and physical fight over property issue followed by his grandfather's death. The precipitating factor activated the patients underlying predisposing mechanisms such as less stress tolerance, poor problem solving and less coping ability.

**Precipitating Factors**
- Familial fight over property issue.

**Predisposing factors**
- Less stress tolerance, poor problem solving ability and less coping ability.

**Perpetuating factors**
- More fluid intake at evening, deep sleep, strained family environment and critical comments of parents.

**Consequences**
- Social withdrawal
- Poor social interaction
- Irritability
- Poor academic performance
- Low self esteem
- Anger outburst
- Housebound
- Inattentive to parents
- Stop playing with other friends
- Feeling of helplessness and

**Protective Factors**
- High level motivation of parents and patient to participate in therapeutic process.
- Patient's ability to cooperate with professional.

**Figure - 1**
These predisposing factors also perpetuate the problem behaviour with additional perpetuating factors in the form of personal, environmental and familial. The two way interaction of perpetuating factors with the enuresis problem can be seen in following diagram (Figure-1) as how perpetuating factors increases the frequency of enuresis behaviour and on other side enuresis behaviour increases the frequency of perpetuating behaviour leads exacerbation of the enuresis problem behaviour. These precipitating, predisposing and perpetuating factors responsible for enuresis behaviour of the present case reflected in his significant impairment of personal, social and academic life. High motivation for treatment and ability to understand and cooperate for treatment were protective factors for this case, suggests good prognosis of the case.

**Cognitive behaviour therapy formulation**

Cognitive behaviour therapy works to changes the cognition and behaviour both responsible for maladaptive behaviour. The enuresis problem of present case was viciously maintained by the negative thought and maladaptive behaviour (figure-2). This could be managed by the cognitive behaviour therapy approach.

![Cognitive Behaviour Therapy Diagram](image)

**Thought**

(i am crep, i want have control on my bladder and its highly embarrassing)

**Physical Reaction**

(feel tired, weak, lack of bladder control)

**Emotion**

(i am worthless, helpless about bladder control and nothing wana help me out)

**Behaviour**

(avoid other peers and social interaction)
Cognitive behaviour therapy intervention of present case

Brief descriptions of therapeutic intervention of present case are:

Session 1 Psycho-Education and activity scheduling:
After collection of detail case history and assessment of the problem behaviour, the both patient and parents were informed about the nature, origin, causing, maintaining and vulnerability factors of enuresis (figure – 1). They were also informed about the prevalence, epidemiology and management of the enuresis. The detail information about the planned therapeutic sessions, their role and cooperation in therapy were given to them. Thereafter patient's current activities revived and scheduled his activities for next one week.

Session 2 Socialization of Cognitive Behaviour therapy formulation and reviewed activity scheduling with adding waking schedule:
The patient has been socialized about the cognitive behaviour therapy formulation (figure -2) with the detail explanation about the vicious cycle between thought, emotion, behaviour and physical reaction. He was explained that how the thought and behaviour was responsible for his mood and physiological changes with the example of an alternative thought developed for dysfunctional thought and its effects on changing emotion, behaviour and physiological reaction. Then his activity schedule was revived with adding waking schedule on it. For waking schedule parents were told that make a list of waking schedule during night time which will help them to controlling the frequency of bedwetting of the patient. The waking list contained to woke the child with the gap two hour at night for four to five times. The parents were guided to help the patient initially and when he learned the behaviour then the patient can do it by him with the help of alarm clock. Regular follow up of parents suggested to avoid the violating the waking schedule by patient. The parents were suggested that this will continue till the time the patient learned to follow the waking schedule by him without any assistance of the parents.

Session 3 Retention control technique:
In this session initially the activity scheduling and waking schedule were revived and significant changes has been done according to the patient and parents demands and suggestions. Thereafter the retention control technique has been introduced to them to increase the bladder holding capacity of the patient. They were explained that this technique additionally would help the patient to gain his self-control and self-esteem back. They can use this technique at any free time of one hour, before two hours of meal or after one hour of meal and before two to three hour of sleep. They were told about this technique that the patient need to drink four litter water at a time under observation of parents and he has to wait till the time he felt urinating and then he need to hold the urination as much as he can and when uncontrollable then go to urination. Parents would note down the time between he felt urinating to the urinate. Parents were suggested when the patient successfully complete the session then rewards him by his favourite thing. This would be repeated till the time his holding capacity reached to more than half hour. Reward the patient whenever the holding capacity time increases but deprive him from rewards whenever his holding capacity time decreases and again starts reward when the time reached to previous holding time.

Session 4 Cleanliness training:
The activity schedule was revived with addition and subtraction of activities as per presenting demand of parents and patient. The waking schedule hours were increases from two to three due to reduced frequency of bedwetting. The doubts and difficulties about retention control techniques had been discussed with the patient and parents. In this session a new technique added with the suggestion to parents that an immediate negative consequence of any behaviour reduces the frequency of behaviour. So the parents were suggested that they should immediately woke the child to change and clean his own clothes and bed sheets followed by every single incidence of bedwetting to create the immediate
aversive consequence for decreasing or stopping the frequency of bedwetting.

**Session 5 Graded Task Assignment:**
Patients poor academic performance causing pressure on him, make him feel inferior in front of other students and also leads critical comment of parents. He wants to do study but unable to perform it due to pathology and over burden of study material. Therefore this technique was used to reduce his over burden of study material by dividing into small portion and small targets. The sense of accomplishment to achieve the small targets positively reinforced him to do his studies.

**Session 6 Problem solving skill:**
In this session significant reduction in bedwetting problem had been informed by the parents and patient. To prevent the relapse and maintain the dry bed period predisposing factors were targeted in this session. The poor problem solving ability is a significant predisposing factor maintaining his problem behaviour. So he was taught to see the multidimensional aspects of a problem with generating multi-solution and picking the one who had less loss and more gain. This procedure was repeated with two real life problems of the patient to help him to find out the suitable solution of the problem and to generalize and consolidate the problem solving skill.

**Session 7 Conflict resolutions:**
The conflicting situations also works as predisposing factor and can cause to relapse. Therefore he was introduced the advantage and disadvantage technique to solve the conflict and take suitable decision with less losses. This technique also practiced with the patient for his two real life conflicts.

**Session – 8 Anger management:**
The distraction and generating alternative thought techniques has been taught to the patient to control his anger. Two real life anger situations analysed by using generating alternative thought to make the patient understand and the way to use technique.

**Session – 9 Ending session:**
The whole therapeutic process was reviewed and found to be significant improvement in his bedwetting problem with only two wet nights in initial therapeutic sessions. The patient and parents were suggested to follow the waking schedule with the gap of four hours and practice the problem solving skill, conflict resolution skill and anger management skills. They were informed that the therapeutic sessions has been over and they can immediately visit after any relapse or follow-up session after one month.

**Session 10 Follow – up session:**
After one month patient was visited with his father and he was maintaining well without any relapse. He was suggested to keep practicing and using the skills he taught to prevent his relapse and also told to visit immediately after any relapse or next follow – up session after three months.

**Session 11 Follow – up session:**
After three months patient visited alone and communicated that he was maintaining well without any incidence of relapse. In this session he was suggested to keep using skills he taught and immediately visit after relapse.

**Conclusion**
The enuresis problem of present case was precipitated by the exposure of significant familial stressful event but exacerbated by the predisposing and perpetuating factors. The initial target of CBT intervention was perpetuating factors followed by predisposing factors to prevent the relapse. The follow up sessions findings suggest that the CBT intervention for enuresis is an effective treatment approach to reduce and stop the frequency of bedwetting with reduce chances of relapse.
References


MUCUNAP RURIENS (KONCH BEEJ) INDUCED MANIA

Abstract

Some herbs in indigenous preparations used indiscreetly as 'over the counter medications' can precipitate many psychiatric symptoms due to their role in the dopamine pathway. We present a case of a 40 year old male developing mania after consumption of one such herbal preparation containing 'Mucuna Pruriens (Konch Beej)' which is known to contain L-DOPA, a precursor of dopamine.

Keywords:
Mucuna Pruriens, mania, L-DOPA

Introduction

Mucuna pruriens, a tropical legume also known by many names like velvet bean, cowage, lacuna bean, konch beej, is a native to Africa and tropical Asia and widely naturalized and cultivated (1)(2). The seed of this plant is most often employed therapeutically (3). The seeds contain about 3.1–6.1% L-DOPA, with trace amounts of serotonin, nicotine, and bufotenine (4). Mucuna pruriens has been used in ayurvedic medicine for over two thousand years because of its medicinal properties. It’s different preparations (from the seeds) are used frequently for management of rheumatoid arthritis, diabetes, atherosclerosis, nervous disorders and male infertility (5). The ancient Indian system used Mucuna pruriens to treat disorders such as Parkinson's disease (3)(6). Widely used in ayurvedic preparations, Mucuna Pruriens, is indiscriminately utilised in many over the counter herbal products and is injudiciously considered safe by the general population. Unrestricted unsupervised use of herbal preparations containing Mucuna Pruriens can precipitate various psychiatric symptoms. In this case report, we present a case of a mania following the consumption of indigenous preparation containing Mucuna Pruriens.

Case report

We report a case of a 40 year old male who presented with H/O, anger outbursts, decreased sleep, excessive thinking, over activity, and elevated self-esteem which appeared 3 months back according to family members. Patient also developed increased libido. On mental status examination patient appeared overconfident; affect was elated and grandiose ideas were present at times. Further, evaluation did not reveal any significant past or family history or substance abuse history. However, it was reported that patient had started taking “DIVYA Youvnamrit Vati” an over the counter herbal (indegenious) product for decreased libido and contains Ashwagandha (12mg), Konch Beej (12mg), shtavar (22mg), safedmusli (12mg), javitri (12mg), jaifal (17.50mg), shush kuchla (2mg), afrakash (12mg), jundvedastar (12mg), svarnbhakam (0.25mg), pradalpishti (0.25mg), vangbharm (5.50mg), shilajitshudh (5.50mg), chaanrss (0.5mg), and also...
“Shilajit capsules” containing shilajit satrsookh(390mg), aamlarasayan(50mg). The herbs present in the drug are used in indigenous practice to treat sexual disorders. The patient had been consuming these medications for last 3 and half months to treat erectile problems. Based on psychiatric evaluation, diagnosis of Mania (F 30.1) was made as per ICD-10. Patient was advised to stop the herbal product. Treatment with Quetiapine 100mg, Zolpidem 5mg sos was started. On the follow up visits Quetiapine was optimized to 100mg OD and 50mg HS, and Divalproex sodium 500mg HS, and Propranolol 40mg OD were added. The patient showed significant improvement and improved rapidly over course of 2 weeks. With regular follow ups for 6months patient has remained asymptomatic and we gradually tapered all the medications 6 months into follow up patient had remained asymptomatic.

Discussion

In 1500 BC, Ayurvedic texts first described Mucuna as a treatment for Kampavata (paralysis agitans), a neurologic disease with similar symptoms to Parkinson's Disease. Mucuna extract is still used in India as an ayurvedic treatment of Parkinson's disease. In contemporary medicine, Mucuna remains a plant of interest since its L-DOPA content and its use in treatment of Parkinson's Disease continues to be evaluated in biochemical research. Detail literature search regarding other herbal constituents in the aforementioned preparation did not reveal any evidence of precipitation of psychiatric symptoms. Thus, except for mucunapruriens none of the other herbs was considered for precipitation of the affective symptoms. Herbal products with ayurvedic medicinal composition are widely used across the Indian population because they are considered natural and free from any side effects. This leads to their indiscriminate unsupervised use without adequate knowledge about their chemical composition and biological changes that they bring about in the body after consumption.

As in our case, the herbal preparation “DIVYA YouvnamritVati” contained multiple ayurvedic compounds which also included Mucunapruriens (konchbeej) which is rich in L-DOPA. These preparation are also used as 'over the counter products' for improving sexual functioning in males as mucuna was used for male infertility and as an aphrodisiac in Ayurveda practise. L-DOPA is transported across the blood brain barrier and is decarboxylated into Dopamine within the brain. This increase in dopamine levels in the brain precipitate psychotic symptoms. S K Matto et al reported a case of a 60 year old female with Parkinson's disease maintaining well on Syndopa along with other medications who started complaining of hearing of voices and suspiciousness and persecutory delusions. She was later diagnosed with Schizophrenia. A study of M. pruriens causing toxicity was reported in November 1989 in the remote district of Memba (Mozambique). District authorities reported 203 cases of acute toxic psychosis over 6-week period where patients were confused, agitated, displayed hallucinatory behaviour and paranoid delusions. All patients recovered within 2 weeks, more rapidly after administration of intravenous chlorpromazine. A similar case was reported by us, where an adolescent male developed manic symptoms abruptly, with history of ingestion of an ayurvedic preparation containing mucunapruriens. Thus, the above mentioned research supports our case report, and brings to attention that the hyperdopaminergic drive due to mucunapruriens can lead to affective symptoms in an individual.

To conclude, this case reports reminds the need of imbibing more knowledge of alternative medicines in the health care professionals and also to bring awareness among the general population, of the possible adverse effects of the so called 'safe' herbal and over the counter preparations.
Bibliography


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