Article *NIMHANS Journal*

Post Schizophrenic Obsessive Compulsive Disorder: A Case Report

Volume: 15 Issue: 01 January 1997 Page: 61-66

G Satheesh Kumar, C K Murali, - *Department of Psychiatry, National Institute of Mental Health & Neuro Sciences, Bangalore 560 029, India*
S Ramachandra &, - *Department of Clinical Psychology, National Institute of Mental Health & Neuro Sciences, Bangalore 560 029, India*
Santosh K Chaturvedi -

Reprints request
,
- *Department of Psychiatry, National Institute of Mental Health & Neuro Sciences, Bangalore 560 029, India*

Abstract

Different aspects of the relationship between the obsessive compulsive disorder and schizophrenia have been reported. Although there are reports on subjects with obsessive compulsive disorder developing schizophrenia with obsessive compulsive symptoms, in none of the reports is there a mention of an obsessive compulsive disorder developing after the remission of a schizophrenic illness. We describe our experience with a patient who developed obsessive compulsive disorder in the post schizophrenic phase. Obsessive compulsive symptoms leading on to obsessive compulsive disorder appeared after the complete remission of psychotic symptoms. The maintenance dose of anti psychotic medication was continued and obsessive compulsive disorder was treated with fluoxetine and behaviour therapy. We find this presentation exceptional which raises more questions about the neurotransmitter hypotheses for schizophrenia and obsessive compulsive disorder, than it answers.

Key words -
Schizophrenia,
Obsessions OCD,
Psychosis

The relationship of obsessive compulsive disorder (OCD) to psychosis has been an area of interest for more than a century. There have been case reports of psychotic symptoms in patients presenting with prominent obsessions and compulsions [1], [2], [3]. Drawing evidences from their own study and previous studies, Insel and Akiskal [4] postulated that obsessive compulsive disorder represents a psychopathological spectrum varying along a continuum of insight. Patients at the severe end of the spectrum are best described as having an obsessive compulsive psychosis.

The frequency of obsessions and compulsions in patients who have psychotic disorders has also been studied [5], [6]. Summers et al [7] in their study of neurotic symptoms in the post acute phase of schizophrenia, found the incidence of obsessive compulsive symptoms to be higher than that in the normal population. Obsessive compulsive symptoms are currently being noted in schizophrenic patients treated with clozapine [8], [9].

Though there are reports on the comorbidity of obsessive compulsive disorder and schizophrenia i.e. subjects with obsessive compulsive disorder developing schizophrenia, and the occurrence of obsessive compulsive symptoms in schizophrenia, in none of the reports is there a mention of an obsessive compulsive disorder developing in an individual after remission from schizophrenic illness treated with conventional antipsychotics. In this case report, we describe a schizophrenic patient who developed obsessive compulsive disorder in the post-schizophrenic phase.

Case Report

Mr. S, a 31 year old physician, presented to our psychiatric centre at the National Institute of Mental Health & Neuro Sciences (NIMHANS), Bangalore, India, initially with a 2 year history of being suspicious of others and withdrawn, inability to relate to people, irritability, and feelings of sadness, which were gradually progressive. These symptoms, which started after he had finished his medical graduation and had joined post graduation, subsequently increased in intensity leading to inability in continuing further studies. Once he assaulted one of his friends as he suspected that person to be "poisoning the mind of his professor against him". Mr. S, had a strong family history of affective disorders. One of his brothers had committed suicide and another brother who was treated for depression, later committed suicide. Two second degree relatives also had history of bipolar affective disorder and were being maintained on lithium. One cousin had bipolar affective disorder while another cousin had generalised epilepsy. Both parents had hypertension. Premorbidly, he was a shy, introverted and very meticulous person but no other anakastic traits were noted. He did not have any identifiable personality disorder on axis II of DSM-III R [10].

He was prescribed a tricyclic antidepressant (dothiepin 75 mg/day) as symptoms of depression were more prominent. There was some improvement in his sleep and mood, but suspiciousness, lack of confidence and other symptoms continued. He reported feeling dull and disinterested and it was difficult to differentiate whether these features were negative symptoms of schizophrenia or due to depression. Later, on examination he has found to have delusion of persecution, though diffusion, and elementary (ringing sounds) auditory hallucinations and was prescribed antipsychotics (Injection Fluphenazine decanoated 25 mg IM every two weeks and Trifluoperazine 10 mg/day). Later he was hospitalised for three weeks. The psychometry test programme included Multiphasic Questionnaire, 16 Personality Factors test, Sentence Completion test, Object Sorting test, Thematic Apperception test, and Rorschach Inkblot tests. Different parameters on psychometry which included both objective and projective tests confirmed a schizophrenic illness. Routine investigations were within normal limits. Following treatment with antipsychotic medications, symptoms decreased markedly and he was discharged from the hospital and asked to continue trifluoperazine 10 mg/day.

He was subsequently followed up by his local psychiatrist. Eventually he passed his postgraduation in Medicine started his clinical practice. Later antipsychotic was changed from trifluoperazine to pimozide (8 mg/day), probably because of some adverse side effects with trifluoperazine, but no psychotic symptoms were noted during this period. He then had a relapse of symptoms with suspiciousness, self-referential ideations, restlessness, tremors and violent behaviour and was brought back to our hospital. On examination, he had thought broadcast, thought insertion and akathisia, and hence was rehospitalised for two months. Diazepam was prescribed to control akathisia. Since thought alienation phenomena and formal thought disorders became severe, after the akathisia had subsided, he was treated with 7 ECTs and thioridazine 100 mg/day. There were recurrences of extrapyramidal

symptoms but were manageable with trihexyphenedyl 4 mg/day. After the psychotic symptoms subsided, he was discharged on thioridazine 100 mg/day. He remained asymptomatic for 10 months on these medications and continued his clinical practice and had regular follow ups with his local psychiatrist.

After a few months, patient developed repetitive thoughts of "raping" female patients and "slapping" male bystanders of patients in his clinic. These symptoms were very minimal initially but later increased in intensity and duration so that he found it very difficult to carry on with his job in his clinic. He had to suspend his clinic for 3-4 days in a week as these thoughts were preventing him from attending to the patients. However, sleep and appetite were normal. His local psychiatrist started clomipramine 75 mg/day for this complaint, but there was no improvement.

He consulted us with these complaints and on examination he was found to have obsessive impulses (to slap male bystanders; to assault women, to touch their body parts) and obsessive doubts (whether he has locked his clinic, has he cleaned his face) which were interfering with his work. There were no psychotic symptoms. The content of the obsessions were dissimilar from the content of his hallucinations, delusions or thought disorders in the previous history. The psychological test programme included administration of Bender Gestalt test, Rorschach Inkblot test, Thematic Apperception test, and Leyton's Obsessional Inventory.

During this repeat psychometry there was no evidence of psychosis, but indicated the presence of repetitive aggressive thoughts. Antipsychotic medication was changed from thoridazine to trifluoperazine 10 mg/day and clomipramine 75 mg already started by his local psychiatrist was continued. He continued clomipramine for 2 weeks and since no improvement was evident, he himself changed it to fluoxetine 60 mg/day initially and increased it to 120 mg/day later. There was worsening in his obsessions which severely impaired his occupational functioning. Subsequently, trifluoperazine was changed to flupenthixol 20 mg IM every 3 weeks for the ease of administration and to decrease extrapyramidal symptoms which were mild, but fluoxetine was continued. There was no relief in his symptoms and he developed depressive features with occasional death wish which resulted in almost complete stoppage of his work in his clinic. He later developed nausea and vomitting, probably fluoxetine induced adverse effects.

In view of these features he was hospitalised for one month. A detailed assessment confirmed the presence of obsessive ideas, obsessive impulses and obsessive doubts mentioned above, and a compulsive checking (lock in his clinic) behaviour which was not severe. No psychotic symptoms were noted. Diagnosis of paranoid schizophrenia (remitted), currently with obsessive compulsive disorder with predominantly obsessions was made based on axis I of DSM III-R [10]. Fluoxetine was reduced to 80 mg/day and behaviour therapy was started. Behaviour therapy consisted of exposing him to specific situations which were anxiety provoking and to listen to the cassette containing a detailed account of his obsessions. In the hospital the intensity and frequency of obsessional thoughts decreased and he was able to concentrate on studying for an examination. Patient was discharged on flupenthixol 20 mg IM every 3 weeks with fluoxetine 80 mg/day. Since discharge, for more than two years the patient has been maintaining improvement, doing his routine clinical work without any psychotic or obsessive symptoms. Mr. S. was informed about publishing his case history maintaining confidentiality, to which he granted his consent.

Discussion

The obsessive compulsive disorder in this case appeared during the post schizophrenic phase after the remission of all psychotic symptoms, a phenomenon which may be difficult to explain with the neurochemical explanations. Further, there was no similarity between the content of obsessive symptoms and other schizophrenic perceptual or thought disorders, which the patient had in the earlier part of the illness.

The hypothesis that schizophrenia is caused by the over activity of dopaminergic neurotransmission has been accepted for a long time, though later researchers turned away from this over-simplified hypothesis, focusing instead on the alteration specific to stage of illness treatment and selected region of brain [11], [12], [13]. Concurrently, researchers found evidences to suggest that other monoamines like nor-epinephrine and serotonin (5-HT) are also involved. Meltzer [14] noting the effect of clozapine, hypothesized that the primary abnormality in schizophrenia could be with serotoninergic system.

The serotonin hypothesis in obsessive compulsive disorder was drawn initially from the effectiveness of serotonin uptake inhibitors in the condition [15]. A correlation was also found between the clinical response and decrease in the cerebrospinal fluid level of 5-hydroxy indole acetic acid and serotonin content in platelets [16], [17]. The blockade of post serotonin receptors using metergoline during treatment was found to have reversed the obsessive symptoms [17]. In spite of all these evidences the precise nature of serotonin dysfunction remains unclear. It seems unlikely that a solitary disturbance in serotonin function can fully account for the pathophysiology of obsessive compulsive disorder. Amphetamine induced stereotypes [18], stereotypes induced by other dopaminergic drugs [19], [20] obsessive compulsive symptoms in post encephalitic parkinsonism [21] and obsessive compulsive symptoms in Tourette disorder [22] point to the involvement of dopaminergic system in obsessive compulsive disorder. The Dopamine-Serotonin balance as the pathophysiological basis of these disorders explain the effectiveness of the novel antipsychotics like clozapine and serotonin uptake inhibitors in certain resistant cases of schizophrenia and neuroleptics in a few obsessive compulsive disorders. To say that serotonin-dopamine dysregulation is the cause of both the disorders will be highly specualtive, but there are ample evidences of dopamine-serotonin dysregulation in schizophrenia and obsessive compulsive disorder.

Thus, it is quite difficult for us to explain the occurrence of obsessive compulsive disorder (a probable serotoninergic system disorder) in an individual who has just had paranoid schizophrenia (a probable dopaminergic system disorder). Our subject did not receive clozapine, which has recently been implicated in the occurrence of obsessive compulsive symptoms in schizophrenia [8], [9]. There is no report of such a phenomenon occurring due to thioridazine or trifluoperazine which our subject was taking prior to development of obsessive compulsive disorder. Hence, we find this presentation exceptional which raises more questions about the neurotransmitter hypotheses for schizophrenia and obsessive compulsive disorder, than it answers.

1.Kringlen E, Obsessional neurotics, a long term follow up
British Journal of Psychiatry Page: 111: 709-22, 1965
2.Rosenberg C M, Complications of obsessional neurosis
British Journal of Psychiatry Page: 114: 477-8, 1968
3.Mirza Hussain K A, Chaturvedi S K, Obsessive Compulsive Disorder with psychotic features - A case report

Indian Journal of Psychiatry Page: 30: 117, 1988
4.Insel T R, Akiskal H S, Obsessive compulsive disorder with psychotic features. A phenomenologic Analysis
American Journal of Psychiatry Page: 143: 1527-33, 1986
5.Rosen I, The clinical significance of obsessions in schizophrenia
Journal of Mental Sciences Page: 103: 773-85, 1957
6.Fenten W S, McGlashan T H, The prognostic significance of obsessive compulsive symptoms in schizophrenia
American Journal of Psychiatry Page: 143: 437-41, 1986
7.Summers F, Harrow M, Westermeyer J, Neurotic symptoms in the post acute phase of schizophrenia
Journal of Nervous & Mental Diseases Page: 171: 216-21, 1983
8.Patil V J, Development of transient obsessive compulsive symptoms during treatment with clozapine
American Journal of Psychiatry Page: 149: 272, 1992
9.Baker R W, Chengappa K N R, Baird J W, Steingard S, Christ M A G, Schooler N R, Emergence of Obsessive compulsive symptoms during treatment with clozapine
Journal of Clinical Psychiatry Page: 53: 439-42, 1992
10.American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders, 3rd Edition, Revised, Washington D. C., A.P.A.
1987
11.Pickar D, Breier A, Hsiao J K, et al, Cerebrospinal fluid and plasma monoamine metabolites and their reaction to psychosis
Archives of General Psychiatry Page: 47: 641-8, 1990
12.Pickar D, Perspectives on a time dependant model of neuroleptic action
Schizophrenia Bulletin Page: 14: 256-68, 1988
13.Meltzer H V, Biological studies in schizophrenia
Schizophrenia Bulletin Page: 13: 109-11, 1987
14.Meltzer H V, Clinical studies on the mechanism of action of clozapine, the dopamine serotonin in hypothesis of Schizophrenia
Psychopharmacology Page: 99, (Suppl) S18-S27, 1989
15.De-Veaugh-Geiss J, Landau P, Katz R, Treatment of obsessive compulsive disorder with clomipramine
Psychiatric Annals Page: 19: 97-101, 1989
16.Flament M F, Rapoport J L, Murphy D L, et al, Biochemical changes during clomipramine treatment of childhood obsessive compulsive disorder
Archives of General Psychiatry Page: 44: 219-25, 1987
17.Benkelfat C, Murphy D L, Zohar J, et al, Clomipramine in obsessive compulsive disorders, further evidence for a serotonin mechanism of action
Archives of General Psychiatry Page: 46: 23-28, 1989
18.Wallah M B, Drug induced stereotyped behaviour, Similarities and differences
In:Usdin A. ed. Neuropsycho-pharmacology of monoamines and their regulatory enzymes, New York, Page: 241-60, 1974
19.Loew D M, Vigouret J M, Jaton A, Neuropharmacology of bromocryptine and dihydroergotoxine
In: Goldstein ed. Ergot Compounds and Brain Function, Neuroendocrine and Neurophyschiatric aspe Page: 63-74, 1980
20.Wallah M B, Gershan S, A Neuro-psychopharmacological comparison of d-amphetamine, L-dopa and cocaine

Neuro-pharmacology Page: 10: 743-52, 1971
21.Jellife S E, Psychopathology of forced movements in Occulo gyric crises
Journal of Nervous & Mental Diseases Page: 151: 550-2, 1932
22.Pitman R K, Green R C, Jenike M A et al, Clinical comparison of Tourette's disorder and obssessive compulsive disorder
American Journal of Psychiatry Page: 144: 1166-71, 1987