Original Article

Neuropsychological Assessment in Obsessive-Compulsive Disorder

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ABSTRACT

Background: Neuropsychological deficits in obsessive-compulsive disorder (OCD) have been encouraged by brain imaging studies suggesting a putative fron to- striatial biological basis of the condition. Studies of neuropsychological functions in OCD have documented deficits in several cognitive domains, particularly with regard to visuospatial abilities, executive functioning, motor speed and memory. The Aim of the present study was to assess neuropsychological profile of patients with OCD. Objectives of the study were to assess and compare the neuropsychological profile of patients with OCD and matched healthy controls. **Materials and Methods:** Twenty clinically stable outpatients with ICD-10 diagnosis of OCD and equal number of normal controls matched for age, education, gender and handedness were studied using a battery of neuropsychological tests. The tests consisted of verbal and performance tests of intelligence, memory, perceptual motor functions, set test and Wisconsin Card Sorting Test (WCST). **Results:** On perceptual-motor functions, verbal fluency, executive functions (WCST), intelligence and memory patients with OCD did not show impairments comparable to healthy controls. An attempt to correlate the test findings with the duration of illness, stability of illness and the average drug dose was made and it was found that there was no correlation between the two. **Conclusion:** The present study does not provide evidence for a localized neuropsychological/cognitive impairment in OCD in cases that are stable for at least three months. Absence of impairments in perceptual-motor functions, verbal fluency, executive functions (WCST), intelligence, and memory does not agree with the results of other studies using these tests.

Key words: *Executive functions, neuropsychological, OCD*

INTRODUCTION

Obsessive Compulsive Disorder (OCD) is a highly debilitating neuropsychiatric condition with estimated lifetime prevalence of 2-3 percent.[1] However unlike other neuro-psychiatric conditions; relatively little is understood about the etiology, neural substrate and cognitive profile of OCD. Until recently OCD was described as a neurotic illness[2] or a manifestation of psychodynamic conflict. Current approaches to OCD suggest that neurobiological abnormalities are involved in its pathogenesis. Moreover renewed interest in demonstrating neuropsychological deficits in OCD has been encouraged by brain imaging studies suggesting a putative fronto-striatial biological basis for the condition.[3,4] Functional neuroimaging studies has demonstrating an abnormal neuronal activity in the orbitofrontal cortex, anterior cingulate cortex, dorsolateral prefrontal cortex, caudate nucleus, and thalamus evidence for frontostriatal dysfunction in OCD.[5] They have generally reported

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hyperactivation of the orbitofrontal cortex, cingulate gyrus, and caudate nucleus.[6] However, structural neuroimaging studies have been less consistent, with some investigations reporting abnormal volumes of the caudate nucleus and orbitofrontal cortex relative to healthy controls.[7] Research on the neuropsychology of OCD has contributed to a better understanding of the neurobiological basis of this disorder. Studies on neuropsychological functioning in OCD have documented deficits in several cognitive domains, particularly visuospatial abilities, executive functioning, motor speed and memory.[8] Clinical observations have also suggested the presence of fundamental processing deficits but abnormalities in several other cognitive domains including executive functions, memory, and visuospatial skills are inconsistent.[9] There is controversy regarding executive functioning: Several studies suggested that the performances of OCD patients on executive functioning tasks were similar to those of healthy controls,[10] whereas others have reported poorer performance for OCD patients on the same measures.[11] For instance, several studies suggest impaired performances on WCST by OCD patients,[12-14] while others[15] found otherwise. Whereas a number of studies failed to find evidence of verbal memory deficits,[16,17] others found significant impairments in measures of free recall and recognition of the verbal material.[18] Patients with OCD also exhibit performance deficits on tests of visual spatial memory and verbal memory[11,19] but they did not demonstrate impaired performance on all the memory functions.[20] It has also been suggested that verbal memory problems in OCD were mediated by impaired organisational strategies used during the learning process secondary to executive dysfunction.[18,21]

From the above literature it is evident that further research is needed to corroborate neuropsychological assessments revealing inconclusive patterns of impairments among patients with OCD. Although there are studies on the neuropsychological profile, and its correlates, of OCD from India but they are mainly from a major centre in South India.[22-28] Cultural variations in a country like India assume[29,30] their own relevance, even in the field of neuropsychology with relative lack of data along with contradictions in assisting literature prompted us to carry out the present research. Additionally, the demonstration of cognitive deficits also has implications for the development of new treatment strategies that is, cognitive remediation and behavioural interventions.[31]

Aim and objectives

The aim of the study was to assess the neuropsychological profile of patients with OCD, and the specific objectives were:

1. To assess the neuropsychologial profile of patients with OCD using various neuropsychological tests and
2. To compare the neuropsychological profile of patients with OCD and matched healthy controls.

MATERIAL AND METHODS

Sample: The sample consisted of two sets that is patients and normal healthy controls. Description of each group is given below:

Clinical group (patients)

Twenty patients with OCD diagnosed according to ICD-10 criteria by a consultant psychiatrist attending the outpatient clinic of Department of Psychiatry, Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh comprised the study group. They were stable for at least for three months (that is 'no clear cut exacerbation of positive or negative symptoms for the last three months on amnestic recall and scrutiny of medical records and on a stable dose of psychotropics where not requiring more than 50% hike or reduction of doses of psychotropics during this period'). They were in the age range of 20-55 years, of either gender, and had at least 10 years of formal education. Co-morbid psychiatric illness, substance abuse or presence of major physical illness served as an exclusion criteria.

Healthy control group

For comparative purpose, 20 normal healthy controls, matched for age, education, gender, and handedness[32] were recruited from staff members of the institute who volunteered for participation in the study. The controls were administered General Health Questionnaire-12[33] for ensuring their health status. A score of ≤2 was deemed necessary for ensuring absence of psychological problems.

Design: Cross-sectional.

INSTRUMENTS

The following tools were used to measure different neuro-cognitive functions in patients and healthy controls:

1. *Verbal Adult Intelligence Scale*[34] (VAIS): It consists of four verbal subtests viz. information, digit span, arithmetic and comprehension. Norms are available for age, sex, and education for the Indian population. The test has adequate reliability and validity.
2. *Bhatia Battery of Performance tests of Intelligence*[35] (BSS): It consists of two tests-Koh's Block Design and Pass Along test measuring spatial, abstract and

practical ability respectively. It is standardised on Indian population aged 20-59 years.

3. *PGI Memory Scale*[36] (PGIMS): It consists of ten subtests viz. remote memory, recent memory, mental balance, attention and concentration, delayed recall, immediate recall, retention of similar pairs, retention of dissimilar pairs, visual retention, and recognition. PGIMS is a reliable and valid measure of memory.
4. *Bender Visual Motor Gestalt test*[37] (BVMGT): It measure perceptual motor skills and consists of eight geometrical drawings to be copied on a plain sheet of paper. Norms are available for the Indian population.[27]
5. *Set test*[38]: It measures the verbal fluency, memory and mental efficiency. It consists of four categories (names of animals, colours, fruits and, cities) to be recalled within 2 minutes each.
6. *Wisconsin Card Sorting Test*[39] (WCST): WCST was administered as a test of executive functions.

Procedure

Both the study groups were assessed on the battery of neuropsychological tests outlined above. Assessment was carried out over 1-2 sessions. All these tests were administered and scored by trained clinical psychologists.

Statistical analysis

Descriptive statistics in the form of mean, standard deviation (SD), and frequency (%) were computed for all the variables. The two groups were subjected to chi-square test for dichotomous variables and t-test test for continuous variables. As a large number of tests of association for 27 neuropsychological variables, Bonferroni correction was employed and statistical significance was revised to 0.002 (0.05/27).

Ethical considerations

The study was approved by the Institute Ethics Committee. Additionally, written informed consent was sought from all the subjects before recruiting them for the study.

RESULTS

In the present study, performance on various individual cognitive tasks rather than summary scores is reported and comparison is made with healthy controls. The result section is divided into socio-demographic characteristics, clinical profile of patients and results pertaining to comparison between clinical and healthy control population on all cognitive/ neuropsychological tests:

1. Socio-demographic Profile of Clinical Sample: Of the 20 patients with OCD, 60% were males and 50% were single. With regard to occupational status, 20% were unemployed, 20% were housewives, 30% were students, and 30% were from other occupational background. 70% were Hindus, 60% belonged to nuclear families and were of urban background each. The mean age of the patients was 32.75 years. All patients had completed at least 10 educational years and were right handed.
2. Socio-demographic Profile of Healthy controls: Twenty healthy controls pair wise matched for age, sex, education and handedness were included in the study. 35% were single and 70% were married. 25% were students, 5% were housewives and 70% belonged to other occupational categories; none were unemployed. 85% were Hindus and 15% were Sikhs. 80% of them were from nuclear, 15% from joint, and 10% from other family types. The mean age of healthy controls was 33.10 years. All healthy controls had completed at least 10 educational years. All were from urban background.
3. Clinical Profile: With regard to the clinical profile, the mean duration of illness was 96.28 (SD = 25.28) months. The mean stability of illness was 9.73 (SD = 6.05) months. Mean drug dose was 84.01 mg of fluoxetine equivalents per day.
4. Intelligence: On all subtests of VAIS and performance tests of Intelligence (BSS) there were no statistically significant differences between the both groups [Table 1].
5. Memory: On PGIMS [Table 2] both groups were comparable.
6. Visuomotor Functions: On BVMGT, the mean errors of OCD patients and normal controls were not significantly different.
7. Verbal fluency: No significant differences in verbal fluency between patients with OCD and healthy controls was seen [Table 3].
8. Executive functions: On WCST [Table 4], results did not show any significant differences between patients and healthy controls on any of the parameters.

DISCUSSION

Aim of the present study was to assess cognitive/

Table 1: Mean, SD, t-values of VAIS & BSS

Subtests	OCD'S (*N* = 20) Mean (SD)	Normal (*N* = 20) Mean (SD)	*t*-value
Information	95.30 (34.93)	102.10 (19.79)	0.76
Digit-span	106.95 (17.23)	105.95 (19.91)	0.07
Arithmetic	93.30 (14.36)	95.00 (13.13)	0.39
Comprehension	102.20 (13.56)	112.20 (13.64)	2.33
Verbal Quotient (VQ)	101.40 (10.77)	105.30 (15.26)	0.93
Block design	9.15 (3.54)	10.25 (4.30)	0.88
Pass along test	9.95 (4.12)	10.60 (2.98)	0.57
Performance Quotient (PQ)	104.95 (14.33)	107.70 (16.99)	0.55

Table 2: Mean, SD and t-values on PGI Memory Scale

Subtests	OCD'S (*N* = 20) Mean (SD)	Normal (*N* = 20) Mean (SD)	*t*-value
Remote memory	5.50 (0.68)	5.80 (0.41)	−1.67
Recent memory	4.90 (0.31)	4.95 (0.22)	0.59
Mental balance	7.25 (1.37)	7.55 (1.40)	0.69
Attention-concentration	10.25 (1.92)	10.10 (2.02)	0.24
Delayed recall	8.30 (1.38)	8.85 (1.23)	1.33
Immediate recall	7.00 (1.86)	8.40 (2.10)	2.17
Retention-similar pairs	4.50 (0.88)	7.25 (10.30)	1.19
Retention-dissimilar pairs	10.45 (3.97)	12.70 (3.13)	1.99
Visual-retention	11.20 (1.94)	11.30 (2.43)	0.14
Recognition (visual)	9.00 (1.38)	9.65 (0.59)	1.94
Percentile	42.20	52.20	2.64

Table 3: Mean, SD and t-values on Set Test

Variables	OCD'S (*N* = 20) Mean (SD)	Normal (*N* = 20) Mean (SD)	*t*-values
Animals	14.90 (3.16)	16.70 (4.10)	−1.56
Colours	13.70 (3.51)	15.60 (4.94)	−1.40
Fruits	13.25 (2.85)	13.50 (3.91)	−0.23
Cities	22.90 (5.74)	23.65 (6.23)	−0.40

Table 4: Mean, SD and t-values of scores on WCST

Variables	OCD's (*N* = 20) Mean (SD)	Normal Controls (*N* = 20) Mean (SD)	t-values
Trials administered	122.75 (16.17)	119.80 (17.24)	0.56
Correct responses	65.60 (17.30)	72.70 (10.84)	−1.56
Errors	57.15 (23.06)	46.95 (18.13)	1.56
Perseverative response	47.20 (35.94)	31.10 (17.07)	1.81
Perseverative error	38.21 (26.03)	26.80 (12.99)	1.72
% CLR	41.85 (22.22)	45.25 (16.34)	−.550
Categories completed	3.15 (1.78)	3.85 (1.73)	−1.27
Trials at 1st category	32.50 (36.70)	19.20 (9.49)	1.57

neuropsychological functioning of OCD patients who were clinically stable with the aim to assess whether enduring neuropsychological deficits are core characteristics of patients with OCD or not.

Intelligence

On VAIS and its subtests there were no significant differences between the clinical and normal sample. On digit span test, which is part of VAIS, similar results were replicated by Bédard.[40] On performance tests of Intelligence (BSS) there were no statistically significant differences either on Pass along or Koh's block design test. Other studies also did not find significant differences between OCD and controls on Koh's Block design and Alexander Pass along subtests of WAIS.[41] Our results are in similar lines with literature which suggests that deficit in intelligence is not a core characteristic in OCD.[42]

Memory

On PGIMS, there were no significant differences between patients with OCD and healthy controls which could be due to the following reasons- clinical sample chosen for the study was not having any active symptomatology at the time of assessment, with presence of at least 6 months of clinical stability. One may speculate that visual retention and verbal memory problems (immediate nonverbal memory) in OCD appear only when patients have an active illness and failure in organizational strategies that mediate the recall process may ultimately lead to deficits in visual retention and verbal memory.[8] OCD and healthy controls use different strategies at encoding level of information processing. In verbal memory, OCD patients code words during objective verbal memory tests, whereas healthy individuals tend to use an organisational strategy, such as semantic relationships. OCD patients have difficulty formulating an organisational strategy, but are able to implement one once formulated[8] Inability to formulate an organisational strategy for information coding might also be related to executive dysfunction. As per Penades *et al*.,[43] nonverbal memory deficits appear to have less to do with memory per se and more to do with the degree of organisational strategies necessary to complete the task. The memory (including immediate recall and new learning) dysfunction in OCD seems to be largely mediated by organisational deficits during the encoding phase that in turn makes recall more difficult. Therefore, it can be argued that the memory impairment in OCD might be due to the failure of organisational strategies. But in the present study stability of illness with lack of active symptomatology might have led onto non significant differences in verbal memory. Executive functions as measured by WCST are also part of organisational strategy but no significant differences between patients and healthy controls. Hence, findings on WCST also suggest that there were no deficits in terms of organisational strategies which might have accounted for non significant differences in memory.

Visuomotor functions

According to published studies, visuospatial coping abilities do not seem impaired in young children nor adolescents with OCD, but these deficits may become apparent in adulthood.[44,45] On BVMGT, the mean errors of OCD's and healthy controls were not significantly different. Overall, general slowness was seen in patients with OCD's compared to healthy controls, which could be attributed to intrusive thoughts and meticulousness.

Verbal fluency

The ability to generate words when given the first letter of the word is an executive function called verbal fluency.

Present study did not show significant differences in verbal fluency between patients with OCD and normal controls. Similar findings have been reported by Aycicigi *et al*.,[15] Rao *et al*.,[25] and in both child and the majority of adult OCD studies, there is little evidence to support impairment in verbal fluency.[41,46]

Executive functions

On WCST, results did not show significant differences between OCD's and normal controls. There are different possible reasons for these non significant differences in OCD's and normal controls. First, studies citing significant differences between patients with OCD and normal controls may be attributed to the possibility that executive dysfunction among patients with OCD is associated with coexistent depression or subclinical depressive symptoms.[10] Depression has been associated with volume reduction in the frontal lobe and medial orbitofrontal cortex and atrophy in these areas was associated with disturbances in memory and executive functions.[47] Further, neuroimaging evidence found lower cerebral glucose metabolism in the caudate, thalamus and hippocampus in participants with both OCD and major depression than in subjects with a primary diagnosis of OCD[48] indicating the negative impact of the presence of co-morbidity on executive functions.[49] Due to the present study not including patients with co morbid depression, the findings are not indicative of significant differences between patients with OCD and healthy controls. Secondly prior studies that have reported performance deficits on tests of executive functions in OCD were associated with other co morbid conditions (e.g., personality disorders). In prior works, it was observed that OCD patients who did poorly on executive function tasks obtained high scores on a measure of schizotypal personality.[50] Such co morbid personality disorders were not part of the present study. Thirdly, the results of tests of executive functioning have been inconsistent in the literature and were difficult to interpret. Reasons being that WCST is the most widely used test for executive functions but is criticized as involving many different cognitive processes,[51] including concept formation, set shifting, abstraction and matching-to-sample. Further, it can be argued that the WCST is not a pure measure of set-shifting; rather, it likely taps many other processes (inhibition, working memory, and the maintenance of attentional processes)[52] and may be more sensitive to the dorsolateral prefrontal cortex (DLPFC) than to orbitofrontal cortex dysfunction. Performance on WCST is consistent with more generalized cognitive impairments which are nonspecific to executive functions. Other studies also reported non significant WCST scores between OCD's and healthy controls.[15] Indian studies have been mainly from one centre in south India that is NIMHANS. Kashyap *et al*.,[28] recently reported presence of deficits in encoding of non-verbal memory and executive dysfunction in patients with OCD with YBOCS score of at least 20 and various co-morbid illness. Possible reasons for differences in results obtained additionally could be-different illness duration, different educational years, lack of clinical stability and variation in use of anti-obsessional agents.

However, our study results should be viewed with the following limitations in mind viz. absence of structured measurement of symptom severity (YBOCS), small sample size, lack of detailed clinical profile of patients, neuropsychological battery being different and not being that comprehensive.

CONCLUSIONS

The current study assessed the neuropsychological functioning of patients with OCD. In comparison to available literature the present study has certain strengths viz:

a. A clinically stable patient population was studied which aimed at minimizing transient state-like effects associated with acute exacerbations and the associated treatment.
b. A reasonably comprehensive neuropsychological evaluation was carried out, and
c. Demographic characteristics (age, sex, education and handedness) known to confound or effect the neuropsychological profile were controlled for.

The present study does not provide evidence for a localized neuropsychological/cognitive impairment in patients with OCD that are clinically stable at least for three months. Absence of impairments in verbal fluency, WCST, and perceptual-motor functions does not agree with the results of other studies using these tests. Additionally, memory deficits in OCD are also not evident. To conclude, more work is needed in this area where differences in research findings out through the similarities obtained till date.

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