Original Article

Soft Neurological Signs and Cognitive Function in Obsessive-compulsive Disorder Patients

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ABSTRACT

Objective: Modern research on obsessive-compulsive disorder (OCD) indicates that the primary cause of OCD, which was earlier explained only on basis of psychoanalytical theories, is biological. Our study attempts to investigate the neurobiological signs in form of soft neurological signs and cognitive function in OCD. **Methods:** A cross sectional study was conducted at psychiatric facility of Seth G.S. Medical College and KEM Hospital. **Materials and Method:** 50 OCD patients and age- and education-matched controls were selected for the study. Established instruments were used to assess the neurological soft signs (NSS) and the cognitive deficits. **Results:** OCD patients had significant more NSS in tests for motor coordination, sensory integration, complex motor tasks, hard signs, and right/left and spatial orientation. Cognitive deficits in the domains of visuospatial ability, executive function, attention, and working memory were significantly more in OCD patients compared to controls. **Conclusion:** Our study highlights the role of biological factors in form of soft neurological signs and cognitive dysfunction in the development of the OCD.

Key words: *Cognitive function, obsessive-compulsive disorder, neurological soft signs*

INTRODUCTION

In last two decades following advances in neuroimaging studies, there has been growing interest in studying the neurological soft signs (NSS) and cognitive function in obsessive-compulsive disorder (OCD), a disorder which was earlier explained only on basis of psychoanalytical theories. Dysfunction in brain functioning implies that OCD should be characterized by neurological abnormalities which can be either "hard" or "soft." Hard signs refer to impairment in basic motor, sensory, and reflex behaviors. In contrast, NSS are described as nonlocalizing neurological abnormalities that cannot be related to impairment of a specific brain region or are not believed to be part of a well-defined neurological syndrome.[1] However, even if they are nonlocalizing, some NSS can suggest dysfunction in particular neural networks and, thus, can give additional information concerning abnormalities in the functional organization that characterize some psychiatric diseases. Cognition denotes a relatively high level of processing of specific information including thinking, memory, perception,

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motivation, skilled movements, and language. A constellation of these core cognitive deficits in various combinations and severity also have a role in the explaining the neuroanatomy and psychopathology of the disease.[2,3] Efforts for assessment of NSS in OCD have met with variable success with some studies reporting significantly higher NSS in the domains of complex motor coordination, involuntary movements, mirror movements, and primitive reflexes in patients with OCD.[4,5] However, there has been lack of consistency and specificity in the findings of the studies.[6] There is also growing evidence for cognitive dysfunction in OCD. Studies have reported impairment in visuospatial ability, executive function, attention, concentration, and working memory in OCD subjects.[7-10] These deficits further lead to impairment in social and occupational functioning leading to increased distress and disability in OCD patients. Although western studies have looked at these issues, there are few studies from India investigating the role of NSS and cognitive deficits in adult OCD.

MATERIALS AND METHODS

The study was conducted in the Outpatient Department of Psychiatry in a tertiary care general public hospital after obtaining permission of the Institutional Review Board. The study group comprised fifty subjects with the International Classification of Diseases-10 diagnosis of OCD, with age more than 18 years. Subjects with intellectual subnormality, organic mental disorders, and spectrum of psychotic disorders (schizophrenia, schizoaffective disorder, and bipolar disorder) as the major axis I disorder were excluded from the study. The control group comprised fifty subjects matched with study group for age and education with no past, present, or family history of major psychiatric disorder in first-degree relatives. All participants gave written informed consent to a protocol approved by the Institutional Review Board.

NSS were assessed using Heidelberg soft neurological signs scale, a 16-item scale. A sufficient internal reliability and interrater reliability has been established for this scale.[11] It assess five factors: Motor coordination (ozeretski test, diadochokinesis, pronation-supination, finger-to-thumb opposition, and speech articulation), sensory integration (gait, tandem walking, and 2-point discrimination), complex motor tests (finger-to-nose test, fist-edge-palm test), right/left and spatial orientation (right/left orientation, graphesthesia, face-hand test, and stereognosis), and hard signs (includes arm-holding test, mirror movements).

The cognitive function was assessed using the Montreal cognitive assessment for mild cognitive dysfunction.[12] It is a one-page, 30-point test. It assesses six cognitive domains: Short-term memory recall task (two learning trials of five nouns and delayed recall after approximately 5 min), visuospatial abilities (clock-drawing task and a three-dimensional cube copy), executive functions (alternation, phonemic fluency, and verbal abstraction task), attention, concentration and working memory (sustained attention task, a serial subtraction, digits forward and backward), language (naming, repetition, aforementioned fluency task), and orientation to time and place.

All analyses were performed with the Statistical Package for Social Sciences software (SPSS Inc., Chicago, IL, USA). Mann–Whitney U-test for comparison of mean between the study and control group was used for the analyses. For correlational analyses, Pearson correlation (p) was used, $P < 0.05$ were considered statistically significant.

RESULTS

Sociodemographic variables

The mean age of OCD subjects was 30.62 years with range between 18 and 59 years. Among them, 64% of the subjects were males, 56% of the subjects were college educated, 34% were up to secondary level, 6% were primary educated, and 4% were illiterate.

Comparison of neurological soft signs

As revealed in Table 1, scores were significantly higher in OCD subjects in all the tests for motor coordination (Ozeretski's test, $P = 0.001$; diadochokinesis, $P < 0.001$; pronation-supination, $P = 0.004$; finger-to-thumb opposition, $P < 0.001$; and speech articulation, $P < 0.001$), sensory integration (gait, $P = 0.002$; tandem walking, $P < 0.001$; and 2-point discrimination, $P < 0.001$), complex motor tasks (finger-to-nose test, $P < 0.001$ and fist-edge-palm test, $P < 0.001$), and hard signs (arm-holding test, $P < 0.001$ and mirror movements, $P < 0.001$). OCD subjects scored significant higher in all the tests of right/left and spatial orientation (right/left orientation, $P < 0.001$; graphesthesia, $P = 0.007$; and stereognosis, $P < 0.001$) except for test of sensory extinction (face-hand test, $P = 1.000$).

Comparison of cognitive assessment for mild cognitive dysfunction

As shown in Table 2, OCD patients fared worse on all subsets of Montreal Cognitive Assessment compared to controls. OCD patients showed significant deficits in short-term memory task ($P < 0.001$), visuospatial

Table 1: Comparison of neurological soft signs

	Group	Mean	SD	*P*
Gait	Study	0.32	0.587	0.002
	Control	0.04	0.198	
Tandem walking	Study	0.38	0.490	<0.001*
	Control	0.02	0.141	
Right/left orientation	Study	0.96	0.968	<0.001*
	Control	0.02	0.141	
Arm-holding test	Study	0.92	0.665	<0.001*
	Control	0.04	0.198	
Finger-to-nose test	Study	0.82	0.825	<0.001*
	Control	0.00	0.000	
Ozeretski's test	Study	1.52	0.646	0.001
	Control	1.02	0.654	
Diadochokinesis	Study	0.96	0.727	<0.001*
	Control	0.46	0.579	
Pronation-supination	Study	0.24	0.517	0.004
	Control	0.02	0.141	
Finger-to-thumb opposition	Study	0.50	0.614	<0.001*
	Control	0.00	0.000	
Mirror movements	Study	0.74	0.600	<0.001*
	Control	0.00	0.000	
Two-point discrimination	Study	0.22	0.418	<0.001*
	Control	0.00	0.000	
Graphesthesia	Study	0.30	0.614	0.007
	Control	0.04	0.198	
Face-hand test	Study	0.02	0.141	1.000
	Control	0.02	0.141	
Stereognosis	Study	0.38	0.530	<0.001*
	Control	0.06	0.240	
Fist-edge-palm test	Study	1.34	0.772	<0.001*
	Control	0.22	0.465	
Speech and articulation	Study	0.60	0.606	<0.001*
	Control	0.02	0.141	
Total score	Study	10.22	0.606	<0.001*
	Control	1.98	0.141	

Mann–Whitney U-test. $P<0.05$ – Significant; *$P<0.001$ – Highly significant. SD – Standard deviation

Table 2: Comparison of cognitive assessment for mild cognitive dysfunction

Domains	Group	Mean	SD	*P*
Short term memory task	Study	3.72	1.278	<0.001*
	Control	4.54	0.579	
Visuospatial ability	Study	2.02	1.059	<0.001*
	Control	3.20	1.107	
Executive function	Study	2.28	0.607	<0.001*
	Control	2.86	0.833	
Attention, concentration, and working memory	Study	3.46	1.528	<0.001*
	Control	4.80	1.429	
Language	Study	4.26	0.803	<0.001*
	Control	4.84	0.422	
Orientation	Study	5.66	0.745	0.573
	Control	5.76	0.591	
Total score	Study	21.84	3.466	<0.001*
	Control	26.40	3.232	

Mann–Whitney U-test, $P<0.05$ – Significant; *$P<0.001$ – Highly significant. SD – Standard deviation

ability ($P < 0.001$), executive function ($P < 0.001$), attention, concentration and working memory ($P < 0.001$), and language ($P < 0.001$). There was no significant difference for orientation ($P = 0.53$) between OCD patients and controls.

DISCUSSION

NSS were present to a significantly greater extent in OCD patients than controls in our study. These findings are in accordance with numerous prior studies reporting significantly higher total NSS scores in OCD subjects.[4,5,13] NSS in our OCD patients were significantly higher in all the subscales of motor coordination, sensory integration, complex motor tests, right/left and spatial orientation, and hard signs. Higher scores for motor coordination, complex motor tasks, and hard signs seen here are also reported by other studies.[4,5] Although the initial study by Hollander 1990 did not report higher scores in sensory integration and right/left and spatial orientation, subsequent studies by Bolton 1998 and Guz 2004 have reported higher scores also on these subtests as well. Bolton 1998 also demonstrated that OCD subjects had neurological signs in certain categories such as motor coordination, sensory integration, hard signs, tardive dyskinesia, catatonic, and extrapyramidal signs similar to patients with schizophrenia.

The significant impairment in cognition observed in our study in the domains of visuospatial ability, executive function, attention, and working memory is in accordance to other studies. Visuospatial and visuoconstructional deficits in tests using ability to draw complex figures are the most consistent findings in OCD.[7,9] Executive function deficits have also been reported in several studies among OCD patients.[8,14,15] OCD patients have shown impairment on both, verbal memory on measures of new learning such as the California verbal learning and nonverbal memory such as visual reproduction and delayed recognition of figures.[14,16] These findings may suggest impairment in encoding and retrieval of new information in OCD patients. Similarly, the finding of short-term memory task deficit in this study may be explained on the above basis as well as the impaired attention seen in our patients. The observation of language deficit in our study needs further evaluation. Although recent research has reported deficit in verbal fluency in OCD,[17,18] there is scanty literature studying other language deficits such as repetition and naming.

The structural and functional imaging studies in OCD pathogenesis have identified dysfunctional frontostriatal circuits.[19-22] The orbitofrontal cortex and

basal ganglia are the most consistently associated with OCD in imaging studies, OCD symptoms attributed to be caused by the hyperactive orbito-fronto-thalamic circuit.[23] These imaging findings are corroborated by the finding that disrupting connections between OFC, ACC, thalamus, and basal ganglia by means of anterior capsulotomy or cingulotomy result in a symptomatic improvement in most OCD patients.[24-26] Further OCD symptoms are reported damage to the basal ganglia, especially the caudate and orbitofrontal cortex.[27,28] Also, dysfunction of the basal ganglia secondary to a streptococcal infection[29] or encephalitis lethargic[30] has also been associated with the development of OCD. Although NSS are described as nonlocalizing neurological abnormalities, neuroimaging studies have suggested associations of NSS with activation changes in the sensorimotor cortex, supplementary motor area, cerebellum, basal ganglia, and thalamus.[11,31] Similarly, the cognitive deficits, namely, visuospatial, executive, attention, concentration, and memory observed in the study are indicative of the underlying neuroanatomical and neurophysiological changes, mainly in the frontal lobe.

The presence of confounding factors such as comorbid depressive or psychotic symptoms is known to influence NSS and cognition. However, study by Trivedi *et al.* comparing OCD patients without any other comorbid axis I disorder with healthy controls reported significant impairment in executive function, sustained attention abilities, and spatial working memory in OCD patients.[32] Similarly, Rao *et al*. reported significant neuropsychological deficits in thirty recovered OCD patients in comparison with thirty matched healthy controls.[33] These findings are indicative that neuropsychological deficits are possibly stated independent and are indicative of underlying neuroanatomical deficit. As this was a cross-sectional study, the effect of therapeutic interventions on the scores of NSS and cognitive functions could not be assessed.

CONCLUSION

The presence of NSS and cognitive deficits in OCD is indicative of underlying neuroanatomical and neurophysiological dysfunction and that OCD is a brain disease. OCD affects the younger population and is known to cause significant impairment in individual social and occupational productivity. This disability can now be linked to the defective higher mental functions secondary to neurological dysfunction and not just the obsessive symptomatology as thought previously.

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Conflicts of interest
There are no conflicts of interest.

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