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Neurological Soft Signs, Cognition and Sensory Phenomena in Obsessive Compulsive Disorder

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Abstract

Background: Neurological soft signs (NSS), cognitive deficits, and sensory phenomena are increasingly recognized in obsessive-compulsive disorder (OCD), suggesting neurobiological underpinnings. However, their interrelationships and clinical significance remain unclear. This study examines NSS, cognition, and sensory phenomena in OCD patients compared to healthy controls.

Methods: A cross-sectional study was conducted on 35 OCD patients and 35 matched healthy controls. Neurological soft signs were assessed using the Neurological Evaluation Scale (NES), cognitive function was evaluated with the Montreal Cognitive Assessment (MoCA), and sensory phenomena were measured using the University of São Paulo Sensory Phenomena Scale (USP-SPS). The Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) assessed OCD severity, and the Hamilton Depression Rating Scale (HDRS) evaluated depressive symptoms. Statistical analyses included group comparisons and correlation analyses.

Results: OCD patients exhibited significantly higher NSS scores (21.49 ± 6.98) compared to controls (16.63 ± 3.83, $p = 0.002$), with pronounced deficits in sensory integration ($p = 0.003$) and sequencing of motor acts ($p = 0.009$). Cognitive impairment was evident, with OCD patients scoring lower in MoCA total scores (26.74 ± 2.05 vs. 27.71 ± 0.93, $p = 0.019$), particularly in visuospatial/executive function ($p = 0.034$) and memory ($p = 0.045$). Sensory phenomena were more frequent in OCD patients, with 48.6% reporting abnormal sensory experiences ($p = 0.041$). NSS and sensory phenomena correlated positively with OCD severity ($r = 0.28$, $p = 0.041$), while depressive symptoms also showed a significant association with OCD severity ($r = 0.52$, $p < 0.001$).

Conclusion: OCD patients exhibit increased neurological soft signs, cognitive deficits, and sensory phenomena compared to healthy controls. The association between

NSS, sensory phenomena, and OCD severity highlights potential neurobiological dysfunctions in OCD. Future studies should explore underlying neural mechanisms and their therapeutic implications.

Keywords: Obsessive-compulsive disorder, Neurological soft signs, Yale-Brown Obsessive-Compulsive Scale, Montreal Cognitive Assessment, University of São Paulo Sensory Phenomena Scale.

Introduction

Obsessive-Compulsive Disorder (OCD) affects approximately 2–3% of the global population and remains underdiagnosed and undertreated, particularly in low- and middle-income countries [1,2]. It is characterized by persistent obsessions and compulsions, significantly impairing daily functioning. While traditionally understood through cognitive-behavioral models, recent research highlights the role of neurological soft signs (NSS), cognitive deficits, and sensory phenomena (SP) in its pathophysiology, offering new perspectives for understanding and managing the disorder [3,4].

NSS are minor neurological abnormalities, such as motor coordination deficits, sensory integration issues, and difficulties in complex movement sequencing. These signs suggest dysfunctions in cortico-striato-thalamo-cortical circuits implicated in OCD[5]. Studies report a higher prevalence of NSS in OCD patients compared to healthy controls, supporting a neurodevelopmental component in its pathogenesis [6].

Cognitive dysfunctions, particularly in executive functioning, memory, and attention, contribute to OCD symptom persistence and severity. Impaired response inhibition, cognitive flexibility, and decision-making hinder the ability to suppress intrusive thoughts and resist compulsive behaviors[7]. Neuropsychological assessments, such as the Wisconsin Card Sorting Test and Stroop Test, frequently show poorer performance in OCD patients, reinforcing cognitive rigidity as a core feature of the disorder [8]. SP, described as “just-right” perceptions or “not-just-right experiences” (NJRE), refer to uncomfortable sensory urges that precede compulsive behaviors, distinguishing them from anxiety-driven compulsions. Approximately 60–70% of individuals with OCD experience SP, which is strongly associated with symptom severity, particularly in compulsions related to symmetry, ordering, and arranging [9,10]. Heightened sensitivity to tactile, auditory, or visual stimuli amplifies distress, reinforcing compulsions to alleviate sensory discomfort [11].

Understanding the interplay between NSS, cognitive deficits, and SP in OCD may refine diagnostic approaches and contribute to targeted interventions. This study aimed to explore these associations, providing insights into the neurobiological mechanisms of OCD and their potential implications for treatment.

Materials and Methods

Study Design and Setting

The present study was a cross-sectional study conducted in the Department of Psychiatry, Vardhman Mahavir Medical College and Safdarjung Hospital over a period of 18 months from April 2023 to October 2024. Institutional ethical committee approval was obtained prior to the commencement of the study (IEC/VMMC/SJH/THESIS/2023/CC-270, dated 27/03/2023), and written informed consent was secured from all participants.

Sample Size

The sample size was determined based on a reference study conducted by Dhuri et al., [12,45]. In the study group of OCD patients, the mean value was 10.22 ± 1.212, while in the control group, it was 1.98 ± 0.282. Using a power of 80% (β=0.2) and a 95% confidence interval (α=0.05), the minimum required sample size was

calculated to be 30 per group. Considering a 10% error rate, the final sample size was set at 35 participants in each group, leading to a total of 70 subjects.

Study Participants

The study included patients aged 18-60 years diagnosed with obsessive-compulsive disorder (OCD) as per DSM-5 criteria, who were able to read Hindi or English. Exclusion criteria for the patient group comprised individuals unable to provide a detailed history due to neurodevelopmental disorders or psychiatric comorbidities, as well as those with suicidal ideation. The control group consisted of age- and education-matched healthy individuals with no personal or family history of major psychiatric disorders in first-degree relatives, who could read Hindi or English. Controls with neurodevelopmental disorders or psychiatric comorbidities were excluded.

Assessment Tools

Several validated scales were employed to assess neurological soft signs, cognition, and sensory phenomena in OCD patients. The Neurological Evaluation Scale (NES) was used to measure neurological soft signs, assessing sensory integration, motor coordination, and sequencing of complex motor acts, with acceptable psychometric properties. The Montreal Cognitive Assessment (MoCA) evaluated cognitive impairment across domains such as visuospatial function, attention, memory, and executive functioning. The Hamilton Depression Rating Scale (HDRS) was used to assess depressive symptoms, categorizing severity into mild, moderate, and severe levels. OCD severity was measured using the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS), which quantifies the intensity of obsessive-compulsive symptoms. Additionally, the University of Sao Paulo Sensory Phenomena Scale (USP-SPS) was employed to evaluate sensory phenomena before or during compulsive behaviors, assessing frequency, distress, and interference in functioning.

Statistical Analysis

Data was coded and recorded in an MS Excel spreadsheet, and statistical analysis was performed using SPSS version 21.0. Descriptive statistics were presented as means with standard deviations for continuous variables, while categorical variables were expressed as frequencies and percentages. Group comparisons for continuous data were conducted using an independent sample t-test for two groups, and One-Way ANOVA for more than two groups, followed by Tukey's HSD test for post-hoc analysis. If data were found to be non-normally distributed, non-parametric tests such as the Wilcoxon test or Kruskal-Wallis test were applied. The chi-square test was used for categorical comparisons, and Fisher's exact test was employed when expected frequencies were low. Correlations between continuous variables were analyzed using Pearson's correlation for normally distributed data and Spearman's correlation for non-normally distributed data. Statistical significance was set at $p < 0.05$.

Results

The study included 35 OCD cases and 35 matched controls. The mean age was comparable between cases (35.37 ± 13.01 years) and controls (35.20 ± 8.13 years) ($p = 0.601$), with a similar male-to-female ratio ($p = 0.701$). Educational level, occupation, marital status, and family type showed no significant differences. However, socioeconomic status differed significantly ($p = 0.010$), with more OCD cases in the upper middle class. The mean duration of OCD was 2.91 ± 3.16 years. The mean Y-BOCS score was 26.91 ± 4.14, with 62.9% classified as severe. The mean HDRS score was 14.21 ± 6.53, indicating mild-to-moderate depression (Table 1).

Table 1: Demographic and Clinical Characteristics of OCD Cases and Controls.

Parameter	Cases (n = 35)	Controls (n = 35)	p-value
	Frequency (%)/Mean ± SD		
Age (years)	35.37 ± 13.01	35.20 ± 8.13	0.601
Sex			
Male	23 (65.7%)	25 (71.4%)	0.701
Female	12 (34.3%)	10 (28.6%)	
Education			
No Formal Education	5 (14.3%)	5 (14.3%)	1.000
Primary School	0 (0.0%)	0 (0.0%)	
Middle School	1 (2.9%)	1 (2.9%)	
High School	2 (5.7%)	2 (5.7%)	
Intermediate	6 (17.1%)	6 (17.1%)	
Graduate/Postgraduate	20 (57.1%)	20 (57.1%)	
Diploma (Civil)	1 (2.9%)	1 (2.9%)	
Occupation			
Unemployed	1 (2.9%)	2 (5.7%)	0.869
Homemaker/Housewife	9 (25.7%)	8 (22.9%)	
Private Job	8 (22.9%)	12 (34.3%)	
Government Job	9 (25.7%)	6 (17.1%)	
Business	2 (5.7%)	2 (5.7%)	
Student	6 (17.1%)	5 (14.3%)	
Marital Status			
Unmarried	20 (57.1%)	15 (42.9%)	0.232
Married	14 (40.0%)	20 (57.1%)	
Widowed	1 (2.9%)	0 (0.0%)	
Type of Family			
Nuclear Family	24 (68.6%)	22 (62.9%)	0.6153
Joint Family	11 (31.4%)	13 (37.1%)	
Socioeconomic Status			
Upper	0 (0.0%)	0 (0.0%)	0..010
Upper Middle	8 (22.9%)	3 (8.6%)	
Lower Middle	27 (77.1%)	32 (91.4%)	
Upper Lower	0 (0.0%)	0 (0.0%)	
Lower	0 (0.0%)	0 (0.0%)	

Duration of OCD (years)	2.91 ± 3.16	_	_
Y-BOCS Total Score	26.91 ± 4.14	_	_
Y-BOCS Score Categories			
Mild (<16)	3 (8.6%)	-	-
Moderate (16-23)	10 (28.6%)	-	-
Severe (>23)	22 (62.9%)	-	-
HDRS Total Score	14.21 ± 6.53	-	-

The OCD group exhibited significantly higher NES scores compared to controls, indicating greater neurological soft sign impairments. Sensory integration deficits were more pronounced in the OCD group (5.29 ± 1.79) than in controls (4.17 ± 0.71, p=0.003), suggesting difficulties in processing and responding to sensory stimuli. Similarly, impairments in sequencing of motor acts were observed in the OCD group (5.43 ± 1.69) compared to controls (4.34 ± 1.28, p=0.009). The "other domain" score was also significantly elevated in the OCD group (5.69 ± 1.76) relative to controls (4.34 ± 1.41, p=0.005). Although motor coordination scores were higher in the OCD group (5.31 ± 1.76) compared to controls (4.46 ± 0.95), the difference was not statistically significant (p=0.065). The total NES score was significantly higher in individuals with OCD (21.49 ± 6.98) than in controls (16.63 ± 3.83, p=0.002), reinforcing the presence of subtle neurological abnormalities in OCD, potentially reflecting underlying neurodevelopmental disruptions (Table 2).

Table 2: Neurological Examination Score (NES) in OCD and Control Groups.

NES Domains	OCD Group (n=35)	Control Group (n=35)	p-value
	Mean ± SD		
Sensory Integration	5.29 ± 1.79	4.17 ± 0.71	0.003
Motor Coordination	5.31 ± 1.76	4.46 ± 0.95	0.065
Sequencing of Motor Acts	5.43 ± 1.69	4.34 ± 1.28	0.009
Other domain	5.69 ± 1.76	4.34 ± 1.41	0.005
Total NES Score	21.49 ± 6.98	16.63 ± 3.83	0.002

OCD patients had significantly lower total MoCA scores than controls (26.74 ± 2.05 vs. 27.71 ± 0.93, p=0.019), with deficits in visuospatial/executive function (4.2 ± 1.1 vs. 4.8 ± 0.6, p=0.034) and memory (3.9 ± 1.0 vs. 4.5 ± 0.8, p=0.045). Although attention and language scores were lower in the OCD group, differences were not significant. Notably, 20% of OCD patients had MoCA scores in the 11–25 range, indicating mild cognitive impairment (p=0.011) (Table 3).

Table 3: Montreal Cognitive Assessment (MoCA) Scores in OCD and Control Groups

Variables	OCD Group (n=35)	Control Group (n=35)	p-value
	Frequency (%)/Mean ± SD		
MoCA Domains			
Visuospatial/Executive	4.2 ± 1.1	4.8 ± 0.6	0.034

Attention	5.6 ± 0.9	5.9 ± 0.5	0.112
Memory	3.9 ± 1.0	4.5 ± 0.8	0.045
Language	2.7 ± 0.6	2.9 ± 0.3	0.078
Total MoCA Score	26.74 ± 2.05	27.71 ± 0.93	0.019
MOCA Category			
11 to 25 (n=7)	7 (20.0%)	0 (0.0%)	0.011
26 to 30 (n=63)	28 (80.0%)	35 (100.0%)	

Sensory and perceptual phenomena were assessed using the USP-SPS domains. Among physical sensations, tactile sensations (A1) were reported by 34.3% of OCD patients, while other sensory sensations (A2) were present in 48.6%. Unspecified sensory perceptions (B3) were uncommon, seen in only 2.9%. In the Just Right Perception category, cognitive (B1), motor (B2), and general (C1) "just right" perceptions were reported by 11.4%, 8.6%, and 8.6%, respectively. Energy dysregulation was less frequent, with 5.7% reporting increased (D) and decreased (E) energy levels. Regarding Urge-Only Type, the majority (88.6%) exhibited no urge-only symptoms, while 8.6% had Type E and 2.9% had Type F (Table 4).

Table 4: Distribution of Sensory and Perceptual Phenomena (USP-SPS Domains) and Urge-Only Type in OCD Patients.

USP-SPS Domain	Present	Absent
	Frequency (%)	
Physical Sensations		
A1 (Tactile Sensation)	12 (34.3%)	23 (65.7%)
A2 (Other Sensory Sensations)	17 (48.6%)	18 (51.4%)
B3 (Unspecified Sensory Perception)	1 (2.9%)	34 (97.1%)
Just Right Perception		
B1 (Cognitive "Just Right")	4 (11.4%)	31 (88.6%)
B2 (Motor "Just Right")	3 (8.6%)	32 (91.4%)
C1 (General "Just Right")	3 (8.6%)	32 (91.4%)
Energy Dysregulation		
D (Increased Energy)	2 (5.7%)	33 (94.3%)
E (Decreased Energy)	2 (5.7%)	33 (94.3%)
Urge-Only Type	n (%)	
None	31 (88.6%)	-
Type E	3 (8.6%)	-
Type F	1 (2.9%)	-

The analysis showed no significant differences in Y-BOCS scores based on most USP-SPS domains. However, individuals with unspecified sensory perception (B3) had the highest Y-BOCS score (30.00 ± 0.00), while those with general "just right" perception (C1) also had slightly higher scores (28.33 ± 1.15).

Energy dysregulation (D & E) showed lower Y-BOCS scores (22.00 ± 0.00) but was not statistically significant (p = 0.080). Urge-only Type E and F had higher Y-BOCS scores (29.00 ± 1.00 and 29.00 ± 0.00, respectively) compared to those without urges (26.65 ± 4.32). Cognitive performance (MoCA Category) was not significantly linked to symptom severity (p = 0.207). However, higher NES scores were significantly correlated with increased Y-BOCS scores (p = 0.038), with the high NES group (>40) having the highest score (29.14 ± 4.32). Similarly, increasing HDRS scores were significantly associated with greater OCD severity (p = 0.012), with severely depressed individuals (>23 HDRS score) having the highest mean Y-BOCS score (30.75 ± 3.94), indicating a strong relationship between depressive symptoms and OCD severity (Table 5).

Table 5: Association of Y-BOCS Total Score with USP-SPS Domains, MoCA Category, NES Score, and HDRS Score.

Variable	Y-BOCS Total Score (Mean ± SD)	p-value
USP-SPS Domains		
Physical Sensation		
A1 (Tactile Sensation) - Present	26.42 ± 4.38	0.453
A1 (Tactile Sensation) - Absent	27.17 ± 4.09	
A2 (Other Sensory Sensations) - Present	26.41 ± 4.17	0.493
A2 (Other Sensory Sensations) - Absent	27.39 ± 4.17	
Just Right Perception		
B1 (Cognitive "Just Right") - Present	27.00 ± 1.15	0.979
B1 (Cognitive "Just Right") - Absent	26.90 ± 4.39	
B2 (Motor "Just Right") - Present	27.33 ± 1.15	0.953
B2 (Motor "Just Right") - Absent	26.88 ± 4.32	
B3 (Unspecified Sensory Perception) - Present	30.00 ± 0.00	0.345
B3 (Unspecified Sensory Perception) - Absent	26.82 ± 4.17	
C1 (General "Just Right") - Present	28.33 ± 1.15	0.554
C1 (General "Just Right") - Absent	26.78 ± 4.30	
Energy Dysregulation		
D (Increased Energy) - Present	22.00 ± 0.00	0.080
D (Increased Energy) - Absent	27.21 ± 4.08	
E (Decreased Energy) - Present	22.00 ± 0.00	0.080
E (Decreased Energy) - Absent	27.21 ± 4.08	
Urge-Only Type		
None	26.65 ± 4.32	-
Type E	29.00 ± 1.00	-
Type F	29.00 ± 0.00	-

MoCA Category		
11 to 25	25.29 ± 2.75	0.207
26 to 30	27.32 ± 4.36	
NES Score Category		
Low (<30)	24.80 ± 3.95	0.038
Moderate (30-40)	26.90 ± 3.75	
High (>40)	29.14 ± 4.32	
HDRS Score Category		
Minimal (0-7)	23.20 ± 4.12	0.012
Mild (8-16)	25.76 ± 3.89	
Moderate (17-23)	28.22 ± 4.08	
Severe (>23)	30.75 ± 3.94	

Pearson's correlation analysis revealed that NES Total Score (r = 0.04, p = 0.757) and MoCA Total Score (r = 0.05, p = 0.804) had weak and non-significant correlations with OCD severity. However, USP-SPS Total Score showed a mild but significant positive correlation (r = 0.28, p = 0.041), indicating a potential link between sensory phenomena and OCD severity. Notably, HDRS Score exhibited a moderate to strong positive correlation (r = 0.52, p < 0.001), suggesting that greater depressive symptom severity is significantly associated with increased OCD severity (Table 6).

Table 6: Correlation of NES, MoCA, USP-SPS, and HDRS Scores with OCD Severity.

Variable	Correlation Coefficient (r)	p-value
NES Total Score	0.04	0.757
MoCA Total Score	0.05	0.804
USP-SPS Total Score	0.28	0.041
HDRS Score	0.52	<0.001

Discussion

The present study investigated the relationship between neurological soft signs (NSS), cognitive function, and sensory phenomena in patients with obsessive-compulsive disorder (OCD). Our findings indicate significant differences in NSS domains, cognitive performance, and sensory experiences between individuals with OCD and healthy controls.

Patients with OCD demonstrated significantly higher Total NSS Scores (21.49 ± 6.98 vs. 16.63 ± 3.83, p = 0.002), particularly in Sensory Integration (p = 0.003), Sequencing of Motor Acts (p = 0.009), and Other Domain (p = 0.005), suggesting greater neurodevelopmental abnormalities. Prior studies by Lee et al., and Vicente et al., have similarly reported increased NSS in OCD, associating these deficits with dysfunctional cortico-striato-thalamo-cortical (CSTC) circuits [13,14]. Elevated NSS scores may reflect aberrant motor coordination, perceptual integration deficits, and impaired execution of motor sequences, highlighting potential cerebellar and basal ganglia dysfunction in OCD pathophysiology [15].

The cognitive assessment using the Montreal Cognitive Assessment (MoCA) revealed significantly lower total

scores in the OCD group compared to controls (26.74 ± 2.05 vs. 27.71 ± 0.93, p = 0.019). Specific deficits were observed in Visuospatial/Executive functioning (p = 0.034) and Memory (p = 0.045). These findings align with prior research by Fajnerova et al., and Liu et al., indicating executive dysfunction in OCD, likely due to impaired prefrontal-striatal connectivity [16,17]. Cognitive impairments in OCD patients have been attributed to excessive cognitive rigidity, deficits in set-shifting, and altered decision-making, which may exacerbate compulsive behaviors [18,19]. The presence of mild cognitive impairment in OCD further supports the hypothesis of frontostriatal dysregulation contributing to obsessive thoughts and compulsive acts.

The sensory phenomena associated with OCD, measured through the USP-SPS scale, demonstrated notable findings. A substantial proportion of patients reported physical sensations (A1: 34.3%, A2: 48.6%) and "just right" perceptions (B1: 11.4%, B2: 8.6%, C1: 8.6%). Sensory phenomena, particularly "just right" perceptions, have been implicated in the pathogenesis of compulsions, where patients experience an overwhelming urge to repeat behaviors until a subjective sense of completion is achieved [20,21].

A significant correlation was found between USP-SPS Total Score and OCD severity (r = 0.28, p = 0.041), indicating that greater sensory abnormalities are linked to more severe compulsions. These findings support the theory that sensory processing dysfunctions, possibly mediated by abnormal somatosensory and premotor cortical activity, play a crucial role in OCD symptomatology [22].

Depression frequently coexists with OCD, as reflected in our study, where HDRS scores were significantly correlated with OCD severity (r = 0.52, p < 0.001). This strong correlation is consistent with previous studies by Vellucci et al., and McGovern et al., that suggest shared neurobiological substrates between OCD and depression, particularly dysfunctions in serotonergic transmission[23,24]. The presence of comorbid depression in OCD patients may exacerbate cognitive impairments, sensory hypersensitivity, and compulsive behaviors, further complicating treatment outcomes.

The findings of this study highlight the need for a multidimensional approach in OCD assessment and management. The presence of significant NSS and sensory processing abnormalities suggests that incorporating motor and sensory-based rehabilitation strategies may benefit patients with OCD[25]. Additionally, cognitive training programs targeting executive dysfunction could improve symptom control and daily functioning[26,27]. The strong correlation between depressive symptoms and OCD severity further underscores the importance of addressing comorbid mood disorders in therapeutic interventions [28,29,30].

Limitations and Future Directions

This study has some limitations, including a relatively small sample size, which may limit the generalizability of the findings. Additionally, the cross-sectional design prevents establishing causality between NSS, cognitive deficits, sensory phenomena, and OCD severity. Future research should focus on longitudinal studies with larger cohorts and neuroimaging techniques to better understand the underlying neurobiological mechanisms of these associations.

Conclusion

This study provides valuable insights into the neurocognitive and sensory alterations in OCD, emphasizing the role of NSS, cognitive impairments, and sensory phenomena in symptom severity. The observed correlations between USP-SPS scores, HDRS scores, and OCD severity suggest that these factors contribute

significantly to the disorder's clinical expression. Integrating these findings into clinical practice may enhance diagnostic accuracy and treatment efficacy for individuals with OCD.

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