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Cognitive Dysfunctions and Neurological Soft Signs in Drug-Naïve Schizophrenia: A Cross-Sectional Study

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Abstract

Background: Cognitive impairments and neurological soft signs (NSS) are common in schizophrenia, often evident prior to treatment. This study investigates the relationship between cognitive deficits and NSS in drug-naïve schizophrenia patients.

Methods: A cross-sectional study was conducted with 60 drug-naïve schizophrenia patients. Cognitive performance was evaluated using the Montreal Cognitive Assessment (MoCA), with scores ≤26 indicating cognitive impairment. NSS were assessed via the Neurological Evaluation Scale (NES). Pearson Chi-square tests analysed associations between MoCA scores and specific NSS, with significance set at $p < 0.05$.

Results: Of the 60 patients, 39 (65%) showed cognitive impairment (MoCA ≤26). Significant correlations were found between cognitive deficits and several NSS, including Audio-Visual Integration ($p = 0.001$), graphesthesia ($p = 0.027$), fist-ring test ($p = 0.001$), and fist-edge-palm test ($p = 0.001$). Additional associations were noted with memory ($p = 0.001$), rhythm tapping test part A ($p = 0.001$), and rapid alternating movements ($p = 0.001$). No significant correlations were observed for cerebral dominance ($p = 0.435$), stereognosis ($p = 0.459$), or extinction ($p = 0.459$). Patients exhibited an average of 4.6 NSS, with 86.7% showing at least one NSS.

Conclusion: Cognitive impairments in drug-naïve schizophrenia patients are significantly associated with specific NSS, suggesting shared neurobiological underpinnings. These findings emphasize the need for early cognitive and neurological evaluations to guide treatment. Longitudinal studies are required to examine the progression of these deficits.

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Introduction

Schizophrenia is a profound mental disorder marked by symptoms such as hallucinations, delusions, negative symptoms, and cognitive impairments. Cognitive deficits, a central characteristic of schizophrenia, often emerge at the illness's onset and persist despite treatment [1]. These impairments disrupt attention, memory, executive functioning, and processing speed, profoundly affecting patients' daily functioning and quality of life [2]. Alongside cognitive challenges, schizophrenia is linked to neurological soft signs (NSS), subtle neurological irregularities that indicate dysfunction in sensory integra-

tion, motor coordination, and task sequencing [3]. NSS are commonly observed in schizophrenia and are viewed as potential endophenotypes, signalling underlying neurodevelopmental disruptions.[4] These signs include challenges in motor coordination (e.g., Fist-Edge-Palm Test), sensory processing (e.g., Audio-Visual Integration), and graphesthesia. Notably, NSS often appears in drug-naïve patients, suggesting they are inherent to the disorder rather than a consequence of antipsychotic treatment [5]. The presence of NSS in first-episode, untreated patients supports the neurodevelopmental hypothesis, which attributes schizophrenia to early brain development anomalies [6].

Globally, schizophrenia affects approximately 24 million people, or 0.32% of the population (1 in 300), with a slightly higher prevalence among adults at 0.45% (1 in 222) [7]. The Global Burden of Disease (GBD) Study 2019 reports that from 1990 to 2019, the raw prevalence of schizophrenia increased by over 65%, from 14.2 million to 23.6 million, though age-standardized prevalence rates remained stable [8]. Schizophrenia ranks among the top 15 leading causes of disability worldwide, contributing significantly to disability-adjusted life years (DALYs) due to its chronic nature and associated impairments [8]. In India, the National Mental Health Survey (NMHS) conducted between 2015 and 2016 across 12 states reported a lifetime prevalence of schizophrenia spectrum disorders at 1.41% and a current prevalence of 0.42%, with a substantial treatment gap of 72% [9]. Another study analyzing data from 2008 to 2017 indicated a 19% increase in schizophrenia cases in India, from 2.91 million to 3.46 million, with higher prevalence among males and individuals aged 25–49 [10]. These figures highlight the significant burden of schizophrenia in India, where genetic, environmental, and cultural factors may influence its clinical presentation and outcomes [9]. The connection between cognitive impairments and NSS in schizophrenia has attracted increasing research interest. Studies suggest NSS may indicate cognitive deficits, likely due to shared neurobiological disruptions in frontocerebellar and subcortical pathways [11]. However, this relationship remains underexplored in drug-naïve patients, particularly in India, where genetic and cultural factors may shape clinical manifestations [12]. This study examines the association between cognitive impairments, assessed by the Montreal Cognitive Assessment (MoCA; score ≤26 indicating dysfunction) [13], and NSS, evaluated using the Neurological Evaluation Scale (NES) [14], in drug-naïve schizophrenia patients. By focusing on untreated individuals, the study aims to clarify the inherent link between cognitive and neurological deficits.

Methodology

This cross-sectional study involved 60 drug-naïve patients diagnosed with schizophrenia as per the International Classification of Diseases (ICD-10) criteria. The sample size was calculated as follows.

Sample Size

The sample size was estimated using the formula

$$n = \frac{Z^2 S^2}{d^2}$$

where

n is denoted for the required minimum sample size

z = 1.96 at 95% confidence interval

S = Standard deviation of GAF total score 4.18

d = 3% Relative error of mean (mean of GAF Total score=36.54)

d = 1.09

$$n = \frac{(1.96)^2 * 4.18 * 4.18}{(1.09)^2} = 59 \text{ minimum sample}$$

Participants were enrolled from the outpatient department of a tertiary care center in India from July 2023 to March 2025. Eligibility criteria included ages 18–45 years, no prior antipsychotic treatment, and no history of neurological conditions, substance dependence, or significant head trauma. The institutional ethics committee approved the study, and all participants provided informed consent. Cognitive performance was evaluated using the montreal cognitive assessment (MoCA), a validated instrument for identifying mild cognitive impairment as shown in Table 1. The MoCA assesses domains such as attention, memory, language, visuospatial abilities, and executive function, with scores ranging from 0 to 30. A score of ≤26 indicated cognitive impairment, as recommended for psychiatric populations [13]. The MoCA was con-

Table 1: Distribution of cognitive function based on MoCA scores

MoCA score	*Frequency*	*Percent*
< 26	39	65.0
> or = 26	21	35.0
Total	60	100.0

Table 2: Distribution of patients according to number of soft signs present

Number of Soft signs Present	*Frequency*	*Percent*
0	8	13.3
1	10	16.7
2	6	10.0
3	1	1.7
4	0	0
5	7	11.7
6	8	13.3
7	5	8.3
8	8	13.3
9	4	6.7
10	2	3.3
11	1	1.7
Total	60	100.0

ducted in a controlled, distraction-free setting. Neurological soft signs (NSS) were assessed using the neurological evaluation scale (NES), a validated tool for detecting subtle neurological irregularities in psychiatric patients in India [14-16]. The NES evaluates sensory integration (e.g., Audio-Visual Integration, Graphesthesia), motor coordination (e.g., Fist-Ring Test, Fist-Edge-Palm Test), and domains like memory and rhythm tapping. Tests were scored based on abnormality presence and severity, with higher scores indicating greater impairment. Neurologists, unaware of MoCA scores, performed NES assessments to ensure objectivity. Data analysis was conducted using SPSS version 25.0. Descriptive statistics summarized cognitive impairment and NSS prevalence as shown in Table 2. Pearson Chi-square tests explored associations between MoCA scores (≤26 vs. >26) and specific NSS, with significance set at $p < 0.05$. The average NSS per patient was calculated by dividing the total NSS by the sample size.

Note

The Study has been reviewed and approved by the Institutional Ethics Committee. (No.IEC/2023/7335-70)

Results

Relevant Sociodemographic Details

The study comprised 60 participants with a mean age of approximately 29.5 years (range 18-45). The largest age group was 26 to 35 years (45.0%), followed by 18 to 25 (43.3%). Most were male (58.3%) and single (53.3%), with Hinduism as the predominant religion (80.0%). Educationally, half had middle or high school qualifications (50.0%). Unemployment was the most common occupation (33.3%), and the highest income bracket was ₹4110 to 8219 (33.3%). Nuclear families prevailed (45.0%), with urban residence slightly more common (51.7%). Illness duration was mostly 7 to 12 months (53.3%), family psychiatric history was absent in most cases (81.7%), and tobacco was the leading substance abuse (46.7%). The majority of participants (65%) demonstrated cognitive impairment on the Montreal Cognitive Assessment (MoCA), with scores below 26, while 35% exhibited normal cognitive function (scores ≥26).

Number of soft signs present in the patients as determined by testing on according to NES scale.

Correlations between cognitive dysfunction (MoCA ≤26) and specific NSS were analyzed using Pearson Chi-square tests, as shown in Table 3. Correlation between cognitive dysfunctions and soft signs present (Based on MoCA rating interpretation and NES)

Pearson Chi-square analyses revealed significant associations (p <0.05) between lower MoCA scores (<26, indicating cognitive impairment) and poorer performance across multiple neurological domains in a sample of 60 participants. Specifically, those with impaired MoCA scores showed higher rates of errors or disruptions in:

Sensory Integration and Perception

Audio-visual integration (p = 0.001), graphesthesia (p=0.027), right/left confusion (p = 0.001), and gaze persistence (p=0.01).

Table 3a: Cognitive dysfunctions and difficulties with sensory integration

Neurological evaluation scale tests		*MoCA score*			*Pearson Chi-Square Tests*
Test	Rating	<26	> OR = 26	Total	*p-value*
Cerebral dominance	Right	28 (62.2%)	17 (37.8%)	45 (100.0%)	0.435
	Left	11 (73.3%)	4 (26.7%)	100.0%)	
Audio-visual integration	0 = no error	3 (12.5%)	21 (87.5%)	24 (100.0%)	0.001
	1 = one error	14 (100.0%)	0 (0.0%)	14 (100.0%)	
	2 = two or more errors	22 (100.0%)	0 (0.0%)	100.0%)	
Stereognosis	0 = no errors	38 (64.4%)	21 (35.6%)	59 (100.0%)	0.459
	1 = one error	1 (100.0%)	0 (0.0%)	(100.0%)	
Graphesthesia	0 = no errors	28 (57.1%)	21 (42.9%)	49 (100.0%)	0.027
	1 = one error	5 (100.0%)	0 (0.0%)	5 (100.0%)	
	2 = more than one error	6 (100.0%)	0 (0.0%)	6 (100.0%)	
Extinction (Face-Hand Test)	0 = no errors	38 (64.4%)	21 (35.6%)	59 (100.0%)	0.459
	1 = one error	1 (100.0%)	0 (0.0%)	1 (100.0%)	
Right/left confusion	0 = no errors	13 (38.2%)	21 (61.8%)	34 (100.0%)	0.001
	1 = one error	14 (100.0%)	0 (0.0%)	14 (100.0%)	
	2 = two or more errors	12 (100.0%)	0 (0.0%)	12 (100.0%)	
Gaze impersistence	0 = no deviation from fixation	26 (55.3%)	21 (44.7%)	47 (100.0%)	0.01
	1 = deviation from fixation after 20 secs	6 (100.0%)	0 (0.0%)	6 (100.0%)	
	2 = deviation from fixation before 20 secs	7 (100.0%)	0 (0.0%)	7 (100.0%)	

Motor Coordination and Sequencing

Fist-ring test (p=0.001), fist-edge-palm test (p=0.001), Ozeretski test (p=0.001), rapid alternating movements (p = 0.001), and rhythm tapping (Parts A and B, both p=0.001).

Memory

Immediate word recall (p=0.001).

No significant differences were observed in cerebral dominance (p=0.435), stereognosis (p=0.459), or extinction/face-hand test (p=0.459). Overall, these findings suggest that cognitive impairment correlates strongly with deficits in sensory-motor integration, praxis, and memory functions.

Discussion

This study reveals a strong association between cognitive impairments and neurological soft signs (NSS) in drug-naïve schizophrenia patients. The 65% prevalence of cognitive dysfunction (MoCA ≤26) in drug-naïve schizophrenia patients is consistent with recent research highlighting cognitive deficits as a core feature of the disorder, often present at illness onset. For instance, Ugwuonye *et al.*[17] reported an 82.9% prevalence of neurocognitive deficits in first-episode schizophrenia patients using the SCIP tool. Similarly, Goonathilake *et al.* [18] found an 89.3% prevalence of cognitive impairment in schizophrenia patients. Tang *et al.* [19] demonstrated significant cognitive deficits in first-episode drug-naïve schizophrenia patients, with improvements post-treatment. Deng *et al.*[20] revealed significant cognitive impairments in first-episode drug-naïve male schizophrenia patients associated with BMI. Additionally, Khedr *et al.* [21] showed lower MoCA scores in schizophrenia patients compared to controls, underscoring the early onset of these deficits. The average of 4.6 NSS per patient and the 86.7% prevalence of at least one NSS reinforce that

Table 3B: Cognitive dysfunctions and difficulties with sequencing of complex motor tasks

Fist-Ring Test	*0 = no major disruption of motion after first repetition*	*1 (11.1%)*	*8 (88.9%)*	*9 (100.0%)*	*0.001*
	1 = no major disruption of motion after first repetition or complete breakdown of motion	9 (42.9%)	12 (57.1%)	21 (100.0%)	
	2 = major disruption of movement or complete breakdown of motion, or more than four fist ring hesitations or confusions.	29 (96.7%)	1 (3.3%)	30 (100.0%)	
Fist-Edge-Palm Test	0 = no major disruption of motion after first repetition	1 (25.0%)	3 (75.0%)	4 (100.0%)	0.001
	1 = no major disruption of motion after first repetition or complete breakdown of motion	1 (7.1%)	13 (92.9%)	14 (100.0%)	
	2 = major disruption of movement or complete breakdown of motion, or more than four hesitations or position confusions.	37 (88.1%)	5 (11.9%)	42 (100.0%)	
Ozeretski Test	0 = no major disruption of motion after first repetition	0 (0.0%)	2 (100.0%)	2 (100.0%)	0.001
	1 = no major disruption of motion after first repetition or complete breakdown of motion	1 (11.1%)	8 (88.9%)	9 (100.0%)	
	2 = major disruption of movement or complete breakdown of motion, or more than four hesitations or position confusions.	38 (77.6%)	11 (22.4%)	49 (100.0%)	

NSS are inherent to schizophrenia, independent of antipsychotic treatment effects [5].

The study highlights a strong link between cognitive impairments and neurological soft signs (NSS) in drug-naïve schizophrenia patients, suggesting shared neurobiological underpinnings rooted in disrupted brain connectivity and neurodevelopmental abnormalities, including dysfunction in frontocerebellar, frontoparietal, and corticostriatal circuits implicated in sensory integration, motor coordination, and executive function. Recent research by Petrescu et al. [22] supports these findings, reporting higher NSS prevalence in drug-naïve patients with deficits in motor coordination and sensory integration tied to prefrontal and cerebellar changes, aligning with observed correlations in tasks like the Fist-Ring and Fist-Edge-Palm Tests. Similarly, Zhang et al. [23] identified pronounced cognitive deficits in first-episode patients, particularly in processing speed and attention, linked to negative symptoms and reduced cortical thickness in frontal and parietal regions, consistent with graphesthesia impairments. Tsapakis et al. [24] further reinforce this, noting NSS as trait markers reflecting cerebellar and basal ganglia atrophy, underscoring schizophrenia's neurodevelopmental basis with NSS and cognitive impairments as endophenotypes. Dong et al. [25] confirm severe deficits in working memory and verbal learning in first-episode cases, while Petrescu et al. [26] highlight associations with negative symptoms independent of antipsychotics.

Significant differences in Rhythm Tapping and Rapid Alternating Movements (p = 0.001) underscore schizophrenia-specific motor timing deficits, likely tied to cerebellar dysfunction.[27] The absence of correlation with Extinction (p = 0.459) may indicate its lesser relevance to cognitive deficits, possibly due to distinct neural pathways like the temporoparietal junction.[28] These findings suggest that cognitive dysfunction and NSS are integral to schizophrenia, detectable before treatment. Early detection could inform tailored interventions, such as cognitive remediation or motor-focused therapies [29]. NSS may also serve as endophenotypes, aiding in risk identification and genetic research [4]. The drug-naïve focus minimizes medication-related confounds, but the cross-sectional design limits insights into impairment progression. Longitudinal studies and larger, diverse samples are needed to explore these dynamics. Cul-

Table 3C: Cognitive dysfunctions and difficulty in motor coordination

(a) Rhythm Tapping Test Part A	*0 = no errors*	*2 (10.5%)*	*17 (89.5%)*	*19 (100.0%)*	*0.001*
	I = one error of either non-discrimination between soft and hard sounds, rhythm, or error in number of taps	3 (50.0%)	3 (50.0%)	6 (100.0%)	
	2 = more than one error	34 (97.1%)	1 (2.9%)	(100.0%)	
1.(b) Rhythm Tapping Test Part B	0 = no errors	3 (14.3%)	18 (85.7%)	21 (100.0%)	0.001
	I = one error	4 (57.1%)	3 (42.9%)	7 (100.0%)	
	2 = more than one error	32 (100.0%)	0 (0.0%)	32 (100.0%)	
Rapid Alternating Movements	0 = no major disruption of motion, hesitation, or mistake in hand placement	2 (14.3%)	12 (85.7%)	14 (100.0%)	0.001
	1= no major disruption of motion or one to two hesitations or mistakes in hand placement	17 (65.4%)	9 (34.6%)	26 (100.0%)	
	2 = major disruption of motion or three or more hesitations or mistakes in hand placement.	20 (100.0%)	0 (0.0%)	20 (100.0%)	

Table 3D: Cognitive dysfunctions and difficulties with memory

Memory	*0 = Subject remembers all words*	*7 (25.9%)*	*20 (74.1%)*	*27 (100.0%)*	*0.001*
	1 = Subject remembers three words	13 (92.9%)	1 (7.1%)	14 (100.0%)	
	2 = Subject remembers fewer than three words	19 (100.0%)	0 (0.0%)	19 (100.0%)	

tural and genetic factors, such as dopamine pathway variations in Indian populations, may influence these impairments, warranting further investigation [30].

Conclusion

In conclusion, this study underscores the intrinsic connection between cognitive impairments and neurological soft signs in drug-naïve schizophrenia patients, reinforcing the neurodevelopmental origins of the disorder through shared disruptions in fronto-cerebellar and subcortical pathways. Clinically, these insights advocate for early, integrated assessments to inform personalized interventions, potentially improving patient outcomes, while highlighting the potential of neurological soft signs as biomarkers for risk stratification and monitoring. However, the study's limitations include its cross-sectional design, which precludes causal inferences; a relatively small sample size, limiting generalizability; reliance on a single cognitive screening tool, potentially overlooking domain-specific deficits; and recruitment from a single center in India, which may not capture broader sociocultural or genetic variations. Future research should prioritize longitudinal designs with larger, multicultural cohorts to track impairment trajectories and treatment impacts. There are no conflicts of interest.

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