Section: Psychiatry

Original Research Article

NEUROLOGICAL SOFT SIGNS AS PREDICTORS OF COGNITIVE DYSFUNCTION IN DRUG-NAÏVE SCHIZOPHRENIA

Amit Singh[1], *Aman Gupta*[2]

[1]*Department of Psychiatry, Owner - Neuro Mind Care Clinic, Aligarh, Uttar Pradesh, India*
[2]*Assistant Professor, Department of Psychiatry, Muzaffarnagar Medical College, Begrajpur, Muzaffarnagar, Uttar Pradesh, India*

Received *: 08/07/2026*
Received in revised form *: 15/08/2026*
Accepted *: 28/08/2026*

Corresponding Author:
Dr. Aman Gupta,
Assistant Professor, Department of Psychiatry, Muzaffarnagar Medical College, Begrajpur, Muzaffarnagar, Uttar Pradesh, India.
Email: aman111aman@gmail.com

DOI: 10.70034/ijmedph.2026.3.814

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (3); 5289-5295

ABSTRACT

Background: Cognitive dysfunction and neurological soft signs are increasingly recognized as core neurobiological features of schizophrenia. Their presence before antipsychotic exposure may reflect underlying neurodevelopmental and neural-network abnormalities. This study assessed the pattern of cognitive dysfunction and neurological soft signs in drug-naïve schizophrenia and examined their relationship with clinical and sociodemographic variables.

Materials and Methods: This analytical cross-sectional study included 129 patients with drug-naïve schizophrenia attending a tertiary-care psychiatric centre. Sociodemographic and clinical characteristics were recorded using a structured proforma. Psychopathology was assessed using the Positive and Negative Syndrome Scale, cognitive functioning using the Brief Assessment of Cognition in Schizophrenia, and neurological soft signs using the Neurological Evaluation Scale. Correlation and multiple linear regression analyses were performed to identify factors associated with cognitive performance.

Results: The participants had a mean age of 30.8 ± 7.1 years, and 78 (60.5%) were male. Clinically relevant global cognitive impairment was identified in 86 (66.7%) participants. Verbal memory was impaired in 97 (75.2%), attention and processing speed in 95 (73.6%), and working memory in 89 (69.0%). At least one neurological soft sign was present in 114 (88.4%) participants, while 49 (38.0%) had a high soft-sign burden. Cognitively impaired participants had significantly higher total Neurological Evaluation Scale scores than those with relatively preserved cognition (16.0 ± 5.7 vs 7.8 ± 6.1; $p<0.001$). Total neurological soft-sign scores showed a strong inverse correlation with the BACS composite score ($r=-0.733$; $p<0.001$). Neurological soft-sign severity was the strongest independent predictor of poorer cognition ($\beta=-0.591$; $p<0.001$), followed by negative-symptom severity and increasing age, whereas higher education predicted better cognitive performance.

Conclusion: Cognitive dysfunction and neurological soft signs were highly prevalent and strongly interrelated in drug-naïve schizophrenia. Neurological soft-sign severity independently predicted cognitive performance, suggesting that structured neurological assessment may provide an accessible clinical marker of cognitive vulnerability. Early recognition of these abnormalities may support targeted cognitive remediation and functional rehabilitation.

Keywords: Antipsychotic-naïve; cognitive dysfunction; drug-naïve schizophrenia; neurological evaluation scale; schizophrenia.

INTRODUCTION

Schizophrenia is a severe and disabling psychiatric disorder characterized by disturbances in thought, perception, affect, behaviour, and social functioning. It affects approximately 24 million people worldwide and commonly begins in late adolescence or early adulthood, resulting in substantial educational,

occupational, and social impairment.[1] Although positive and negative symptoms form the basis of clinical diagnosis, cognitive dysfunction is increasingly recognized as a core feature of the disorder.[2]

Cognitive deficits in schizophrenia commonly involve attention, working memory, processing speed, verbal and visual learning, executive functions, reasoning, and social cognition.[3] These impairments may be present before the onset of overt psychosis and often persist despite improvement in positive symptoms. Meta-analytic evidence suggests that drug-naïve patients with first-episode schizophrenia show moderate-to-severe deficits across multiple cognitive domains, particularly in verbal memory, processing speed, and working memory.[4,5] Cognitive dysfunction is also a major determinant of treatment adherence, independent living, occupational performance, interpersonal functioning, and overall quality of life.[6]

Neurological soft signs are another important neurobiological feature of schizophrenia.[7] These are subtle neurological abnormalities that cannot be attributed to a localized structural lesion and typically include impaired motor coordination, motor sequencing, sensory integration, balance, and disinhibition.[8] Neurological soft signs have been reported in nearly 50–65% of patients with schizophrenia and may be evident even during the first episode and before exposure to antipsychotic medication.[8] Their presence in unaffected first-degree relatives further supports their possible role as markers of neurodevelopmental vulnerability or candidate endophenotypes.[9,10]

Cognitive dysfunction and neurological soft signs may represent related manifestations of disrupted frontoparietal, frontocerebellar, corticostriatal, and corticothalamic networks. However, their relationship remains incompletely understood, particularly in drug-naïve patients. Studying untreated patients minimizes the confounding effects of antipsychotic medication, chronic illness, hospitalization, and extrapyramidal adverse effects. Therefore, the present study aimed to assess cognitive dysfunction and neurological soft signs in drug-naïve patients with schizophrenia and examine the association between these two clinical dimensions.

MATERIALS AND METHODS

Study Design and Setting: This analytical cross-sectional study was conducted in the Department of Psychiatry, a tertiary-care teaching hospital in North India, over a period of 2 years from January 2023 to December 2024. Patients attending the psychiatric outpatient department or admitted to the psychiatry inpatient unit were screened for eligibility. The study aimed to assess cognitive dysfunction and neurological soft signs among patients with drug-naïve schizophrenia and to examine the relationship between these two clinical dimensions.

Study Participants and Eligibility Criteria: Patients aged 18–50 years who fulfilled the diagnostic criteria for schizophrenia according to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision were included. The diagnosis was established by a qualified psychiatrist through detailed clinical evaluation and confirmed using the Structured Clinical Interview for DSM-5 Disorders–Clinician Version. Only patients who had never received antipsychotic medication were enrolled. Drug-naïve status was confirmed through interviews with the patient and accompanying family members and review of available prescriptions and medical records.

Patients were required to be clinically stable, cooperative, and capable of understanding the assessment procedures. Those with intellectual disability, dementia, delirium, epilepsy, cerebrovascular disease, Parkinsonism, central nervous system infection, significant head injury, or any other neurological disorder affecting cognition or motor performance were excluded. Patients with schizoaffective disorder, bipolar disorder, major depressive disorder with psychotic features, substance-induced psychosis, psychosis due to a medical condition, or current substance-use disorder other than nicotine or caffeine use were also excluded. Patients with severe agitation, aggression, catatonia, marked behavioural disturbance, or inability to complete the assessments were not enrolled.

Sample Size and Sampling: The sample size was calculated to detect a moderate correlation between neurological soft-sign severity and cognitive performance. Assuming an expected correlation coefficient of 0.35, a two-sided alpha error of 5%, and 80% statistical power, the minimum required sample was 112 participants. After allowing for approximately 10% incomplete assessments, the target sample was increased to 129. Eligible patients were recruited through consecutive sampling until the required sample size was achieved.

Sociodemographic and Clinical Assessment: Sociodemographic and clinical information was recorded using a predesigned case-record form. Variables included age, sex, residence, marital status, educational attainment, occupation, socioeconomic status, age at illness onset, duration of illness, duration of untreated psychosis, mode of onset, family history of psychiatric illness, and nicotine use. Duration of untreated psychosis was defined as the interval between the onset of the first persistent psychotic symptom and the date of assessment. Information was obtained from the patient, a reliable caregiver, and available clinical records.

Assessment of Psychopathology: Psychopathological severity was assessed using the Positive and Negative Syndrome Scale. The scale contains 30 items divided into positive symptoms,

negative symptoms, and general psychopathology subscales. Each item is rated from 1 to 7, with higher scores indicating greater symptom severity. Positive, negative, general psychopathology, and total scores were calculated for each participant.

Assessment of Cognitive Function: Cognitive functioning was evaluated using the Brief Assessment of Cognition in Schizophrenia. The battery assesses verbal memory, working memory, motor speed, verbal fluency, attention and processing speed, and executive functioning. It includes list learning, digit sequencing, token motor, semantic and letter fluency, symbol coding, and Tower of London tasks. Testing was conducted individually in a quiet, well-lit room using standardized instructions. Raw scores were recorded for each domain and converted into standardized scores using available age- and education-adjusted normative values. A composite cognitive score was calculated by averaging the standardized domain scores, with lower scores indicating poorer cognitive functioning.

Assessment of Neurological Soft Signs: Neurological soft signs were assessed using the Neurological Evaluation Scale. The scale evaluates sensory integration, motor coordination, sequencing of complex motor acts, and other neurological abnormalities. Tasks included tandem walking, rapid alternating movements, finger–thumb opposition, finger-to-nose testing, stereognosis, graphesthesia, right–left orientation, fist–edge–palm sequencing, and audiovisual integration. Each item was scored according to standardized instructions, and subscale and total scores were calculated. Higher scores represented greater neurological abnormality. Abnormalities attributable to pain, musculoskeletal limitation, visual impairment, or identifiable neurological disease were not classified as soft signs.

Assessment Procedure and Bias Control: All assessments were completed before initiation of antipsychotic treatment and, whenever possible, during a single visit. After diagnostic confirmation and informed consent, sociodemographic details and PANSS scores were recorded, followed by cognitive and neurological assessment. Assessors were trained in the administration of all instruments. Wherever feasible, the investigator performing the cognitive assessment was unaware of the neurological soft-sign score. Standardized instructions, consecutive recruitment, and exclusion of sedated or clinically unstable patients were used to minimize measurement and selection bias.

Ethical Considerations: Ethical approval was obtained from the Institutional Ethics Committee. Written informed consent was obtained from all participants. When decision-making capacity was impaired, assent was obtained from the patient along with consent from a legally authorized representative. Confidentiality was maintained using coded identifiers, and participation did not delay or interfere with clinical treatment.

Statistical Analysis: Data were analysed using IBM SPSS Statistics version 25.0. Categorical variables were expressed as frequency and percentage, whereas continuous variables were presented as mean ± standard deviation or median with interquartile range. Normality was assessed using the Shapiro–Wilk test. Group comparisons were performed using the independent-samples t-test, Mann–Whitney U test, one-way analysis of variance, Kruskal–Wallis test, chi-square test, or Fisher's exact test, as appropriate. Correlations between cognitive scores, neurological soft-sign scores, and PANSS scores were examined using Pearson's or Spearman's correlation. Multiple linear regression was used to identify independent predictors of cognitive performance after adjusting for age, sex, education, illness duration, duration of untreated psychosis, and symptom severity. A two-tailed p-value of less than 0.05 was considered statistically significant.

RESULTS

The mean age of the participants was 30.8 ± 7.1 years, with the largest proportion belonging to the 26–35-year age group (61, 47.3%). Males constituted 78 (60.5%) participants, while 69 (53.5%) were urban residents and 83 (64.3%) were unmarried, separated, or widowed. The mean duration of education was 11.4 ± 3.1 years, and 49 (38.0%) participants were unemployed. A family history of psychotic illness was present in 27 (20.9%), and 45 (34.9%) reported current tobacco use. The mean age at illness onset was 28.5 ± 7.2 years. The median duration of illness and duration of untreated psychosis were 9.1 (5.8–14.2) months and 6.7 (4.0–10.7) months, respectively. The mean PANSS total score was 91.9 ± 10.0 [Table 1].

Table 1: Sociodemographic and Clinical Characteristics of Participants With Drug-Naïve Schizophrenia (N=129).

Characteristic	Frequency (%)/mean ± SD/median (IQR)
Age (years)	30.8 ± 7.1
Age group	
18–25 years	29 (22.5)
26–35 years	61 (47.3)
36–45 years	39 (30.2)
Gender	
Male	78 (60.5)
Female	51 (39.5)
Residence	
Urban	69 (53.5)
Rural	60 (46.5)

Marital status	
Unmarried/separated/widowed	83 (64.3)
Married	46 (35.7)
Education (years)	11.4 ± 3.1
Educational attainment	
≤8 years	17 (13.2)
9–12 years	56 (43.4)
>12 years	56 (43.4)
Occupational status	
Employed	42 (32.6)
Unemployed	49 (38.0)
Student	18 (14.0)
Homemaker	20 (15.5)
Family history of psychotic disorder	27 (20.9)
Current tobacco use	45 (34.9)
Age at onset (years)	28.5 ± 7.2
Duration of illness (months)	9.1 (5.8–14.2)
Duration of untreated psychosis (months)	6.7 (4.0–10.7)
PANSS positive score	25.0 ± 4.5
PANSS negative score	22.0 ± 5.3
PANSS general psychopathology score	44.9 ± 7.2
PANSS total score	91.9 ± 10.0

IQR: Interquartile range; PANSS: Positive and Negative Syndrome Scale; SD: Standard deviation.

Significant cognitive deficits were observed across all BACS domains compared with the normative mean of zero (all $p<0.001$). The greatest impairments were found in verbal memory (−1.53 ± 0.87) and attention and processing speed (−1.51 ± 0.82), with clinically relevant impairment observed in 97 (75.2%) and 95 (73.6%) participants, respectively. Working-memory impairment was present in 89 (69.0%), followed by executive dysfunction in 79 (61.2%), verbal-fluency impairment in 67 (51.9%), and motor-speed impairment in 52 (40.3%). The mean BACS composite score was −1.30 ± 0.66, and overall cognitive impairment was identified in 86 (66.7%) participants [Table 2].

Table 2: Cognitive Performance on the Brief Assessment of Cognition in Schizophrenia.

BACS cognitive domain	Standardized z-score mean ± SD	Participants with z-score ≤−1 Frequency (%)	p-value†
Verbal memory	−1.53 ± 0.87	97 (75.2)	<0.001
Working memory	−1.31 ± 0.93	89 (69.0)	<0.001
Motor speed	−0.85 ± 0.94	52 (40.3)	<0.001
Verbal fluency	−1.21 ± 0.92	67 (51.9)	<0.001
Attention and processing speed	−1.51 ± 0.82	95 (73.6)	<0.001
Executive functioning	−1.22 ± 0.90	79 (61.2)	<0.001
BACS composite score	−1.30 ± 0.66	86 (66.7)	<0.001

BACS: Brief Assessment of Cognition in Schizophrenia; SD: Standard deviation. A standardized z-score ≤−1 indicated clinically relevant impairment. †The p-values were obtained using a one-sample Student's t-test comparing the mean standardized score with the normative mean of zero.

Neurological soft signs were highly prevalent, with at least one abnormal sign detected in 114 (88.4%) participants. Motor-coordination abnormalities were the most frequent, occurring in 113 (87.6%), followed by sensory-integration and complex motor-sequencing abnormalities, each present in 111 (86.0%). Other neurological signs were observed in 106 (82.2%) participants. The mean total NES score was 13.2 ± 7.0, with scores ranging from 0 to 33. Regarding overall burden, 56 (43.4%) participants had moderate neurological soft signs and 49 (38.0%) had a high burden, whereas only 15 (11.6%) had no neurological soft signs [Table 3].

Table 3: Distribution and Severity of Neurological Soft Signs According to the Neurological Evaluation Scale.

NES domain	Score (mean ± SD)	Observed score range	Participants with ≥1 abnormal sign Frequency (%)
Sensory integration	3.4 ± 2.1	0–9	111 (86.0)
Motor coordination	3.8 ± 2.6	0–12	113 (87.6)
Sequencing of complex motor acts	4.1 ± 2.5	0–9	111 (86.0)
Other neurological signs	1.9 ± 1.4	0–6	106 (82.2)
Total NES score	13.2 ± 7.0	0–33	114 (88.4)
Total NES score category			
No neurological soft signs	15 (11.6)		
Low burden, score 1–7	9 (7.0)		
Moderate burden, score 8–15	56 (43.4)		
High burden, score ≥16	49 (38.0)		

NES: Neurological Evaluation Scale; SD: Standard deviation. The total NES burden was descriptively categorized as absent, score 0; low, score 1–7; moderate, score 8–15; and high, score ≥16.

Participants with cognitive impairment were significantly older than those with relatively preserved cognition (31.9 ± 7.1 vs 28.8 ± 6.7 years; p=0.017) and had fewer years of education (10.7 ± 3.1 vs 12.9 ± 2.3 years; p<0.001). They also had higher PANSS negative scores (23.0 ± 5.3 vs 20.1 ± 5.0; p=0.004), higher total PANSS scores (93.5 ± 9.4 vs 88.9 ± 10.5; p=0.017), and markedly higher total NES scores (16.0 ± 5.7 vs 7.8 ± 6.1; p<0.001). At least one neurological soft sign was present in 85 (98.8%) cognitively impaired participants compared with 29 (67.4%) participants with relatively preserved cognition (p<0.001). Gender, family history, illness duration, duration of untreated psychosis, and PANSS positive and general psychopathology scores did not differ significantly between groups [Table 4].

Table 4: Comparison of Participants With and Without Cognitive Impairment.

Variable	Cognitive impairment (n=86)	Relatively preserved cognition (n=43)	Test statistic	p-value
	Frequency (%)/mean ± SD			
Age (years)	31.9 ± 7.1	28.8 ± 6.7	t=2.42	0.017
Male Gender	56 (65.1)	22 (51.2)	χ^2=1.79	0.181
Education (years)	10.7 ± 3.1	12.9 ± 2.3	t=4.42	<0.001
Family history of psychosis	16 (18.6)	11 (25.6)	χ^2=0.47	0.491
Illness duration (months)	9.6 (5.9–15.4)	8.5 (5.2–13.0)	U=1586.0	0.195
Duration of untreated psychosis (months)	6.9 (4.3–11.8)	6.4 (3.7–9.9)	U=1625.0	0.264
PANSS positive score	25.2 ± 4.5	24.8 ± 4.5	t=0.41	0.684
PANSS negative score	23.0 ± 5.3	20.1 ± 5.0	t=2.97	0.004
PANSS general psychopathology score	45.3 ± 6.6	43.9 ± 8.2	t=1.00	0.319
PANSS total score	93.5 ± 9.4	88.9 ± 10.5	t=2.42	0.017
Total NES score	16.0 ± 5.7	7.8 ± 6.1	t=7.43	<0.001
At least one neurological soft sign	85 (98.8)	29 (67.4)	χ^2=24.45	<0.001

Cognitive impairment was defined as a BACS composite z-score ≤−1. BACS: Brief Assessment of Cognition in Schizophrenia; IQR: Interquartile range; NES: Neurological Evaluation Scale; PANSS: Positive and Negative Syndrome Scale; SD: Standard deviation; t: Independent-samples Student's t-test; U: Mann–Whitney U test; χ^2: Chi-square test.

The total NES score demonstrated significant inverse correlations with all cognitive domains assessed using the BACS. The strongest domain-specific correlation was observed with attention and processing speed (r=−0.595, p<0.001), followed by verbal fluency (r=−0.572), working memory (r=−0.538), executive functioning (r=−0.536), verbal memory (r=−0.509), and motor speed (r=−0.493); all correlations were statistically significant at p<0.001. A strong inverse correlation was observed between the total NES score and BACS composite score (r=−0.733, p<0.001), indicating that greater neurological soft-sign severity was associated with poorer overall cognitive performance [Table 5].

Table 5: Correlation Between Neurological Soft Signs and Cognitive Performance.

Cognitive measure	Correlation with total NES score, r	p-value
Verbal memory	−0.509	<0.001
Working memory	−0.538	<0.001
Motor speed	−0.493	<0.001
Verbal fluency	−0.572	<0.001
Attention and processing speed	−0.595	<0.001
Executive functioning	−0.536	<0.001
BACS composite score	−0.733	<0.001

BACS: Brief Assessment of Cognition in Schizophrenia; NES: Neurological Evaluation Scale; r: Pearson's correlation coefficient. Negative coefficients indicate worsening cognitive performance with increasing neurological soft-sign severity.

In the multivariable linear regression analysis, the total NES score emerged as the strongest independent predictor of poorer cognitive performance (standardized β=−0.591; B=−0.056, 95% CI: −0.067 to −0.044; p<0.001). Higher educational attainment was independently associated with a better BACS composite score (β=0.254; p<0.001). Increasing age (β=−0.154; p=0.009) and greater negative-symptom severity (β=−0.169; p=0.004) were independently associated with lower cognitive scores. In contrast, PANSS positive symptoms (p=0.970) and duration of untreated psychosis (p=0.088) were not independently associated with the BACS composite score [Table 6].

Table 6: Multiple Linear Regression Analysis of Factors Associated With BACS Composite Score.

Predictor	Unstandardized coefficient, B	Standard error	Standardized β	95% CI for B	p-value
Total NES score	−0.056	0.006	−0.591	−0.067 to −0.044	<0.001
Education (years)	0.055	0.012	0.254	0.031 to 0.079	<0.001

Age (years)	−0.014	0.005	−0.154	−0.025 to −0.004	0.009
PANSS negative score	−0.021	0.007	−0.169	−0.035 to −0.007	0.004
PANSS positive score	−0.0003	0.008	−0.002	−0.017 to 0.016	0.97
Duration of untreated psychosis (months)	−0.010	0.006	−0.098	−0.022 to 0.002	0.088

B: Unstandardized regression coefficient; BACS: Brief Assessment of Cognition in Schizophrenia; β: Standardized regression coefficient; CI: Confidence interval; NES: Neurological Evaluation Scale; PANSS: Positive and Negative Syndrome Scale. Negative coefficients indicate an inverse association with cognitive performance.

DISCUSSION

The present study demonstrated a substantial burden of cognitive dysfunction and neurological soft signs (NSS) among drug-naïve patients with schizophrenia. Clinically relevant global cognitive impairment was present in 86 (66.7%) participants, while 114 (88.4%) exhibited at least one neurological soft sign. These abnormalities were observed before exposure to antipsychotic treatment, supporting the concept that cognitive impairment and NSS are intrinsic neurobiological features of schizophrenia rather than consequences of medication, chronic hospitalization, or extrapyramidal adverse effects.

The participants were predominantly young adults, with a mean age of 30.8 ± 7.1 years and mean age at illness onset of 28.5 ± 7.2 years. Males constituted 60.5%, while 64.3% were unmarried, separated, or widowed, and 38.0% were unemployed. These findings reflect the typical onset of schizophrenia during the period of educational, occupational, and interpersonal development. The median duration of untreated psychosis was 6.7 months, indicating a considerable delay in treatment initiation. However, duration of untreated psychosis was not independently associated with cognitive performance, suggesting that cognitive deficits may already be established during the early course of illness.

Significant impairment was observed across all Brief Assessment of Cognition in Schizophrenia domains. Verbal memory was the most frequently impaired domain, affecting 97 (75.2%) participants, followed by attention and processing speed in 95 (73.6%), working memory in 89 (69.0%), executive functioning in 79 (61.2%), verbal fluency in 67 (51.9%), and motor speed in 52 (40.3%). The mean composite BACS score was −1.30 ± 0.66. These findings are consistent with meta-analyses by Lee et al., and Fatouros-Bergman et al., of antipsychotic-naïve and first-episode schizophrenia, which have demonstrated moderate-to-severe deficits in verbal learning, processing speed, working memory, attention, and executive functioning.[11,12] Verbal-memory impairment may reflect abnormalities in hippocampal–prefrontal networks involved in encoding, organization, and retrieval. Reduced processing speed may result from inefficient frontoparietal connectivity and white-matter dysfunction, while working-memory and executive deficits may be associated with dorsolateral prefrontal cortical abnormalities.[11,12]

NSS were highly prevalent, with motor-coordination abnormalities observed in 113 (87.6%), sensory-integration and complex motor-sequencing abnormalities in 111 (86.0%) each, and other neurological signs in 106 (82.2%). Moreover, 43.4% of participants had a moderate NSS burden and 38.0% had a high burden. Previous studies by Gunasekaran et al., Nathani et al., and Dutta et al., have similarly reported a high prevalence of NSS in first-episode and drug-naïve schizophrenia, although estimates vary according to the instrument, scoring threshold, illness stage, and sample characteristics.[13-15] The predominance of motor-coordination and sequencing abnormalities may indicate dysfunction within cerebellar, basal ganglia, premotor, supplementary motor, and prefrontal circuits. Sensory-integration deficits may additionally reflect impaired parietal, temporal, and thalamocortical processing.[16,17]

Participants with cognitive impairment were older, had fewer years of education, higher negative-symptom scores, higher total PANSS scores, and markedly greater NSS severity than those with relatively preserved cognition. The mean NES score was 16.0 ± 5.7 in the cognitively impaired group compared with 7.8 ± 6.1 in the preserved-cognition group. Furthermore, 98.8% of cognitively impaired participants had at least one NSS, compared with 67.4% of those with relatively preserved cognition. These findings indicate considerable overlap between neurological abnormalities and cognitive dysfunction.

Total NES scores showed significant inverse correlations with all cognitive domains. The strongest domain-specific relationship was observed with attention and processing speed ($r=-0.595$), followed by verbal fluency ($r=-0.572$), working memory ($r=-0.538$), executive functioning ($r=-0.536$), verbal memory ($r=-0.509$), and motor speed ($r=-0.493$). The correlation with the BACS composite score was particularly strong ($r=-0.733$, $p<0.001$). Similar associations have been reported previously in studies by Solanki et al., and Peng et al., suggesting that NSS and cognitive deficits may represent parallel manifestations of widespread neurodevelopmental dysconnectivity.[18,19]

In multivariable analysis, total NES score remained the strongest independent predictor of cognitive performance ($\beta=-0.591$, $p<0.001$). Higher educational attainment predicted better cognition, supporting the role of cognitive reserve, whereas increasing age and greater negative-symptom severity were associated with poorer performance.

Positive symptoms and duration of untreated psychosis were not independently associated with cognition. The association with negative symptoms may reflect shared prefrontal and cerebello-thalamo-cortical dysfunction, while positive symptoms may be more closely related to acute dopaminergic and salience-processing abnormalities.[20,21]

Limitations: The study had several limitations. Its cross-sectional design prevented assessment of causality or temporal changes in cognition and neurological soft signs. Recruitment from a single tertiary-care centre may limit generalizability. The absence of a healthy control group restricted direct comparison with normative performance. Cognitive testing may also have been influenced by education, motivation, and symptom severity despite statistical adjustment. Longitudinal multicentre studies are needed to confirm these findings.

CONCLUSION

Cognitive dysfunction and neurological soft signs were highly prevalent in drug-naïve schizophrenia, with verbal memory and attention-processing speed being the most affected cognitive domains. Neurological soft-sign severity showed a strong inverse relationship with overall cognitive performance and remained its strongest independent predictor after adjustment for clinical and sociodemographic variables. Higher education was associated with better cognition, whereas older age and greater negative-symptom severity predicted poorer performance. Routine evaluation of cognitive deficits and neurological soft signs may help identify vulnerable patients early and guide individualized cognitive remediation, rehabilitation, and functional recovery strategies. Their assessment may also provide accessible markers of underlying neurodevelopmental dysfunction in routine psychiatric practice.

REFERENCES

1. Hany M, Rizvi A. Schizophrenia. Treasure Island (FL): StatPearls Publishing; 2026.
2. Zhu Y, Womer FY, Leng H, et al. The Relationship Between Cognitive Dysfunction and Symptom Dimensions Across Schizophrenia, Bipolar Disorder, and Major Depressive Disorder. Front Psychiatry. 2019;10:253.
3. Bowie CR, Harvey PD. Cognitive deficits and functional outcome in schizophrenia. Neuropsychiatr Dis Treat. 2006;2(4):531-536.
4. Zhang T, Wei Y, Tang X, et al. Cognitive Impairments in Drug-Naive Patients With First-Episode Negative Symptom-Dominant Psychosis. JAMA Netw Open. 2024;7(6):e2415110.
5. Liu H, Pan X, Huang X, et al. Do interpersonal trust and social avoidance mediate the association between psychotic symptoms and social functioning in chronic schizophrenia patients? Front Psychiatry. 2025;16:1433763.
6. Cappadona I, Pagano M, Anselmo A, et al. Cognitive and Psychosocial Recovery in Schizophrenia: Evidence from a Case Study on Integrated Rehabilitation. Psychiatry Int. 2026;7(1):6.
7. Rathod B, Kaur A, Basavanagowda DM, et al. Neurological Soft Signs and Brain Abnormalities in Schizophrenia: A Literature Review. Cureus. 2020;12(10):e11050.
8. Tsapakis EM, Mitkani CA, Fountoulakis KN. Neurological soft signs and schizophrenia. CNS Spectr. 2023;28(6):657-661.
9. Lavanya T, A Balaji A, Jayakrishnaveni. Neurological soft signs in non psychotic siblings of psychotic patients: A cross-sectional study. Int J Psychiatry Res. 2026;8(4):07-12.
10. Herold CJ, Duval CZ, Lässer MM, et al. Neurological soft signs and cognitive impairment in chronic schizophrenia. Schizophr Res Cogn. 2019;16:17–24.
11. Lee M, Cernvall M, Borg J, et al. Cognitive function and variability in antipsychotic drug-naïve patients with first-episode psychosis: a systematic review and meta-analysis. JAMA Psychiatry. 2024;81(5):468–476.
12. Fatouros-Bergman H, Cervenka S, Flyckt L, Edman G, Farde L. Meta-analysis of cognitive performance in drug-naïve patients with schizophrenia. Schizophr Res. 2014;158(1–3):156–162.
13. Gunasekaran V, Venkatesh VMK, Asokan TV. A study of soft neurological signs and its correlates in drug-naïve patients with first-episode psychosis. Indian J Psychol Med. 2016;38(5):408–413.
14. Nathani YL, Faye A, Kirpekar V, et al. A study of neurological soft signs and cognition in schizophrenia. Cureus. 2023;15(12):e50925.
15. Dutta M, Nath K, Baruah A, Naskar S. A clinical study of neurological soft signs in patients with schizophrenia. J Neurosci Rural Pract. 2016;7(3):393–399.
16. Chan RCK, Xu T, Heinrichs RW, Yu Y, Wang Y. Neurological soft signs in schizophrenia: a meta-analysis. Schizophr Bull. 2010;36(6):1089–1104.
17. Samson GD, Lahti AC, Kraguljac NV. The neural substrates of neurological soft signs in schizophrenia: a systematic review. Schizophrenia (Heidelb). 2022;8(1):42.
18. Solanki RK, Swami MK, Singh P. An examination of relationship between neurological soft signs and neurocognition. Asian J Psychiatr. 2012;5(1):43–47.
19. Peng XJ, Hei GR, Yang Y, et al. The association between cognitive deficits and clinical characteristics in first-episode drug-naïve patients with schizophrenia. Front Psychiatry. 2021;12:638773.
20. Chan RCK, Geng FL, Lui SSY, et al. Course of neurological soft signs in first-episode schizophrenia: relationship with negative symptoms and cognitive performances. Sci Rep. 2015;5:11053.
21. Rodriguez M, Knížková K, Keřková B, et al. The relationships between cognitive reserve, cognitive functioning and quality of life in first-episode schizophrenia spectrum disorders. Psychiatry Res. 2022;310:114479.