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ORIGINAL RESEARCH

Impact of cannabis use in patients with schizophrenia- a cross sectional comparative study

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Background: Cannabis has become one of the key substances attracting increasing attention in recent times, particularly for its association with psychotic disorders. Neurological soft signs (NSS) are subtle motor and sensory integration abnormalities observed in schizophrenia, reflecting underlying neurodevelopmental dysfunction. Cannabis use is increasingly prevalent in schizophrenia and may influence the clinical and neurobiological phenotype, including NSS.

Aim: To compare NSS and psychopathology in schizophrenia patients with and without cannabis use.

Materials and methods: A comparative cross-sectional study was conducted among 60 male patients diagnosed with schizophrenia (DSM-5 criteria), divided equally into two groups: those with (n = 30) and without (n = 30) cannabis use. Cannabis use of more than 2 years' duration was confirmed using the Cannabis Use Disorder Identification Test–Revised (CUDIT-R) and the Severity of Dependence Scale (SDS). The Neurological Evaluation Scale (NES), Scale for the Assessment of Positive Symptoms (SAPS), Scale for the Assessment of Negative Symptoms (SANS), and Brief Psychiatric Rating Scale (BPRS) were administered. Statistical analysis was performed using SPSS v26.0.

Results: Cannabis users showed significantly lower scores in the NES domain "sequencing of complex motor acts" ($p = 0.042$), suggesting better motor coordination. No significant differences were observed in total NSS or other subdomains. Cannabis users had significantly higher SAPS ($p = 0.036$) and BPRS scores ($p = 0.014$) and lower SANS scores ($p = 0.010$), indicating greater positive symptoms, overall psychopathology, and lesser negative symptoms. Among users, the mean CUDIT-R score was 6.26 ± 7.32, and the SDS score was 3.16 ± 3.52. Half of the users initiated cannabis between ages 16 and 20, and 43.4% used it weekly.

Conclusion: Schizophrenia patients with cannabis use exhibited milder deficits in certain NSS domains and a higher positive symptom burden. These findings suggest a distinct clinical profile and possible neurodevelopmental variation, underlining the importance of screening for cannabis use.

Keywords: schizophrenia, cannabis, neurological soft signs, psychopathology, substance use

Introduction

Schizophrenia is a chronic, debilitating psychiatric disorder that significantly impairs cognition, behavior, and functioning (1). It presents with a wide array of symptoms, including hallucinations, delusions, disorganized speech and behavior, and negative symptoms. The etiology of schizophrenia is multifactorial, with genetic, neurobiological, and environmental factors contributing to its onset and progression (2). While clinical features have been extensively studied, recent focus has shifted toward more subtle neurological abnormalities known as neurological soft signs (NSS), which may precede or coexist with the illness.

Neurological soft signs (NSS) are minor, non-localizing neurological abnormalities without evidence of structural

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lesions. They manifest as impairments in sensory integration, motor coordination, and sequencing of complex motor acts (3). Prior research has identified NSS as potential biological markers, or endophenotypes, of schizophrenia, supported by findings of their presence in unaffected first-degree relatives and in prodromal phases of illness (4, 5).

Substance use, particularly cannabis, is another important factor in the etiopathogenesis of schizophrenia. Epidemiological studies have consistently shown that cannabis use, especially during adolescence, increases vulnerability to psychosis and schizophrenia (6, 7). Moreover, cannabis use has been associated with earlier onset of schizophrenia (8, 9).

Despite evidence linking both NSS and cannabis use independently to schizophrenia, their interaction remains insufficiently explored. Cannabis users with schizophrenia may exhibit a distinct neurobiological profile, with some studies suggesting fewer cognitive deficits but more prominent NSS, possibly due to the neurodevelopmental impact of cannabis exposure (10, 11). Conversely, some findings indicate that cannabis may preserve certain cognitive functions, complicating its relationship with NSS (12). Kuepper et al. (12) reported higher NSS in schizophrenia patients who used cannabis, while Murray et al. (13) found that cannabis use could exacerbate NSS-related cognitive impairments. These findings suggest a complex interplay between cannabis use and neurodevelopmental abnormalities in schizophrenia that warrants further investigation.

In this context, the present study aims to compare NSS and psychopathology in schizophrenia patients with and without cannabis use. This exploration is expected to contribute to a better understanding of the differential neurodevelopmental trajectories and clinical presentations in schizophrenia influenced by cannabis use.

Materials and methods

This hospital-based comparative cross-sectional study was conducted at a tertiary mental health care institution in South India among 60 patients diagnosed with schizophrenia as per DSM-5 criteria. Participants were recruited from inpatient and outpatient services using consecutive sampling and divided into Group A (with cannabis use) and Group B (without cannabis use). Inclusion criteria for both groups included age between 18 and 60 years, a DSM-5 diagnosis of schizophrenia, and willingness to participate, while additional criteria included cannabis use for more than 2 years for Group A and illness duration greater than 2 years for Group B. Patients with current intoxication, dependence on substances other than nicotine, or a history of neurological illness or significant head injury were excluded. Data were collected using a semi-structured proforma for sociodemographic, clinical, and substance use details; NSS were assessed using the Neurological Evaluation Scale (NES), while symptom severity was evaluated using the Brief Psychiatric Rating Scale (BPRS), the Scale for the Assessment of Positive Symptoms (SAPS), and the Scale for the Assessment of Negative Symptoms (SANS). Cannabis-related problems and dependence were assessed in the cannabis-using group using the Cannabis Use Disorder Identification Test–Revised (CUDIT-R) and the Severity of Dependence Scale (SDS). All instruments used had established validity and reliability. Following written informed consent, participants underwent detailed clinical interviews, and cannabis use history was corroborated by patient and informant reports. The study was approved by the Institutional Ethics Committee and Scientific Committee. Data were analyzed using IBM SPSS Statistics version 26, employing appropriate descriptive and inferential statistical methods.

TABLE 1 | Socio-demographic characteristics of the study participants (N = 60).

Variable	Category	Frequency	Percentage (%)
Age	≤25 years	11	18.3
	26–35 years	29	48.3
	36–45 years	13	21.7
	>45 years	7	11.7
Gender	Male	60	100.0
	Female	0	0.0
Educational Status	Primary school	3	5.0
	Middle school	19	31.6
	High school	15	25.0
	Higher secondary/Diploma	13	21.7
	Graduate/Postgraduate	10	16.7
Occupation	Unemployed	41	68.3
	Unskilled worker	12	20.0
	Others	7	11.7
Marital Status	Married	12	20.0
	Unmarried	48	80.0
Religion	Hindu	46	76.6
	Muslim	7	11.7
	Christian	7	11.7
Habitat	Urban	19	31.7
	Rural	41	68.3
Social Support	Weak	4	6.7
	Moderate	26	43.3
	Strong	30	50.0
Living Arrangement	With family	57	95.0
	Alone	3	5.0

TABLE 2 | Clinical and cannabis use characteristics of the study participants (N = 60).

Variable	Category	Frequency	Percentage (%)
Age of Onset of Schizophrenia	≤20 years	24	40.0
	21–25 years	18	30.0
	26–30 years	13	21.7
	>30 years	5	8.3
History of Antipsychotic Treatment	Atypical	51	85.0
	Typical	2	3.3
	Both Typical and Atypical	7	11.7
Duration of Illness	≤3 years	10	16.7
	4–6 years	16	26.7
	7–9 years	12	20.0
	≥10 years	22	36.6
Family History of Schizophrenia	Positive	22	36.7
	Negative	38	63.3
History of Tobacco Use	Yes	34	56.7
	No	26	43.3
Age of Onset of Cannabis Use (*n = 30*)	≤15 years	6	20.0
	16–20 years	15	50.0
	>20 years	9	30.0
Duration of Cannabis Use (*n = 30*)	≤5 years	13	43.3
	6–10 years	11	36.7
	>10 years	6	20.0
Frequency of Cannabis Use (*n = 30*)	Weekly	13	43.4
	Monthly	10	33.3
	Occasionally	7	23.3

Results

Among the participants, nearly half were aged between 26 and 35 years, and all 60 participants were male. Most participants had below higher secondary education, comprising those educated up to middle school or high school, while about two-fifths had higher secondary education or above. About two-thirds of were unemployed, and socioeconomic distribution showed that 63.3% belonged to the lower or lower-middle class. Rural residents comprised two-thirds of the study participants (**Table 1**).

Age of onset of schizophrenia was ≤20 years in 40%, and illness duration was less than 10 years in nearly two-thirds of participants, while just over one-third had a duration of 10 years or more. A positive family history of schizophrenia was reported by 36.7% of participants. Tobacco use was present in nearly half of the study population. Among cannabis users (n = 30), 50% initiated use at ages 16–20 years, 20% at ≤15 years, and 30% after 20 years. The duration of cannabis use was 10 years or less in about four-fifths of users, while one-fifth had more than 10 years of use; in terms of frequency, nearly half reported weekly use (**Table 2**).

Mean scores of assessment tools were NES: 21.46 ± 5.69, Brief BPRS: 41.93 ± 6.29, SAPS: 35.85 ± 16.00, SANS: 34.46 ± 21.48, CUDIT-R: 6.26 ± 7.32, and SDS: 3.16 ± 3.52. A chi-square test assessing the association between family history of schizophrenia and cannabis use revealed no statistically significant difference between the groups ($\chi = 0.287$, $p = 0.592$). Cannabis users were significantly younger than non-users (mean age = 29.33 ± 6.45 vs. 36.46 ± 9.60; $Z = -3.042$, $p = 0.002$). Duration of illness was also significantly shorter in users (6.86 ± 4.83 years) compared to non-users (11.40 ± 7.15 years; $p = 0.011$). However, no significant differences were observed in age at onset of schizophrenia (22.40 vs. 25.06 years; $p = 0.304$) or mean antipsychotic dosage (411.93 mg vs. 453.00 mg; $p = 0.355$).

Total NES scores were lower among cannabis users (mean = 20.36 ± 4.36) than non-users (22.56 ± 6.66), but the difference was not statistically significant ($p = 0.205$). Similarly, no significant differences were found in subdomains of sensory integration (2.01 vs. 2.80; $p = 0.157$) or motor coordination (2.50 vs. 2.16; $p = 0.445$). However, the sequencing of complex motor acts subscale showed significantly lower scores in cannabis users (5.03 ± 2.45) compared to non-users (6.73 ± 2.31; $p = 0.005$), indicating a specific difference in this domain. Total SANS scores were significantly lower in cannabis users (26.83 ± 20.29) than non-users (42.10 ± 20.16; $Z = -3.150$, $p < 0.001$). Affective flattening/blunting was significantly lower in users (6.13 ± 9.71) than non-users (15.30 ± 10.89; $p < 0.001$). Alogia scores were also significantly lower in users (2.56 ± 5.17) compared to non-users (5.80 ± 5.16; $p = 0.005$). No significant differences were observed in avolition-apathy (8.96 vs. 10.70; $p = 0.108$), anhedonia-asociality (8.00 vs. 9.96; $p = 0.106$), or attentional impairment (1.36 vs. 1.70; $p = 0.836$). Total SAPS scores were significantly higher among cannabis users (42.40 ± 15.01) than non-users (29.30 ± 14.38; $Z = 3.061$, $p = 0.002$). Delusions (13.80 vs. 10.03; $p = 0.031$), bizarre behavior (8.90 vs. 5.33; $p = 0.002$), and positive formal thought disorder (7.63 vs. 4.16; $p = 0.012$) were significantly higher among users. Hallucination scores (12.06 vs. 9.10) did not show statistical significance ($p = 0.180$). BPRS total scores were significantly higher in cannabis users (43.86 ± 6.10) compared to non-users (40.00 ± 5.95; $Z = 2.673$, $p = 0.007$), indicating greater psychiatric symptom severity in the user group (**Table 3**).

Discussion

The majority of participants in this study were aged 26–35 years, aligning with global data showing that schizophrenia

TABLE 3 | Association of cannabis use with clinical and demographic variables.

Variable	Cannabis users (n = 30) mean ± SD or n (%)	Cannabis non-users (n = 30) mean ± SD or n (%)	Test used/Z-value	p-value
Family History of Schizophrenia	12 (40%) Positive 18 (60%) Negative	10 (33.3%) Positive 20 (66.7%) Negative	Chi-square = 0.287	0.592
Age (years)	29.33 ± 6.45	36.46 ± 9.60	Z = −3.042	0.002*
Age of Onset of Schizophrenia	22.40 ± 3.54	25.06 ± 7.70	Z = −1.027	0.304
Duration of Illness (years)	6.86 ± 4.56	11.40 ± 8.05	Z = −2.515	0.011*
Neuroleptic Dosage (mg/day)	411.93 ± 340.13	453.00 ± 366.01	Z = −0.923	0.355
NES – Total Score	20.36 ± 4.36	22.56 ± 6.66	Z = −1.267	0.205
Sensory Integration	2.01 ± 1.05	2.80 ± 1.71	Z = −1.415	0.157
Motor Coordination	2.50 ± 1.52	2.16 ± 1.53	Z = 0.763	0.445
Sequencing of Complex Motor Acts	5.03 ± 2.45	6.73 ± 2.31	Z = −2.769	0.005*
SANS – Total Score	26.83 ± 20.29	42.10 ± 20.16	Z = −3.150	<0.001*
Affective Flattening/Blunting	6.13 ± 9.71	15.30 ± 10.89	Z = −3.833	<0.001*
Alogia	2.56 ± 5.17	5.80 ± 5.16	Z = −2.756	0.005*
Avolition – Apathy	8.96 ± 2.97	10.70 ± 3.72	Z = −1.606	0.108
Anhedonia – Asociality	8.00 ± 4.94	9.96 ± 4.90	Z = −1.613	0.106
Attentional Impairment	1.36 ± 2.18	1.70 ± 4.58	Z = 0.206	0.836
SAPS – Total Score	42.40 ± 15.01	29.30 ± 14.38	Z = 3.061	0.002*
Hallucinations	12.06 ± 5.10	9.10 ± 7.88	Z = 1.339	0.180
Delusions	13.80 ± 5.62	10.03 ± 6.14	Z = 2.152	0.031*
Bizarre Behavior	8.90 ± 4.43	5.33 ± 3.77	Z = 3.018	0.002*
Positive Formal Thought Disorder	7.63 ± 5.71	4.16 ± 5.55	Z = 2.488	0.012*
BPRS – Total Score	43.86 ± 6.10	40.00 ± 5.95	Z = 2.673	0.007*

*$p < 0.05$, statistically significant

often emerges in early adulthood. This is consistent with large meta-analyses indicating that the median age of onset is around 24 years for males and 28 years for females, reflecting a vulnerable neurodevelopmental period for psychosis onset (14–16). Although the all-male sample limits generalizability, it is in line with findings that men are diagnosed more frequently, exhibit earlier onset, and display more severe positive symptoms (17, 18). Gender differences in symptom expression and substance use patterns have been highlighted in recent studies, emphasizing the need for inclusive research (19).

Half of the sample reported cannabis use, which was significantly associated with younger age, earlier illness onset, shorter illness duration, and greater severity of positive symptoms. This supports existing evidence that cannabis use is a risk factor for earlier and more severe psychotic presentations (20–22). It also echoes findings that cannabis may precipitate psychotic relapse and worsen clinical outcomes in patients with schizophrenia (23). The observed mean CUDIT-R and SDS scores reflected a broad spectrum from recreational to problematic use.

Interestingly, no significant association was found between cannabis use and family history of schizophrenia in this sample, although a numerically higher proportion of users reported a positive history. While some studies suggest cannabis use interacts with genetic vulnerability to increase risk (24), others propose that this relationship is complex and may not be apparent in cross-sectional samples due to sample size or unmeasured genetic factors such as polygenic risk scores (25).

Cannabis users were significantly younger than non-users and also had an earlier age at illness onset. This is consistent with research showing that cannabis is more prevalent among younger individuals and those from socioeconomically disadvantaged backgrounds (9, 26). The shorter illness duration among users may reflect earlier cannabis exposure or accelerated onset of schizophrenia following use.

Cannabis users had lower total NES scores, although not significantly so. However, they performed better in the "sequencing of complex motor acts" domain, which may reflect preserved or enhanced executive functioning in certain cannabis-using individuals. Prior studies suggest that cannabis may have differential effects on cerebellar and prefrontal systems that mediate complex motor and cognitive tasks (27, 28).

Negative symptoms, as assessed by SANS, were significantly less severe in cannabis users. This paradoxical finding supports earlier reports that cannabis may transiently reduce negative symptoms such as anhedonia or affective flattening, possibly due to its dopaminergic effects (29, 30). However, this potential benefit is likely short-term,

with chronic use often resulting in long-term cognitive and motivational impairments.

Conversely, cannabis use was associated with significantly higher SAPS scores, particularly in delusions, bizarre behavior, and formal thought disorder. These results corroborate a growing body of evidence linking cannabis to exacerbation of positive symptoms via dopaminergic sensitization mechanisms (31, 32). Similarly, BPRS scores were also higher among cannabis users, indicating greater overall psychiatric symptom burden. Recent studies have associated cannabis use with heightened severity of not only psychotic features but also affective and behavioral dysregulation (33, 34). Taken together, these findings underscore cannabis as a potential aggravator of schizophrenia symptoms, particularly in the domains of positive symptoms and general psychopathology.

Conclusion

This study highlights the impact of cannabis use on schizophrenia in the Indian context, suggesting that substance exposure may contribute to clinical and neurodevelopmental heterogeneity. Cannabis use was associated with more severe positive symptoms but fewer negative symptoms and relatively better performance on certain motor sequencing tasks, indicating complex neurobiological interactions. These findings raise the possibility that cannabis-associated schizophrenia may represent a distinct clinical subtype or lie along a continuum shaped by environmental influences.

Limitations

This study included only male participants, limiting generalizability across genders, and was conducted at a single center with a demographically skewed sample, potentially affecting external validity. Although the sample size was statistically adequate, a larger and more diverse cohort would enhance generalizability. The cross-sectional design restricts causal inference, and reliance on self-reported cannabis use introduces the risk of recall and social desirability biases. Additionally, absence of biological verification [e.g., tetrahydrocannabinol (THC) assays] limits the objectivity of substance use data.

Future directions

Future research should include female participants to explore gender-specific associations and adopt longitudinal designs to clarify causal links and the trajectory of NSS. Incorporating biological confirmation of cannabis use and quantifying potency across different consumption methods will improve reliability. Neuroimaging studies could further elucidate the structural and functional brain changes associated with cannabis use in schizophrenia.

Data availability statement

Data available on reasonable request from the corresponding author.

Ethics statement

Approved by Institutional Ethics Committee. Written informed consent was obtained from all participants.

Competing interests

The authors declare no competing interests.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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