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Scientific publishing in the era of artificial intelligence

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In recent years, artificial intelligence (AI) and large language models have rapidly become integrated into many stages of scientific production. AI can be used in various phases of the publication process, including language editing, summarization, literature searches, draft generation, and the creation of visual content. (1). However, each of these applications carries distinct ethical risks. While language-level editorial assistance may be considered relatively low risk, the use of AI in content generation, methodological contribution, or data interpretation raises far more complex concerns. At this point, excluding these technologies entirely from academic publishing is neither realistic nor practical. The core debate is no longer whether AI should be used, but rather how it should be used, for what purposes, and within which ethical boundaries.

In a survey of more than 2,000 medical researchers across 95 countries, 44.5% reported using AI-based tools in their research processes (1). In another survey of over 2,300 researchers from diverse disciplines, regions, and career stages, this rate reached 76% (2). These findings indicate that AI use in academic production is increasingly widespread. In a global survey of approximately 5,000 researchers, most participants considered language-related uses such as language editing and translation ethically acceptable, yet expressed greater caution when AI was used to draft an initial version of a manuscript (3). Although more than 90% regarded AI-assisted editing and translation as ethically permissible, only 28% reported using AI for manuscript editing and 8% for translation (3). The discrepancy between ethical acceptance and self-reported use raises the possibility that AI use may be more widespread than openly declared.

AI-assisted language editing is widely accepted (4). However, while some journals require explicit disclosure of such use, others do not. The boundary between minor linguistic refinement and substantive content modification is often unclear, complicating transparency requirements. Transparency remains one of the most contested areas of AI integration. In psychiatry and mental health journals, only 39% have an official AI policy, with this proportion rising to 56% among Q1 journals and falling to 20% among Q4 journals (4). Furthermore, there is no consensus regarding where AI use should be disclosed within a manuscript (e.g., Methods section or Acknowledgements). Although readers arguably have the right to know whether AI was involved, in an environment where reliable AI-detection remains limited, mandatory disclosure may paradoxically increase the risk of incomplete or inaccurate reporting.

Editorial processes also face significant challenges. At present, reliably detecting AI-generated text appears difficult. Experimental studies show that AI-generated scientific abstracts cannot be perfectly distinguished by human reviewers or automated tools. Gao et al. reported that abstracts generated by a generative AI system were correctly identified by human reviewers only 68% of the time, while AI detection tools, despite relatively high discriminatory performance, were not error-free (5). An experimental study focusing on psychiatric publications similarly demonstrated that both free and paid detection tools produced substantial rates of false positives and false negatives (6). Pratama et al. further noted serious limitations regarding accuracy and bias, particularly the disproportionate flagging of texts written by non-native English

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authors as AI-generated (7). Moreover, current detection tools are unable to determine not only whether AI was used but also the purpose or extent of that use. Strict detection-based policies may therefore risk producing unequal and potentially unjust outcomes.

Beyond issues of detection and transparency, the growing use of AI introduces new risks to scientific reliability. Large language models are known to generate inaccurate or fabricated information. This increases the burden of reference verification and places additional responsibility on editors and reviewers. AI-generated manuscripts may include fabricated data or non-existent results, and distinguishing real from fabricated data is not always straightforward (8). In one study, an AI model was instructed to generate a rheumatoid arthritis study purportedly based on a licensed and restricted-access health database from 2012–2020. Although the model had no direct access to proprietary databases and its training data were limited to content up to 2019, it produced a manuscript containing detailed descriptions as if 2020 data had been analyzed. This example illustrates how AI can present inaccessible data and results as though they were genuine, posing serious risks to scientific publishing (8).

These uncertainties and risks make it increasingly important to clarify how AI should be positioned within academic publishing. An emerging consensus in the scientific community is that AI cannot qualify as an author (9). Authorship requires critical thinking, scientific judgment, ethical accountability, and responsibility, all of which are uniquely human capacities. AI systems lack legal personas and cannot be held accountable for the content they generate. When inaccuracies, bias, insufficient citation, or flawed interpretation occur, responsibility rests solely with human authors (9). Accordingly, AI should be conceptualized not as an author but as a tool that supports human researchers.

Can AI generate hypotheses or interpret findings in a genuinely scientific sense? Large language models learn statistical associations between words and concepts from vast text corpora. Although their outputs may appear coherent, they are based on pattern recognition rather than conscious reasoning. (9). Experimental work suggests that while AI performs well on narrow, well-defined tasks, it remains limited in areas requiring genuine scientific reasoning, such as hypothesis generation and revising interpretations in light of new evidence. Experiments with a large language model-based generative AI system have shown that the model can provide plausible interpretations but struggles to generate alternative explanations and flexibly update its hypotheses (10). The distinction between producing an interpretation and critically re-evaluating it underscores a fundamental difference between current AI systems and human researchers. To address these limitations, neuro-symbolic approaches seek to integrate current large language models—primarily driven by data-based pattern learning—with symbolic components that more closely resemble human cognition, such as logical rules, causal relationships, and explicit reasoning processes (11). The aim is to move beyond responses grounded solely in statistical similarity and to develop AI systems capable of rule-based inference and more consistent adaptation to novel situations, thereby enhancing reliability and flexibility.

Several organizations, including the Committee on Publication Ethics (COPE), the International Committee of Medical Journal Editors (ICMJE), and the World Association of Medical Editors (WAME), have issued guidance on the use of AI in scholarly publishing (12–14). In this context, Perkins and Roe examined the policy documents of more than 100 publishers to evaluate their approaches to artificial intelligence. Their study identified several recurring themes, including the restriction of authorship to humans, the attribution of ultimate responsibility to authors, the requirement for transparent disclosure of AI use, and the adaptable nature of these policies. (15). However, substantial inconsistencies persist across journals and publishers concerning the scope and manner of disclosure. The CANGARU initiative, involving major publishers such as Elsevier, Springer Nature, Wiley, and COPE, aims a consensus-based framework for AI standards in research and publishing (16). Given the variability in existing guidelines, a widely accepted, standardized AI framework would

provide valuable guidance for researchers, editors, and reviewers.

In conclusion, the debate surrounding artificial intelligence is not merely a methodological issue, but also a matter of responsibility in knowledge production. Science is not simply the generation of plausible sentences; it requires accountability, the willingness to revise claims in light of evidence, and the capacity to justify interpretations. AI is not a person capable of bearing such responsibility. Therefore, AI should not be positioned as a substitute for human reasoning, but rather, under the guidance of critical judgment, as a careful assistant in the process of knowledge production. For this reason, publication policies must establish clear, applicable, and enforceable frameworks.

Could you truly determine whether the text you have just read was written by a human or by artificial intelligence? Perhaps the more important question is not whether we can tell the difference, but how we preserve scientific responsibility in a world where that distinction becomes increasingly blurred.

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Clinical reflections of sensation seeking in antisocial personality disorder

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SUMMARY

Objective: Sensation seeking is defined as the pursuit of varied, novel, complex, and intense experiences, accompanied by a willingness to take physical, social, legal, and financial risks to attain such experiences. The present study aims to examine the relationship between sensation seeking behavior and both the dimensions of psychopathology and clinical features in individuals diagnosed with Antisocial Personality Disorder (ASPD).

Method: The study included 60 individuals diagnosed with ASPD according to DSM-5 criteria and 60 healthy controls. The level of sensation seeking was evaluated using the Zuckerman Sensation Seeking Scale (SSS). Clinical Reflections were assessed using the Novelty Seeking subscale of the Temperament and Character Inventory (TCI-NS), the Addiction Profile Index (API) and its Clinical Form (API-C), and the Criminal Thinking Scale (CTS). Data were statistically analyzed using appropriate methods.

Results: The ASPD group showed significantly higher scores than the control group in the SSS subdimensions of disinhibition, experience seeking, boredom susceptibility, and total score. Additionally, in the ASPD group, the total SSS score was positively correlated with the total TCI-NS (novelty seeking) score, the total API (addiction severity) score, the API-C novelty seeking subscale score, and the entitlement and power orientation subscales of the CTS. Furthermore, the boredom susceptibility subscale of the SSS showed a positive correlation with the number of suicide attempts.

Discussion: In individuals with antisocial personality disorder, sensation seeking behavior is positively associated with the number of suicide attempts, novelty seeking, severity of addiction, and criminal thinking patterns. These findings highlight the importance of considering sensation seeking in the clinical assessment of ASPD.

Key Words: Antisocial personality disorder, Sensation seeking, Novelty seeking, Substance use, Criminal thinking

INTRODUCTION

Sensation seeking is a multidimensional personality trait defined by an individual's willingness to engage in novel, complex, and intense experiences, along with a tendency to take physical, social, or legal risks to obtain such experiences (1). Although the term is often confused with the concept of novelty seeking, the two differ both theoretically and clinically. For example, in Cloninger's theory, novelty seeking emphasizes cognitive curiosity and behavioral responsiveness to novel stimuli, whereas sensation seeking reflects a biologically driven need for arousal (2). While McCourt et al. acknowledged a relationship between the two concepts, they demonstrated that sensation seeking and novelty seeking represent independent motivational systems and may have distinct implications in psychopathological contexts (3).

The behavioral spectrum of sensation seeking is quite broad and includes not only adaptive or neutral expressions but also maladaptive forms (4). In some individuals, sensation seeking may contribute to adaptive outcomes such as athletic success or leadership; however, numerous studies—particularly in the domain of externalizing psychopathologies—have shown that this trait is associated with risky and unhealthy behaviors. High levels of sensation seeking have been linked to various problematic behaviors, including uncontrolled substance use, impulsive aggression, reckless driving, gambling, self-injury, and criminal acts (5,6).

Antisocial Personality Disorder (ASPD) is a severe personality disorder characterized by pervasive dis-

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regard for the rights of others, failure to conform to social norms, impulsivity, and aggression (7). Individuals diagnosed with ASPD exhibit significantly higher levels of impulsivity and risk-taking compared to the general population, suggesting that sensation seeking traits may be particularly elevated in this clinical group (8). In fact, ASPD may be considered a clinical phenotype in which sensation seeking is intensely manifested. Previous studies have shown that individuals with ASPD exhibit higher overall levels of sensation seeking compared to healthy controls, and that this may be associated with disinhibition, difficulties in emotional regulation, and the presence of blame-oriented cognitive schemas (9-11). However, these behavioral tendencies have often been examined in different samples and as independent variables, with limited focus on their co-occurrence or interactions within the same population. For example, studies have typically focused on either substance use or aggressive behaviors, while neglecting a comprehensive assessment of other maladaptive traits that may be associated with sensation seeking (12-15).

This study aims to fill these gaps by comprehensively examining sensation seeking tendencies in individuals diagnosed with ASPD. We tested the hypothesis that individuals with ASPD would demonstrate significantly higher total sensation seeking scores, as well as elevated scores in specific subdimensions, compared to a healthy control group. Additionally, we hypothesized that these sensation seeking tendencies would be associated with clinical indicators such as increased severity of substance dependence, suicide attempts, self-harming behaviors, and criminal cognitive schemas. By integrating theoretical perspectives and multidimensional assessment tools, we aim to provide novel and clinically meaningful insights into whether sensation seeking may serve as a transdiagnostic determinant specific to externalizing disorders within the context of ASPD.

METHODS

Selection and Description of Participants

This study was conducted using a cross-sectional and comparative clinical research design, comparing patients diagnosed with ASPD to healthy controls. The research was carried out at the Department of Psychiatry, Gulhane Training and Research Hospital, between January 2022 and April 2022. Before the study began, approval was obtained from the Scientific Research Ethics Committee of the University of Health Sciences (Decision No: 2021/414, dated 16.12.2021). Written informed consent was obtained from all participants, and the study was conducted in accordance with the Declaration of Helsinki and Good Clinical Practice (GCP) guidelines.

The sample comprised 60 adult male patients diagnosed with ASPD, based on DSM-5 criteria, and 60 age- and sex-matched healthy male individuals. The ASPD group was established through simple random sampling from individuals presenting to the psychiatry outpatient clinic. Of 85 potential candidates selected using random number tables, 14 declined participation and 11 were excluded due to insufficient data on substance use or suicidal history, resulting in a final ASPD group of 60 male participants. Diagnosis confirmation for all ASPD candidates involved the administration of the Structured Clinical Interview for DSM-5 Personality Disorders (SCID-5-PD) (16, 17). These face-to-face interviews were conducted by two psychiatrists (Ö.U. and Y.S.), each possessing a minimum of three years of diagnostic experience. The control group was randomly selected from hospital staff and patient companions, with their current and past absence of psychiatric diagnoses verified through structured interviews.

Data Collection and Measurements

The Sociodemographic and Clinical Information Form, developed by the research team, was used to assess participants' basic demographic and clinical characteristics, including age, education level, employment status, and marital status. The form also included questions regarding tobacco, alcohol, and illicit substance use, past suicide attempts, self-harming behaviors, and criminal record. Substance use was defined as problematic consumption of alcohol, drugs, or prescription medications within the past 12 months. Detailed information was col-

lected through semi-structured interviews, and data was corroborated, where possible, through medical records and family reports.

Structured Clinical Interview for DSM-5 Personality Disorders (SCID-5-PD): This semi-structured interview, designed to evaluate personality disorders based on DSM-5 criteria, was employed to confirm ASPD diagnoses. Experienced clinicians conducted face-to-face interviews to ensure standardized assessment of diagnostic criteria.

Zuckerman Sensation Seeking Scale (SSS): The fifth version of the SSS, developed by Zuckerman, measured sensation-seeking levels (18, 19). This 40-item scale, supported by extensive validity and reliability data, comprises four subscales assessing distinct arousal tendencies: The thrill and adventure seeking subscale reflects the desire to engage in physically risky activities and have unusual experiences. The experience seeking subscale includes preferences for an unconventional lifestyle, interest in various forms of art and music, and a desire for non-monotonous experiences. The disinhibition subscale refers to the tendency to disregard social and moral restrictions, act impulsively, and engage in socially inappropriate behavior, particularly under the influence of substances. This subscale is also referred to as loss of conditioned inhibition. The boredom susceptibility subscale is characterized by intolerance to monotony, becoming easily bored with repetitive routines, and constantly seeking new stimuli. Higher scores on each subscale indicate a stronger presence of the relevant trait. The Turkish adaptation by Öngen demonstrated psychometric validity and reliability in a university student sample (20).

Novelty Seeking subscale of the Temperament and Character Inventory (TCI-NS): Developed by Cloninger et al., this subscale assesses impulsive and exploratory responses to novel stimuli (2). It comprises four features: Exploratory excitability refers to the tendency to be curious and responsive to new stimuli. Impulsiveness represents acting without considering the consequences during decision-making processes. Extravagance reflects impulsive spending, exaggerated emotional reactions, and a general inclination toward excess. Disorderliness denotes a dislike of rules, a lack of planning, and chaotic behavioral tendencies. Higher scores on the TCI-NS subscale indicate a greater tendency to seek and react to environmental novelty. The Turkish version, adapted by Köse et al., was found to be psychometrically reliable (21).

Addiction Profile Index (API) and its Clinical Form (API-C): Developed by Ögel et al., these tools evaluated the severity of addiction and associated psychopathological features (22, 23). The API is a comprehensive self-report scale assessing substance use patterns, consequences, and diagnostic criteria for substance dependence. The API-C is a complementary clinician-completed form focusing on clinically significant symptoms, including difficulties in anger control, novelty seeking, impulsivity, depression, and anxiety. A high API-C score signifies clinically significant psychosocial symptoms accompanying substance dependence.

Criminal Thinking Scales (CTS): Developed by Knight et al., the CTS assessed cognitive distortions and thinking styles predisposing individuals to criminal behavior (24). It identifies cognitive schemas sustaining antisocial behavior by measuring thought patterns that legitimize or rationalize criminal acts. Key subdimensions include: Entitlement reflects the individual's tendency to perceive behaviors that violate the rights of others as personal privileges and to view oneself as above the rules. Power orientation represents the desire to have dominance and control over others. Criminal rationalization refers to the inclination to perceive criminal acts as reasonable or inevitable and to attribute the motivation for such behavior to external factors (e.g., “Anyone would have done the same”). Cold heartedness is characterized by a lack of empathy and indifference toward the impact of criminal acts on victims. Justification involves attempts to legitimize one’s criminal behavior or to shift the responsibility onto the victim or environmental circumstances as a means of avoiding moral accountability. Personal irresponsibility describes a tendency to avoid taking responsibility for one’s own actions and to disregard rules and obligations. High scores on the CTS indicate a stronger presence of the corresponding cognitive distortion. Psychometric evaluations of the Turkish

version have shown that the instrument is both valid and reliable for use in the local population (25).

Statistics

Prior to data collection, an a priori power analysis was performed using G*Power software to ascertain the requisite sample size. Based on effect sizes from comparable studies and assuming a medium effect size (Cohen's d≈0.5), a total sample size of approximately 128 participants (64 per group) was determined to achieve 80% power at $\alpha=0.05$. The final sample of 120 participants in this study closely approximated this estimated minimum, providing largely sufficient statistical power.

Data analysis was performed using IBM SPSS Statistics version 25. The Shapiro–Wilk test was employed to assess the distribution of continuous variables. Since the assumption of normality was violated for certain variables, non-parametric tests were used for group comparisons. Specifically, the Mann–Whitney U test was applied to continuous variables, while the Chi-square test was used for categorical variables. Effect sizes were reported as r for non-parametric tests and as Cramér's V for categorical data. The interpretation of r values was as follows: 0.10–0.29 indicated a small effect size, 0.30–0.49 a medium effect size, and ≥ 0.50 a large effect size. Spearman's rank correlation coefficient was employed to examine associations between continuous variables, with the direction and magnitude of each correlation qualitatively interpreted using the same thresholds. Multiple linear regression analyses were then conducted to identify predictors of sensation seeking subscale scores. Prior to regression, key assumptions were tested, including linearity, independence of residuals, homoscedasticity, normal distribution of residuals, and multicollinearity. All assumptions were met. In particular, all Variance Inflation Factor (VIF) values were below 2, indicating no significant multicollinearity. The level of statistical significance for all tests was set at $p < 0.05$. To control for Type I error due to multiple comparisons, the Holm–Bonferroni method was applied to adjust p values.

RESULTS

Sociodemographic and Clinical Characteristics

Table 1 presents a comparison of baseline sociodemographic and clinical variables between the ASPD and control groups. No statistically significant difference was found in mean age between the groups (Mann-Whitney U=1432.5, p=0.052, r=0.18). However, a significant difference was observed in years of education: individuals in the ASPD group had significantly fewer years of formal education compared to the control group (U=270.0, p=0.001) and had a large effect size (r=0.70). Employment status and residential environment also differed significantly between the groups. The ASPD group demonstrated higher rates of unemployment and greater likelihood of residing in rural areas (Fisher's exact test, p=0.018, V=0.26 for unemployment; $x^2=6.316$, p=0.027, V=0.23 for rural residence).

Clinically, the ASPD group exhibited a significantly more adverse risk profile. Cigarette smoking was significantly more prevalent in this group (U=299.0, p=0.001, r=0.80), as were alcohol use ($x^2=$

Table 1. Comparative sociodemographic and clinical profile of ASPD and control groups

Variable	ASPD (n=60)	Control (n=60)	Statistical analysis	df	p
Age; years, Mean –SD	25.20±6.03	24.55±2.02	U=1432.5	118	0.052
Years of education; years, Mean –SD	9.543±4.30	16.00±0.00	U=270.0	118	<0.001
Marital status; n (%)			x^2 =5.116	2	0.091
Single/Separated	45 (75.0)	43 (71.7)			
Married	15 (25)	17 (28.3)			
Employment status; n (%)			x^2 =8.090	2	0.018
Not working	20 (33.3)	18 (30)			
Working	40 (66.6)	42 (70.0)			
Residence; n (%)			Fisher□s exact test	1	0.027
Urban	54 (90)	60 (100)			
Rural	6 (10)	0			
Smoking, pcs/day; Mean –SD	31.30±21.74	1.92±5.45	U=299.0	118	<0.001
Alcohol use; n (%)	31 (51.7)	13 (21.7)	x^2 =11.627	1	0.001
Substance use; n (%)	22 (36.7)	0	Fisher□s exact test	1	<0.001
Number of suicide attempts; Mean –SD	1.15±1.31	0	U =750.0	118	<0.001
Self-mutilation attempts; Mean –SD	1.35±2.91	0	U =1380.0	118	<0.001
Number of criminal history; Mean –SD	1.09±1.85	0	U =1080.0	118	<0.001

Mean – SD: Mean – standard deviation, ASPD: Antisocial personality disorder, U: Mann Whitney U test, x^2: Chi-square test
Note: Fisher□s exact test was used for anycell frequencies less than five

Table 2. Group differences in sensation seeking, novelty seeking, addiction severity, and criminal thinking scores

Subscales	ASPD (n=60) Mean –SD	Control (n=60) Mean –SD	Statistical analysis	*df*	*p*
Zuckerman Sensation Seeking Scale					
SSS-DIS	5.03±3.11	2.63±2.09	*U*=1000.0	118	<0.001
SSS-TAS	4.96±2.46	5.26±2.42	*U*=1691.0	118	0.564
SSS-ES	4.98±2.09	3.95±2.07	*U*=1304.5	118	0.009
SSS-BS	5.45±2.47	2.55±1.84	*U*=654.0	118	<0.001
SSS-Total	20.43±6.73	14.40±6.54	*U*=937.0	118	<0.001
Temperament and Character Inventory-Novelty Seeking					
TCI-NS1 Exploratory excitability	5.36±1.32	6.13±1.09	*U*=1073.5	118	<0.001
TCI-NS2 Impulsiveness	5.10±1.56	4.00±1.28	*U*=1046.0	118	<0.001
TCI-NS3 Extravagance	4.46±1.30	5.13±1.18	*U*=1307.0	118	0.008
TCI-NS4 Disorderliness	4.46±1.23	4.65±1.36	*U*=1691.5	118	0.558
TCI-NS Total	19.60±2.65	19.91±2.68	*U*=1742.0	118	0.759
Addiction Profile Index					
API-Substance use	1.88±1.76	0.11±0.39	*U*=514.0	118	<0.001
API-Diagnostic criteria	7.05±6.29	0.10±0.58	*U*=618.0	118	<0.001
API- Effect on everyday life	12.70±11.78	0.40±1.73	*U*=688.0	118	<0.001
API-Craving	5.18±4.84	0.08±0.27	*U*=592.5	118	<0.001
API-Motivation for substance use	4.71±4.80	0.25±0.91	*U*=806.5	118	<0.001
API-Total	6.25±5.04	0.22±0.50	*U*=503.5	118	<0.001
Addiction Profile Index-Clinical Version					
API-C Anger control failure	4.83±1.64	0.93±1.05	*U*=194.5	118	<0.001
API-C Lack of safe behavior	5.93±3.05	3.55±1.84	*U*=947.5	118	<0.001
API-C Novelty-seeking behavior	3.11±2.00	1.70±1.52	*U*=1051.0	118	<0.001
API-C Impulsivity	4.06±1.68	2.01±1.39	*U*=688.0	118	<0.001
API-C Depression	5.30±2.24	2.58±1.21	*U*=641.5	118	<0.001
API-C Anxiety	3.48±1.88	1.21±0.88	*U*=588.5	118	<0.001
Criminal Thinking Scales					
CTS- Entitlement	27.33±9.78	16.50±5.24	*U*=645.0	118	<0.001
CTS- Criminal rationalization	32.97±9.63	28.25±7.32	*U*=1306.5	118	0.009
CTS- Power orientation	35.72±11.54	20.50±5.88	*U*=489.0	118	<0.001
CTS- Cold heartedness	24.25±10.97	17.62±4.47	*U*=1154.5	118	0.001
CTS- Justification	37.38±8.53	33.77±7.35	*U*=1365.0	118	0.022
CTS- Personal irresponsibility	29.66±12.03	20.00±7.47	*U*=932.5	118	<0.001

U: Mann Whitney U test, Mean ± SD: Mean – standard deviation, SSS: Zuckerman Sensation Seeking Scale Form V, DIS: Disinhibition, TAS: Thrill and Adventure Seeking, ES: Experience Seeking, BS: Boredom Susceptibility, TCI-NS: Temperament and Character Inventory- Novelty Seeking dimension, API: Addiction Profile Index, API-C: Addiction Profile Index-Clinical Version, CTS: Criminal Thinking Scales

11.627, p=0.001, V=0.31) and illicit drug use (Fisher's exact test, p=0.001, V=0.47). A history of suicide attempts was also significantly more common in the ASPD group compared to the control group (U=750.0, p=0.001, r=0.57), as was the prevalence of self-harming behaviors (U=1380.0, p =0.001, r=0.27). Moreover, the presence of a criminal record was significantly higher in the ASPD group (U=1080.0, p=0.001, r=0.41).

Group Differences in Sensation Seeking and Related Scales

Table 2 summarizes group comparisons in psychological scale scores. The total score on the Zuckerman SSS was significantly higher in the ASPD group compared to the control group (U= 937.0, p=0.001, r=0.45). In particular, the ASPD group scored significantly higher on the subscales of disinhibition (r=0.41), experience seeking (r= 0.26), and boredom susceptibility (r=0.59), with medium to large effect sizes. However, no significant group differences were found on the thrill and adventure seeking subscale (p=0.564).

Regarding the TCI-NS, no significant difference was found in total scores between the two groups (p =0.759). However, differences emerged at the subscale level. Individuals in the ASPD group scored significantly higher in the impulsiveness (U= 1046.0, p=0.001, r=0.41) and extravagance subscales (p=0.008, r=0.27), while the control group scored significantly higher in exploratory excitability (p=0.001, r=0.49).

In the API and its clinical form (API-C), the ASPD group scored significantly higher on the overall score and all subscales compared to the control group (all p<0.001), typically with large effect sizes.

The CTS yielded similar results. The ASPD group scored significantly higher across all subdimensions (all $p < 0.001$). Notable differences were observed in entitlement (U=645.0, p=0.001, r=0.44), power orientation (r=0.53), and personal irresponsibility (r=0.46), highlighting the deeply rooted nature of antisocial cognitive schemas. Medium-sized differences were also observed in other domains such as criminal rationalization and cold-heartedness.

Table 3. Associations of sensation seeking dimensions with clinical variables

Variable		Zuckerman Sensation Seeking Scale				
		SSS -DIS	SSS-TAS	SSS -ES	SSS-BS	SSS-Total
Age	*r*	-0.060	0.085	0.037	-0.207	-0.086
	p	1.0	1.0	1.0	1.0	1.0
Years of education	*r*	-0.260	0.148	-0.139	-0.337	-0.252
	p	0.945	1.0	1.0	0.192	1.0
Number of suicide attempts	*r*	0.172	0.045	0.279	0.420	0.332
	p	1.0	1.0	0.681	0.025	0.207
Number of self-mutilative actions	*r*	0.021	-0.169	0.210	0.230	0.101
	p	1.0	1.0	1.0	1.0	1.0
Number of criminal history	*r*	-0.019	-0.042	-0.004	-0.022	-0.068
	p	1.0	1.0	1.0	1.0	1.0

r: correlation coefficient, SSS: Zuckerman Sensation Seeking Scale Form V, DIS: Disinhibition, TAS: Thrill and Adventure Seeking, ES: Experience Seeking, BS: Boredom Susceptibility
Note: p-values were adjusted for multiple comparisons using the Holm–Bonferroni method.

Correlations between Sensation Seeking and Clinical Variables

Table 3 presents bivariate correlations between subscale scores of the sensation seeking scale and key clinical variables. Age and education level were not significantly correlated with any sensation seeking subdimension (all $p>0.05$). Correlations between sensation seeking and suicide attempts were also noteworthy. In the ASPD group, the number of past suicide attempts was positively correlated with boredom susceptibility scores ($r=0.420$, $p=0.025$), but was not significantly correlated with total sensation seeking scores ($r=0.332$, $p=0.207$).

Table 4 provides detailed correlations between the sensation seeking subscales and related clinical variables such as impulsivity, addiction severity, and criminal cognition. The disinhibition subscale showed moderate positive correlations with several adverse clinical variables. Individuals with higher disinhibition scores also had higher TCI total scores ($r=0.422$, $p=0.005$), and their addiction severity (API total score) increased in parallel ($r=0.454$, $p=0.005$). Craving scores from the API-C were also strongly correlated with disinhibition ($r = 0.566$, $p=0.005$). Similarly, disinhibition was highly correlated with API-C novelty-seeking scores ($r=0.673$, $p=0.005$). In terms of criminal cognitive patterns, individuals with higher levels of disinhibition also tended towards entitlement thinking styles (CTS Entitlement, $r=0.333$), with a statistically significant correlation ($p=0.036$).

Total sensation seeking scores also correlated significantly with several clinical variables. Notably, total sensation seeking was positively associated with addiction severity (API total score, $r=0.374$, $p=0.012$), suggesting that individuals with higher sensation seeking tendencies are more likely to exhibit pronounced substance use behaviors. Furthermore, total sensation seeking scores were significantly correlated with the power orientation subscale of the CTS ($r=0.371$, $p=0.016$), indicating that individuals with elevated sensation seeking may also possess a strong drive to dominate or control others. Additionally, total sensation seeking was strongly correlated with API-C novelty seeking ($r=0.679$, $p=0.005$).

Regression Analyses: Predictors of Sensation Seeking Subdimensions

Table 5 summarizes the results of multiple linear regression models used to identify predictors of sensation seeking subdimensions. Each subdimension—disinhibition, experience seeking, boredom susceptibility, and total score—was treated as a dependent variable, with relevant clinical variables entered as predictors in the model.

The most significant predictors for the disinhibition subdimension were total TCI-NS, substance use severity (API substance use subscale), craving (API-C craving), and API-C novelty seeking. Each

Table 4. Correlations of sensation seeking scores with novelty seeking, addiction severity, and criminal thinking

Subscales		Zuckerman Sensation Seeking Scale (SSS)				
		SSS -DIS	SSS-TAS	SSS -ES	SSS-BS	SSS- Total
Temperament and Character Inventory-Novelty Seeking						
TCI-NS1 Exploratory excitability	*r*	0.178	0.056	-0.102	0.102	0.086
	p	0.869	1.0	1.0	1.0	1.0
TCI-NS2 Impulsiveness	*r*	0.313	-0.115	0.073	0.455	0.283
	p	0.06	0.764	0.764	**0.005**	0.087
TCI-NS3 Extravagance	*r*	0.234	0.149	0.175	0.000	0.205
	p	0.36	0.542	0.542	0.999	0.464
TCI-NS4 Disorderliness	*r*	0.079	-0.032	0.079	0.086	0.107
	p	1.0	1.0	1.0	1.0	1.0
TCI-NS Total	*r*	0.422	-0.003	0.102	0.329	0.353
	p	**0.005**	0.979	0.874	**0.03**	**0.024**
Addiction Profile Index						
API-Substance use	*r*	0.501	0.030	0.191	0.404	0.442
	p	**0.005**	0.819	0.286	**0.005**	**0.005**
API-Diagnostic criteria	*r*	0.523	-0.077	0.209	0.403	0.425
	p	**0.005**	0.556	0.218	**0.005**	**0.005**
API-Effect on everyday life	*r*	0.384	-0.108	0.094	0.355	0.295
	p	**0.01**	0.826	0.826	**0.02**	0.066
API-Craving	*r*	0.566	-0.148	0.249	0.462	0.442
	p	**0.005**	0.259	0.11	**0.005**	**0.005**
API-Motivation for substance use	*r*	0.218	-0.007	-0.107	0.206	0.158
	p	0.475	0.957	0.828	0.475	0.687
API-Total (addiction severity)	*r*	0.454	-0.039	0.123	0.381	0.374
	p	**0.005**	0.766	0.702	**0.012**	**0.012**
Addiction Profile Index-Clinical Version						
API-C Anger control failure	*r*	0.225	-0.301	0.116	0.279	0.130
	p	0.252	0.095	0.646	0.124	0.646
API-C Lack of safe behavior	*r*	-0.023	-0.286	0.058	0.208	-0.023
	p	1.0	0.135	1.0	0.44	1.0
API-C Novelty-seeking behavior	*r*	0.673	0.072	0.486	0.516	0.679
	p	**0.005**	0.583	**0.005**	**0.005**	**0.005**
API-C Impulsivity	*r*	0.234	-0.159	0.313	0.451	0.301
	p	0.142	0.226	0.06	**0.005**	0.06
API-C Depression	*r*	0.012	-0.072	0.073	0.213	0.073
	p	1.0	1.0	1.0	0.51	1.0
API-C Anxiety	*r*	0.117	-0.158	0.123	0.318	0.130
	p	0.966	0.916	0.966	0.065	0.966
Criminal Thinking Scales						
CTS- Entitlement	*r*	0.333	-0.091	0.110	0.426	0.312
	p	**0.036**	0.806	0.806	**0.005**	**0.036**
CTS- Criminal rationalization	*r*	0.312	0.030	0.037	0.323	0.283
	p	0.06	1.0	1.0	0.06	0.084
CTS- Power orientation	*r*	0.300	0.086	0.141	0.444	0.371
	p	0.06	0.564	0.564	**0.005**	**0.016**
CTS- Cold heartedness	*r*	0.249	-0.105	0.256	0.211	0.221
	p	0.24	0.424	0.24	0.27	0.27
CTS- Justification	*r*	0.113	0.054	0.034	0.271	0.174
	p	1.0	1.0	1.0	**0.18**	0.736
CTS- Personal irresponsibility	*r*	0.211	-0.295	0.032	0.514	0.202
	p	0.318	0.088	0.807	**0.005**	0.318

SSS: Zuckerman Sensation Seeking Scale Form V, DIS: Disinhibition, TAS: Thrill and Adventure Seeking, ES: Experience Seeking, BS: Boredom Susceptibility, TCI-NS: Temperament and Character Inventory- Novelty Seeking dimension, API: Addiction Profile Index, API-C: Addiction Profile Index-Clinical Version, CTS: Criminal Thinking Scales
Note: p-values were adjusted for multiple comparisons using the Holm–Bonferroni method.

Table 5. Multivariate predictors of sensation seeking subdimensions: Regression analysis results

Dependent variable	Predictor variables	*Beta*	Std.Error	95 % CI		*t*	*p*
SSS- Disinhibition	TCI-NS total	0.496	0.145	0.205	0.788	3.423	0.001
	API-substance use	0.669	0.317	0.031	1.306	2.110	0.040
	API- craving	0.271	0.125	0.019	0.522	2.160	0.036
	API-C-novelty-seeking behavior	0.514	0.174	0.163	0.864	2.946	0.005
SSS- Experience seeking	API-novelty-seeking behavior	0.462	0.134	0.194	0.730	3.456	0.001
SSS- Boredom susceptibility	CTS- personal irresponsibility	0.086	0.026	0.034	0.138	3.326	0.002
SSS- Total	API substance use	1.887	0.746	0.386	3.388	2.531	0.015
	API-C novelty-seeking behavior	1.704	0.457	0.785	2.623	3.732	0.001

Statistically significant results are given; CI = confidence interval
SSS: Zuckerman Sensation Seeking Scale Form V, DIS: Disinhibition, TAS: Thrill and Adventure Seeking, ES: Experience Seeking, BS: Boredom Susceptibility, TCI-NS: Temperament and Character Inventory- Novelty Seeking dimension, API: Addiction Profile Index, API-C: Addiction Profile Index-Clinical Version, CTS: Criminal Thinking Scales

of these variables demonstrated a statistically significant predictive effect within the model (all $p < 0.05$). Collectively, they accounted for a substantial proportion of the variance in disinhibition scores, indicating that the combination of these variables strongly predicted disinhibitory tendencies. In the regression model for experience seeking, only one variable emerged as a significant predictor: API-C novelty seeking. The API-C novelty seeking score significantly predicted experience seeking scores (standardized $\beta=0.46$, $p=0.001$), while other variables did not contribute significantly. Scores on the CTS personal irresponsibility subscale significantly predicted boredom susceptibility ($\beta=0.09$, $p=0.002$). Other variables, such as entitlement or power orientation, did not significantly contribute to the model.

Finally, the regression model for total sensation seeking identified two significant predictors: API substance use severity and API-C novelty seeking. The API substance use subscale ($\beta=1.887$, $p=0.015$) and the API-C novelty seeking subscale ($\beta=1.704$, $p = 0.001$) each exerted significant and independent predictive effects. Notably, these two predictors represent both behavioral (substance use) and motivational/emotional (novelty/impulsivity) dimensions. Overall, the model explained a substantial portion of the variance in total sensation seeking scores and was statistically significant (overall model $p< 0.001$).

DISCUSSION

This study provides an original contribution to the literature by comprehensively examining the psychopathological implications of sensation seeking tendencies in individuals diagnosed with ASPD. Our findings revealed that the ASPD group scored significantly higher than healthy controls not only in overall sensation seeking levels but also in specific subdimensions, particularly disinhibition, experience seeking, and boredom susceptibility. Moreover, this personality trait was found to be associated with various maladaptive patterns in ASPD individuals. High sensation seeking was significantly related to adverse clinical variables such as substance use, suicidal behaviors, and criminal cognitive schemas. These results suggest that sensation seeking should not be considered an isolated personality trait, but rather a mediating variable that interacts with other risk factors and potentially shapes the clinical course of psychopathology.

Our findings align with Zuckerman's multidimensional theoretical model of sensation seeking (1), revealing a distinct pattern within the ASPD group regarding its subcomponents. Individuals with ASPD scored significantly higher in disinhibition, experience seeking, and boredom susceptibility, while no significant difference was found between groups in the thrill and adventure seeking dimension. This pattern is consistent with previous research and offers crucial insights into the qualitative nature of heightened sensation seeking in ASPD (8,12,26,27). In our study, disinhibition emerged as the most distinguishing dimension for the ASPD group, indicating these individuals' difficulty in suppressing their impulses. This trait is thought to predispose them to outcomes like criminal behavior or substance use (8,12,26). Furthermore, the elevated experience seeking scores recorded in the cases suggest a propensity for unusual, socially deviant, or marginal experiences (27). The increased boredom susceptibility observed in the ASPD group reflects an intolerance

for routine and monotony (11,13,28). In our analysis, this subdimension showed the strongest association with suicidal behavior, suggesting that individuals experiencing internal distress and possessing a low arousal threshold may resort to extreme behaviors like self-harm to alleviate this state. In essence, not all subdimensions of sensation seeking reflect pathological tendencies. The absence of group differences in thrill and adventure seeking reminds us that sensation seeking does not always equate to maladaptive risk-taking (29). This finding suggests that the heightened sensation seeking observed in ASPD manifests more through impulsive and maladaptive expressions rather than socially sanctioned risks.

Although the terms "sensation seeking" and "novelty seeking" are frequently used interchangeably, our study reinforces their theoretical and clinical distinctness. As highlighted by McCourt et al., sensation seeking primarily reflects an amplified need for stimulation, whereas novelty seeking is more closely related to cognitive and behavioral responsiveness to novel stimuli (3,30,31). While both traits were generally elevated in the ASPD group compared to controls, analyses at the subdimension level elucidated the specific nature of this elevation. In individuals with ASPD, sensation seeking was characterized by increased disinhibition and boredom susceptibility. Conversely, novelty seeking in this group primarily manifested through heightened impulsiveness and extravagance. Interestingly, the exploratory excitability subdimension of novelty seeking was significantly higher in the control group. These findings suggest a differential manifestation of novelty seeking across groups: ASPD individuals exhibit novelty seeking via behavioral disorganization and impulsivity, while healthy individuals demonstrate a more curiosity-driven and cognitively exploratory pattern. This distinction holds particular relevance within the context of substance use. Prior research indicates that individuals with elevated sensation-seeking tendencies may initiate substance use to achieve sensory stimulation, whereas those with high novelty seeking may perpetuate substance use in a habitual and compulsive manner (32,33). The simultaneous elevation of both sensation seeking and novelty seeking could therefore lead to more chronic and entrenched patterns of risky behavior (31). Our findings corroborate this perspective, as sensation seeking subdimensions showed strong correlations with both substance dependence severity and diagnostic indicators of substance use disorders.

The observed association between sensation seeking and self-injurious behaviors (i.e., suicide attempts and self-harm) in our study reinforces the theoretical link between this personality trait and difficulties in emotion regulation. Within the ASPD group, individuals who scored higher on boredom susceptibility were also more likely to report past suicide attempts. This finding suggests that impulsivity and low arousal tolerance may predispose individuals to engage in extreme behaviors under emotional strain (34). In literature, this tendency is often explained by the concept of "negative urgency," which is frequently observed in externalizing disorders (35-37). Negative urgency refers to the tendency to act rashly and potentially harmfully during moments of intense emotional distress. This implies that self-injurious behavior may serve as a maladaptive coping mechanism for emotional dysregulation (35,38). Our findings are consistent with this view: ASPD participants who reported more suicide attempts were also those who demonstrated the lowest tolerance for monotony and the strongest drive for stimulation.

Another key finding of our study is the strong association between sensation seeking and substance use. Previous research has demonstrated that individuals high in sensation seeking tend to have heightened sensitivity within the brain's reward systems and exhibit greater motivation for the pleasurable effects of substance use (39, 40). Our results are in line with this evidence: among individuals with ASPD, total sensation seeking scores were strongly correlated with indicators of addiction severity derived from the API and API-C instruments. In other words, individuals high in sensation seeking may not only be more inclined to begin using substances but may also possess internal motivational traits (e.g., impulsivity, craving for novelty) that perpetuate substance use over time (41,42).

The significant correlations observed between sensation seeking and patterns of criminal thinking in our study underscore the crucial role of cognitive processes in the development and persistence of

antisocial behavior. Individuals with elevated sensation-seeking tendencies demonstrated higher levels of entitlement, power orientation, and personal irresponsibility schemas that not only reflect distorted beliefs but also indicate a motivational drive for heightened arousal (36). These findings suggest that criminal behavior in such individuals may not solely result from momentary disinhibition or external provocation. Instead, their actions may be internally rationalized through entrenched belief systems, whereby criminal acts are perceived as acceptable, deserved, or even necessary (43). In this context, sensation seeking appears to function not merely as a personality trait but as a cognitive-affective framework that influences the justification and internal logic behind antisocial behaviors. This interpretation is consistent with Walters' model of criminal thinking, which posits that antisocial cognitions serve to legitimize risky and unlawful behaviors (44,45).

Limitations

Several limitations should be considered when interpreting the findings of this study. First, due to its cross-sectional design, the study does not allow for causal inferences. The correlations we identified cannot determine which variable precedes or influences the other; therefore, our findings should be understood at an associative level only. Second, our sample included only male participants. While this choice was deliberate to control for the potential confounding effects of gender on sensation seeking and antisocial behaviors, it limits the generalizability of the findings to female ASPD populations. Future research incorporating female antisocial samples would be valuable in exploring possible gender-specific dynamics. Third, variables such as substance use, suicide attempts, and criminal history were partially based on self-reports, which may be subject to recall bias or social desirability effects. Although we attempted to verify this information through medical records and reports from close relatives whenever possible, the inherent limitations of subjective data collection could not be entirely eliminated. Lastly, although our final sample size was close to the number suggested by power analysis, it fell slightly short of the optimal size for conducting complex regression models. This limitation may have reduced the statistical power of some regression results. Indeed, certain marginal findings in our multivariate models warrant replication in studies with larger and more diverse samples. Therefore, the regression outcomes in particular should be interpreted with caution and confirmed through future research.

This study revealed that individuals diagnosed with ASPD exhibit significantly elevated levels of sensation seeking, and that this elevation is associated with several clinically relevant behavioral and cognitive patterns. In particular, the disinhibition and boredom susceptibility subdimensions were found to be linked to high-risk behaviors such as substance use and suicide attempts within the ASPD group. While the cross-sectional nature of the study precludes causal interpretation, the findings suggest that sensation seeking may play a central role in the clinical presentation of ASPD. A multidimensional evaluation of sensation seeking may aid in identifying at-risk individuals and developing tailored intervention strategies in clinical practice. In addition, by distinguishing the unique characteristics of sensation seeking and novelty seeking, our study emphasizes the importance of evaluating these traits independently in clinical assessments. Sensation seeking showed strong associations with reward sensitivity, impulsivity, and antisocial cognitions, while subdimension analyses of novelty seeking revealed its heterogeneous nature, distinguishing impulsive tendencies from cognitive curiosity. In this context, evaluating the subdimensions of both sensation seeking and novelty seeking could provide a more detailed understanding of an individual's risk profile and behavioral tendencies.

In conclusion, the present findings underscore the importance of sensation seeking in the context of ASPD and support its consideration as a transdiagnostic risk factor. However, it must be reiterated that all associations observed in this study were correlational and based on cross-sectional data. Future longitudinal research is needed to clarify the developmental trajectory of sensation seeking and its temporal interactions with related variables.

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Psychometric properties of the Turkish version of the Posttraumatic Maladaptive Beliefs Scale

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SUMMARY

Objective: Previous studies have shown that maladaptive cognitions following trauma are closely associated with post-traumatic stress symptoms and depression. The aim of the present study was to examine the psychometric properties of the Turkish version of the Posttraumatic Maladaptive Beliefs Scale (PTMBS).

Method: Data were collected from 338 adults (M age = 32.95, SD = 11.43; 56.2% female) residing in regions affected by the February 6, 2023 earthquakes in Türkiye. Participants completed the PTMBS along with the International Trauma Questionnaire and the International Depression Questionnaire.

Results: Confirmatory factor analysis indicated that the one-factor model showed poor fit, whereas the three-factor model (Threat of Harm, Self-Worth and Judgment, Reliability of Others) demonstrated good fit indices. Criterion validity was supported by significant positive correlations of the scale with post-traumatic stress and depressive symptoms. Reliability analyses, including Cronbach's alpha, item–total correlations, and Spearman–Brown split-half coefficients, indicated acceptable to high internal consistency for the total and dimension scores.

Discussion: Findings demonstrate that the Turkish version of the PTMBS adequately reflects the original three-dimensional structure and shows the expected associations with PTSD and depression symptoms. In particular, the stronger associations of the Threat of Harm subscale with psychopathology highlight the clinical relevance of "shattered assumptions" following trauma. In conclusion, the Turkish PTMBS is a valid and reliable tool for assessing maladaptive cognitions after trauma and can be used for clinical diagnosis, treatment planning, and monitoring therapeutic progress.

Key Words: Trauma, maladaptive beliefs, psychometric evaluation, PTSD, depression

INTRODUCTION

A traumatic event is defined as an experience that threatens an individual's life, physical integrity, or sense of safety, and exceeds their ordinary coping resources (1). Such events can range widely, from natural disasters (e.g., earthquakes, floods, or fires) and serious accidents to human-caused acts of violence such as war, torture, sexual assault, and childhood abuse. Data on the prevalence of traumatic events indicate that these experiences are highly common on a global scale (2–5). For instance, findings from the World Mental Health Surveys conducted across 26 countries reported that approximately 70% of participants had experienced at least one traumatic event during their lifetime, and 30% had been exposed to four or more (6).

Although traumatic events constitute a significant risk factor for psychopathology, only a portion of those exposed to trauma develop trauma-related mental disorders. Various meta-analyses and epidemiological findings have shown that approximately 30 - 40% of individuals exposed to trauma develop posttraumatic stress disorder (PTSD), depression, anxiety, or somatization disorders (7-9). This rate may vary depending on factors such as the type, intensity, and duration of the trauma, as well as individual and environmental protective or risk factors (10,11).

Previous research has identified numerous risk factors that may contribute to the development of mental health problems following a traumatic event. For example, studies in the literature have shown that various factors such as difficulties in emotion regulation (12), deficits in executive functions (13), and low self-esteem (14) serve as risk

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factors for the development of psychopathology after trauma exposure. Among the important determinants of posttraumatic psychopathology are cognitive processes. Recent studies (15,16) have highlighted the relationship between posttraumatic maladaptive cognitions and mental health problems.

From a theoretical perspective, the cognitive impact of trauma has been conceptualized through frameworks such as Janoff-Bulman's "shattered assumptions" theory (17,18) and, more specifically, Herman's emphasis on the disruption of self and interpersonal trust following trauma (19). Consistent with these perspectives, research has shown that maladaptive posttraumatic cognitions are particularly concentrated in domains such as safety, self-esteem, control, trust, and intimacy (20–22). Building on this body of work, Ehlers and Clark's cognitive model (23) proposes that such negative appraisals play a central role in maintaining trauma-related symptoms. Furthermore, treatment studies have demonstrated that the restructuring of maladaptive cognitions typically precedes symptom improvement (24), thereby suggesting that these cognitions may play a causal role in posttraumatic recovery (25,26). Taken together, these findings indicate that posttraumatic maladaptive cognitions are not merely correlated with symptoms but also represent one of the key obstacles to psychological recovery.

In light of these theoretical frameworks, the Posttraumatic Maladaptive Beliefs Scale (PTMBS) was developed as a systematic measurement tool to assess dysfunctional cognitions following trauma (27). The scale measures cognitive distortions across domains such as safety, self-esteem, control, trust, and intimacy, and therefore is widely used in both clinical and research contexts. Specifically, encompassing dimensions such as Threat of Harm, Reliability/Trustworthiness of Others, and Self-Worth and Judgment, the PTMBS has demonstrated good psychometric reliability and validity, as well as strong associations with posttraumatic stress and depressive symptoms (28–32). Accordingly, the PTMBS stands out as a theoretically grounded instrument that facilitates understanding of individuals' cognitive profiles after trauma, guides therapeutic interventions, and enables the monitoring of treatment progress.

When considered within the sociocultural context of Türkiye, large-scale adversities such as earthquakes and other natural disasters, forced displacement, war-related refugee experiences, and collective or sociopolitical traumas constitute highly prevalent phenomena. In this respect, the systematic assessment of maladaptive cognitions that emerge following trauma represents a critical need for both clinical evaluation and empirical research. More specifically, the comprehensive evaluation of maladaptive posttraumatic beliefs among Turkish-speaking populations would not only support a more detailed understanding of posttraumatic psychopathology but also enable the monitoring and optimization of trauma-focused therapeutic interventions.

Furthermore, the PTMBS has demonstrated cross-cultural applicability beyond Western contexts. For example, Vöhringer et al. (28) employed the scale in a refugee sample and reported that it effectively captured the maladaptive belief systems that develop in the aftermath of traumatic experiences. Such findings underscore the scale's theoretical and practical relevance in culturally diverse settings and highlight the importance of its adaptation into Turkish. A Turkish version of the PTMBS would provide a psychometrically sound tool for identifying posttraumatic maladaptive cognitions, evaluating the efficacy of trauma-focused treatments, and contributing to the expansion of culturally sensitive trauma research. Taken together, a valid and reliable instrument in the Turkish literature that comprehensively assesses cognitive and belief-based processes associated with posttraumatic adaptation represents a gap. Therefore, the present study aims to translate and psychometrically evaluate the Turkish version of the PTMBS to establish its utility in both clinical and research contexts.

METHOD

Participants

The study sample consisted of 338 individuals (aged 18–59) residing in the provinces of Hatay, Kahramanmaraş, and Adıyaman, which were

among the regions most affected by the February 6, 2023 earthquakes in Türkiye. Participants were recruited using a convenience sampling method. Prior to data collection, ethical approval was obtained from the Mersin University Social Sciences Ethics Committee (Approval No: 2025/146). The inclusion criteria were as follows: (a) residing in one of the provinces at the epicenter of the earthquake, (b) being 18 years of age or older, and (c) being literate. The exclusion criteria included the presence of any self-reported psychiatric diagnosis or substance use disorder. Both inclusion and exclusion criteria were assessed based on participants' self-reports.

Measures

Sociodemographic Information Form: The Sociodemographic Information Form was developed by the researchers to collect data on participants' age, gender, marital status, educational level, substance use, and history of any psychiatric diagnosis or disorder.

International Trauma Questionnaire (ITQ): The ITQ is a self-report measure designed to assess PTSD symptoms in accordance with ICD-11 diagnostic criteria (33). The scale consists of six items covering three symptom clusters: (1) re-experiencing the traumatic event in the here and now, (2) deliberate avoidance of reminders, and (3) a persistent sense of current threat. Participants rate how much they have been bothered by each symptom over the past month. Three additional items assess the degree of functional impairment associated with these symptoms. All items are rated on a five-point Likert scale ranging from 0 (Not at all) to 4 (Extremely). According to ICD-11 diagnostic rules, a probable PTSD diagnosis is met if the respondent endorses at least one symptom from each cluster and at least one item reflecting functional impairment. A symptom or impairment is considered "present" when rated ≥ 2 (Moderately). The Turkish adaptation of the ITQ was conducted by Gündoğmuş et al. (34), and the Turkish version demonstrated high internal consistency (α=.91). In the present study, internal reliability was also satisfactory (α= .86).

International Depression Questionnaire (IDQ): The IDQ is a self-report instrument developed to assess depressive symptoms in line with ICD-11 criteria (35). Respondents indicate how often they experienced each symptom during the past two weeks on a five-point Likert scale ranging from 0 (Never) to 4 (Every day). Total scores range from 0 to 36, with higher scores indicating greater symptom severity. The Turkish adaptation was validated by Alpay et al. (36), who reported excellent internal consistency (ω=.94). In the present study, the Cronbach's alpha coefficient for the IDQ was also high (α= .90).

Posttraumatic Maladaptive Beliefs Scale (PTMBS): The PTMBS, originally developed by Vogt et al. (27), is a 15-item measure assessing dysfunctional beliefs that may develop following exposure to traumatic events. The scale comprises three dimensions: Threat of Harm (e.g., "Something bad could happen at any time"), Self-Worth and Judgment (e.g., "What happened to me shows that I am inadequate"), and Reliability/Trustworthiness of Others (e.g., "It is dangerous to trust people"). Each item is rated on a seven-point Likert scale ranging from 1 (Not at all true for me) to 7 (Completely true for me). The total score ranges from 15 to 105, and each dimension score ranges from 5 to 35, with higher scores indicating greater endorsement of maladaptive posttraumatic beliefs. In the original study, the PTMBS demonstrated good psychometric properties, with Cronbach's alpha coefficients of α=.86 for the total score and between .75 and .84 across the dimensions.

Procedure

Following permission from the original developers of the scale, the translation and cultural adaptation of the PTMBS into Turkish were carried out in several stages. First, the original English version of the scale was translated into Turkish by two experts with doctoral degrees in clinical psychology. Both translators were native speakers of Turkish and fluent in English. Subsequently, a blind back-translation procedure was conducted by an independent linguist who had not seen the original items. The back translator held a Ph.D. degree in English Language and Literature, had advanced training in

translation pedagogy, and possessed substantial experience with psychological terminology. Their native language was Turkish and their second language was English.

The blind back-translation method was chosen to minimize researcher bias and semantic drift. After the translation and back-translation phases, the translators and the linguist met to discuss discrepancies and reach consensus on the final Turkish version using a consensus-based reconciliation process. Three primary criteria guided the evaluation of each item: (1) conceptual equivalence with the original meaning, (2) linguistic clarity and naturalness in Turkish, and (3) appropriateness of psychological terminology. Discrepancies were iteratively discussed until full agreement was reached among all translators, and feedback from the original developers was sought when necessary. The finalized Turkish version was subsequently approved by the original development team.

A pilot study was then conducted with 12 participants (6 women, 6 men; aged 19–42) to evaluate the linguistic clarity, comprehensibility, and cultural relevance of the items. Based on participants' feedback, minor linguistic adjustments were made to produce the final Turkish version of the scale. During the data collection phase, undergraduate psychology students from Mersin University and Toros University (3rd and 4th year) conducted face-to-face interviews in the regions where they resided. Prior to data collection, all interviewers received a one-day training session conducted by the researchers, which covered ethical and methodological considerations, including informed consent, trauma-sensitive communication, and confidentiality.

Interviews were conducted individually in container settlements, homes, and community centers where participants were residing. Each interview lasted approximately 20 minutes. Data collection took place between June 12 and August 21, 2025. Before participating, all individuals were provided with an informed consent form, and voluntary participation was confirmed. No monetary or material compensation was provided for participation in the study.

Statistical Analysis

After the data collection process was completed, the data were transferred to a statistical software program for analysis. First, missing data were examined, and it was found that no variable had more than 10% missing values. Missing values were replaced with the mean of the corresponding variable. Multivariate outliers were identified using the Mahalanobis distance method. A significance threshold of $p < .001$ was adopted, and the degrees of freedom were calculated based on the number of scale items ($k=15$). Nineteen participants whose values fell below this threshold were identified as outliers and excluded from the dataset. Initially, data were collected from 357 participants; after removing outliers, analyses were conducted with a dataset of 338 participants. To test the assumption of normality, skewness and kurtosis coefficients were examined, and all variables were found to be within the acceptable range of –2 to +2 (37).

The factor structure of the PTMBS was examined using Confirmatory Factor Analysis (CFA). Model fit was evaluated based on χ^2, Comparative Fit Index (CFI), Tucker–Lewis Index (TLI), Root Mean Square Error of Approximation (RMSEA), and Goodness-of-Fit Index (GFI). The following criteria were adopted for acceptable model fit: CFI and TLI ≥ .90 (preferably ≥ .95), GFI ≥ .95, RMSEA ≤ .06–.08, and a χ^2/df ratio below 3 indicating good fit and below 5 indicating acceptable fit (38,39). Criterion-related validity of the PTMBS was examined by analyzing the relationships between the total and dimension scores and depressive and PTSD symptoms using Pearson correlation analysis.

The validity and reliability of the scale were analyzed in several steps. For each dimension and for the total scale, (i) corrected item–total correlations, (ii) Cronbach's alpha coefficients for internal consistency, and (iii) split-half reliability coefficients were calculated. All analyses were conducted using SPSS version 26, while the CFA was performed in R Studio using the "lavaan" package.

RESULTS

The mean age of participants was 32.95 years with

a standard deviation of 11.43. Among the total sample, 56.2% (n=190) were women and 43.8% (n = 148) were men. Regarding marital status, 43.3% (n=146) of participants were married, 49.0% (n = 165) were single, 4.2% (n=14) were divorced, and 3.6% (n=12) had lost their spouse. In terms of educational background, a very small proportion of participants (0.3%; n=1) were illiterate, while 3.8% (n=13) were literate without formal schooling, 8.3% (n=28) had completed primary school, and 11.2% (n = 38) had completed secondary school. The majority of participants had completed high school (35.5%; n = 120) or held a bachelor's degree (34.6%; n = 117), while 6.2% (n = 21) had pursued graduate-level education.

Validity Analyses

Construct Validity: Confirmatory Factor Analysis

In this study, the factor structure of the PTMBS was examined using CFA. Wetested two competing models: a three-factor model proposed by Vogt et al. (27), comprising Threat of Harm, Reliability/Trustworthiness of Others, and Self-Worth and Judgment, and a single-factor model in which all items were loaded onto a single latent construct. The results of the two CFA models are presented in Table 1.

Table 1. Fit indices for the three-factor and unidimensional CFA models of the Turkish version of the PMBS.

CFA	x^2	sd	x^2/sd	CFI	GFI	TLI	RMSEA
1 factor	653.936	90	7.26	.682	.629	.722	.184
3 factor	291.337	87	3.35	.973	.961	.968	.083

According to the analysis results, the single-factor model demonstrated poor model fit, with indices falling below acceptable thresholds [$x^2(90)$= 653.94, p< .001, x^2/df = 7.26, CFI = .68, GFI = .63, TLI=.72, RMSEA=.18]. In contrast, the three-factor model exhibited acceptable to good fit indices, indicating a satisfactory representation of the data [$x^2(87)$ = 291.34, p < .001, x^2/df = 3.35, CFI = .97, GFI = .96, TLI=.97, RMSEA= .08].The standardized factor loadings and model structure of the three-factor solution are illustrated in Figure 1.

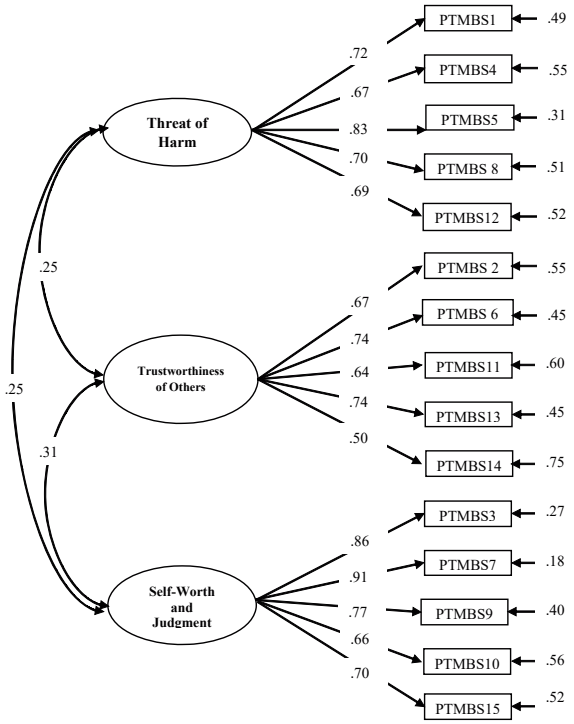

Figure 1. Confirmatory factor analysis results testing the three-factor structure of the Turkish version of the PTMBS.

Criterion-Related Validity

Descriptive statistics and Pearson correlation coefficients for the variables included in the study are presented in Table 2. According to the correlation analyses, significant and positive associations were found between the dimensions of the PTMBS and both PTSD and depressive symptoms. The Threat of Harm dimension showed moderate positive correlations with PTSD (r=.41, p<.01) and depressive symptoms (r=.50, p<.01). The Self-Worth and Judgment dimension was correlated with PTSD (r = .13, p<.01) and depressive symptoms (r=.24, p < .01). Similarly, the Reliability/Trustworthiness of Others dimension demonstrated significant positive correlations with PTSD (r=.16, p<.01) and depressive symptoms (r=.37, p< .01).

The total PTMBS score was also found to be posi-

Table 2. Descriptive statistics and correlations among the TPMBS subscales, PTSD, and depressive symptoms

Variable	M	Sd	1	2	3	4	5	6
1. Threat of Harm	18.65	7.07	-					
2. Self-Worth and Judgment	18.50	7.00	.14**	-				
3. Trustworthiness of Others	13.20	5.11	.21**	.34**	-			
4. PTSD	9.91	5.38	.41**	.13*	.16**	-		
5. Depressive Symptoms	21.52	8.70	.50**	.24**	.37**	.63**	-	
6. TPMBS □Total	50.35	13.36	.68**	.73**	.67**	.35**	.53**	-

Note: PTSD = Posttraumatic Stress Disorder; TPMBS = Turkish Posttraumatic Maladaptive Beliefs Scale.
*p < .05, **p < .01 (two-tailed).

Table 3. Factor Loadings, Item Means, Standard Deviations, and Corrected Item□Total Correlations of the Posttraumatic Maladaptive Beliefs Scale (PTMBS) (N = 338)

Item	Factor Loading	Mean	SD	Item-total correlation
Factor 1. Self-Worth and Judgment				
7. I trust my own judgment.*	.907	3.03	1.71	.785
3. I am a good person*	.855	2.58	1.62	.735
9. I have lost respect for myself.	.773	2.91	1.71	.632
15. I comfort myself very well when I□m upset*	.695	3.08	1.74	.589
10. I dont feel confident that I can make good decisions for myself.	.663	2.74	1.57	.550
Factor 2. Reliability and Trustworthiness of Other				
13. I feel as though I can depend on other people.*	.744	3.97	1.91	.622
6. It is possible for me to have close and loving feelings with other people.*	.743	3.36	1.95	.569
2. Other people can be genuinely loving toward me*.	.673	3.90	1.97	.530
11. Some people can be trusted.*	.636	3.57	2.00	.510
14. Most people are basically caring.*	.499	3.75	2.00	.388
Factor 3. Threat of Harm				
5. I dont trust anyone anymore.	.829	4.10	1.95	.664
1. I dont feel safe anywhere anymore.	.715	3.30	2.09	.578
8. I avoid other people because they might hurt me.	.702	3.67	1.97	.578
12. Because I dont feel able to protect myself, I have lost my sense of freedom.	.693	3.09	1.98	.569
4. The world is very dangerous.	.669	4.41	1.87	.590

*Reverse items

tively correlated with both PTSD (r=.35, p< .01) and depressive symptoms (r=.53, p<.01). Furthermore, strong positive correlations were observed among the PTMBS dimensions themselves, indicating a coherent internal structure. Item means, standard deviations, factor loadings, and corrected item–total correlations for the PTMBS are presented in Table 3.

Reliability

Reliability analyses for the total scale and dimensions of the PTMBS were conducted based on Cronbach's alpha coefficients and item–total correlations. The Cronbach's alpha coefficient for the total score of the scale was found to be .77, with item-total correlations ranging from .25 to .41. At the dimension level, the Threat of Harm dimension yielded a Cronbach's alpha of .81, with item-total correlations between .57 and .66. The Self-Worth and Judgment dimension demonstrated a Cronbach's alpha of .83, and item–total correlations ranged from .55 to .79. For the Reliability/Trustworthiness of Others dimension, Cronbach's alpha was .82, with item-total correlations varying between .39 and .62. These findings indicate that the PTMBS possesses acceptable to high internal consistency both at the total scale and dimension levels. Moreover, the fact that all item-total correlations exceeded .30 supports the reliability of the items in reflecting their respective constructs.

Finally, the Guttman Split-Half reliability coefficient was examined to further assess internal consistency. The split-half reliability coefficient was .79, and after applying the Spearman–Brown correction, the coefficient increased to .81. Taken together, these results demonstrate that the Turkish version of the PTMBS exhibits high internal consistency reliability and that the dimensions contribute consistently to the overall construct measured by the instrument.

DISCUSSION

This study examined the psychometric properties of the Turkish version of the Posttraumatic Maladaptive Beliefs Scale (PMBS). The findings indicated that the three-factor structure (Threat of Harm, Self-Worth and Judgment, and Trustworthiness of Others) was valid in a Turkish-speaking sample. The poor fit indices obtained for the single-factor model suggest that posttraumatic maladaptive cognitions cannot be adequately conceptualized as a unidimensional construct, and that a multidimensional structure provides a better representation of these beliefs. This finding is consistent with the original results reported by Vogt et al. (27) and Nickerson et al. (28). Moreover, a previous study conducted in a Turkish community sample similarly supported the three-factor structure, yielding results comparable to those of the present study (40).

Criterion-related validity analyses demonstrated that the PMBS dimensions were positively associated with both PTSD and depressive symptoms. In

particular, the moderate associations between the Threat of Harm dimension and PTSD and depressive symptoms appear to support the assumptions of Janoff-Bulman's (17,18) shattered assumptions theory, which posits that disruptions in fundamental beliefs about the safety of the world constitute a major risk factor for psychopathology following trauma. Likewise, the association between the Trustworthiness of Others dimension and psychopathological symptoms is consistent with Herman's (19) emphasis on the central role of damaged interpersonal trust in the recovery process. Taken together, these findings corroborate the core premise of Ehlers and Clark's cognitive model (23), which emphasizes the pivotal role of negative appraisals in the maintenance of posttraumatic symptoms.

The findings further indicated that the Self-Worth and Judgment dimension was associated with depressive symptoms. This result is consistent with Beck's cognitive theory (4) and with previous evidence demonstrating close links between negative self-related schemas following trauma and depressive symptomatology (20,29). However, the relatively weaker magnitude of these associations suggests that this dimension may be more strongly related to depressive symptoms than to PTSD symptoms, or that it may be more closely linked to the persistence of depression in longitudinal designs. This pattern warrants further investigation in future prospective studies.

Reliability analyses revealed high internal consistency for both the total scale score and the dimension scores. Cronbach's alpha coefficients exceeding .70 met commonly accepted thresholds for reliability in psychological measurement (41). In addition, strong split-half reliability coefficients further supported the structural consistency of the scale. These findings align with prior research indicating that the PMBS is a reliable measurement instrument across different cultural contexts (27,28).

The present findings also have important implications for clinical practice. In cognitive - behavioral therapies, the identification and restructuring of maladaptive beliefs constitute a core treatment target (43). The observed strong associations between maladaptive cognitions and both PTSD and depressive symptoms are consistent with prior studies demonstrating that changes in trauma-related cognitions precede and predict symptom improvement during treatment (24,44,45). Accordingly, the Turkish version of the PMBS offers clinicians a useful tool for assessing posttraumatic cognitive distortions and monitoring therapeutic progress.

Item–total correlation analyses for the total score indicated that Item 4 ("The world is a very dangerous place") exhibited a relatively low item-total correlation (approximately .25). This finding may be related to culturally shared meanings of threat and safety in the aftermath of collective traumas. In the Turkish cultural context, the belief that "the world is a dangerous place" may reflect a broader worldview rather than a trauma-specific maladaptive cognition unique to trauma-exposed individuals, thereby reducing item variance and its correlation with the total score. More broadly, this result suggests that the target construct, negative core assumptions about world safety, may manifest differently across cultural contexts. In collectivist societies such as Türkiye, perceptions of danger are often interpreted not solely in terms of individual vulnerability, but in relation to communal solidarity, social and institutional trust, and fatalistic beliefs. Kağıtçıbaşı's (46) model of autonomous–related self emphasizes that the self and interpersonal relationships are organized within a framework of interdependence. From this perspective, individuals may evaluate "danger" not only through personal defenselessness, but also through the availability and functioning of family, neighborhood, and social support networks. Accordingly, the Trustworthiness of Others dimension in collectivist contexts may reflect norms of communal solidarity, belongingness, and mutual support rather than the personal boundaries and individual autonomy emphasized in individualistic cultures. Such semantic shifts may have reduced the shared variance between this item (and, to some extent, the dimension) and the total score, thereby weakening the item–total correlation. Additional factors may also have contributed to the reduced discriminative power of this item, including (i) ceiling effects resulting from widespread and recent collective stressors such as earthquakes, leading to uniformly high endorsement of general danger beliefs; (ii) the

broad and absolute wording of the item ("world," "very"), which may have increased conceptual breadth and content diffusion; (iii) the limited representation of culturally salient cues (e.g., institutional trust, economic instability, disaster preparedness) within a single item; and (iv) potential response tendencies such as acquiescence or patterned responding. Together, these findings highlight the importance of examining the cultural appropriateness of scale items and the culturally specific manifestations of posttraumatic maladaptive beliefs. Future research should address these issues by testing measurement invariance across groups using multi-group confirmatory factor analysis, examining differential item functioning through item response theory analyses, and clarifying the local meaning of "danger" through cognitive interviewing techniques. Where necessary, culturally sensitive adaptations incorporating more contextual and relational components of threat (e.g., institutional trust, neighborhood support, disaster preparedness) may strengthen cultural validity. Such an integrative approach would enhance both the cultural sensitivity of the measurement tool and theoretical understanding of how posttraumatic maladaptive beliefs are structured within the Turkish context.

These findings further highlight the importance of assessing posttraumatic maladaptive beliefs in the Turkish context. Türkiye is a country with high trauma exposure due to natural disasters (e.g., the 2023 Kahramanmaraş earthquakes), migration, and collective social adversities. Evaluating trauma-related maladaptive cognitions is therefore critical not only for research purposes but also for the cultural adaptation of psychosocial interventions.

Several limitations of the present study should be acknowledged. First, the sample was recruited using convenience sampling from a specific community, which increases the risk of selection bias and limits the generalizability of the findings to other trauma types, clinical populations, or cultural contexts. Future studies should test the validity and reliability of the scale in more heterogeneous samples and across different trauma exposures. Second, the cross-sectional design precludes causal inferences. Longitudinal studies are needed to clarify whether posttraumatic maladaptive beliefs contribute to the development of psychopathology or whether ongoing symptoms reinforce these beliefs over time. Third, the presence of psychiatric diagnoses or substance use was assessed solely through self-report, without objective clinical verification, which may weaken the certainty of exclusion criteria. Fourth, only convergent validity was examined, whereas discriminant validity was not assessed. This limitation raises the possibility that the scale may capture general psychological distress rather than trauma-specific maladaptive beliefs. Future research should explicitly test discriminant validity. Fifth, data were obtained exclusively through self-report measures, introducing potential biases such as social desirability and recall bias. Incorporating clinician-rated assessments or multi-informant approaches may enhance measurement objectivity. Additionally, the sample was drawn exclusively from provinces affected by the earthquake (Hatay, Kahramanmaraş, and Adıyaman), which limits conclusions regarding the scale's psychometric performance in other trauma contexts or in the general population. Finally, test-retest reliability could not be assessed. Although temporal stability is a critical psychometric indicator for a scale measuring relatively stable posttraumatic beliefs, logistical challenges and field constraints prevented follow-up assessments. Future studies should examine temporal reliability across different time intervals. Moreover, although the Turkish version demonstrated satisfactory reliability, cross-cultural measurement equivalence has not yet been tested, highlighting the need for future measurement invariance analyses across cultural groups.

In conclusion, this study demonstrates that the Turkish version of the PMBS is both a valid and reliable instrument. The findings indicate that posttraumatic maladaptive beliefs are closely associated with PTSD and depressive symptoms, supporting the utility of this scale as a robust tool for trauma-related research and clinical assessment in Türkiye.

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Comparative evaluation of psychopathological characteristics, alexithymia, and quality of life in adolescents with somatic symptom disorder and functional neurological symptom disorder

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SUMMARY

Objective: Somatic Symptom Disorder (SSD) and Functional Neurological Symptom Disorder (FNSD) are common presentations of somatic symptom and related disorders in adolescence. Despite shared emotional and functional impairments, these disorders differ in clinical and psychological characteristics. This study aimed to compare SSD and FNSD with each other and with healthy controls in terms of clinical profiles, psychopathology, quality of life, and alexithymia.

Method: The sample consisted of 120 adolescents aged 12–17 years, equally divided into three groups: SSD (n = 40), FNSD (n = 40), and healthy controls (HC; n=40). All participants underwent comprehensive clinical assessment using the semi-structured diagnostic interview. Self-report measures included the Brief Symptom Inventory (BSI), Pediatric Quality of Life Inventory (PedsQL), and Toronto Alexithymia Scale (TAS-20). Clinical severity was rated using the Clinical Global Impression–Severity scale (CGI-S).

Results: Compared to HCs, both SSD and FNSD groups showed significantly higher symptom burden, lower health-related quality of life, and elevated alexithymia. The SSD group demonstrated higher alexithymia total scores, particularly in Difficulty Identifying Feelings and Difficulty Describing Feelings subscales, relative to both FNSD and HC groups. FNSD cases were more frequently associated with motor symptoms and neurological consultations, whereas SSD cases reported longer symptom duration and more extensive somatic evaluations.

Discussion: Although SSD and FNSD share psychosocial risk factors such as reduced quality of life and increased alexithymia, SSD is characterized by greater psychological distress and emotional unawareness. Systematic assessment of alexithymia may help refine diagnosis and guide interventions in adolescents with somatic presentations.

Key Words: Somatic symptom disorder, Functional neurological disorder, adolescents, alexithymia, psychopathology, quality of life

INTRODUCTION

Somatic symptom disorder (SSD) is characterized by the presence of one or more physical symptoms in the absence of an identifiable organic condition. Core features include persistent and disproportionate concerns and thoughts about these symptoms, excessive time and energy devoted to them, and significant impairment in daily functioning and psychosocial well-being (1,2). Similarly, functional neurological symptom disorder (FNSD)—formerly referred to as conversion disorder—is characterized by motor, sensory, or cognitive symptoms that are increasingly understood to arise from altered functioning within brain networks, rather than from any detectable structural damage or lesion in

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the nervous system. In the DSM-5, SSD and FNSD has been reclassified under the broader category of Somatic Symptom and Related Disorders (SSRD), along with illness anxiety disorder (IAD) and factitious disorder (FD). According to the DSM-5, these symptoms do not necessarily have to be medically unexplained; rather, the emphasis is placed on the individual's maladaptive response to the symptoms (1).

Building on this diagnostic framework, recent empirical research has provided valuable insights into the prevalence and clinical presentation of SSD and FNSD in pediatric populations. A recent meta-analysis investigating the prevalence of somatoform symptoms and disorders in children and adolescents reported a global prevalence of 31% for somatoform symptoms and 3.3% for somatoform disorders (3). In the literature, the most frequently reported somatic symptoms among children and adolescents with SSD are headache and abdominal pain, with adolescents in particular tending to experience multiple somatic complaints (4,5). Furthermore, factor analyses conducted in general populations have identified three main clusters of somatic symptoms: gastrointestinal, pain-related, and cardiopulmonary (6). In contrast, the most commonly reported presentations of FNSD among children and adolescents include dystonia, motor weakness, abnormal gait, psychogenic non-epileptic seizures (PNES), and sensory disturbances (7). Both disorders are reported to be more prevalent during adolescence and among females (7,8). Indeed, a five-year prospective cohort study investigating predictive factors associated with the persistence of functional somatic symptoms in adolescents identified female sex, the presence of baseline depressive symptoms, and initial presentation with multiple somatic complaints as significant predictors (9).

Both SSD and FNSD can have serious consequences at the individual, familial, and ultimately societal levels. Both disorders are closely associated with increased risk of internalizing symptoms, low self-esteem, poor social adjustment, and avoidance of school and social activities, all of which can lead to significant functional impairment (7,10). Recurrent hospital visits are often associated with school absenteeism and a decline in academic achievement. Among adolescents and their families, a lack of a clear explanatory framework and the persistent belief that “something has been overlooked” may intensify feelings of frustration, anger, and mistrust. At the same time, receiving explicit or implicit messages that the symptoms are “purely psychological” may reinforce the adolescent’s sense of being dismissed and devalued (11). Furthermore, frequent emergency room visits, hospitalizations, diagnostic overutilization, and increased medicalization place a growing financial burden on healthcare systems and may exacerbate inequities in service delivery, particularly in resource-limited countries (8). For these reasons, it is of critical importance to recognise these disorders early and take a comprehensive approach that addresses not only somatic symptoms, but also psychological and contextual factors.

Although SSD and FNSD are classified under the same overarching category, they differ significantly in terms of symptom characteristics, assessment criteria, and diagnostic approaches. In SSD, the commonly observed symptoms are often chronic, widespread, and subjective, leading individuals to seek medical care frequently and repeatedly. In contrast, FNSD typically presents with observable neurological symptoms and is often classified as a neurological emergency due to its sudden and dramatic onset, with diagnosis relying on the clinical inconsistency of motor or sensory findings during neurological examination—such as a positive Hoover sign. While individuals with SSD generally experience significant distress related to their symptoms, those with FNSD may demonstrate a striking lack of concern about the severity of their symptoms—a presentation often referred to in the literature as "la belle indifférence." In addition, comorbidity with psychosomatic disorders appears to be more prevalent in SSD than in FNSD (12). Furthermore, studies examining the differences between these two disorders in terms of sociodemographics, clinical features, phenomenology and psychosocial factors remain notably limited in the current literature.

Alexithymia is a multifaceted construct involving difficulties in identifying, describing, and expressing emotions; challenges in distinguishing between emotions and bodily sensations; and a tendency towards externally oriented thinking. Individuals with clinically significant levels of alexithymia are

considered to be particularly vulnerable to psychiatric disorders characterized by affective dysregulation, due to persistent difficulties in processing and regulating emotions at the cognitive level, impairments in perspective-taking and understanding others' emotional states, and reduced awareness of internal bodily sensations (13). Several studies have shown that patients diagnosed with FNSD exhibit higher levels of alexithymia compared to healthy controls (14,15), while other studies have reported similar findings in patients with SSD (16–18). Furthermore, alexithymia has been associated with symptom severity and the clinical course, particularly in patients with SSD (16). In addition, comorbid psychiatric symptoms are thought to play a mediating role in the association between alexithymia and the severity of somatic symptoms (19,20). Alexithymia has also been shown to negatively affect quality of life, both directly and indirectly through co-occurring psychiatric symptoms (21,22). Although psychopathology, alexithymia, and quality of life have each been examined separately, studies that address these constructs together in an integrative and comparative framework are still limited in pediatric populations. Given the limited number of studies in this field, our findings contribute valuable insights into the identification of both shared and distinct features of these disorders in adolescents, particularly highlighting the role of quality of life and alexithymia as potentially important differentiating variables. Findings from such work have important implications for both diagnostic processes and the development of targeted therapeutic strategies.

Building upon these findings, the primary aim of this study is to conduct a comparative analysis of alexithymia, quality of life, and psychiatric symptoms among adolescents with SSD, FNSD, and healthy controls. A secondary aim is to provide a comprehensive evaluation of the sociodemographic and clinical characteristics of the study groups.

METHODS

Study Design and Ethics

This cross sectional case-control study is conducted with the ethical approval of the Ethics Committee of Usak University (Date: April 10, 2025/No: 617-617-15). This study has been carried out following the Declaration of Helsinki. Comprehensive verbal information was provided regarding the study's methodology and procedures, participants' responsibilities, the duration of the assessment, the potential benefits and risks, and the confidentiality of the data. Written informed consent was also obtained from all the adolescents and their parents.

Participants, Inclusion and Exclusion Criteria

The study sample consisted of 120 adolescents aged between 12 and 17 years, divided into three groups: 40 participants diagnosed with Somatic Symptom Disorder (SSD), 40 diagnosed with Functional Neurological Disorder (FNSD), and 40 age- and gender-matched healthy controls (HC).

The case groups consisted of adolescents who presented to the Child and Adolescent Psychiatry Outpatient Clinic of X and Y Research and Training Hospitals between April 15, 2025, and July 25, 2025 and were diagnosed with SSD or FNSD based on DSM-5 criteria. The control group included age matched 40 healthy children between the ages of 12 and 17 who did not have any chronic disease and did not receive any psychiatric diagnosis after the diagnostic evaluation. The healthy control group consisted of adolescents who had applied to these clinics for general psychiatric counselling, including support for families regarding typical developmental issues of adolescence, as well as guidance related to acute stressors, such as family disagreements or difficulties in peer or romantic relationships. They underwent a semi-structured psychiatric interview to confirm that they did not meet the diagnostic criteria for any psychiatric disorder. Participants who presented to the child and adolescent psychiatry outpatient clinics during the specified recruitment period and met the inclusion criteria were consecutively included in the study. Recruitment for each clinical group and the healthy control group, was terminated once the target sample size of 40 had been reached. This study did not include participants with a diagnosis of any neurological or chronic medical condition, or other psychiatric disorders such as psychotic disorders, bipolar disorder, neurodevelopmental disorders.

Data Collection

After obtaining informed consent, the parents of the participants were interviewed using a sociodemographic and clinical data form developed by the researchers. This form gathered information on the child's age and gender, parental age and education levels, presence of physical or psychiatric illnesses in the family.

All participants underwent a comprehensive psychiatric diagnostic assessment using the Kiddie Schedule for Affective Disorders and Schizophrenia – Present and Lifetime Version (K-SADS-PL).

In the FNSD and SSD groups information regarding the nature of symptoms, age of symptom onset, duration of symptoms, medical departments visited due to symptoms, as well as invasive and non-invasive diagnostic procedures and treatments administered, was collected through a review of hospital records and patient-reported data.

In both the FNSD and SSD groups, illness severity was also assessed using the Clinical Global Impression–Severity scale (CGI-S). The CGI-S is a clinician-rated instrument that evaluates the overall severity of a patient's mental illness on a 7-point scale, ranging from 1 (normal, not at all ill) to 7 (among the most extremely ill patients). This scale provides a standardized measure of global symptom burden based on the clinician's impression following diagnostic evaluation and clinical observations (23). All CGI-S assessments were conducted by a single clinician for each participant.

Following the completion of clinical evaluations, all participants were administered the Brief Symptom Inventory (BSI), the Pediatric Quality of Life Inventory (PedsQL), and the 20-item Toronto Alexithymia Scale (TAS-20) as standardized self-report measures to quantitatively assess psychopathological symptom burden, health-related quality of life, and alexithymic features, respectively.

K-SADS-PL is a semi-structured diagnostic interview designed to assess current and past psychiatric disorders in children and adolescents. Originally developed by Kaufman et al. (24) and updated for DSM-5 in 2016, its Turkish adaptation was validated by Ünal et al. (25).

The Brief Symptom Inventory (BSI) is a multidimensional self-report measure developed to screen for a range of psychological symptoms in both adult and adolescent populations (26), and has been shown to be a reliable and valid tool in Turkish samples (27). It includes five subscales: Anxiety, Depression, Negative Self, Somatization, and Hostility. Higher total scores reflect greater levels of psychological distress.

The revised version of the Toronto Alexithymia Scale (TAS-20), developed by Bagby et al. (28), consists of 20 items rated on a five-point Likert scale and assesses three dimensions of alexithymia: Difficulty Identifying Feelings (DIF), Difficulty Describing Feelings (DDF), and Externally Oriented Thinking (EOT). Higher total scores reflect greater alexithymic traits. The Turkish adaptation and validation of the scale was conducted by Gulec et al. (29), demonstrating acceptable psychometric properties for use in adolescent and adult populations.

The Pediatric Quality of Life Inventory (PedsQL) is used to assess health-related quality of life in children and adolescents (30). The scale includes Physical Health Summary Score (PHSS) and Psychosocial Health Summary Score (PSHSS), with higher scores indicating better quality of life. Turkish adaptation and validation of the scale was conducted by Memik et al. (31), demonstrating acceptable psychometric properties for use in adolescent population.

Statistical Analysis

Since no prior study with an identical design was found in the literature, a priori power analysis was conducted using GPower 3.1.9.7*. Assuming a medium effect size (f = 0.30), an alpha level of 0.05, and a statistical power of 0.80, the required sample size for a one-way ANOVA comparing three groups ("ANOVA: Fixed effects, omnibus,

Table 1: Comparison of demographic characteristics between groups

		SSD (N = 40)	FNSD (N = 40)	HC (N = 40)	Statistics*		Post-hoc Differences
					KW *or* x†	p	
Age *(years)*	Mean (SD)	15.2 (1.8)	15.3 (1.3)	14.6 (1.9)	2.987[a]	.225	-
	Mdn (IQR)	16.0 (3.0)	15.0 (1.0)	14.0 (4.0)			
Gender, *n (%)*							
Girl		33 (82.5%)	37 (92.5%)	25 (62.5%)	11.318[b]	.003	SSD = FNSD>HC
Boy		7 (17.5%)	3 (7.5%)	15 (37.5%)			
Mother□s Age *(years)*	Mean (SD)	45.2 (5.4)	43.0 (4.3)	43.5 (4.2)	5.430[a]	.066	-
	Mdn (IQR)	45.0 (9.0)	42.0 (7.0)	43.0 (3.0)			
Mother□s Education *(years)*	Mean (SD)	9.6 (3.9)	10.4 (4.5)	10.6 (4.7)	1.419[a]	.492	-
	Mdn (IQR)	12.0 (7.0)	12.0 (11.0)	12.0 (11.0)			
Father□s Age *(years)*	Mean (SD)	47.6 (5.1)	45.1 (4.3)	47.7 (5.5)	5.304[a]	.071	-
	Mdn (IQR)	48.0 (8.0)	45.0 (5.0)	45.0 (11.0)			
Father□s Education *(years)*	Mean (SD)	11.6 (3.1)	11.6 (3.7)	11.6 (4.2)	.144[a]	.930	-
	Mdn (IQR)	12.0 (0.0)	12.0 (8.0)	12.0 (8.0)			
Family History with Psychiatric Illness, *n (%)*		16 (40.0%)	28 (70.0%)	1 (2.5%)	39.040[b]	≤.001	FNSD>SSD >HC

Note. SD = standard deviation, Mdn = median, IQR = Interquartile range. *Continuous variables were analyzed using the Kruskal-Wallis test (a), and categorical variables were analyzed using Pearson's Chi-Square test (b). A Bonferroni correction was applied to adjust for multiple comparisons across groups (alfa = .05/3 = .017).

one-way") was calculated as 111 participants. With a total of 120 participants included in the study, the statistical power was calculated to be 0.83.

All statistical analyses were conducted using the Statistical Package for the Social Sciences (SPSS) version [26.0] and Stata version [17.0]. Descriptive statistics were calculated and reported as mean (M), standard deviation (SD), median (mdn), and interquartile range (IQR) for continuous variables, and frequency with percentages (%) for categorical variables. The normality of distribution was assessed using the Shapiro–Wilk test. Between-group differences for continuous variables were examined using the Kruskal–Wallis test due to non-normally distributed data. Post-hoc pairwise comparisons were performed using the Mann–Whitney U test with a Bonferroni correction to control for Type I errors (adjusted alpha: $\alpha = .05/3 = .017$). Categorical variables were analyzed using Pearson's Chi-square test, and Fisher's Exact test was applied when expected frequencies were below 20.0%. Significant overall results from categorical analyses were followed by post-hoc comparisons, also adjusted using Bonferroni correction. Effect sizes were computed to evaluate the practical significance of the observed differences. Cohen's d values were calculated for pairwise group comparisons, interpreted as small (0.20), medium (0.50), and large (0.80). Odds ratios (OR) were also calculated for categorical comparisons where applicable.

RESULTS

Table 1 presents the demographic characteristics of the SSD, FNSD, and HC groups. Groups did not differ significantly in mean age. However, gender distribution varied significantly ($p = .003$), with more girls in the SSD (82.5%) and FNSD (92.5%) groups than in the HC group (62.5%). No significant group differences were found in parental age or education. Family history of psychiatric illness differed significantly across groups ($p < .001$), being most prevalent in the FNSD group (70.0%), followed by SSD (40.0%) and HC (2.5%).

Figure 1 illustrates symptom profiles in the SSD and FNSD groups. In the SSD group, the most common symptoms were abdominal pain (27.5%), nausea (25.0%), shortness of breath (25.0%), and

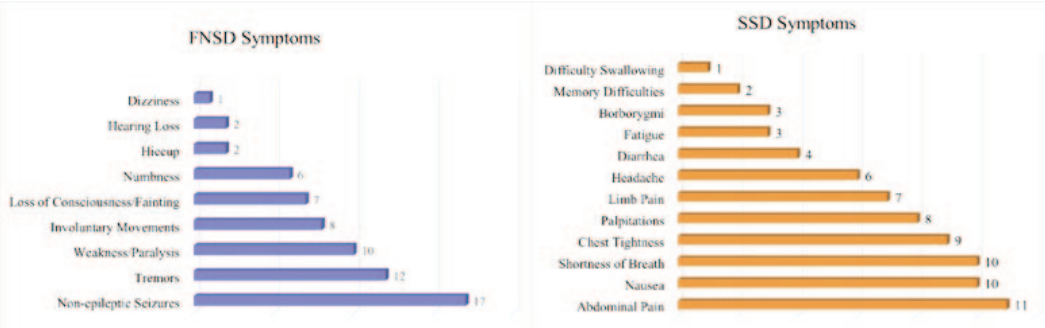

Figure 1: Nature of Symptoms

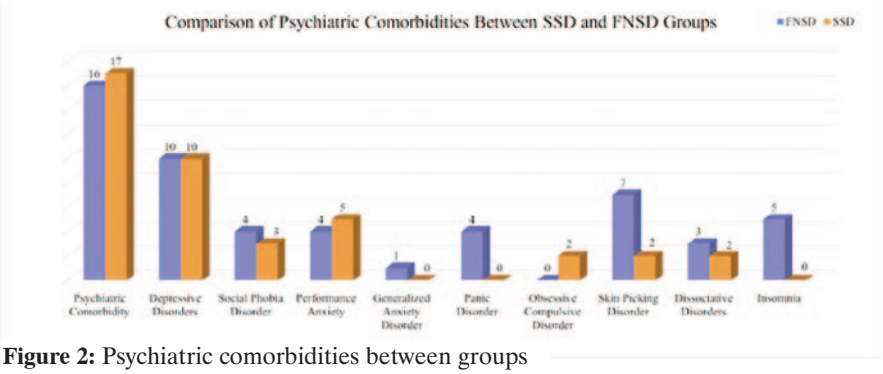

Figure 2: Psychiatric comorbidities between groups

chest tightness (22.5%), with less frequent complaints including palpitations, limb pain, headache, and others. In the FNSD group, non-epileptic seizures (42.5%), tremors (30.0%), weakness/paralysis (25.0%), and involuntary movements (20.0%) were most prevalent, followed by less common symptoms such as fainting, numbness, and dizziness.

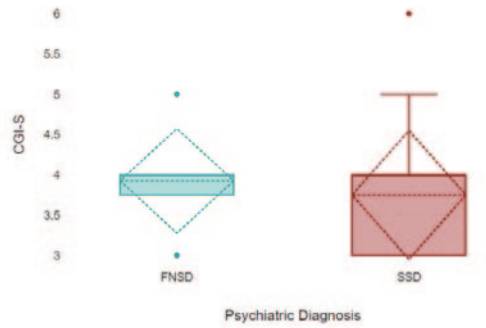

Figure 3: Distribution of Clinical Global Impression-Severity (CGI-S) Scores in SSD and FNSD Groups

Figure 2 displays psychiatric comorbidities in the SSD and FNSD groups, with similar overall rates (SSD: 42.5%, FNSD: 40.0%) and identical prevalence of depressive disorders (25.0%). Social phobia and performance anxiety were comparably distributed. Certain conditions—panic disorder, GAD, dissociative disorders, skin picking, and insomnia—were observed only in the FNSD group, whereas OCD appeared only in the SSD group. However, none of these differences reached statistical significance ($p > .05$). A comprehensive description of psychiatric comorbidity patterns and overall comorbidity burden in the SSD and FNSD groups is presented in Table 4.

Table 2 summarizes clinical comparisons between the SSD and FNSD groups. While age of symptom onset did not differ significantly (p=.296), symptom duration was longer in the SSD group (M=7.9 months) than in the FNSD group (M=4.4 months; $p < .001$). Clinic visits varied notably: FNSD cases

Table 2: Comparison of clinical characteristics between groups

		SSD (N = 40)	FNSD (N = 40)	Statistics*		Effect Size
				U/Z *or* x†	p	
Age of Symptom Onset *(years)*	Mean (SD)	14.5 (1.8)	14.9 (1.3)	691.5 / -1.045[a]	.296	-
	Mdn (IQR)	15.3 (2.9)	14.8 (1.4)			
Duration of Symptoms *(months)*	Mean (SD)	7.9 (2.2)	4.4 (1.9)	143.5 / -6.373[a]	<001	Cohen d = 1.70
	Mdn (IQR)	8.0 (3.0)	4.0 (2.0)			
Clinics Visited Due to Symptoms, *n (%)*	Pediatrics	38 (95.0%)	40 (100.0%)	-[b]	.494	-
	Pediatric Neurology	7 (17.5%)	36 (90.0%)	42.288[c]	<001	OR = 42.43
	Pediatric Gastroenterology	13 (32.5%)	-	15.522[c]	<001	OR = 39.76
	Pediatric Cardiology	7 (17.5%)	-	7.671[c]	.006	OR = 18.13
	Pediatric Surgery	8 (20.0%)	-	8.889[c]	.003	OR = 21.19
	Otolaryngology	-	6 (15.0%)	-[b]	.026	OR = 15.15
Emergency Department Visit Due to Symptoms, *n (%)*		27 (67.5%)	32 (80.0%)	1.614[c]	.204	-
Number of Emergency Department Visits Due to Symptoms				766.0 / -332[a]	.740	-
Non-invasive Diagnostic Procedures Performed Due to Symptoms, *n (%)*	Blood Test	38 (95.0%)	40 (100.0%)	-[b]	.494	-
	Urine Analysis	7 (17.5%)	-	-[b]	.012	OR = 18.13
	Audiometry	-	2 (5.0%)	-[b]	.494	-
	EEG	-	17 (42.5%)	21.587[c]	<001	OR = 60.32
	X-ray	15 (37.5%)	2 (5.0%)	12.624[c]	<001	OR = 11.40
	CT	14 (35.0%)	34 (85.0%)	20.833[c]	<001	OR = 10.52
	MRG	6 (15.0%)	38 (95.0%)	51.717[c]	<001	OR = 107.67
	USG	22 (55.0%)	-	30.345[c]	<001	OR = 98.51
Invasive Diagnostic Procedures Performed Due to Symptoms, *n (%)*	Endoscopy	5 (12.5%)	-	-[b]	.055	-
	Colonoscopy	4 (10.0%)	-	-[b]	.116	-
	Appendectomy	2 (5.0%)	-	-[b]	.494	-
Medical Treatments Performed Due to Symptoms, *n (%)*		36 (90.0%)	37 (92.5%)	-[b]	1.000	-

Note. SD = standard deviation, Mdn = median, IQR = Interquartile range. *Continuous variables were analyzed using the Mann - Whitney U test (a), and categorical variables were analyzed using Fisher's Exact Test (b) or Pearson Chi-square Test (c).

Table 3: Comparison of BSI, PedsQL, and TAS-20 subscales scores between groups

		SSD (N = 40)		FNSD (N = 40)		HC (N = 40)		Statistics*		Cohen d	Post-hoc Differences
		Mean (SD)	Mdn (IQR)	Mean (SD)	Mdn (IQR)	Mean (SD)	Mdn (IQR)	KW	p		
BSI	Somatization	2.2 (1.1)	2.2 (1.9)	1.4 (0.8)	1.4 (0.9)	0.3 (0.3)	0.3 (0.3)	60.445	<001	d1=0.83, d2=2.36, d3=1.82	SSD>FNSD>HC
	Obsessive-Compulsive	2.5 (1.0)	2.5 (1.5)	1.7 (0.9)	1.7 (1.5)	0.8 (0.5)	0.7 (0.5)	51.229	<001	d1=0.84, d2=2.15, d3=1.24	SSD>FNSD>HC
	Interpersonal Sensitivity	2.4 (1.1)	2.5 (1.6)	2.0 (1.2)	2.2 (2.0)	0.5 (0.6)	0.2 (0.3)	45.873	<001	d1=0.35, d2=2.14, d3=1.58	SSD=FNSD>HC
	Depression	2.5 (1.1)	2.5 (1.6)	2.1 (1.1)	2.2 (1.3)	0.5 (0.4)	0.4 (0.4)	55.154	<001	d1=0.36, d2=2.42, d3=1.93	SSD=FNSD>HC
	Anxiety	2.3 (1.1)	2.5 (1.8)	1.6 (1.1)	1.0 (1.3)	0.4 (0.3)	0.3 (0.3)	62.074	<001	d1=0.64, d2=2.36, d3=1.49	SSD>FNSD>HC
	Hostility	2.7 (1.1)	3.2 (2.0)	2.4 (1.1)	2.5 (2.2)	0.6 (0.5)	0.6 (0.5)	62.939	<001	d1=0.27, d2=2.46, d3=2.11	SSD=FNSD>HC
	Phobic Anxiety	1.6 (1.0)	1.8 (1.4)	1.1 (0.9)	1.0 (1.2)	0.3 (0.4)	0.2 (0.4)	41.194	<001	d1=0.53, d2=1.71, d3=1.15	SSD=FNSD>HC
	Paranoid Ideation	2.3 (1.2)	2.8 (1.4)	1.6 (1.1)	1.4 (1.2)	0.6 (0.5)	0.4 (0.7)	43.291	<001	d1=0.61, d2=1.85, d3=1.17	SSD>FNSD>HC
	Psychoticism	2.0 (1.1)	2.0 (2.0)	1.4 (0.9)	1.2 (1.6)	0.4 (0.4)	0.2 (0.4)	52.659	<001	d1=0.6, d2=1.93, d3=1.44	SSD=FNSD>HC
	GSI	2.3 (0.9)	2.5 (1.6)	1.7 (0.8)	1.7 (1.2)	0.5 (0.3)	0.4 (0.2)	62.104	<001	d1=0.7, d2=2.68, d3=1.99	SSD>FNSD>HC
	PSDI	120.3 (50.3)	135.5 (83.2)	88.8 (45.3)	88.0 (65.0)	25.6 (17.4)	20.0 (13.0)	62.345	<001	d1=0.66, d2=2.52, d3=1.84	SSD>FNSD>HC
	PST	2.7 (0.7)	3.0 (1.1)	2.2 (0.7)	2.0 (1.1)	1.2 (0.3)	1.2 (0.5)	61.972	<001	d1=0.71, d2=2.79, d3=1.86	SSD>FNSD>HC
PedsQL	Total	50.0 (19.7)	47.8 (29.4)	61.5 (19.2)	65.2 (17.4)	83.4 (8.0)	83.7 (16.3)	55.243	<001	d1=0.59, d2=2.22, d3=1.49	HC>SSD=FNSD
	PHSS	54.0 (22.8)	53.1 (35.2)	66.0 (19.6)	70.3 (23.4)	88.0 (4.6)	87.5 (9.4)	54.248	<001	d1=0.56, d2=2.07, d3=1.55	HC>FNSD>SSD
	PSHSS	47.8 (19.4)	45.0 (29.2)	59.0 (20.3)	60.0 (29.2)	81.0 (10.3)	82.5 (20.0)	52.393	<001	d1=0.56, d2=2.14, d3=1.37	HC>SSD=FNSD
TAS-20	Total	63.9 (12.4)	61.0 (12.2)	59.7 (10.0)	61.5 (13.5)	42.5 (6.5)	42.0 (8.0)	61.682	<001	d1=0.37, d2=2.16, d3=2.04	SSD=FNSD>HC
	DIF	25.1 (7.0)	27.0 (11.0)	20.4 (7.1)	20.0 (12.2)	9.9 (3.9)	9.0 (4.2)	66.485	<001	d1=0.67, d2=2.68, d3=1.83	SSD>FNSD>HC
	DDF	17.5 (4.7)	19.0 (7.2)	14.7 (4.3)	14.0 (7.0)	9.8 (2.6)	9.5 (3.0)	49.684	<001	d1=0.62, d2=2.03, d3=1.38	SSD>FNSD>HC
	EOT	23.0 (7.6)	23.5 (7.2)	24.6 (3.0)	25.0 (3.0)	22.8 (3.4)	24.0 (3.0)	5.480	.065	d1=0.28, d2=0.03, d3=0.56	-

Note. SD=standard deviation, Mdn=median, IQR=Interquartile range, BSI=Brief Symptom Inventory, DIF=Difficulty Identifying Feelings, DDF=Difficulty Describing Feelings, EOT = Externally Oriented Thinking, TAS -20=Toronto Alexithymia Scale-20, PedsQL = Pediatric Quality of Life Inventory, PHSS = Physical Health Summary Score, PSHSS = Psychosocial Health Summary Score * Kruskal-Wallis test was used. A Bonferroni correction was applied to adjust for multiple comparisons across groups (alfa=.05/3 =.017). Cohen's d values represent effect sizes between groups; specifically, d1 indicates the effect size between the SSD and FNSD groups, d2 indicates the effect size between the SSD and HC groups, and d3 indicates the effect size between the FNSD and HC groups.

more frequently visited Pediatric Neurology (90.0% vs. 17.5%), whereas SSD cases more commonly consulted Pediatric Gastroenterology, Cardiology, and Surgery (all $p < .01$). Diagnostic procedures such as urine analysis, X-ray, and ultrasound were more frequent in the SSD group, while EEG, CT, and MRI were more common in the FNSD group (all $p < .001$). Otolaryngology consultations occurred only in the FNSD group ($p = .026$). No group differences were found for emergency visits, number of visits, invasive procedures, or medical treatments (all $p > .05$).

Figure 3 displays the CGI-S scores for the SSD and FNSD groups. Both had the same median (4), reflecting comparable central symptom severity. The SSD group had a slightly lower mean (M= 3.75, SD=0.81) than the FNSD group (M=3.93, SD=0.66), with greater variability (range=3–6, IQR=1 vs. range=3–5, IQR=1) and visible outliers, indicating a wider severity spectrum. The difference between groups was not statistically significant ($p = .179$).

Table 3 summarizes group comparisons for BSI, PedsQL, and TAS-20 subscales. SSD scored significantly higher than FNSD and HC on all BSI subscales except TAS-20-EOT ($p = .065$), particularly on Somatization, Obsessive-Compulsive, Anxiety, Paranoid Ideation, GSI, PSDI, and PST. Interpersonal Sensitivity, Depression, Hostility, Phobic Anxiety, and Psychoticism were elevated in both SSD and FNSD compared to HC. PedsQL Total and PSHSS scores were highest in HC, followed by FNSD and SSD (all $p < .001$). TAS-20 total scores showed greater alexithymia in SSD and FNSD than HC ($p < .001$), with SSD > FNSD > HC on DIF and DDF (both $p < .001$). EOT scores did not differ significantly ($p=.065$).

Table 4: Comprehensive description of psychiatric comorbidity patterns and comorbidity burden in SSD and FNSD groups

	Patient-level psychiatric comorbidity patterns (all observed patterns)	Number of comorbid diagnoses per participant, n (%)	Mean (SD)	Median (Min–Max)
SSD	DD; OCD; PA-only; SPD; PA-only + OCD; DD + Dissociative Disorder; DD + SPD + Dissociative Disorder	1 diagnosis: 12 (63.2%) 2 diagnoses: 5 (26.3%) 3 diagnoses: 2 (10.5%)	1.47 (0.77)	1 (1–3)
FNSD	DD + SPD; SAD + Insomnia; SAD + DD; DD + Dissociative Disorder; DD + Dissociative Disorder + Panic Disorder; DD + Dissociative Disorder + PA-only; DD + Panic Disorder + SPD; Panic Disorder + SPD + Insomnia; SPD + PA-only; GAD + Panic Disorder; PA-only + Insomnia; PA-only + OCD + Insomnia	1 diagnosis: 0 (0%) 2 diagnoses: 6 (42.9%) 3 diagnoses: 8 (57.1%)	2.43 (0.65)	3 (2–3)

Note. SSD = Somatic Symptom Disorder; FNSD = Functional Neurological Symptom Disorder; DD = Depressive Disorders; OCD = Obsessive–Compulsive Disorder; SAD = Social Anxiety Disorder; SPD = skin picking disorder; PA -only = performance anxiety-only; GAD = Generalized Anxiety Disorder; SD = standard deviation.

DISCUSSION

In this study, we conducted a comparative analysis of adolescents with SSD, FNSD, and HC in terms of sociodemographic and clinical features, psychiatric comorbidities and symptoms, quality of life, and alexithymia. Both the SSD and FNSD groups exhibited elevated levels of alexithymia compared to HC. However, adolescents with SSD showed significantly higher alexithymic characteristics than those with FNSD, particularly in terms of difficulty in identifying and describing feelings. SSD group also showed a significantly lower quality of life and an overall greater burden of psychiatric symptoms. Notably, the elevated psychiatric symptom burden in the SSD group was particularly pronounced in specific domains, such as somatization, obsessive-compulsive symptoms, and anxiety. A predominance of the female gender was observed in both clinical groups. The SSD group had a longer symptom duration, whereas a positive family history of psychiatric disorder was significantly more prevalent in the FNSD group compared to the SSD and HC groups.

Consistent with our findings, alexithymia has consistently emerged as a relevant construct in relation to both FNSD and SSD (32,33). On the other hand, to our knowledge, no previous study has directly compared alexithymia in adolescents with SSD and FNSD, nor reported higher alexithymic traits in the SSD group. One possible explanation for this finding is that alexithymia-related characteristics, such as reduced awareness of bodily sensations and an exaggerated, selective and inaccurate interpretation of these sensations during emotional arousal, may increase vulnerability to the development of somatic symptoms (33). Consistent with this argument, previous neurobiological studies have suggested that alexithymia and chronic somatic symptoms share vulnerabilities related to emotional awareness, the processing of internal sensations, and self-regulation (34). At the same time, electrophysiological studies have proposed a possible mismatch, referred to as 'decoupling,' between subjective emotional experiences and physiological stress responses, particularly in individuals who have difficulty identifying and expressing their emotions. Although these individuals may exhibit increased anticipatory anxiety in response to environmental stressors, they may not exhibit physiological stress responses when encountering the stressor directly. This dissociation between emotional and physiological processes has been suggested to coincide with exaggerated or inaccurate interpretations of internal states and insufficient cognitive processing of emotional distress (35,36). The classical phenomenon of 'la belle indifférence,' traditionally associated with FNSD, may provide an additional framework for understanding the comparatively lower alexithymia scores observed in this group. In one of the few studies examining this phenomenon, individuals with la belle indifférence demonstrated physiological arousal in response to emotionally evocative stimuli despite limited subjective emotional awareness, a pattern interpreted as reflecting the operation of psychological defense mechanisms, particularly suppression (37). These psychophysiological findings may help contextualize our findings of comparatively lower alexithymia scores in the FNSD group. Rather than reflecting a

primary deficit in identifying or describing emotions, they may suggest that emotional arousal in FNSD could remain predominantly at the physiological level and be modulated through defense mechanisms, potentially limiting its translation into conscious emotional awareness. Given that the present study did not directly examine neurobiological or psychophysiological mechanisms, these interpretations should be considered theoretical possibilities rather than explanatory mechanisms. Although alexithymia levels were comparatively higher in the SSD group, alexithymia represents a transdiagnostic risk factor across SSRD and may contribute meaningfully to both diagnostic assessment and the development of effective therapeutic strategies.

Despite comparable psychiatric comorbidity rates, the SSD group exhibited a greater burden of psychiatric symptoms, longer symptom duration, and lower perceived quality of life, highlighting a more severe clinical presentation and greater functional impairment in adolescents with SSD. A tendency to experience multiple somatic symptoms in adolescents, disproportionate and exaggerated thoughts about these symptoms, and illness behaviors reinforced by frequent medical consultations may be associated with longer symptom duration in SSD (8,10). The high psychiatric symptom burden, frequently reported in the literature and consistently observed in our sample, may further complicate the clinical course (38, 39). In this context, the lower perceived quality of life among adolescents with SSD is not unexpected, considering the chronic nature of the symptoms and the associated elevated levels of psychiatric distress. On the other hand, the sudden-onset, neurologically appearing symptoms observed in FNSD are typically perceived as acute medical conditions, prompting more immediate referral to healthcare services. Conversely, the more common and non-specific symptoms seen in SSD, such as headache or abdominal pain, may be perceived as less urgent or serious by both families and clinicians. This perception may contribute to delays in diagnosis and a more persistent course of symptoms. Given these considerations, early identification and intervention in SSD are essential in preventing chronicity and minimizing functional impairment. Raising awareness of SSD symptoms among both families and healthcare professionals is crucial to ensuring timely and effective clinical management.

Interestingly, although the FNSD group had a higher prevalence of anxiety-related psychiatric comorbidities, the SSD group reported higher levels of anxiety symptoms. Clinician-made psychiatric diagnoses require symptoms of a certain duration and intensity and reflect a current or lifelong clinical picture. In contrast, self-report instruments reflect an individual's current subjective experience and perceived symptom severity. In this context, particularly in the SSD group, excessive focus on bodily sensations, disproportionate and exaggerated thoughts about these symptoms, and health-related anxiety may have led to an increased perception of anxiety and higher scores on the self-report scales. Furthermore, the higher prevalence of anxiety-related psychopathologies in the FNSD group may have increased the likelihood of these patients receiving psychotherapy or psychotropic medication, which may have contributed to the relatively lower anxiety scores on self-report measures. Additionally, consistent with the findings of our study, longer symptom duration, frequent medical visits, and lower perceived quality of life were observed in the SSD group. These clinical characteristics suggest that general psychological distress may be higher in SSDs, and thus higher scale scores may be related to untreated or inadequately treated anxiety symptoms. Finally, self-report bias, a fundamental limitation of self-report instruments, may also have contributed to this finding. For these reasons, it is possible to conclude that the differences between diagnostic categories and self-reported symptom levels may reflect the combined influence of methodological factors, the current clinical presentation, and treatment-related variables.

A marked female predominance was observed in both the SSD and FNSD groups. This finding is consistent with previous literature indicating that both disorders are more frequently diagnosed in females during adolescence (4,7). The literature emphasizes that internalizing symptoms can serve as both a risk factor for and a potential outcome of somatic complaints (40). Since females are particularly prone to internalizing psychopathology during adolescence, it can be concluded that female adolescents represent a more vulnerable group for SSRD (40, 41). Another possible explanation for the observed sex differences is the hormonal fluctuations associated with the onset of menstruation, which are thought to increase pain sensitivity (42).

This increased sensitivity may, in turn, may provide a biological basis for the development of somatic symptoms. From a sociocultural perspective, especially in societies shaped by traditional norms, expressing emotions is often discouraged; indeed, it has been noted that some languages even lack specific vocabulary for certain emotional states. Within such cultural contexts, females may be disproportionately exposed to pressures arising from rigid socioeconomic and gender role expectations. They are often expected to be compliant, calm, and reserved, and are frequently given domestic responsibilities at a young age. Such sociocultural constraints may hinder the direct communication of emotional distress, thereby increasing the likelihood of its manifestation through somatic symptoms (43).

Despite the valuable findings of this study, several limitations should be acknowledged. First, the cross-sectional design does not allow for conclusions about causality. The sample was drawn from a single geographic region, which may limit the generalizability of the findings. Data were collected through self-report measures, which may be particularly prone to bias in adolescents with limited emotional awareness, such as those with alexithymic traits. The overrepresentation of female participants is another factor that may restrict the generalizability of the findings. Another limitation of this study is the gender imbalance between the groups. Both the FNSD and SSD groups have a higher proportion of females compared to the healthy control group. This discrepancy introduces a potential confounding factor, as comparisons between patient groups and controls may reflect gender differences rather than disorder-specific effects. Future studies should aim to ensure comparable gender distributions across all groups to strengthen the validity of group comparisons. Furthermore, although physical trauma was assessed, other trauma types—such as emotional, sexual, or neglect-related experiences—were not systematically investigated. Future research should aim to evaluate trauma history in greater depth, ensure a more balanced gender distribution, and incorporate multi-informant data sources to enhance the robustness of findings. In addition, as each participant was evaluated by a single clinician, inter-rater reliability could not be calculated, which is a methodological limitation of single-rater assessment protocols. Finally, the absence of assessments targeting social cognition, particularly performance-based tasks and multi-informant measures, represents another important limitation of the study, given the relevance of these measures to alexithymia and somatic symptom expression. Incorporating behavioral tasks and parent reports in future research would strengthen the validity and interpretability of the findings.

This study highlights both overlapping and distinct features of SSD and FNSD in adolescents. Shared characteristics include a predominance of female gender, a family history of psychiatric disorders, higher alexithymic characteristics, and impairments in psychosocial functioning. However, SSD was associated with a higher frequency of psychiatric symptoms, longer symptom duration, and a more chronic clinical course compared to FNSD. Moreover, while alexithymic traits were present in both groups, SSD group showed comparatively higher levels of alexithymia scores than those with FNSD. Identifying the disorder-specific features of SSD and FNSD is essential for accurate diagnosis and tailored treatment planning. In light of the findings, the systematic assessment of alexithymia, comorbid psychiatric symptoms and quality of life—particularly among adolescents presenting with somatic complaints—appears to be critical and may represent a valuable target for clinical intervention in both conditions.

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Data Availability: The datasets analyzed during the current study are available from the corresponding author upon reasonable request.

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Sleep disturbances, chronotype, and functional impairment in inattentive and restrictive presentations of attention-deficit/hyperactivity disorder

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SUMMARY

Objective: This study examined functional impairment, sleep disturbances, and chronotype characteristics in children with the Predominantly Inattentive Presentation of ADHD (ADHD-PI), the Restrictive ADHD phenotype (ADHD-R), and typically developing controls.

Method: A cross-sectional study was conducted with 104 children aged 7–12 years: ADHD-PI (n=34), ADHD-R (n=36), and Control group (n=34). Parent-rated measures included the CSHQ (The Children's Sleep Habits Questionnaire), CCQ (Children's Chronotype Questionnaire) , and WFIRS-P (Weiss Functional Impairment Rating Scale – Parent Form). All participants had an IQ above 80 and were free of comorbid psychiatric or medical conditions. Subgroup analyses were performed based on functional impairment status, and group comparisons were made on sleep-related and chronotype variables.

Results: Functional impairment was significantly more common in both ADHD groups (ADHD-PI: 67%, ADHD-R: 61%) compared to control group (5.9%) ($p < .001$). Both clinical groups showed greater daytime sleepiness. Although no statistically significant group differences were found in eveningness scores, the ADHD-R group exhibited higher eveningness tendencies compared to both the ADHD-PI and healthy control groups. In ADHD-PI, functional impairment was associated with delayed sleep onset, parasomnia, and sleep-disordered breathing. In ADHD-R, higher sleep disturbance, parasomnia, and longer sleep duration were observed in the impaired group. However, these variables did not significantly predict impairment. Oppositional defiant behavior scores significantly predicted functional impairment within the ADHD-R group.

Discussion: Sleep problems—particularly parasomnias—are associated with greater functional impairment in ADHD. Addressing sleep disturbances may contribute to improving daily functioning and quality of life in affected children.

Key Words: ADHD subtypes, Restrictive ADHD, Sleep disorders, Functionality, Chronotype

INTRODUCTION

The clinical presentation of ADHD has historically been categorized into three distinct subtypes: inattentive (ADHD-PI), hyperactive/impulsive (ADHD-H), and combined (ADHD-C) (1). Among children presenting to clinics, the most common type of ADHD is ADHD-C, whereas in population-based samples, ADHD-PI has been reported as the predominant subtype (2). The Restrictive phenotype has been proposed to describe children who exhibit few or no hyperactivity symptoms, distinguishing it from the Inattentive subtype" (3,4). There is growing evidence indicating that the ADHD-R phenotype may differ from ADHD-PI in its genetic, neuropsychological, and neurobiological features (3,5).

Research on sleep problems in children with ADHD has often involved heterogeneous participant groups (6-10). Consequently, existing knowledge primarily reflects findings from children with ADHD-C (5). Evidence suggests that children with ADHD experience significant difficulties not only in attention and behavior but also in their sleep patterns. Both objective measurements and parent

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reports highlight a range of sleep problems in these children, including delayed sleep onset, shorter sleep duration, nighttime awakenings, difficulty waking in the morning, excessive daytime sleepiness, and sleep-disordered breathing (11,12). Among ADHD-affected children, disrupted sleep contributes to greater behavioral difficulties, impaired cognitive performance, and lower overall quality of life (13,14). One study highlighted that unaddressed sleep problems can substantially intensify behavioral symptoms in children with ADHD, negatively impacting their daily functioning and overall family quality of life (15). Furthermore, poor sleep quality has been shown to significantly impact executive functioning, which is already frequently impaired in children with ADHD (16,17). Research examining whether sleep disturbances differ among ADHD subtypes has produced inconsistent findings. Although certain studies report no subtype-related differences, some findings suggest that children with Inattentive presentation may exhibit greater daytime sleepiness than those with the combined type (6,18-22). Increases in inattention and sluggish cognitive tempo (SCT) symptoms have also been linked to increased daytime sleepiness (23). Moreover, youth with ADHD-PI often present with a drowsy and tired demeanor and tend to exhibit co-occurring internalizing disorders, both of which have been significantly linked to sleep disturbances (24). Evaluating sleep disturbances in children with ADHD-PI is important, as such problems have been shown to predict functional impairment in this population (13,21,22,25-27). Chronotype describes a person's innate tendency to be more active at certain times of the day and is commonly classified as morning, evening, or intermediate type. ADHD has been associated with a preference for eveningness (28,29). Evidence suggests that eveningness is more common among individuals with ADHD, particularly those with inattentive features, and this tendency is related to reduced sleep quality and heightened symptom severity (30-32). Findings regarding circadian rhythm disturbances across ADHD subtypes are mixed. While some studies argue that eveningness is more pronounced in ADHD-C, others associate circadian rhythm disruptions and evening chronotype with the inattentive subtype (31). Each ADHD subtype is associated with increased levels of functional impairment relative to typically developing peers (33). Studies examining the ADHD–functionality relationship have demonstrated that both core symptoms and comorbid psychopathologies contribute to functional outcomes (33-39). Craig et al. found that sleep disturbances accounted for about 12% of the variance in functional impairment (13). Those diagnosed with ADHD-PI are particularly susceptible to sleep difficulties such as daytime sleepiness, trouble falling asleep, and poor sleep quality (20,23). Furthermore, these individuals are more likely to experience circadian rhythm disturbances and a preference for eveningness, both of which may negatively affect academic performance and daily functioning (30, 31). Recognizing sleep problems is crucial for improving functionality and quality of life in children who exhibit an inattentive presentation. Previous studies have reported a significant association between eveningness preference and inattentive symptoms(31,40). It has been proposed that eveningness tendency might be particularly associated with the inattentive presentation of ADHD (31). Investigating the effects of chronotype preferences and sleep disturbances on functional outcomes in ADHD-PI and ADHD-R subtypes may provide valuable insights that could contribute to improving functionality through targeted clinical interventions.

We hypothesized that children with ADHD-PI and ADHD-R who exhibit functional impairment would display higher sleep disturbance scores and a stronger eveningness preference compared to those without impairment. However, the existing literature provides limited evidence regarding the relationship between eveningness tendency, sleep disturbances, and functional impairment in these subtypes. The present study aimed to investigate distinctions in sleep disturbances and eveningness preference between ADHD-PI and ADHD-R groups, as well as their impact on functional outcomes, while controlling for factors such as comorbid psychiatric disorders, increased CDS (Cognitive Disengagement Syndrome) symptoms, psychostimulant use, and cognitive ability.

METHOD

Sample

Seventy children aged 7–12 years with a diagnosis of ADHD (34 with ADHD-PI and 36 with ADHD-R) were consecutively recruited according to the inclusion and exclusion criteria from patients either newly admitted to the clinic or already under follow-up for ADHD-PI or ADHD-R, with no psychotropic medication use in the last 3 months. All participants were primary or secondary school students. ADHD subtype classification was determined based on DSM-5 criteria following clinical interviews and scale assessments completed by both parents and teachers. Using the Turgay DSM-IV based ADHD rating Scale (T-DSM-IV-S). According to the T-DSM-IV-S scale, children exhibiting six or more symptoms of inattention along with two or fewer symptoms of hyperactivity/impulsivity were classified into the ADHD-R group, whereas those with three to five symptoms of hyperactivity/impulsivity were classified into the ADHD-PI group. In the T-DSM-IV-S, symptom scores of two or higher were considered clinically significant. Children with comorbid psychiatric (excluding Oppositional Defiant Disorder-ODD) or chronic medical conditions and those using psychostimulants were excluded. Thirty-four typically developing children who presented to the clinic and were not given any psychiatric diagnosis after evaluation constituted the control group. Informed consent forms were received from the participants and their caregivers.

Statistical Analyses

The sociodemographic characteristics of the ADHD groups and the control group were analyzed using one-way ANOVA. Chi-square tests were employed to compare chronotype preferences, the prevalence of sleep disturbances, and the proportion of individuals with functional impairment among the groups. The severity of inattention, hyperactivity/impulsivity, oppositional defiant, and conduct disorder symptoms, as well as Barkley functional impairment scores, were compared using the Kruskal-Wallis test due to non-normal distributions. As the WISC-R (Wechsler D.: Manual for the Wechsler Intelligence Scale for Children-Revised) verbal, performance, and total IQ scores were normally distributed, they were analyzed using one-way ANOVA. For variables showing significant group differences, post-hoc analyses (Bonferroni test) were conducted to identify which groups differed from each other. In both the ADHD-R and ADHD-PI groups, participants were divided into two subgroups based on the presence or absence of functional impairment, and comparisons were made regarding sleep duration, sleep onset times, total and subscale scores of the CSHQ, chronotype scores, and behavioral symptom scores. To identify predictors of functional impairment based on total WFIRS-P scores, binary logistic regression analyses were conducted separately for the ADHD-R and ADHD-PI groups. The regression models included CSHQ total score, sleep duration, parasomnias, sleep-disordered breathing, inattention and oppositional defiant symptom scores, and the Barkley functional impairment score. For each variable, odds ratios (Exp(B)) and 95% confidence intervals were reported. All analyses were conducted using SPSS version 25.0.

Sample Characteristics

Specific learning disorder, intellectual functioning, and psychiatric comorbidities (excluding ODD) were evaluated using the Schedule for Affective Disorders and Schizophrenia for School-Age Children – Present and Lifetime Version (K-SADS-P) and the WISC-R (41-43). Children scoring ≥23 on the Barkley Child Attention Scale, which screens for CDS, were excluded.

Of the total sample (n = 104), 57% were male and 43% were female. The mean ages were 9.53 ± 1.9 years in the ADHD-PI group, 9.67 ± 1.6 years in the ADHD-R group, and 9.18 ± 1.42 years in the control group. The distribution of participants was as follows: 32% ADHD-PI, 34% ADHD-R, and 32% controls.

ADHD Symptoms. ADHD symptoms were assessed using a T-DSM-IV-S . This instrument translates DSM-IV diagnostic criteria into question format and consists of forty-one items: nine assess-

ing inattention, six hyperactivity, three impulsivity, eight oppositional defiant disorder , and fifteen conduct disorder (CD) (43). It is completed by parents or teachers of children suspected to have ADHD. Items are rated on a 4-point Likert scale (0 = none, 1 = mild, 2 = moderate, 3 = severe) The Turkish validity and reliability study was conducted by Ercan et al. (2001) (44-46)

Cognitive Disengagement Syndrome (CDS). The Barkley Child Attention Scale was used to screen for CDS symptoms. Developed by Russell Barkley, the 12-item scale is completed by parents or teachers . The cutoff score was determined to be 23. In the Turkish validity and reliability study conducted by Fırat and colleagues, the Cronbach's alpha coefficient was found to be 0.86, indicating that the scale is valid and reliable (47,48)

Sleep and Chronotype. CSHQ is a 33-item parent-report measure that evaluates the child's sleep behaviors over the past week. It assesses various domains including bedtime resistance, sleep onset delay, sleep duration, sleep anxiety, night wakings, parasomnias, sleep-disordered breathing, and daytime sleepiness (49). The Turkish adaptation of the scale was validated by Perdahlı Fiş et al. in 2010 (50). Chronotype was assessed using the CCQ (50). CCQ is completed by parents. Items 17, 18, 24, and 25 are reverse-coded. Chronotype was determined using the total score from 10 items, with scores of 23 or below indicating a morning type, scores between 24 and 32 indicating an intermediate type, and scores of 33 or above indicating an evening type. Average sleep duration was estimated based on reported sleep onset and wake times. Napping was included in the total sleep duration. Sleep duration was averaged across the week by calculating five times the school day value plus two times the free day value, divided by 7 .The Turkish version was validated by Dursun et al. (51).

Functionality. Functional impairment was assessed using the WFIRS-P (52). Parents rated their child's functioning in six domains—family, school, life skills, self-concept, social activities, and risky behaviors—on a scale from 0 to 4. A mean score of 1.5 or higher in any domain was considered indicative of impairment in that area. Children who showed impairment in at least two domains were classified as having overall functional impairment. The adaptation conducted in our country by Tarakçıoğlu et al. yielded a Cronbach's alpha of 0.93 for the total scale, indicating excellent reliability (53).

Intelligence. WISC-R was used to assess cognitive functioning (42). In Turkey, validity and reliability studies conducted by Savaşır and Şahin led to the adaptation of the WISC-R to Turkish culture (42).

Psychiatric Comorbidities. Comorbid psychiatric diagnoses were screened using the (K-SADS-PL), and those with additional diagnoses were excluded from the study. The validity and reliability study in Turkey was conducted by Gökler and colleagues. (41).

RESULTS

Age, height, weight, and the ages of the mother and father did not differ significantly between the groups (p>.05); however, significant differences were observed in parental education levels, with both maternal and paternal education reaching statistical significance (p=.003 and p=.001, respectively). (Table 1). The groups did not differ significantly in developmental milestones such as age of first word, first sentence, walking, or toilet training (p>.05). Likewise, no significant differences were found in birth weight (p=.510) or breastfeeding duration (p=.393).

Table 1. Sociodemographic Characteristics of Children with ADHD-PI, ADHD-R, and Controls

Sociodemographic Variables	ADHD-PI (n = 34)	ADHD-R (n = 36)	Control (n = 34)	p-value	P (Bonferroni)	n^2p
Age (years), M – SD	9.5 ± 1.9	9.6 ± 1.6	9.1 ± 1.4	0.507	0.507	0.01
Height (cm), M – SD	139.7 ± 15.9	138.2 ± 12.8	136.9 ± 9.8	0.770	0.770	0.01
Weight (kg), M – SD	35.3 ± 13.9	36.8 ± 13.8	33.9 ± 11.9	0.691	0.691	0.01
Maternal age, M – SD	38.9 ± 5.2	38.4 ± 5.3	37.1 ± 4.0	0.302	0.302	0.02
Paternal age, M – SD	42.5 ± 5.7	42.6 ± 6.7	37.1 ± 4.0	0.590	0.590	0.01
Maternal education (years), M – SD	12.5 ± 3.1	11.2 ± 3.2	13.8 ± 2.9	**0.003**	**0.021**	0.09
Paternal education (years), M – SD	14.2 ± 3.1	12.3 ± 3.2	14.8 ± 2.3	**0.001**	**0.007**	0.12

Note. M = Mean; SD = Standard deviation. *p*-values indicate statistically significant results. Note. Analyses were conducted using a one-way ANOVA across three groups. A total of seven variables were examined, and Bonferroni correction was applied for multiple comparisons (adjusted significance level: p < .007). Effect size was reported as partial eta squared (n^2p).

Table 2. Chronotype Preferences and Presence of Sleep Disturbances in Children with ADHD-PI, ADHD-R, and Control Groups

	ADHD-PI(n =34)	ADHD-R (n = 36)	Control (n = 34)	p-value	x†	df	q (FDR)	Cramer□s V
Chronotype Preference				0.64	0.87	4	0.647	0.09
Morning type	6 (17.6%)	3 (8.3%)	4 (11.8%)	0.42			0.63	0.10
Intermediate type	18 (52.9%)	17 (47.2%)	18 (52.9%)	0.83			0.83	0.04
Evening type	10 (29.4%)	16 (44.4%)	12 (35.3%)	0.46			0.63	0.12
Sleep Disturbances				0.08	5.03	2	0.12	0.23
Absent	11 (32.4%)	12 (33.3%)	19 (55.9%)					
Present	23 (67.6%)	24 (66.7%)	15 (44.1%)					
Daytime Sleepiness	58.88	58.42	39.85	**0.01***	8,98			

Note. Sleep disturbances are classified based on a total CSHQ score ≥ 41. Note. Analyses were conducted using Pearsons Chi-square test. Group comparisons for the Daytime Sleepiness subscale of the CSHQ were conducted using the Kruskal-Wallis test. Multiple comparisons were adjusted using the Benjamini□Hochberg False Discovery Rate (FDR) correction (two tests; q< 0.05). Effect size was reported as Cramers V (small = 0.10, medium = 0.30, large = 0.50).

There were no significant group differences regarding chronotype preference or the occurrence of sleep disturbances (p=.64 and p=.08, respectively). (Table 2). Among the CSHQ subscales, only the daytime sleepiness subdomain showed a significant difference. Both the ADHD-PI (p< .001) and ADHD-R (p=.01) groups exhibited greater daytime sleepiness compared to controls, whereas no difference was found between the two ADHD groups (p=.94). However, no significant difference in daytime sleepiness was observed between the ADHD-R and ADHD-PI groups (p=.94).

Comparison of the T-DSM-IV-S

Group comparisons showed significant differences in inattention, hyperactivity, oppositional behavior, and conduct problem scores across the ADHD-PI, ADHD-R and control groups (p<.05 for all). Subsequent post-hoc analyses demonstrated that individuals in the Inattentive presentation group exhibited higher levels of hyperactivity (p=.00), oppositionality (p=.01), and conduct-related issues (p=.03) compared to those in the ADHD-Rgroup. (Table 3). The control group outperformed both ADHD subgroups on verbal, performance, and full-scale IQ scores measured by the WISC-R (Table 3).

Comparison of WFIRS-P Scale Scores

Both ADHD groups demonstrated significantly higher rates of functional impairment compared to the control group in the domains of family functioning (Cramer's V=0.28, moderate effect), school functioning (Cramer's V=0.62, large effect), life skills (Cramer's V=0.49, moderate-to-large effect), risky behaviors (Cramer's V=0.37, moderate effect), and overall functioning, defined as impairment in at least two domains (Cramer's V = 0.56, large effect). (Table 4).

In the ADHD-R group, children with functional impairment had significantly higher scores in total sleep disturbance (CSHQ total score) (p<.00), sleep duration (p=.03), parasomnia (p=.01), inattention (p=.01), oppositional behavior (p<.00),

Table 3. Comparison of Turgay Scale Total Scores (Inattention, Hyperactivity/Impulsivity, Oppositional Defiant, and Conduc Disorder), the Barkley Child Attention Scale Total Score, and WISC -R Scores (Verbal, Performance and Total) among the ADHD-PI, ADHD-R, and Control Groups

	ADHD-PI (n=34)	ADHD-R (n=36)	Control (n=34)	F/x^2	p-value	(Bonferroni)	(n^2 / r)	Post-Hoc Analysis
Inattention Total Score	72.35	63.28	21.24	56.201	0.000**	<.006	0.68	(PI> C, R> C) (p<.00)
Hyperactivity Total Score	77.53	43.18	37.34	37.192	0.000**	<.006	0.55	(PI> C, PI> R) (p<.00)
Oppositional Defiant Total Score	69.60	52.56	35.34	23.399	0.000**	<.006	0.47	(PI> C) (p<.00), (PI> R, R>C)(p=.01)
Conduct Disorder Total Score	64.90	48.78	44.04	18.969	0.000**	<.006	0.39	(PI> R, PI> C) (p<.00)
Barkley	62.84	64.27	28.53	31.723	0.000**	<.006	0.44	(PI> C, R> C)(p<.00)
WISC-R Verbal	93.8 – 13	90.1 – 14	108.5 – 10	19.620	0.000**	<.006	0.32	(C> PI, C> R) (p<.00)
WISC-R Performance	101.9 – 15	103.5– 14	113.5 – 12	11.605	0.000**	<.006	0.28	C> PI (p<.00), C> R (p=.02)
WISC-R Total	97.4 – 13	96.3 – 15	111.7 – 11	13.100	0.000**	<.006	0.35	(C> PI, C> R) (p<.00)

Notes. Kruskal-Wallis test was applied for comparison analyses. * p <0.05, ** p < 0.01. ANOVA was used for WISC-R verbal and total scores. PI=ADHD-PI (Predominantly Inattentive Presentation of ADHD); R = ADHD-R (Restrictive Presentation of ADHD); C = Controls. Multiple comparisons were adjusted using the Bonferroni correction ($alfa_adj$ = 0.00625). Effect sizes were reported as partial eta squared (n^2p) for ANOVA and r for Kruskal -Wallis tests (small = 0.10, medium = 0.30, large = 0.50; Cohen, 1988).

Table 4. Distribution of Functional Impairment Across WFIRS-P Domains in ADHD-PI, ADHD-R, and Control Groups

Functional Impairment (WFIRS-P)	ADHD-PI (n = 34)	ADHD-R (n = 36)	Control (n = 34)	p-value	x^2	q (FDR)	Cramer□s V
Family Functioning				.014*	8.565	.035*	0.28
No Impairment	24 (70.6%)	24 (66.7%)	32 (94.1%)				
Impairment	10 (29.4%)	12 (33.3%)	2 (5.9%)				
School Functioning				.000**	37.136	.003*	0.62
No Impairment	12 (35.3%)	14 (38.9%)	34 (100%)				
Impairment	22 (64.7%)	22 (61.1%)	0 (0%)				
Life Skills				.000**	18.529	.003*	0.49
No Impairment	10 (29.4%)	15 (41.7%)	27 (79.4%)				
Impairment	24 (70.6%)	21 (58.3%)	7 (20.6%)				
Self-Perception				.048*	9.611	.072	0.24
No Impairment	24 (70.6%)	29 (80.6%)	33 (97.1%)				
Impairment	10 (29.4%)	7 (19.4%)	1 (2.9%)				
Social Activities				.085	4.937	.092	0.19
No Impairment	27 (79.4%)	31 (86.1%)	33 (97.1%)				
Impairment	7 (20.6%)	5 (13.9%)	1 (2.9%)				
Risky Behaviors				.003**	11.874	.009*	0.37
No Impairment	26 (76.5%)	34 (94.4%)	34 (100%)				
Impairment	8 (23.5%)	2 (5.6%)	0 (0%)				
Total (≥2 Impaired Domains)				.000**	31.816	.003*	0.56
No Impairment	11 (32.4%)	14 (38.9%)	32 (94.1%)				
Impairment	23 (67.6%)	22 (61.1%)	2 (5.9%)				

Note. Analyses were conducted using Pearsons Chi-square test across seven subdomains. Multiple comparisons were adjusted using the Benjamini□Hochberg False Discovery Rate (FDR) correction (k=7; adjusted significance threshold: q<0.05). Effect sizes were reported as Cramers V (small = 0.10, medium = 0.30, large = 0.50).

and CDS total score (Barkley Child Attention Scale) (p<0.00)(Table 5). Nevertheless, none of these variables were found to be significant predictors of functional impairment in logistic regression analysis: total CSHQ score (p= .14), sleep duration (p=.12), parasomnia (p=.21), inattention (p= .85), and CDS (p=.25). The oppositional behavior total score approached statistical significance (p=.05), indicating a possible trend toward increased risk of functional impairment (Table 6).

In the ADHD-PI group, comparisons between participants with and without functional impairment revealed that those with impairment had significantly higher scores in sleep duration on free days (p=.01), parasomnia (p=.01), and sleep-disordered breathing (p = .04) according to the CSHQ subscales (Table 7). However, binary logistic regression analysis showed that these variables did not significantly predict functional impairment: sleep duration on free days (p= .07), parasomnia (p = .15), and sleep-disordered breathing (p = .14) (Table 8).

Table 5. Comparison of Chronotype scale total score, CSHQ total score and subscale scores, Turgay Scale Total Scores (Inattention, Hyperactivity/Impulsivity, Oppositional Defiant, and Conduct Disorder), the Barkley Child Attention Scale Total Score in relation to overall functional impairment (WFIRS-P) in the ADHD-R group

Variable	Impairment Present (n=22)	No Impairment (n=14)	Z/t	p-value	q (FDR)	(d / r)
Sleep Onset Time on Scheduled Days	9.02	9.68	1.768$^+$	.086	.112	0.28
Sleep Onset Time on Free Days	10.00	10.21	0.552$^+$	.585	.625	0.08
Total Sleep Duration	9.26	9.79	1.566$^+$	.127	.153	0.23
Chronotype Scale Score	32.45	29.43	-1.476$^+$	.149	.170	0.21
CSHQ Total Score	46.91	40.43	-3.122$^+$	.004*	.024*	0.41
Bedtime Resistance	20.70	15.04	-1.596	.111	.135	0.25
Sleep Duration Problems	20.84	14.82	-2.119	.034*	.045*	0.33
Sleep Anxiety	20.11	15.96	-1.173	.241	.267	0.17
Sleep Onset Delay	19.80	16.46	-1.342	.180	.210	0.19
Night Wakings	19.82	16.43	-1.042	.297	.323	0.15
Parasomnias	21.66	13.54	-2.425	.015*	.045*	0.36
Sleep Disordered Breathing	19.95	16.21	-1.507	.132	.158	0.22
Daytime Sleepiness	12.77	11.64	-0.873$^+$	.389	.420	0.14
Inattention Total Score	16.86	12.92	-2.579	.015*	.045	0.36
Hyperactivity/Impulsivity Total Score	20.55	13.69	-1.932	.053	.070	0.30
Oppositional Defiant Total Score	6.77	2.08	-3.733	.001**	.007*	0.49
Conduct Disorder Total Score	19.48	15.50	-1.512	.130	.156	0.22
Barkley Child Attention Scale Total Score	20.29	16.14	-2.630	.006*	.027*	0.38

Note. $^+$t-test was applied for variables marked with a plus sign; Mann-Whitney U test was applied for the others.
Note. Functional impairment was defined as a mean score ≥1.5 on WFIRS-P subscales; impairment in ≥ 2 subscales was classified as overall functional impairment. Multiple comparisons were adjusted using the Benjamini□Hochberg False Discovery Rate (FDR) correction. *p < .05; **p < .01.

Table 6. Binomial logistic regression analysis of CSHQ Total Score, Sleep Duration Problems, Parasomnia Scores, Inattention/Oppositional Defiant Total Score and the Barkley Child Attention Scale Total Score predicting functional impairment in the ADHD-R group

Weiss Total Functional Impairment (Present/Absent)	B	S.E.	Wald	df	Sig.	q (FDR)	Exp(B)	95% C.I. for Exp(B) Lower
CSHQ Total Score	0.254	0.173	2.155	1	.142	.158	1.289	0.918
Sleep Duration Problems	2.378	1.530	2.417	1	.120	.144	10.782	0.538
Parasomnias	1.100	0.893	1.519	1	.218	.218	3.005	0.522
Inattention Total Score	0.037	0.201	0.034	1	.854	.854	0.964	0.650
Oppositional Defiant Total Score	0.475	0.242	3.846	1	**.050**	**.050**	1.608	1.000
Barkley Child Attention Scale Total Score	0.363	0.318	1.304	1	.254	.254	1.438	0.771

Note. *p<.05; *p<.01

DISCUSSION

This study aims to examine the rates of functional impairment in ADHD-R and ADHD-PI groups, as well as factors that may affect this impairment, including ADHD symptom severity, ODD scores, sleep disturbances, and chronotype characteristics. The current definition of ADHD-PI allows inclusion of children with ADHD-C who exhibit subthreshold hyperactivity/impulsivity symptoms. Increasing evidence suggests that the ADHD-R phenotype may differ from ADHD-PI in genetic, neuropsychological, and neurobiological characteristics (3,5). Therefore, it is important to evaluate a more homogeneous group that does not exhibit H/I symptoms separately. Previous studies have linked inattention with eveningness and found that the ADHD-PI group experiences greater daytime sleepiness than other subtypes (6,11,12,18–22,31). This study is the first to subjectively assess functionality, chronotype preferences, and sleep disturbances in children diagnosed with ADHD-R. By controlling confounding variables such as psychiatric comorbidities, elevated CDS symptoms, cognitive level, and medication use, the relationships among chronotype preferences, sleep disturbances, and functional outcomes were comprehensively examined in both ADHD-R and ADHD-PI groups. Previous studies have reported an association between inattentive symptoms and eveningness tendency(31,40). However, the present study, differing methodologically, did not focus on the correlation between inattentive symptoms and eveningness. Instead, we compared the prevalence of eveningness across the ADHD-PI, ADHD-R, and control groups and identified the intermediate chronotype as the most common preference. Although this finding has been reported in some studies, the results in the literature remain inconsistent(40,54,55) Such inconsistency may be attributed to differences in sample age ranges and the predominant focus on the ADHD-C in previous research.

Our findings demonstrated that both ADHD groups exhibited significantly greater functional impairment compared to control group, and that sleep disturbances were associated with specific

Table.7 Comparison of Chronotype scale total score, CSHQ scores, Turgay Scale Total Scores (Inattention, Hyperactivity/Impulsivity, Oppositional Defiant, and Conduct Disorder), the Barkley Child Attention Scale Total Score in relation to overall functional impairment (WFIRS-P) in the ADHD-PI group

Variable	Impairment Present (n=23)	No Impairment (n=11)	Z/t	p-value	q (FDR)	(d / r)
Sleep Onset Time on Scheduled Days(minutes)	9.04	8.73	-0.806^{+}	.426	.455	0.13
Sleep Onset Time on Free Days(minutes)	20.22	11.82	-2.349	.019*	.045*	0.34
Total Sleep Duration	9.39	8.89	-1.388^{+}	.175	.195	0.22
Chronotype Scale Score	30.57	28.82	-0.807^{+}	.426	.455	0.13
CSHQ Total Score	18.28	15.86	-0.664	.507	.525	0.10
Bedtime Resistance	18.50	15.41	-0.860	.390	.412	0.16
Sleep Duration Problems	15.48	21.73	-1.929	.054	.072	0.29
Sleep Anxiety	17.30	17.91	-0.170	.865	.875	0.03
Sleep Onset Delay	16.63	19.32	-0.892	.372	.398	0.14
Night Wakings	17.70	17.09	-0.190	.849	.865	0.03
Parasomnias	20.28	11.68	-2.526	.012*	.032*	0.37
Sleep Disordered Breathing	19.48	13.36	-2.028	.043*	.049*	0.29
Daytime Sleepiness	12.52	11.73	-0.703^{+}	.487	.512	0.11
Inattention Total Score	17.35	16.18	-0.862	.396	.422	0.13
Hyperactivity/Impulsivity Total Score	9.74	10.00	0.190	.530	.555	0.09
Oppositional Defiant Total Score	6.35	5.18	-0.724	.095	.112	0.27
Conduct Disorder Total Score	18.17	16.09	-0.657	.511	.534	0.10
Barkley Child Attention Scale Total Score	17.43	20.55	1.572	.136	.156	0.23

Note. $^{+}$*t-test was applied for variables marked with a plus sign; Mann-Whitney U test was applied for the others.* *p < 0.5; **p<0.01 Note. Functional impairment was defined as a mean score ≥ 1.5 on WFIRS-P subscales; impairment in ≥ 2 subscales was classified as overall functional impairment.

Table 8. Binomial logistic regression analysis of Sleep Onset Time on Free Days, Parasomnia Scores and Sleep -Disordered Breathing Score predicting functional impairment in the ADHD-PI group

Weiss Total Functional Impairment (Present/Absent)	B	S.E.	Wald	df	Sig.	q (FDR)	Exp(B)	95% C.I. for Exp(B) Lower
Sleep Onset Time on Free Days(minutes)	1.063	0.598	3.155	1	.076	.114	2.894	0.896
Parasomnias	1.096	0.768	2.038	1	.153	.183	2.992	0.664
Sleep-Disordered Breathing	1.339	0.927	2.086	1	.149	.183	3.815	0.620

Note. *p<0.5; *p<0.01. Note. Parasomnia scores and Sleep-Disordered Breathing scores are subscales of the CSHQ.

domains of functional impairment. Consistent with prior literature, our study found that children with ADHD-PI tend to exhibit more pronounced daytime sleepiness than those with controls (13,18). However, in our sample, daytime sleepiness was not directly associated with functional impairment.

The absence of a statistically significant difference in the prevalence of sleep disturbances between the ADHD and control groups may be explained by the composition of the control group, which consisted of children recruited from outpatient clinics who may have had subthreshold psychopathologies, increasing their likelihood of experiencing sleep problems. Additionally, previous research suggests that anxiety and CDS symptoms may influence sleep functioning in the ADHD-PI subtype (24). Since children with comorbid anxiety disorders or CDS , as well as those with hyperactive or combined ADHD subtypes, were excluded from the present study, this methodological choice may also account for the lack of significant differences in sleep disturbances between groups.

In terms of functional domains, our study compared the ADHD-R and ADHD-PI groups with the control group across family, school, life skills, self-concept, social activities, and risky behaviors. The ADHD-PI group showed significantly higher functional impairment across all domains compared to controls. Notably, the ADHD-PI group exhibited greater impairment in risky behaviors than the ADHD-R group, which may be explained by hyperactivity/impulsivity symptoms contributing to aggressive or oppositional behaviors (5). Children in the ADHD-R group demonstrated significant impairment in school, life skills, and self-concept compared to controls. Inattentive symptoms in ADHD are associated with poor academic performance due to difficulties in focusing and completing tasks (56). Academic challenges may also contribute to social difficulties, peer rejection, isolation, frustration, and lowered self-esteem (38,57). In our study, functional impairment in at least two domains was found in 67.6% of the ADHD-PI group, 61.1% of the ADHD-R group, and only 5.9% of the control group—highlighting substantial functional deficits in both ADHD subtypes.

There were no significant group differences in the distribution of chronotype categories. However, the ADHD-R group had the highest proportion of evening-type individuals (44%) and was found to have later bedtimes and wake-up times on scheduled days compared to the other groups. This suggests a delayed circadian rhythm possibly associated with eveningness in the ADHD-R group. In the ADHD-R group, children with functional impairment exhibited higher levels of sleep disturbance (CSHQ total score), longer sleep duration, more parasomnias, more pronounced inattention and oppositional symptoms, and higher CDS scores. However, these variables did not independently predict functional impairment in regression analyses. It is possible that the increased total sleep duration—calculated including naps—may reflect higher levels of daytime sleepiness, which could indirectly contribute to functional difficulties in the ADHD-PI subtype. Additionally, parents in the ADHD-R group had significantly lower levels of education compared to other groups. Lower socioeconomic status and lifestyle factors are known to be associated with poor sleep in children (35). Suboptimal parenting practices may also contribute to increased sleep difficulties in this group.In the ADHD-PI group, children with functional impairment had longer sleep durations on free days, more parasomnias, and more sleep-disordered breathing; however, these variables did not significantly predict impairment. The limited number of cases particularly affects the regression analyses, reducing their statistical power and consequently limiting the generalizability of the findings. Variability in sleep patterns on weekdays, possibly due to insufficient sleep hygiene, may have led to compensatory longer sleep on weekends. Of particular interest, parasomnia scores were higher

among functionally impaired children in both ADHD subtypes. This finding underscores the need for more objective sleep assessments in children with ADHD-PI. The key finding of this study is that even after controlling for psychiatric comorbidities, CDS symptoms, intellectual ability, and medication use, sleep disturbances remained significantly associated with overall functional outcomes. Previous studies using the same functional impairment scale (WFIRS-P) support this relationship. Virring et al. found a significant relationship between sleep problems, as measured by the CSHQ, and functional impairment assessed via the WFIRS-P in children with ADHD (22). In line with this, Craig et al. reported that sleep disturbances could predict approximately 12% of the variance in functional difficulties (13). Addressing sleep disturbances is essential to improving the overall quality of life and daily functioning in children with ADHD. Particularly in the ADHD-PI subgroup, where both pharmacological and non-pharmacological treatments may be insufficient, clinicians should routinely assess for sleep problems, parasomnias, and total sleep duration. Interventions aimed at improving sleep hygiene and resolving sleep-related issues hold promise for enhancing functional outcomes in children with ADHD.

Limitations

This study has several strengths, including the use of a homogenous clinical sample, the exclusion of psychiatric comorbidities and medical diagnoses, the inclusion of only psychotropic-naïve participants, and the exclusion of individuals with low IQ. Furthermore, the inclusion of a well-matched healthy control group and the use of well-validated tools to systematically assess sleep, chronotype, and functional impairment enhance the study's methodological rigor. However, certain limitations should be considered. The control group consisted of children who presented to the clinic but did not receive any psychiatric diagnosis. The possible presence of subclinical psychopathology in this group, which may have influenced sleep problems and sleep hygiene, is considered one of the study's limitations. The limited sample size, particularly within subgroups, may have reduced the statistical power of the regression analyses and restricts the genera-lizability of the findings. The exclusion of all psychiatric comorbidities (excluding ODD) in the ADHD group limits the generalizability of the findings, as such 'pure ADHD' cases are rarely encountered in clinical practice. Since the sample was drawn from outpatient clinic admissions, its representativeness of the general population is limited. Another major limitation is the exclusive reliance on subjective parent-reported questionnaires for assessing sleep parameters, without incorporating objective measures such as actigraphy, the Multiple Sleep Latency Test (MSLT), or polysomnography (PSG). However, although actigraphy-based sleep measures did not differ significantly between 212 children with ADHD and 212 healthy controls, parent-reported sleep problems were associated with behavioral difficulties (58). Additionally, chronotype assessment was based on questionnaire data rather than biological markers such as salivary melatonin levels or basal body temperature, which may offer more direct physiological indicators of circadian preference. The rating scales used in this study did not include specific questions regarding the timing and duration of internet use, which could affect the sleep–wake cycle. Moreover, pubertal development was not assessed using the Tanner staging system, and thus adolescents were not excluded from the sample. Given the increased eveningness and greater prevalence of sleep problems associated with hormonal changes during puberty, this may have influenced the results. Finally, the cross-sectional design of this study precludes causal inferences. Longitudinal studies are needed to examine the developmental trajectory of eveningness and sleep disturbances in children with ADHD-R and ADHD-PI.

This is the first study to evaluate functional impairment, chronotype, and sleep disturbances in children with the ADHD-R. Our findings contribute to the growing literature by comparing children with ADHD-PI with and without hyperactivity symptoms, examining their chronotypic and sleep profiles, and highlighting their associations with functional outcomes.

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A case of chorea-acanthocytosis with suicidal ideation and obsessive-compulsive disorder

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SUMMARY

Chorea-acanthocytosis is one of the neuroacanthocytosis syndromes; a rare (1–5 per 1,000,000) and progressive neurodegenerative disorder characterized by abnormalities in the nervous system accompanied by erythrocyte acanthocytosis. Various neurological dysfunctions and psychiatric symptoms coexist, significantly reducing both quality of life and life expectancy. Due to the rarity of the disease, diagnosis can sometimes be delayed; the initial presentation may include vague cognitive or psychiatric symptoms, leading to prolonged misdiagnoses and incorrect management. In middle-aged adults presenting with chorea and tic-like involuntary movements alongside psychiatric disorders, neuroacanthocytosis syndromes should always be considered. A thorough neurological and psychiatric examination should be conducted, and necessary imaging and laboratory tests should be performed.In this case report, we present the detailed diagnostic evaluation process of a patient suspected of having chorea-acanthocytosis with neuropsychiatric symptoms, in light of the existing literature.

Key words: Neuroacanthocytosis, feeding dystonia, movement disorder, obsessive compulsive disorder, vocal tic

INTRODUCTION

Neuroacanthocytosis (NA) is a rare syndrome characterized by the coexistence of abnormalities in the nervous system and erythrocyte acanthocytosis. The estimated prevalence is fewer than 1 to 5 cases per 1,000,000 individuals (1). It leads to a variety of neurological dysfunctions including seizures, movement disorders, peripheral neuropathy, speech–swallowing difficulties, psychosis, and dementia, leading to reduced life expectancy (2).

NA syndromes can be divided into several groups: 1. Core NA syndromes (1a. Chorea-Acanthocytosis, 1b.McLeod syndrome) 2. Degenerative disorders occasionally associated with acanthocytosis 3. Disorders related to decreased blood lipoproteins and acanthocytosis 4. Paroxysmal dyskinetic disorders (3).

Chorea-acanthocytosis (ChAc) is a progressive neurodegenerative disease that may initially present with subtle cognitive or psychiatric symptoms, often years before neurological signs appear (1). Historically, clinicians have focused primarily on the progressive external motor features of the disease; however, increasing evidence indicates that these disorders may also present with significant psychiatric and neurocognitive comorbidities (3).

In this report, we present a case with neuropsychiatric complaints in whom a diagnosis of chorea-acanthocytosis was considered. This case draws attention to the rare coexistence of obsessive–compulsive symptoms and suicidality in a genetically confirmed patient with Chorea-Acanthocytosis, highlighting the need for awareness of psychiatric-onset presentations in neuroacanthocytosis syndromes.

Case Presentation

Clinical Course

A 32-year-old single male was admitted to our inpatient unit due to the presence of vocal tics,

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feeding dystonia, weight loss, obsessive thoughts, involuntary movements, and subsequent suicidal ideation. His complaints had begun five years earlier with intermittent syncopal episodes, which were later accompanied by compulsive reassurance seeking and hoarding behavior. Following his first psychiatric evaluation that same year, he was treated with topiramate and lamotrigine; however, impulse control difficulties, excessive spending, gait disturbance, problems with anger regulation, and depressive symptoms gradually emerged. Within the past year, he had been hospitalized twice at different centers with diagnoses of Generalized Anxiety Disorder and Conversion Disorder but no significant clinical improvement was observed. Six months before admission, he developed orofaciolingual dyskinesia with involuntary lip biting and gasping vocal tics. He required a straw to consume liquids and experienced weight loss; 7 kg in one month, 30 kg overall. Four months earlier, he had been admitted to the intensive care unit following a generalized tonic-clonic seizure, complicated by right arm weakness. Initially confined to the facial region, tics progressed to involve shoulders and hips.

Over time, his obsessive–compulsive symptoms, which had initially appeared as reassurance seeking and repetitive behaviors, progressively worsened; he spent most of his day engaged in ritualistic behaviors, such as stringing beads or painting in a fixed sequence and became anxious and irritable when interrupted. These repetitive acts occupied several hours daily and interfered with his ability to engage in social activities. He repeatedly made identical bracelets using beads of the same color and felt compelled to complete a specific number of them in a particular order or symmetry. He frequently sought reassurance about his illness and treatment, repeatedly asked the same questions, and rechecked his drawings or bead arrangements until they felt "just right." He described feeling a persistent sense of tension and discomfort until these tasks were completed exactly as he imagined. Although aware that these behaviors were excessive, he was unable to resist them, resulting in marked distress and functional impairment. The escalation of vocal tics led to social withdrawal due to embarrassment, while his depressive symptoms intensified, culminating in suicidal ideation. For further diagnostic clarification and treatment planning, he was admitted to our clinic.

Neurological and Psychiatric Findings

On psychiatric examination, the patient was conscious, cooperative, and oriented, with mildly impaired self-care. Speech was dysarthric, hypophonic, and frequently interrupted by vocal tics. The patient showed compulsive reassurance-seeking, depressed mood, mild distractibility, and coherent thought processes. Sleep was reduced, while appetite was preserved but limited by swallowing difficulties. Neurological examination revealed right hand strength of 4+/5, impaired vibration sense in the lower extremities, absent lower and hypoactive upper reflexes, postural instability, and orofacial self-injury with gasping movements. Past medical history included febrile convulsions, obstructive sleep apnea, and epilepsy. Family history revealed no psychiatric or neurological disorders.

The Structured Clinical Interview for DSM-5 Disorders (SCID) was administered as a screening tool. It indicated symptoms consistent with Major Depressive Disorder, Generalized Anxiety Disorder, Obsessive-Compulsive Disorder, and Adult Attention-Deficit/Hyperactivity Disorder. However, based on clinical evaluation, only depression and obsessive-compulsive symptoms were considered diagnostically relevant. Wechsler Adult Intelligence Scale (WAIS) indicated borderline intellectual functioning (Full-Scale Intelligence Quotient [FS-IQ]=77). The Addenbrooke's Cognitive Examination–Revised (ACE-R) yielded a total score of 82/100, with impairment most pronounced in memory and fluency. On the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS), the patient scored 26/40, falling within the severe symptom range.

Laboratory and Genetic Results

Laboratory findings revealed elevated serum creatine kinase (CK: 2360 U/L), myoglobin (450.9 ng/mL), and aspartate aminotransferase (AST: 63 U/L). Other basic biochemical parameters and complete blood count were within normal limits.

Ceruloplasmin levels and 24-hour urinary copper excretion were normal, thereby excluding Wilson's disease. Brain MRI demonstrated caudate nucleus and putaminal atrophy, while EEG findings were unremarkable. EMG revealed reduced sensory nerve action potential amplitude in the right ulnar nerve, with no additional abnormalities. Peripheral blood smear revealed 3–4% acanthocytes. Genetic testing identified two heterozygous, likely pathogenic variants in the VPS13A gene: c.1795_1796del (p.Asn599Ter) in exon 19 and c.4313_4317del (p.Thr1438LysfsTer21) in exon 37.

Treatment and Follow-up

During hospitalization, clomipramine was titrated to 150 mg/day to target depressive and obsessive-compulsive symptoms, and quetiapine 75 mg/day was initiated to address sleep disturbances. The patient had previously failed to respond adequately to multiple SSRI trials; therefore, clomipramine was selected for its anti-obsessional efficacy and prior partial benefit.

Quetiapine was preferred due to its favorable tolerability in the presence of marked movement disorder and was gradually titrated to 400 mg/day to manage agitation and sleep disturbance. Lamotrigine, which had previously shown benefit, was reintroduced to help with persistent depressive symptoms and titrated to 100 mg/day. Over the course of admission, vocal tics, involuntary movements decreased, accompanied by moderate improvement in obsessive thoughts (Y-BOCS score reduced from 26 to 19). Suicidal ideation resolved, and no seizures were observed.

Outpatient follow-up over a three-year period included regular psychiatric and neurological monitoring. His final treatment regimen consisted of clomipramine 150 mg/day, lamotrigine 200 mg/day, diazepam 15 mg/day, quetiapine 400 mg/day, and tetrabenazine 25 mg/day. At follow-up, feeding difficulties improved, vocal tics and involuntary movements decreased, and depressive symptoms showed remission. Suicidal ideation did not recur. Implementation of a dental guard effectively prevented lip biting, and the patient and his family were referred to social services for future planning, given the progressive nature of the disease. He continues to be followed jointly in psychiatry and neurology clinics.

DISCUSSION

Neuroacanthocytosis refers to a group of rare neurodegenerative disorders characterized by orofaciolingual dyskinesia, seizures, psychiatric manifestations, additional movement abnormalities, and the presence of deformed erythrocytes known as acanthocytes in peripheral blood. Although symptom onset most commonly occurs in the third decade of life, cases have been reported across a wide age range, and the disorder is associated with reduced life expectancy (4). In our patient, symptoms began at the age of 26.

The two major NA syndromes are autosomal recessive chorea-acanthocytosis and the X-linked McLeod syndrome (5). Although they share many clinical features, the hallmark of ChAc is the distinctive feeding dystonia, considered pathognomonic, in which the tongue expels food from the mouth upon contact, often accompanied by profound weight loss. Additional features include orofacial dystonia and chorea, manifesting as grimacing, involuntary vocalizations, dysarthria, and tongue–lip biting (6). Consistent with these reports, our patient presented with feeding difficulties, food expulsion, significant weight loss, dependence on a liquid diet, involuntary vocalizations, oral self-mutilation, and dysarthric speech.

The percentage of acanthocytes in peripheral blood varies from 5–50% and does not correlate with disease severity (2). Since acanthocytes may only be detectable in later stages, their absence does not exclude the diagnosis. Repeated smears or saline-diluted wet preparations, as proposed by Feinberg et al. in 1991, may increase diagnostic yield (7,8). Serum CK levels, often moderately or markedly elevated, appear more reliable for diagnostic support. Approximately half of the patients also present with elevated liver enzymes (1). In our case, 3–4% acanthocytes were observed, but saline-diluted smears could not be performed due to laboratory limitations. Laboratory findings demonstrated markedly elevated CK with mildly elevated

AST, consistent with the diagnosis.

Seizures occur in about one-third of patients and may be the initial manifestation. Although systematic studies of seizure semiology are ongoing, most patients appear to meet the criteria for familial temporal lobe epilepsy (6). Peripheral sensorimotor neuropathy, often accompanied by the absence of deep tendon reflexes, is a frequent finding and can resemble motor neuron disease in its presentation (9). Our patient had a history of seizures, absent deep tendon reflexes, vibration sense loss, and reduced sensory nerve action potential amplitude in the right ulnar nerve on EMG.

Neuroimaging studies in ChAc commonly demonstrate caudate atrophy, similar to Huntington's disease. Putaminal atrophy, globus pallidus and striatal iron accumulation, and cerebellar atrophy may also occur (10,11). Metabolic studies have shown reduced metabolism in the caudate nucleus and putamen (12). Consistent with this, our patient's MRI revealed bilateral caudate and putaminal atrophy.

Chorea-acanthocytosis is inherited in an autosomal recessive manner. The VPS13A gene on chromosome 9q21 encodes the protein "chorein" (13). Recent studies have demonstrated that chorein expression is reduced in erythrocyte membranes and other tissues in patients with ChAc, representing an important diagnostic marker (12). In our patient, heterozygous pathogenic variants in exons 19 and 37 of the VPS13A gene were identified.

NA syndromes frequently present with executive dysfunction, obsessive-compulsive disorder, depression, and, less commonly, psychosis. These psychiatric manifestations may precede the onset of overt motor and cognitive decline (13). In ChAc, compulsive behaviors involving control, cleaning, symmetry, binge eating, and hoarding have been reported (3,13,14). Furthermore, characteristic self-injurious behaviors, such as tongue and lip biting, have also been interpreted as potentially related to OCD symptomatology (15). Given the central role of the caudate nucleus in the neurobiology of OCD, it is plausible that patients with ChAc, who typically develop caudate atrophy, manifest comorbid OCD (16,17). Our patient exhibited prominent OCD features, including hoarding, control-related obsessions, behavioral disturbances, anger dysregulation, and suicidal ideation. On SCID evaluation, he met criteria for MDD, GAD, OCD, and Adult ADHD, while WAIS testing indicated borderline intellectual functioning.

NA syndromes and their psychiatric comorbidities severely impair the quality of life of both patients and their families. Given their slowly progressive course, early recognition of psychiatric symptoms and appropriate management are essential for improving outcomes and quality of life. In our case, longstanding distress and diagnostic uncertainty contributed to the exacerbation of depressive symptoms and suicidality. Following diagnostic clarification and adjustment of treatment, notable improvements were observed in sleep, feeding difficulties, involuntary vocalizations, and obsessive symptoms, with complete resolution of suicidal ideation. In addition, consistent with previous recommendations (18), sustained multidisciplinary follow-up including physiotherapy, speech and occupational rehabilitation, and psychological support is strongly advised to optimize long-term outcomes and quality of life.

In conclusion, when evaluating psychiatric conditions with adult-onset movement disorders, clinicians should carefully investigate organic etiologies before assigning a primary psychiatric diagnosis. Educating patients and families regarding potential psychiatric symptoms, and encouraging treating physicians to consider psychiatric consultation when necessary, represent important steps toward comprehensive care.

Informed Consent: Written informed consent was obtained from the patient and his family for publication of this case report.

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Quadruplet pregnancy delusion in schizophrenia: A rare presentation of delusional procreation syndrome

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SUMMARY

Delusional Procreation Syndrome (DPS) encompasses delusions involving various stages of reproduction, including pregnancy, childbirth, and parenthood. Although the delusion of pregnancy (DoP) is the most commonly reported phenomenon, simultaneous delusions spanning multiple reproductive stages are rare. We describe a 39-year-old woman with chronic schizophrenia who presented with concurrent delusions of quadruplet pregnancy, miscarriage, stillbirth, childbirth, and motherhood. This case highlights the broad clinical spectrum of DPS and underscores the importance of distinguishing DoP from related conditions such as pseudocyesis and Couvade syndrome. It further illustrates the role of psychosocial adversity in shaping delusional themes.

Key words: Schizophrenia, Delusion, Pregnancy, Delivery, Delusional Procreation syndrome, Delusion of Pregnancy

INTRODUCTION

Delusional Procreation Syndrome (DPS) encompasses delusions pertaining to one or several possible stages of reproduction, such as the "delusion of having a spouse," "delusion of pregnancy (DoP)," "delusion of giving birth," and "delusion of being a parent (motherhood/fatherhood)" (Figure 1) (1). Although delusions involving various reproductive stages had been described earlier, it was Manjunatha et al. in (2010) who first unified them under a single framework and defined the syndrome as DPS. Later, they expanded the scope of the syndrome by including the delusion of surrogate polygamy (2). Among the delusions within DPS, the most frequently reported is DoP (3). While DoP is more common in women, it can also occur in men. However, an important clinical consideration is to distinguish DoP from pseudocyesis and Couvade syndrome (4).

In the Diagnostic and Statistical and Manuel of Mental Disorders, Fifth Edition (DSM-5), pseudocyesis is classified under Somatic Symptom and Related Disorders, under the subcategory "Other Specified Somatic Symptom and Related Disorder" and is defined as the false belief of being pregnant despite the presence of objective signs and reported symptoms of pregnancy (5). Couvade syndrome, on the other hand, refers to men whose partners are pregnant experiencing some pregnancy-related symptoms, such as changes in appetite, weight gain, and nausea (6).

To the best of the authors' knowledge, no previously reported case has exhibited five distinct delusions associated with DPS simultaneously. In this case report, we present a chronic schizophrenia patient who had long been deprived of treatment and demonstrated the clinical features of DPS. We believe that the coexistence of delusions of pregnancy with quadruplets, childbirth, and motherhood, together with delusions of stillbirth and miscarriage, which have not been previously discussed within the framework of DPS, will contribute to the existing literature on the syndrome.

This case presentation describes a female patient diagnosed with schizophrenia who had been untreated for a long time and simultaneously exhi-

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bited delusions of pregnancy with quadruplets, miscarriage, stillbirth, giving birth, and motherhood. This case is considered to contribute to the literature on DPS, as it encompasses four distinct delusional themes within DPS and uniquely includes the delusions of pregnancy with quadruplet, miscarriage, and stillbirth, which have not been previously reported together.

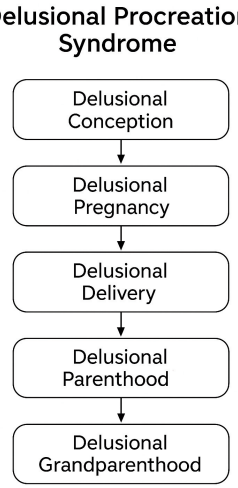
CASE REPORT

F.H., a 39-year-old widowed female patient, who had completed primary school and was unemployed at the time of admission. She resided alone in a container settlement after the 2023 Türkiye earthquakes and had limited social support. Although she had three children, she had not been in contact with them for about 3–4 years. The patient initially presented to an external obstetrics and gynecology outpatient clinic to learn about the fate of the children she believed she had given birth to. Upon evaluation, it was determined that she had no pregnancy follow-up records and no objective medical evidence of pregnancy within the reported time frame. When it became clear that she had not given birth, she was referred to the psychiatry outpatient clinic and subsequently transferred to our hospital for inpatient care.

From her sister's account, it was learned that the patient had divorced one year earlier and had three children aged 23, 20, and 17, with whom she was socially disconnected due to her illness. The children reportedly did not wish to maintain contact with her. Her first psychiatric admission had been approximately seven years earlier, presenting with paranoid, referential, and grandiose delusions. She was hospitalized for 17 days but discontinued follow-up or treatment thereafter. Her delusional symptoms persisted for seven years, with no insight into her illness. A nephew was also under psychiatric follow-up with a diagnosis of psychotic disorder.

At her initial psychiatric examination, the patient appeared to be of her stated age, had adequate self-care, was alert, and fully oriented to person, place, and time. She was cooperative, with a slightly elevated affect. Abstract thinking was intact, but judgment was partially impaired. She reported no perceptual disturbances. Her delusions included referential beliefs ("They keep calling me from the television and giving me important messages from the government"), grandiose delusions (believing she could move objects with her mind and that she was superior to others), and persecutory delusions (believing that neighbors were photographing her and that people at the hospital where she believed she had given birth wanted to harm her babies).

Figure 1.



During her psychiatric history, she stated that she had become pregnant after a relationship with a married man from the container settlement. She reported recognizing her pregnancy in the first week through symptoms such as sensitivity to odors, nausea, and abdominal pain, and that her initial blood test at a local primary care center was negative because it was too early. In the second month, she went to an obstetrics and gynecology clinic, where a blood test and ultrasound were performed. She insisted that she had seen four fetuses on the ultrasound, claiming, "The doctor told me it was just intestinal gas to surprise me, but as a mother, I immediately understood I had four babies."

When she shared the news of her pregnancy with the man she considered the father, she reported that she learned he did not want children. She claimed that in the third month she miscarried one baby. She further alleged that during subsequent hospital visits, doctors withheld the existence of her triplets from her as part of a plan. In the eighth month, she presented to the emergency depart-

ment with abdominal pain and bleeding. She reported that labor began in the emergency room toilet, where she gave birth alone: the first baby, followed by brief fainting, then the second baby. She stated she cut the umbilical cords herself, identified one infant was male and the other female, and expressed joy. According to her, a nurse then took the babies away, promising to transfer them and that she could follow later. At home, she claimed to have delivered the third baby alone, describing it as stillborn. A few days later, when she returned to the hospital to retrieve her babies, she believed the staff had been replaced to conceal them from her.

Upon referral to the gynecology clinic, it was confirmed that she had no elevated beta-hCG levels or any clinical signs of pregnancy in her medical history. She was then referred to psychiatry and admitted to our inpatient unit with family involvement.

During hospitalization, routine laboratory investigations were performed to rule out organic causes. hematological parameters (hemoglobin, white cell count, platelets), biochemical assays (liver and renal function tests, electrolytes, thyroid profile), and hormonal assays (prolactin, FSH, LH, estradiol, TSH) were all within normal reference ranges. A non-contrast brain MRI revealed no structural or ischemic abnormalities. Psychometric assessments showed a Positive and Negative Syndrome scale (PANSS) score of 98 and a Clinical Global Impression (CGI) score of 6. Medical records indicated prior irregular psychiatric consultations for schizophrenia without consistent treatment.

Her treatment was initiated with risperidone, titrated up to 6 mg/day. After four weeks, no clinical improvement was observed, PANSS(88) and CGI(5) scores showed no significant change, and hyperprolactinemia (>200 ng/mL) was detected. Risperidone was cross-tapered to aripiprazole at a dosage of 10 mg/day. After four weeks with no improvement, clozapine was introduced, titrated to 400 mg/day. Partial benefit was observed: she was able to question some of her delusional beliefs but remained convinced of giving birth and continued planning to find her supposed children. Considering treatment-resistant psychosis, electroconvulsive therapy (ECT) was initiated under anesthesia, with 12 sessions administered.

After approximately 12 weeks of treatment, the patient showed enriched thought content, improved social participation, reduced negative symptoms, and subthreshold persistence of positive symptoms. Although she did not spontaneously mention her delusional content, she occasionally expressed doubt about their validity. Psychometric assessments showed improvement, with the PANSS score decreasing to 58 and the CGI score to 4. The patient was discharged with partial remission to outpatient follow-up. At her second- and fourth-week post-discharge visits, she was reported to be adherent to treatment, receiving family support, not engaging in delusion-driven behaviors, and demonstrating partial insight.

Written informed consent was obtained from the patient for publication of this case report.

DISCUSSION

In psychiatry, many delusional syndromes have been described to date. Some examples include Cotard syndrome, Capgras syndrome, Othello syndrome, Fregoli syndrome, and Couvade syndrome. Manjunatha et al. added a new entity to this list by defining the cluster of delusions that can involve one or more stages of reproduction as Delusional Procreation Syndrome (DPS) (1).

DPS can be associated with various psychiatric or neurological disorders such as schizophrenia, schizoaffective disorder, delusional disorder, epilepsy, dementia, and organic brain syndromes (1). In a citation analysis of published cases related to Delusional Procreation Syndrome (DPS), both male and female patients were reported; however, the majority were female. Among patients with DPS, delusion of pregnancy was identified as the most frequently reported delusional theme (87.6%). The most common psychiatric diagnosis associated with DPS was schizophrenia (42%), followed by bipolar disorder (13%). A key distinction must be drawn between DoP and pseudocyesis, the main difference being that physical pregnancy symptoms are present in pseudocyesis (7). This

case also involved a female patient with a diagnosis of schizophrenia.

It has been reported that psychosocial factors play an important role in the development of DoP in schizophrenia. Pregnancy has been described as an experience that reduces feelings of loneliness and helplessness by allowing the mother to form a bond with the fetüs (4). Shankar considered the formation of delusional beliefs as an adaptive mechanism reflecting an individual's conflicts and life experiences. Loss of a valued object, profound loneliness, and the loss of a real or imagined relationship are thought to trigger DoP as a compensatory mechanism (8). Similar psychosocial factors were clearly evident in this case. Being widowed and estranged from her children, the patient lived alone. The post-earthquake container settlement environment with limited social support may have increased her feelings of loneliness and helplessness, thus contributing to the emergence of delusions themed around pregnancy and motherhood. Therefore, the patient's delusional experience appears consistent with psychosocial explanations reported in the literature.

In previously reported DPS cases, one or more stages of reproduction were often present together. A 2013 study described six cases of DPS, including delusions of fatherhood, childbirth (labor and delivery), having a spouse and motherhood, motherhood alone, and having a spouse alone (9). Similarly, the first study that introduced DPS included three cases: delusions of having a spouse and fatherhood, having a spouse and motherhood, and having a spouse, motherhood, and childbirth (1). DPS may also include delusions of multiple pregnancies. For instance, a 2014 case report described a postmenopausal woman with a delusion of giving birth to twins (10). In a case series published in 2024, a female patient with schizophrenia presenting with a delusion of quadruplet pregnancy was reported (11). In this case, delusions of stillbirth and miscarriage, which have not been previously discussed within the framework of Delusional Procreation Syndrome (DPS), coexisted with delusions of pregnancy with quadruplets, childbirth, and motherhood, representing a unique clinical constellation.

In female patients receiving antipsychotic treatment, elevated prolactin levels may be observed. Hyperprolactinemia can lead to symptoms such as amenorrhea and galactorrhea, which are also seen during pregnancy. DoP may occur in the context of antipsychotic use or other conditions that cause hyperprolactinemia, such as prolactinoma. In such cases, discontinuation of the offending antipsychotic has been reported to result in resolution of the delusional beliefs (11).

In the management of DoP, prolactin-sparing antipsychotics such as aripiprazole may be preferred. ECT has been reported to produce only limited and temporary improvement in such cases (12). In addition to pharmacotherapy, non-pharmacological interventions such as psychoeducation, cognitive, and supportive psychotherapy may positively influence treatment outcomes by enhancing insight, strengthening coping abilities, and improving overall functioning (11).

This case, the patient simultaneously exhibited delusions of pregnancy with quadruplets, miscarriage, stillbirth, childbirth (labor and delivery), and motherhood. By encompassing these multiple aspects of reproduction, this case contributes to the literature on DPS as an example of the syndrome's broader spectrum.

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Possible association between immunoglobulin a vasculitis and the development of schizophrenia: A case study

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SUMMARY

This case report describes a 41-year-old woman with a history of childhood immunoglobulin A vasculitis and adult-onset schizophrenia, to the best of our knowledge, representing the first clinical observation suggesting a potential link between these conditions. Although current serological evaluations showed no active vasculitis, the case highlights a possible association, in line with research indicating an increased risk of schizophrenia in individuals with autoimmune disorders, including vasculitis. Mechanistically, immunoglobulin A vasculitis could hypothetically influence neuroimmune processes through transient effects on the blood-brain barrier or microvascular function, potentially contributing to pathways relevant to psychosis. Certain antipsychotic treatments are also known to interact with immune responses, supporting the rationale for further investigation. While causality cannot be inferred from a single case, this report underscores the importance of exploring immunoglobulin A vasculitis as a factor in schizophrenia pathogenesis and encourages longitudinal and mechanistic studies to better understand the potential neuroimmune mechanisms involved.

Key words: Schizophrenia, immunoglobulin A, IgA vasculitis, autoimmune diseases, mental disorders, blood-brain barrier

INTRODUCTION

Schizophrenia is a serious mental disorder marked by psychosis and significant disability (1). Genetic and environmental aspects (2) and immune responses (3) are believed to play a significant role in the development of schizophrenia. (3)

Vasculitis encompasses a diverse group of disorders characterized by inflammation of blood vessel walls. As the index case had a pediatric onset, it is important to note that epidemiological data on childhood vasculitis remains limited and insufficiently defined (4). Immunoglobulin A vasculitis (IgAV) formerly Henoch Schönlein Purpura (HSP) is an IgA-mediated autoimmune vasculitis that impacts multiple organs (5).

Increasing evidence of immune involvement in schizophrenia has prompted research on its links to autoimmune diseases and infections (6,7). Evidence from cerebrospinal fluid analyses indicates an association between blood-brain barrier (BBB) impairment and psychosis (8). Circulating autoantibodies are enabled to penetrate the damaged BBB and influence the central nervous system (7). The immune system's broader role in antipsychotic treatment response is key research. Medications like clozapine (for treatment-resistant schizophrenia) are linked to lower serum immunoglobulin levels (6). The dysregulation of immune-related genes plays a significant role in schizophrenia (9,10).

Although studies link autoimmune diseases like vasculitis to schizophrenia (11–13), the potential correlation with IgAV has not been specifically examined. Our study aims to explore this possible relationship.

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CASE REPORT

We present a case of a 41-year-old single woman with a high school education, no employment history, and a diagnosis of schizophrenia. She lives with her family and has been under regular psychiatric follow-up. Premorbid irritability and poor social adjustment were noted. Her first psychotic symptoms appeared at age 16, including persecutory and referential delusions, social withdrawal, and self-neglect, which persisted for two years. She has no history of hospitalization, suicide attempts, or substance use, and no family history of psychiatric illness.

The patient meets the diagnostic criteria for schizophrenia according to the DSM-5 (Diagnostic and Statistical Manual of Mental Disorders, 5th edition). Current Positive and Negative Syndrome Scale positive, negative, and total scores were 9, 16, and 52, respectively. Global Assessment Scale score was 60. She maintains basic self-care, shows restricted affect with euthymic mood, and has goal-directed thoughts. While some referential ideation persists, no active delusions or hallucinations were noted. Insight is partial. She continues to live with family support. She was initially treated with risperidone but experienced partial symptom control and poor adherence to adjunctive treatments. Olanzapine was discontinued due to side effects. A combination of risperidone 8 mg/day and quetiapine XR 150 mg/day has since provided clinical stability. Despite occasional subthreshold symptom increases, the patient has not experienced a significant relapse or required hospitalization since the initial psychotic episode.

The patient has a significant medical history, including a diagnosis of HSP at age 7, which was found incidentally after purpuric lesions appeared on her hips. She was admitted for three weeks of inpatient care, resulting in full remission. Since then, there have been no recurrences of similar symptoms, and no other major illnesses have been reported during her childhood. About two years ago, she was diagnosed with Hashimoto's thyroiditis and is currently under regular follow-up and treatment with levothyroxine.

Physical examination revealed a BMI of 38.1 kg/m^2 (Class II obesity). Orientation and cooperation were intact. Vital signs were normal, and systemic examinations of cardiovascular, respiratory, abdominal, and dermatological systems were unremarkable.

The neurological exam showed intact cranial nerve functions. Muscle strength was normal in all extremities, with no focal neurological issues found. Sensory testing also showed normal results. Deep tendon reflexes were normal and symmetrical. Cerebellar assessments, specifically the finger-to-nose and heel-to-shin tests, were conducted and yielded no abnormalities in performance. Gait appeared normal.

Initial neuroimaging (cranial MRI and cervical-cranial angiography, 2017) showed no evidence of parenchymal, atrophic, or vascular pathology. Routine laboratory tests, including complete blood count, fasting blood glucose, lipid profile, electrolytes, renal and liver function tests, were within normal limits.

Although the patient currently reports no symptoms suggestive of vasculitis, a vasculitis panel was conducted to investigate potential autoimmune or inflammatory conditions. The results were negative (Table 1). Comprehensive serologic testing showed no evidence of vasculitis, with negative anti-neutrophil cytoplasmic antibodies (ANCA), including both myeloperoxidase (MPO) and proteinase 3 (PR3) subtypes, effectively ruling out ANCA-associated vasculitides such as granulomatosis with polyangiitis and microscopic polyangiitis. Negative antinuclear antibody (ANA) and anti-double-stranded DNA (anti-dsDNA) results argue against systemic lupus erythematosus (SLE)-related vasculitis. Complement levels revealed normal C3 and low-normal C4, indicating no significant complement consumption suggestive of immune complex-mediated vasculitis. The erythrocyte sedimentation rate (ESR) was within normal limits, and although D-dimer was mildly elevated, this finding is non-specific in the absence of supporting clinical features. Finally, a comprehensive antiphospholipid

Table 1: Vasculitis panel results for the patient

Test Name	Result	Reference Range
Anti-beta-2 glycoprotein IgG	<2 RU/mL	≤ 20 RU/mL
Anti-beta-2 glycoprotein IgM	Negative, 7.68 RU/mL	≤20 RU/mL
MPO ANCA	<2 RU/mL	≤ 20 RU/mL
PR3 ANCA	<2 RU/mL	≤ 20 RU/mL
Anti-cardiolipin IgM	Negative, 3.03 PL-IgM-U/mL	<12 PL-IgM- U/mL
Anti-cardiolipin IgG	Negative, 2.53 PL-IgG-U/mL	<12 PL-IgG-U/mL
Anti-ds DNA IFA	Negative	IIF (Crithidia luciliae) <1:10 titre (Negative)
Anti-nuclear antibody (ANA)	Negative	IIF <1/100
Erythrocyte Sedimentation Rate (ESR)	18 mm/hr	2- 20 mm/hr
Anti-phospholipid IgM	Negative, 1.44 IU/mL	Negative <12
Anti-phospholipid IgG	Negative, 4.45 IU/mL	Negative <12
C4	0.15 g/L	0.10- 0.40 g/L
C3	1.47 g/L	0.90- 1.80 g/L
D-dimer	0.61 µg/mL	0- 0.5 µg/mL

Autoimmune and inflammatory markers assessed in the patient. ANA: Anti-nuclear antibody, ANCA: Anti-neutrophil cytoplasmic antibody, dsDNA: Double-stranded DNA, ESR: Erythrocyte sedimentation rate, IFA: Indirect immunofluorescence assay, IgG: Immunoglobulin G, IgM: Immunoglobulin M, MPO: Myeloperoxidase, PR3: Proteinase 3, RU: Relative Units, PL: Phospholipid, IU: International Units.

antibody panel was negative, providing no evidence for antiphospholipid syndrome (APS) as a potential vasculitis mimic.

DISCUSSION

This study aimed to explore the underlying mechanisms of schizophrenia, offering new insights that may enhance our understanding of this complex disorder. Growing evidence suggests that immune dysregulation, particularly in autoimmune conditions, may play a role in schizophrenia's pathogenesis. This case report adds to the growing body of observations suggesting a possible association between schizophrenia and immune system dysfunction, particularly in the context of autoimmune vasculitis such as IgAV.

A Danish population study (N=7,704) of schizophrenia cases diagnosed between 1981-1998 revealed a 45% increased risk of schizophrenia among individuals with any autoimmune disorder, suggesting broader autoimmune associations (11). Another study from the National Health Insurance Research Database included a total of 118,921 participants (10,811 schizophrenia; 108,110 control) shows that there is a significant positive association between hypersensitivity vasculitis and schizophrenia with an ODD ratio of five (12). A follow-up study using the same database of 64,817 patients with autoimmune diseases and an equal number of age-matched controls investigated the association between autoimmune diseases and the development of schizophrenia. Over a maximum follow-up period of 10 years, autoimmune vasculitis (polyarteritis nodosa, hypersensitivity angiitis, Wegener's granulomatosis, giant cell arteritis, thromboangiitis obliterans, Takayasu's disease, acute febrile mucocutaneous lymph node syndrome, Behçet's syndrome) was linked to a significantly elevated risk of schizophrenia development in individuals with autoimmune diseases in the study (13).

Several recent case reports illustrate the clinical intersection of psychosis and autoimmune vasculitis. Gasparinho et al. reported a 42-year-old man with no prior psychiatric history who presented with a first manic episode with psychotic symptoms; subsequent work-up revealed ANCA-negative granulomatosis with polyangiitis (GPA) with central nervous system involvement. Immunosuppressive therapy achieved complete remission of psychiatric symptoms within one year. (14). Another report by Castle et al. detailed a 29-year-old male with schizophrenia who was ultimately diagnosed with c-ANCA-positive GPA initially presenting at the petrous apex (15), and Latvala et al. reported giant cell arteritis presenting with manic-psychotic symptoms (16), highlighting the diagnostic challenges and potential delays in autoimmune detection in psychiatric patients.

While aforementioned autoimmune diseases are associated with an increased risk of schizophrenia, the timing of immune events appears to be crucial. Compelling evidence shows a longitudinal dose-response association between childhood IL-6 levels

and future risk for depression and psychosis (17), coupled with elevated adolescent CRP predicting adult schizophrenia (18). However, the impact of specific entities like IgA vasculitides remains a gap this case aims to address. Though our patient lacked acute neurologic symptoms du-ring IgAV, retrospective studies suggest that headache and behavioral changes (31% of cases) may signify subtle CNS involvement. (19). Indeed, our patient showed premorbid irritability and poor social adjustment during school years, features often linked to EEG abnormalities and transient neurovascular dysfunction (19,20). In our patient, subclinical inflammation from IgAV may have caused lasting blood–brain barrier disruption, faci-litating neuroimmune mechanisms in schizophrenia. EEG, neurocognitive, and CSF assessments were not performed, representing a study limitation; future reports should include these. Although diagnosed with Hashimoto's thyroiditis two years ago, data on initial thyroid tests are lacking, but no psychotic episodes occurred before this diagnosis.

The deposition of IgA1-containing immune complexes in vessel walls is a key pathogenic event that promotes the development of IgAV (21). One study suggests that IgA antibodies may cross-react with host cardiolipin- a mitochondrial phospholipid (22), with increased levels of anticardiolipin antibodies also reported in patients' serum with schizophrenia (23), leading to the formation of immune complexes that contribute to vascular injury (24). Moreover, complement activation has been shown to mediate microvascular injury in IgAV and IgA nephropathy, supporting a mechanism by which IgA-containing immune complexes induce endothelial damage (25). Nailfold videocapillaroscopy has shown significant microvascular abnormalities, including architectural disarrangement and edema, during acute IgAV, with capillary edema persisting at 6-month follow-up despite symptom resolution (26). Analogous processes in childhood IgAV could transiently affect cerebral microvasculature, potentially disrupting the blood-brain barrier and priming long-term neuroimmune effects relevant to psychosis.

The nine-year gap between IgAV onset and psychosis suggests possible coincidence or shared genetic–immune risks that cannot be excluded. Yet, the case meets several of Hill's causality criteria: the immune event preceded psychosis, IgAV's inflammation could affect neurodevelopment, and the link aligns with epidemiological evidence. Though causation remains unproven, this case proposes that childhood IgAV may increase schizophrenia risk, warranting further longitudinal and mechanistic studies.

Another case study correlated IgAV and methylphenidate (27), a stimulant that increases dopamine release (28). These findings suggest a potential metabolic pathway linking IgAV to psychosis, possibly mediated by blood-brain barrier disruption and subsequent neuroinflammation (29).

Patient's D-dimer elevation is unlikely due to inactive childhood IgAV. The elevation may instead be explained by schizophrenia, referencing Geng et al. who reported elevated D-dimer in this disorder (30).

This case may be among the first to suggest a link between IgAV and schizophrenia, supported by biological plausibility and prior evidence on immune-related psychosis. However, as a single report with recall bias and unmeasured variables, it cannot confirm causality. The proposed mechanism of autoantibody passage across a damaged blood–brain barrier is limited by unmeasured factors such as exposure duration, antibody levels, and individual susceptibility. Larger epidemiological and molecular studies are needed to validate these findings. If confirmed, this link could inform precision diagnostics and immunomodulatory strategies for psychosis spectrum disorders.

Consent to participate

Written informed consent for the publication was obtained from the patient and her legal representative.

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Lorazepam use in the treatment of pre-meal anxiety of anorexia nervosa: Three adolescent cases

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SUMMARY

The purpose of this case report was to explore the use of lorazepam, a benzodiazepine, for treating pre-meal anxiety in adolescent patients diagnosed with anorexia nervosa (AN). This study aimed to assess the effects of lorazepam on reducing anxiety, increasing food intake, and improving treatment adherence in these patients.Three cases of adolescent females with AN were presented, highlighting their clinical characteristics, treatment interventions, and outcomes. The patients received a combination of pharmacotherapy (fluoxetine, aripiprazole, and lorazepam) and psychotherapy (cognitive-behavioral therapy) as part of their treatment regimen. The dosage and duration of lorazepam administration varied for each patient based on individual needs. The introduction of lorazepam along with other medications resulted in a reduction in pre-meal anxiety and an increase in food intake among the patients. Furthermore, treatment compliance and motivation improved, leading to weight gain and resumption of menstrual cycles in all cases. Positive effects of lorazepam were observed even after discontinuation of the medication.This case report suggests that the use of lorazepam for treating pre-meal anxiety in adolescent patients with AN may be beneficial in reducing anxiety, enhancing treatment adherence, and facilitating healthy eating habits. However, due to the limited evidence available, benzodiazepines are not recommended as a first-line treatment for AN, and their usage should be cautious due to the potential risks of dependence and withdrawal. Further research is needed to evaluate the efficacy and safety of benzodiazepines, including different types and doses, for treating AN.

Key words: Anorexia nervosa, lorazepam, premeal, anxiety

INTRODUCTION

Anorexia nervosa (AN) is a severe psychiatric disorder marked by self-imposed caloric restriction, intense fear of weight gain, and significantly low body weight (1). It frequently manifests during adolescence and is associated with high morbidity and mortality. AN is often comorbid with psychiatric symptoms such as mood disorders, anxiety, obsessive behaviors, and cognitive rigidity (2). Effective treatment involves multidisciplinary interventions, with nutritional rehabilitation and psychotherapeutic support as the cornerstones (3). Pharmacotherapy is generally limited in efficacy, with SSRIs and antipsychotics offering modest benefits (4).

Benzodiazepines are a class of medications that exert their anxiolytic effects through enhancement of the GABA-A receptor, leading to central nervous system inhibition [5]. While primarily used for anxiety and insomnia [6], benzodiazepines have been considered in AN, particularly for cases complicated by severe pre-meal anxiety that impedes food intake (7,8). Lorazepam, due to its short half-life and relative safety in lower doses, has been proposed as a potential adjunct in this context (9,10).

However, long-term use of benzodiazepines is discouraged due to risks including tolerance, dependence, and withdrawal (5,11). Clinical guidance generally does not recommend them as a first-line treatment for AN [11]. Despite this, selective short-term use may have utility in highly anxious patients struggling with refeeding and meal compliance (12,13).

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This case series describes the clinical course of three adolescent females diagnosed with AN who were administered lorazepam as an adjunctive treatment for pre-meal anxiety. Outcomes related to anxiety reduction, treatment compliance, weight gain, and resumption of menstrual cycles are discussed in light of existing literature. Symptom severity was monitored using the Eating Disorder Examination-Questionnaire (EDE-Q), a validated self-report scale assessing eating disorder-related thoughts and behaviors (14). The EDE-Q scores were recorded at baseline and again at the end of the eating disorder treatment process, following lorazepam discontinuation. This study was conducted in accordance with the Declaration of Helsinki, and ethical approval was obtained from the Ethics Committee of Ankara University Faculty of Medicine (Ethics Committee Decision No: İ06-462-24). Written informed consent was obtained from all adolescents and their parents for participation and publication of the cases.

CASE PRESENTATIONS

Case 1

A 15-year-old female high school student presented with a 13 kg weight loss over 10 months, resulting in a body mass index (BMI) of 15.3 kg/m^2. She followed a restrictive eating pattern, typically consuming only one meal per day, accompanied by binge episodes 3–4 times per week and subsequent self-induced vomiting. While she did not avoid eating in public, she restricted intake during the day and purged more frequently after family dinners, when her anxiety was at its peak. She reported three months of amenorrhea, guilt after consuming carbohydrate-rich foods, and a sense of relief following purging.

She was well-oriented and communicated openly. However, she reported persistent anxiety, distractibility, and fatigue, particularly in recent weeks. Her thought content included excessive concerns about weight gain and body image, consistent with underlying anxiety. She demonstrated partial insight into her condition. Based on DSM-5 criteria, she was diagnosed with anorexia nervosa, binge–purge subtype (1). She had no significant medical comorbidities aside from malnutrition-related endocrine effects, and her laboratory findings were within acceptable limits.She was managed by a multidisciplinary team in an adolescent clinic. Nutritional rehabilitation included a structured diet plan under dietitian supervision, without formula supplementation. Fluoxetine (20 mg/day) was initiated and titrated to 40 mg/day, along with cognitive-behavioral therapy (CBT). Due to persistent purging, the fluoxetine dose was increased to 60 mg/day, and aripiprazole (2.5 mg/day, increased to 5 mg/day) was added.

Despite these adjustments, vomiting after dinner persisted. In CBT sessions, she could resist urges during the day but struggled during family dinners, where heightened anticipatory anxiety impaired her coping. Lorazepam 0.5 mg administered before dinner significantly reduced this anxiety, facilitated meal completion, and decreased vomiting. She gained 3 kg in one month. Lorazepam was tapered after four weeks. She continued fluoxetine and aripiprazole for eight months, ultimately gaining 8 kg, resuming menstruation, and demonstrating a reduction in her EDE-Q score from 45 to 9.

Case 2

A 13-year-old female was referred with a 13 kg weight loss over one year. At presentation, her weight was 35 kg, height 150 cm, and BMI 15.6 kg/m^2. Her diet was limited to water and coffee, occasionally supplemented by a single meal, along with compulsive calorie counting and excessive exercise. She reported binge eating approximately three times per week, typically followed by extended fasting periods. She demonstrated significant body image dissatisfaction, particularly with the appearance of her legs, and had experienced secondary amenorrhea for five months, which she appeared unconcerned about.

She appeared extremely weak, pale, and fatigued. Although well-oriented, she responded minimally during the interview and preferred silence, showing negativistic features. Her affect was dysthymic, and she displayed symptoms of depression. Her thoughts centered around fears of gaining weight and becoming fat. Despite this, she had insight into

her condition.

She was diagnosed with anorexia nervosa, restrictive type [1]. Apart from mild endocrine abnormalities related to malnutrition, she had no other significant medical comorbidities, and laboratory findings were within normal limits, supporting outpatient care. She continued follow-up in the adolescent clinic under a multidisciplinary team. Nutritional rehabilitation was based on a balanced meal plan supervised by a dietitian, without formula supplementation.

Fluoxetine was initiated but discontinued due to side effects. Sertraline was started and titrated to 75 mg/day, which improved social interaction but did not significantly enhance dietary adherence. Aripiprazole (2.5 mg/day, increased to 5 mg/day) was added, leading to partial improvement in dietary compliance. However, restrictive behaviors persisted, and she occasionally vomited after meals.

Lorazepam 0.5 mg was prescribed before lunch and dinner for eight weeks. This led to a notable reduction in pre-meal anxiety, improved adherence to the nutritional plan, and initial weight gain. Menstruation resumed within a few weeks. Lorazepam was tapered without recurrence of significant anxiety.

Over the following 12 months, she remained on sertraline and aripiprazole and continued regular psychiatric and dietary follow-up. She gained a total of 14 kg, and her EDE-Q score decreased from 55 to 10. EDE-Q was recorded at baseline and at the end of treatment, with the reduction aligning with clinical improvements in weight, menstruation, and anxiety symptoms.

Case 3

A 16-year-old 10th-grade female presented with severe weight loss, dropping from 52 kg to 39.5 kg over six months. She initially had a BMI of 22.5 kg/m^2 (72nd percentile). Her dieting began following bullying by male classmates who nicknamed her "Fat." She gradually increased her daily exercise to 40 minutes using online workout videos, while secretly discarding food to avoid detection by her parents. Her BMI declined to the 1.46th percentile, accompanied by secondary amenorrhea, irritability, and resistance to treatment.

On physical examination, her hands were dry and yellowish, and her facial bones were visibly prominent. She wore a coat indoors to conceal her weight loss and appeared younger than her age. She was conscious and oriented but displayed temper tantrums and emotional lability. Her attitude toward the interviewer was hostile, with an irritable affect and depressed mood.

She was diagnosed with severe anorexia nervosa and comorbid major depressive disorder, with an initial EDE-Q score of 69. Apart from malnutrition-related endocrine abnormalities, she had no other medical comorbidities, and her laboratory results were within normal limits, supporting outpatient management.

She was treated by a multidisciplinary team. Nutritional rehabilitation was conducted through a structured diet plan without formula supplementation. Pharmacological treatment included fluoxetine (up to 50 mg/day), olanzapine (2.5 mg/day), and enhanced cognitive-behavioral therapy (CBT-E) involving active family participation.

Lorazepam 0.5 mg was prescribed before each main meal for three months. This intervention effectively reduced anticipatory anxiety and improved meal completion. Over time, she was able to overcome pre-meal anxiety and complete meals more consistently.

After six months, her weight increased to 48 kg (32nd percentile), and menstruation resumed at 46.5 kg. Her EDE-Q score decreased from 69 to 8.

CBT-E focused on restructuring negative cognitions, correcting body image distortions, and addressing affect regulation in relation to eating behaviors. Family support and structured meal monitoring were emphasized throughout treatment, contributing to long-term stabilization.

DISCUSSION

Despite advances in understanding AN, pharmacological treatment remains a challenge. The World Federation of Societies for Biological Psychiatry provides no definitive pharmacologic guidance for AN due to heterogeneous findings and lack of robust evidence (12). Studies examining SSRIs and antipsychotics have shown mixed results, especially regarding weight gain and core eating behaviors (4,13–15).

Traditional psychotropic approaches often overlook one of the most pressing barriers to refeeding: pre-meal anxiety. High levels of anticipatory anxiety are frequently observed in AN and are predictive of poor meal completion (10). A study assessing alprazolam found no benefit in reducing food intake in AN patients (7). However, low-dose lorazepam has shown some promise as a targeted intervention for meal-related anxiety without excessive sedation (9,11).

In this series, all three patients responded positively to lorazepam when used specifically to manage pre-meal anxiety. Notably, benefits extended beyond the treatment window—an important observation consistent with prior case reports (16,17). Lorazepam appeared to serve as a behavioral catalyst, reducing anxiety long enough for patients to develop more adaptive coping strategies via CBT.

These outcomes align with prior work linking pre-meal anxiety and intake deficits (10). Similar benefit was observed in a case of Avoidant/Restrictive Food Intake Disorder (ARFID)—a condition characterized by restrictive or avoidant eating behavior without body-image disturbance—treated with lorazepam for severe feeding avoidance, reinforcing its potential in restricted intake disorders (16).

Still, caution is warranted. Patients with the binge-purge subtype are at increased risk of substance misuse, and prolonged benzodiazepine use carries well-documented risks (5). In this series, lorazepam was used short-term (ranging from one to three months) under strict supervision.

Given these limitations, exploring non-benzodiazepine anxiolytics may be prudent. Buspirone, for instance, has been used in combination with SSRIs in AN patients with obsessive-compulsive traits (17), and another case highlighted its benefit in ARFID (18). While systematic studies are lacking, such alternatives warrant investigation, particularly in adolescents requiring long-term anxiety management.

The therapeutic model in this case series aimed not only to reduce symptoms but to build sustainable treatment behaviors. By targeting pre-meal anxiety during a critical window of nutritional rehabilitation, lorazepam may have facilitated behavioral change. This is consistent with theories suggesting that diminishing anticipatory distress enhances adherence to feeding plans, which can later be internalized (12,13).

This case series suggests that short-term, low-dose lorazepam can be a useful adjunct for managing pre-meal anxiety in adolescents with AN. In these cases, lorazepam improved anxiety symptoms, food intake, treatment adherence, and weight restoration. Importantly, benefits persisted after discontinuation, and no complications related to benzodiazepine use were observed. No adverse effects such as sedation, tolerance, dependence, or withdrawal symptoms were observed during or after lorazepam administration in any of the cases. The reductions in EDE-Q scores closely mirrored clinical improvement in all cases, including weight gain, return of menstruation, and reduction in pre-meal anxiety.

Nevertheless, due to the limited evidence and inherent risks, benzodiazepines should not be used as first-line treatments and should be administered only under strict supervision. Future randomized controlled trials are needed to assess the safety, efficacy, and optimal dosing of benzodiazepines and to explore alternative anxiolytic strategies such as buspirone in this population.

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