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

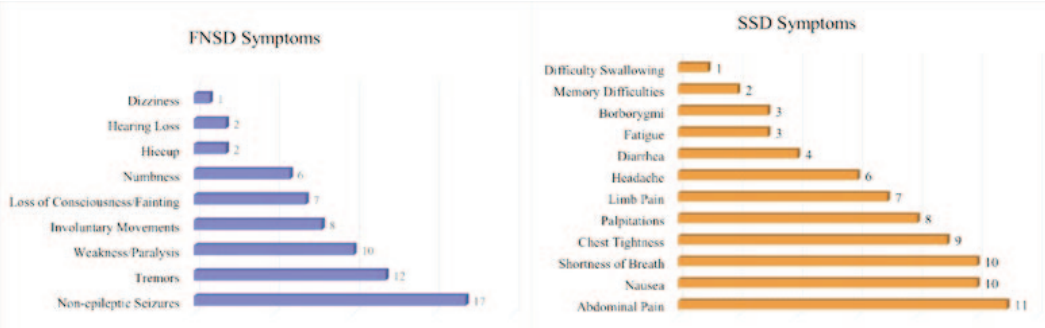

Figure 1: Nature of Symptoms

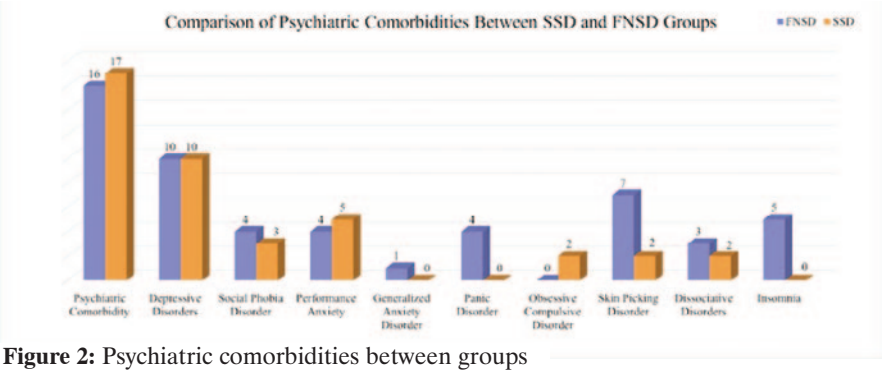

Figure 2: Psychiatric comorbidities between groups

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.

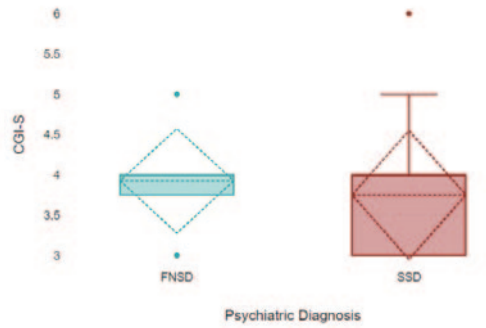

Figure 3: Distribution of Clinical Global Impression-Severity (CGI-S) Scores in SSD and FNSD Groups

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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REFERENCES

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Arlington (VA): American Psychiatric Publishing; 2013.

2. Okur Güney ZE, Sattel H, Witthöft M, Henningsen P. Emotion regulation in patients with somatic symptom and related disorders: A systematic review. PLoS One. 2019;14(6):e0217277.

3. Vesterling C, Schütz-Wilke J, Bäker N, Bolz T, Eilts J, Koglin U, Rademacher A, Goagoses N. Epidemiology of somatoform symptoms and disorders in childhood and adolescence: A systematic review and meta-analysis. Health Soc Care Community. 2023;2023(1):6242678.

4. Gao X, McSwiney P, Court A, Wiggins A, Sawyer SM. Somatic symptom disorders in adolescent inpatients. J Adolesc Health. 2018;63(6):779–84.

5. Andresen JM, Woolfolk RL, Allen LA, Fragoso MA, Youngerman NL, Patrick-Miller TJ, Gara MA. Physical symptoms and psychosocial correlates of somatization in pediatric primary care. Clin Pediatr (Phila). 2011 Oct;50(10):904-9. doi: 10.1177/0009922811406717. Epub 2011 May 16. PMID: 21576182.

6. Tsai CH. Factor analysis of the clustering of common somatic symptoms: A preliminary study. BMC Health Serv Res. 2010;10:1–8.

7. Weiss KE, Steinman KJ, Kodish I, Sim L, Yurs S, Steggall C, Fobian AD. Functional Neurological Symptom Disorder in Children and Adolescents within Medical Settings. J Clin Psychol Med Settings. 2021 Mar;28(1):90-101. doi: 10.1007/s10880-020-09736-2. PMID: 32743729.

8. Malas N, Ortiz-Aguayo R, Giles L, Ibeziako P. Pediatric somatic symptom disorders. Curr Psychiatry Rep. 2017;19(2):11.

9. Janssens KA, Klis S, Kingma EM, Oldehinkel AJ, Rosmalen JG. Predictors for persistence of functional somatic symptoms in adolescents. J Pediatr. 2014;164(4):900–5.

10. Löwe B, Levenson J, Depping M, Hüsing P, Kohlmann S, Lehmann M, Shedden-Mora M, Toussaint A, Uhlenbusch N, Weigel A. Somatic symptom disorder: a scoping review on the empirical evidence of a new diagnosis. Psychol Med. 2022 Mar;52(4):632-648. doi: 10.1017/S0033291721004177. Epub 2021 Nov 15. PMID: 34776017; PMCID: PMC8961337.

11. Dell ML, Campo JV. Somatoform disorders in children and adolescents. Psychiatr Clin North Am. 2011;34(3):643–60.

12. Kleinstäuber M. Somatic symptom and related disorders. In: IsHak WW, editor. Atlas of psychiatry. Cham: Springer; 2023. p. 635–70.

13. Goerlich KS. The multifaceted nature of alexithymia: A neuroscientific perspective. Front Psychol. 2018;9:1614.

14. Demartini B, Petrochilos P, Ricciardi L, Price G, Edwards MJ, Joyce E. The role of alexithymia in the development of functional motor symptoms (conversion disorder). J Neurol Neurosurg Psychiatry. 2014;85(10):1132–7.

15. van Dijl TL, Aben HP, Synhaeve NE, de Waardt DA, Videler AC, Kop WJ. Alexithymia and facial emotion recognition in patients with functional neurological disorder. Clin Neurol Neurosurg. 2024;237:108128.

16. Cerutti R, Spensieri V, Valastro C, Presaghi F, Canitano R, Guidetti V. A comprehensive approach to understand somatic symptoms and their impact on emotional and psychosocial functioning in children. PLoS One. 2017;12(2):e0171867.

17. Gulpek D, Kelemence Kaplan F, Kesebir S, Bora O. Alexithymia in patients with conversion disorder. Nord J Psychiatry. 2014;68(5):300-5.

18. Xu L, Shi J, Li C. Addressing psychosomatic symptom distress with mindfulness-based cognitive therapy in somatic symptom disorder: mediating effects of self-compassion and alexithymia. Front Psychiatry. 2024;15:1289872.

19. Cerutti R, Spensieri V, Presaghi F, Renzi A, Palumbo N, Simone A, Solano L, Di Trani M. Alexithymic Traits and Somatic Symptoms in Children and Adolescents: a Screening Approach to Explore the Mediation Role of Depression. Psychiatr Q. 2020 Jun;91(2):521-532. doi: 10.1007/s11126-020-09715-8. PMID: 32020499..

20. Raffagnato A, Angelico C, Valentini P, Miscioscia M, Gatta M. Using the body when there are no words for feelings: alexithymia and somatization in self-harming adolescents. Front Psychiatry. 2020;11:262.

21. Mattila AK, Saarni SI, Salminen JK, Huhtala H, Sintonen H, Joukamaa M. Alexithymia and health-related quality of life in a general population. Psychosomatics. 2009;50(1):59-68.

22. Tesio V, Di Tella M, Ghiggia A, Romeo A, Colonna F, Fusaro E, Geminiani GC, Castelli L. Alexithymia and Depression Affect Quality of Life in Patients With Chronic Pain: A Study on 205 Patients With Fibromyalgia. Front Psychol. 2018 Apr 4;9:442. doi: 10.3389/fpsyg.2018.00442. PMID: 29670558; PMCID: PMC5893813.

23. Guy W. Clinical Global Impressions. In: ECDEU Assessment Manual for Psychopharmacology, Revised. Rockville: National Institute of Mental Health; 1976. p. 218-22.

24. Kaufman J, Birmaher B, Brent D, Rao U, Flynn C, Moreci P, Williamson D, Ryan N. Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL): initial reliability and validity data. J Am Acad Child Adolesc Psychiatry. 1997 Jul;36(7):980-8. doi: 10.1097/00004583-199707000-00021. PMID: 9204677.

25. Ünal F, Öktem F, Çetin Çuhadaroğlu F, Çengel Kültür SE, Akdemir D, Foto Özdemir D, Çak HT, Ünal D, Tıraş K, Aslan C, Kalaycı BM, Aydos BS, Kütük F, Taşyürek E, Karaokur R, Karabucak B, Karakök B, Karaer Y, Artık A. Okul Çağı Çocukları için Duygulanım Bozuklukları ve Şizofreni Görüşme Çizelgesi imdi ve Ya am Boyu ekli DSM 5 Kasım 2016 Türkçe Uyarlamasının (ÇD G YDSM 5T) Geçerlik ve Güvenirli i [Reliability and Validity of the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version, DSM-5 November 2016-Turkish Adaptation (K-SADS-PL-DSM-5-T)]. Turk Psikiyatri Derg. 2019 Spring;30(1):42-50. Turkish. PMID: 31170306.

26. Derogatis LR. The Brief Symptom Inventory. Baltimore: Clinical Psychometric Research; 1992.

27. Şahin NH, Durak A. Kısa Semptom Envanteri: Türk gençleri için uyarlanması. Türk Psikoloji Dergisi. 1994;9(31):44-56.

28. Bagby RM, Parker JD, Taylor GJ. The twenty-item Toronto Alexithymia Scale--I. Item selection and cross-validation of the factor structure. J Psychosom Res. 1994;38(1):23-32. doi:10.1016/0022-3999(94)90005-1. PMID: 8126686.

29. Güleç H, Köse S, Yazıcı Güleç M, Citak S, Evren C, Borckardt J, Sayar K. Yirmi soruluk Toronto Aleksimi Ölçeği'nin Türkçe uyarlamasının geçerlik ve güvenirliğinin incelenmesi. Klinik Psikofarmakol Bulteni. 2009;19(3):213-9.

30. Varni JW, Seid M, Kurtin PS. PedsQL 4.0: reliability and validity of the Pediatric Quality of Life Inventory version 4.0 generic core scales in healthy and patient populations. Med Care. 2001;39(8):800-12. doi:10.1097/00005650-200108000-00006. PMID: 11468499.

31. Memik NC, Ağaoğlu B, Coşkun A, Karakaya I, Yıldız S. The validity and reliability of the Turkish Pediatric Quality of Life Inventory for children 13–18 years old. Turk Psikiyatri Derg. 2008;19(4):409-17.

32. Hadji-Michael M, McAllister E, Reilly C, Heyman I, Bennett S. Alexithymia in children with medically unexplained symptoms: a systematic review. J Psychosom Res. 2019 Aug;123:109736.

33. Smakowski A, Hüsing P, Völcker S, Löwe B, Rosmalen JGM, Shedden-Mora M, Toussaint A. Psychological risk factors of somatic symptom disorder: A systematic review and meta-analysis of cross-sectional and longitudinal studies. J Psychosom Res. 2024 Jun;181:111608.

34. Kano M, Fukudo S. The alexithymic brain: the neural pathways linking alexithymia to physical disorders. Biopsychosoc Med. 2013;7(1):1.

35. de Timary P, Roy E, Luminet O, Fillee C, Mikolajczak M. Relationship between alexithymia, alexithymia factors and salivary cortisol in men exposed to a social stress test. Psychoneuroendocrinology. 2008;33(9):1160–4.

36. Pollatos O, Werner NS, Duschek S, Schandry R, Matthias E, Traut-Mattausch E, et al. Differential effects of alexithymia subscales on autonomic reactivity and anxiety during social stress. J Psychosom Res. 2011 Jun;70(6):525–33.

37. Rice DG, Greenfield NS. Psychophysiological correlates of la belle indifference. Arch Gen Psychiatry. 1969;20(2):239-45.

38. Liao SC, Ma HM, Lin YL, Huang WL. Functioning and quality of life in patients with somatic symptom disorder: The association with comorbid depression. Compr Psychiatry. 2019;89:1–6.

39. Huang WL, Chiu YT, Löwe B, Wu CS, Liao SC. Psychopathologies and quality of life in mental and functional disorders associated with persistent somatic symptoms. J Affect Disord. 2025 Oct 15;387:119521.

40. Janssens KM, Rosmalen JM, Ormel J, van Oort FA, Oldehinkel AJ. Anxiety and depression are risk factors rather than consequences of functional somatic symptoms in a general population of adolescents: the TRAILS study. J Child Psychol Psychiatry. 2010;51(3):304–12.

41. Romero-Acosta K, Canals J, Hernández-Martínez C, Penelo E, Zolog TC, Domènech-Llaberia E. Age and gender differences of somatic symptoms in children and adolescents. J Ment Health. 2013 Feb;22(1):33–41.

42. Kline CL, Shamshair S, Kullgren KA, Leber SM, Malas N. A review of the impact of sociodemographic factors on the assessment and management of pediatric somatic symptom and related disorders. J Acad Consult Liaison Psychiatry. 2023 Jan-Feb;64(1):58–64.

43. Karaköse S, Sertel Berk Ö. Kadınlarda somatizasyon: Biyopsikososyal yaklaşım perspektifinden bir değerlendirme. KADEM Kadın Araştırmaları Dergisi. 2023;9(1):93–117.