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Comparative analysis of emotional factors in patients with somatic symptom disorder and panic disorder

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ABSTRACT

Objective: This study investigated the emotional symptom profiles and treatment responses in patients exhibiting overlapping physical symptoms to compare differences between Somatic Symptom Disorder (SSD) and Panic Disorder (PD).

Methods: Pharmacotherapy outcomes were analysed in 208 outpatients with SSD ($n=94$) and PD ($n=114$). Stepwise multivariable logistic regression identified predictors of treatment response, considering variables such as the Clinical Global Impression-Severity (CGI-S), Beck Depression Inventory-II (BDI-II), State-Trait Anxiety Inventory, and State-Trait Anger Expression Inventory. Network analysis explored emotional patterns by estimating network structures for each group.

Results: The overall response rate to pharmacotherapy was 23.6% (49/208), with no significant difference between groups. Baseline CGI-S and BDI-II scores were significant predictors of treatment response in both groups, while social phobia score was a significant predictor in PD. Depression and anxiety were related to physical symptoms in both groups, but anger was significantly associated only in SSD. Network analysis revealed that depression was central to other symptoms in SSD, while anxiety was the core symptom in PD, indicating different emotional drivers between the disorders.

Conclusions: This study suggests the differences in emotional symptom profiles between SSD and PD. Findings suggest different mechanisms, considering the role of anger in SSD, highlighting the need for more personalised treatments for each disorder.

KEY POINTS

- Emotional symptom profiles differ between Somatic Symptom Disorder (SSD) and Panic Disorder (PD).
- Depression is central in SSD, while anxiety is the core symptom in PD, with anger specifically associated with SSD.
- Personalised treatments are needed due to the distinct emotional mechanisms driving each disorder.

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Introduction

Somatic Symptom Disorder (SSD) is a relatively common diagnosis in clinical settings, characterised by persistent physical symptoms and heightened health-related anxiety, which often leads to functional impairments in daily life (American Psychiatric Association 2013). Patients with SSD may experience a variety of physical symptoms such as pain, fatigue, palpitations, dyspepsia, breathlessness, and dizziness (Toussaint et al. 2017). These symptoms are frequently accompanied by psychological conditions, particularly depression and anxiety (Liao et al. 2019). Moreover, SSD often coexists with mood and anxiety disorders, sharing overlapping symptoms, diagnostic criteria, and similar psychobiological mechanisms (Hüsing et al. 2018; Löwe et al. 2022). Panic Disorder (PD), on the other hand, is primarily characterised by recurrent, unexpected panic attacks which are sudden episodes of intense fear or discomfort that peak within minutes (American Psychiatric Association 2013). PD present common physical and psychological symptoms include dyspnoea, rapid heart rate, sweating, paresthesias, fear

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of losing control, and fear of dying (American Psychiatric Association 2013). Despite their distinct diagnostic definitions, SSD and PD frequently exhibit similar autonomic and physical symptoms, such as shortness of breath, palpitations, and dizziness, complicating the differential diagnosis between the two conditions (American Psychiatric Association 2013; Hüsing et al. 2018). While SSD and PD have clear diagnostic distinctions, their frequent symptom overlap, such as physical sensations of panic or discomfort, makes diagnosis more challenging (King et al. 1986). In addition to these physical manifestations, both disorders are commonly associated with comorbid emotional symptoms like anxiety and depression (Liao et al. 2019; Manjunatha and Ram 2022). Moreover, treatment strategies, including cognitive-behavioral therapy (CBT) and pharmacotherapy (e.g., antidepressants), are often similarly employed across both conditions due to these shared features (Ziffra 2021; Sauer et al. 2023). According to the literature, the treatment of SSD involves a multifaceted approach. Psychopharmacotherapy, such as antidepressants, can be helpful during the initial phase to address temporary comorbidities, though it carries risks such as side effects (Roenneberg et al. 2019). However, there is limited evidence on which specific symptoms or comorbidities should be targeted. A recent review also highlighted that while psychological interventions, including CBT, are commonly applied for SSD, their effectiveness is not well-supported due to the highly diverse, non-standardised nature of these interventions and the lack of mechanism-specific evidence (Berezowski et al. 2022). For panic disorder, pharmacotherapy with Selective Serotonin Reuptake Inhibitors (SSRIs) and CBT are well-established treatments supported by stronger evidence (Ziffra 2021). However, studies suggest that different approaches may be required depending on comorbid conditions (Caldirola et al. 2018). While some evidence indicates that treatment responses can vary based on comorbidities such as depression or social phobia, this evidence remains weak, and most treatments are still non-specific (Deckert and Erhardt 2019). Therefore, examining whether initiating pharmacotherapy targeting emotional symptoms or sleep disturbances in newly diagnosed SSD or PD influences symptom progression may offer insights to inform initial treatment targets and improve therapeutic strategies for these disorders.

Of particular interest is the subset of SSD patients whose primary symptoms closely mirror those of PD, raising questions about whether treatment responses and emotional predictors differ between these disorders. Although they share overlapping physical symptoms, the underlying mechanisms driving SSD and PD may vary, potentially influencing patient responses to pharmacological treatments. In SSD, psychobiological mechanisms play a significant role, with psychological factors such as heightened health anxiety, dysfunctional cognitive appraisal, and a self-concept of bodily weakness contributing to symptom manifestation (Okur Güney et al. 2019). Studies suggest that patients with SSD may also exhibit altered autonomic reactivity, fronto-striatal circuit dysfunction, and changes in brain regions such as the right temporal and left inferior parietal gyri, indicating atypical pain processing and heightened sensitivity to bodily sensations (Löwe et al. 2022). These psychobiological mechanisms contribute to a persistent focus on somatic symptoms, which in turn amplifies both perceived severity and distress (Schnabel et al. 2022). In contrast, PD involves more pronounced neurochemical imbalances, such as altered serotonin and Gamma-Aminobutyric Acid (GABA) levels, along with hyperactivity in brain regions like the amygdala and hypoactivation in the prefrontal cortex, which lead to heightened fear responses (Kyriakoulis and Kyrios 2023). Additionally, PD is characterised by increased autonomic reactivity, such as heightened sympathetic nervous system activity, resulting in physical symptoms like elevated heart rate and blood pressure (Kyriakoulis and Kyrios 2023). On a cognitive level, PD patients often experience catastrophic thinking and heightened anxiety sensitivity, which further amplify emotional dysregulation and contribute to persistent panic attacks (Oussi et al. 2023). Emotional disturbances, including depression, anxiety, and anger, frequently accompany both conditions; however, how these emotional factors interact to shape treatment outcomes in SSD and PD remains poorly understood.

This study aims to compare the treatment responses, emotional symptom profiles, and key predictors of treatment success between patients with SSD and PD. Specifically, we will investigate whether patients with similar somatic complaints exhibit differences in their responses to pharmacotherapy, reflective of distinct underlying pathophysiologies. Additionally, we will explore the role of emotional dysregulation – particularly anger – in treatment outcomes, hypothesising that anger plays a more prominent role in SSD compared to PD. In previous studies, somatic symptoms of anxiety, such as tachycardia and sweating, have been associated with sympathetic adrenergic arousal (Kyriakoulis and Kyrios 2023). Similarly, anger has been reported to manifest as somatic symptoms through increased sympathetic nervous system activity (Levenson 2014). Notably, Hwa-byung, an anger-related

syndrome recognised as a Korean culture-bound syndrome in the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV) (American Psychiatric Association 1994), is characterised by a range of somatic symptoms, including the sensation of a mass in the epigastrium, heat sensations, palpitations, dyspnoea, and fatigue, as well as emotional symptoms such as dysphoria. These symptoms are commonly attributed to suppressed anger (Mezzich 2000; Koh 2002). A related study demonstrated that anger suppression influences somatic symptoms indirectly through depression and anxiety, underscoring the pivotal role of anxiety in anxiety disorders and both anxiety and depression in somatoform disorders (Koh et al. 2008). Given the shared presentation of sympathetic arousal symptoms between anxiety and anger-related conditions, further research is needed to explore how these emotional states, either independently or in combination, contribute to underlying mechanisms. Such investigations may provide valuable insights for developing more targeted therapeutic strategies. Through this analysis, we aim to provide more insights into the mechanisms behind these disorders and contribute to the development of more personalised treatment strategies for SSD and PD. We hypothesise that even when patients report similar physical symptoms, the predictors of treatment response will differ between SSD and PD due to their distinct underlying mechanisms. Additionally, we expect that anger will have a stronger association with SSD than with PD, indicating differences in emotional processing between the two disorders. By examining these emotional and symptomatic patterns, this study aims to enhance understanding of SSD and PD and support the creation of more tailored treatment approaches for each disorder.

Materials and methods

Study population

This retrospective cohort study involved 208 patients diagnosed with SSD ($N = 94$) and PD ($N = 114$) based on DSM-5 criteria (American Psychiatric Association 2013), who were initiated on pharmacotherapy. The patients were recruited from Seoul National University Bundang Hospital, Republic of Korea, between August 2017 and September 2022. Among the various symptoms of SSD, the study included only those patients who were finally diagnosed with SSD after differentiating it from PD, particularly focusing on primary symptoms that overlap with PD. To assess this, we reviewed the Patient Health Questionnaire-15 (PHQ-15) (Han et al. 2009) completed by the patients. Specifically, we included cases where at least one of the five items overlapping with panic symptoms (chest pain, dizziness, fainting spells, feeling heart pound or race, shortness of breath) scored 2 points ('bothered a lot'). All patients underwent clinical examinations by psychiatrists, and those diagnosed with other major psychiatric disorders – including major depressive disorder, bipolar disorder, psychotic disorder, neurocognitive disorder, or substance use disorder – were excluded. Finally, we included only patients who initiated pharmacological treatments after their first diagnosis. These treatments included prescriptions for either antidepressants or benzodiazepines, which are recognised as first-line pharmacotherapy for both SSD and PD (Somashekar et al. 2013; Ziffra 2021). To ensure a valid comparison of treatment effects, patients receiving additional interventions, such as biofeedback or CBT, were excluded. For the eligible patients, additional demographic variables and clinical data, including psychological assessments and treatment progress, were collected through an Electronic Medical Records (EMR) chart review.

Assessment of the clinical and psychological symptoms

During the initial visit, patients diagnosed with SSD self-rated the severity of their symptoms using the PHQ-15 (Han et al. 2009), while those diagnosed with PD were evaluated with the Albany Panic and Phobia Questionnaire (APPQ) (Kim et al. 2004). Common psychological assessments included the Beck Depression Inventory-II (BDI-II) (Lim et al. 2011), the Spielberger State–Trait Anxiety Inventory (STAI) (Spielberger et al. 1971), and the State–Trait Anger Expression Inventory (STAXI) (Chon et al. 1998). At each subsequent visit, clinicians filled out the Clinical Global Impression-Severity scale (CGI-S) (Guy 1976). The CGI-S was also followed up after 12 weeks of medication. Two clinical psychiatrists administered the CGI-S, with interrater reliability (Cronbach's alpha) for 25 co-rated cases being 0.91 and 0.87 respectively. To reduce interrater variability, a single psychiatrist reviewed the medical records for all participants.

The PHQ-15 is a cost-effective, self-administered tool frequently employed to screen for somatic symptoms across different care settings and is recommended by the DSM-5 Workgroup on SSD for classifying SSD (Narrow et al. 2013). Patients assessed their somatic symptom severity over the past 4 weeks using a 3-point scale, with total severity scores ranging from 0 to 30, classified as minimal (0–4), mild (5–9), moderate

(10–14), or severe (15–30). The APPQ was developed to assess the fear of activities that may trigger physical sensations in patients with PD (Kim et al. 2004). It consists of 27 items rated on a 9-point scale from 0 (not at all) to 8 (extremely), with the total score reflecting the sum of all items. The APPQ is divided into three subscales: agoraphobia, social phobia, and interoceptive fear. We used the Korean version of the APPQ, which demonstrates high internal consistency and test–retest reliability (Kim et al. 2004). The BDI-II is a 21-item self-reporting scale that measures depressive symptoms. Responders rate each item from 0 (not at all) to 3 (very much); thus, the total score can range from 0 to 63 (Lim et al. 2011). Scores higher than 13 are considered clinically significant. The STAI consists of two 20-item self-reporting scales that independently measure anxiety as a temporary emotional state (STAI-S) or a persistent trait of the responder (STAI-T) (Spielberger et al. 1971). Responses range from 1 (not at all) to 4 (very much), and the results are summed to yield a score between 20 and 80. Scores were converted to T-scores, and a cut-off score of 60T was used to indicate clinically significant symptoms reflecting anxiety as a state or trait. The state and traits of anger expression were evaluated using the Korean version of STAXI, consisting of 10 items to measure state anger, 10 items to measure trait anger, and 24 items to measure anger expression style (Chon et al. 1998). The CGI-S is a seven-point scale that requires the clinician to rate the severity of the patient's illness at the time of assessment, relative to the clinician's experience with patients with the same diagnosis. Ratings range from 1 (normal, not at all ill) to 7 (among the most extremely ill patients) (Guy 1976). The CGI-S was completed at every visit by the same clinician for each patient.

Once diagnosed with SSD or PD, clinicians prescribed medications such as antidepressants and/or benzodiazepines, with the type and dose left to the clinician's discretion. The Institutional Review Board of Seoul National University Bundang Hospital approved the protocol of the present study (B-1610-365-004/B-2410-931-103) and waived the requirement of informed consent because this was a retrospective study using an EMR review.

Statistical analysis

In this post-hoc analysis, a CGI-S-based assessment defined patients as responders if they exhibited a decrease of ≥2 points after 12 weeks of pharmacotherapy, following the cut-off criteria from a previous study (Turkoz et al. 2021; Morrens et al. 2022). Antidepressant and benzodiazepine doses were standardised to escitalopram (Hayasaka et al. 2015) and alprazolam (Howard et al. 2014) equivalents, respectively. Differences in characteristics and outcomes between responders and non-responders were analysed using Student's t-test and the χ^2 test for independence, with the Mann-Whitney U test applied for non-normally distributed continuous variables. Missing data were observed only in continuous variables and were imputed with their respective means. The association between emotional symptoms and symptom severity for each diagnosis was analysed using the Pearson correlation. Multivariable logistic regression using the forward stepwise likelihood ratio method was performed to identify predictors of response to 12-week therapies, with significance set at $p < 0.05$. Subgroup analysis by diagnosis was conducted, with multiple comparisons corrected using the Bonferroni method and a significance threshold of $p < 0.025$. All analyses were performed using R version 3.6.1 (R Foundation for Statistical Computing, Vienna, Austria).

Estimation of networks

To analyse the emotional patterns of SSD and PD patients, network structures were estimated for each group. Six nodes measuring depression (BDI-II), anxiety (STAI-T, STAI-S), anger (STAXI-T, STAXI-S), and somatic symptoms (PHQ-15) were included in the SSD network. Similarly, six nodes measuring depression (BDI-II), anxiety (STAI-T, STAI-S), anger (STAXI-T, STAXI-S), and panic symptoms (APPQ) were used in the PD network. The network estimation used the extended Bayesian information criterion (EBIC) Glasso model (Friedman et al. 2014). The EBIC Glasso model is an advanced network analysis method that ensures reliable estimation of variable relationships, offering robustness and flexibility even in the presence of non-Gaussian data distributions (Foygel and Drton 2010). The network visualisation employed the qgraph package (v.1.6.9). Strength, betweenness, and closeness were calculated as centrality indices. To evaluate the stability of the centrality indices, correlation stability coefficients (CS-coefficients) were determined using the R package bootnet (v.1.5). Consequently, the CS-coefficient for strength was the only one exceeding 0.25 (Epskamp et al. 2018), and it was also the most reliable according to the case-dropping subset bootstrap results for the stability of centrality (Supplementary Figure 1). Therefore, in this study, only the strength was interpreted. Strength is a measure of the total connection intensity of a node, indicating how many connections it has with other nodes. Finally, we calculated the

bootstrapped sampling distribution based on 1,000 bootstrap samples and plotted the bootstrapped 95% confidence intervals (CIs) of the estimated edge-weights to assess the accuracy of edge-weights (Supplementary Figure 2).

Results

Demographic and clinical characteristics of patients

A total of 310 eligible patients were screened, and 208 were included for final analysis after excluding 21 patients who started other treatments, such as CBT, simultaneously with pharmacotherapy, 18 patients who had taken medications prior to the initial visit, 27 patients with major psychiatric disorders, 23 patients with incomplete questionnaires, and 13 patients who dropped out before completing 12 weeks of treatment. The overall response rate to pharmacotherapy was 23.6% (49/208), and there was no significant difference in response rate between SSD group (20.2%) and PD group (26.3%). In the comparison between the two disease groups, SSD patients reported higher mean scores for CGI-S, BDI-II, STAI-S, STAI-T, and STAXI-S, whereas the daily dose of antidepressants was higher in the PD group. To control for the influence of symptom severity, an additional analysis of covariance (ANCOVA) was conducted. The analysis revealed that even after adjusting for baseline CGI-S scores, SSD patients exhibited higher levels of state anxiety and state anger compared to PD patients (Supplementary Table 1).

The baseline demographic and clinical characteristics of the responders and the non-responders are presented in Table 1. Overall, when comparing the baseline characteristics between responders and non-responders, the responder group had a significantly higher baseline CGI-S score compared to the non-responder group. In the disease-specific sub-analysis, the responder group had significantly lower mean BDI-II scores compared to the non-responder group in panic disorder.

The most prescribed antidepressants were escitalopram (31.7%), paroxetine (15.3%), and the most common benzodiazepine was alprazolam (42.3%). No statistical association between the depressive symptoms and the prescribed doses of antidepressants was found. However, the daily dose of antidepressants showed a positive correlation with the presence of sleep disturbance ($r=0.14$, $p<0.05$), while the daily dose of benzodiazepines was associated with the presence of sleep disturbance ($r=0.15$, $p<0.05$), STAI-S ($r=0.17$, $p<0.05$), and STAXI-S ($r=0.15$, $p<0.05$). Additionally, the doses of these two drug categories exhibited a strong positive correlation with each other ($r=0.44$, $p<0.001$).

Predictors of treatment response

A multivariable logistic regression analysis was conducted to assess the relationship between the independent variables and treatment response. As presented in Table 2, the baseline CGI-S score consistently showed an association with treatment response in the full logistic regression model. In the forward stepwise logistic regression analysis, both the baseline CGI-S score (Odds Ratio (OR) [95% Confidence Interval (CI)]=4.55 [2.17–9.54], $p<0.001$) and BDI-II score (OR [95% CI]=0.96 [0.93–0.99], $p=0.015$) were significant predictors of treatment response. In the SSD group, the baseline CGI-S score was significantly associated with treatment response (OR [95% CI]=3.56 [1.33–9.54], $p=0.012$). In the PD group, both the baseline CGI-S score (OR [95% CI]=6.09 [2.01–18.41], $p=0.001$) and APPQ-S score (OR [95% CI]=0.95 [0.92–0.99], $p=0.01$) were identified as significant factors associated with treatment response.

Correlates of symptom severity

A correlation analysis was conducted to explore the emotional symptoms associated with the severity of symptoms in each disorder. In the SSD group, PHQ-15 scores showed a positive correlation with all emotional questionnaire scores ($r=0.558$, $p<0.001$ with BDI-II; $r=0.405$, $p<0.001$ with STAI-S; $r=0.410$, $p<0.001$ with STAI-T; $r=0.401$, $p<0.001$ with STAXI-S and $r=0.295$, $p<0.01$ with STAXI-T). In the PD group, APPQ total scores were positively correlated with emotional questionnaire scores except for STAXI ($r=0.414$, $p<0.001$ with BDI-II; $r=0.377$, $p<0.001$ with STAI-S; $r=0.460$, $p<0.001$ with STAI-T).

Network analysis of the emotional domain patterns of SSD and PD

The emotional domain patterns of SSD and PD patients, as calculated through network analysis, are presented in Figure 1. Each edge represents the statistical dependency between nodes, with blue lines indicating positive relationships and red lines indicating negative relationships. Additionally, thicker edges signify stronger connections. Considering Figure 1a in conjunction with Supplementary Figure 2a, it was observed that SSD patients exhibit strong connections between STAI-S and STAI-T, STAXI-S and STAXI-T, BDI-II and STAI-T, and BDI-II and PHQ-15. In PD patients, the

Table 1. Demographics and clinical characteristics of responders vs. non-responders in each patients groups.

	Total ($n=208$)			Somatic symptom disorder ($n=94$)			Panic disorder ($n=114$)		
Variables	Somatic symptom disorder ($n=94$)	Panic disorder ($n=114$)	p	Responder ($n=19$)	Non-responder ($n=75$)	p	Responder ($n=30$)	Non-responder ($n=84$)	p
Demographic data									
Age (year)	44.38 (14.02)	44.78 (12.78)	.726	41.63 (14.79)	45.08 (13.84)	.341	47.37 (10.99)	43.86 (13.3)	.198
Male sex (%)	30 (31.91)	39 (34.21)	.831	8 (42.11)	22 (29.33)	.286	13 (43.33)	26 (30.95)	.220
Clinical data									
Duration of illness (months)	27.33 (46.28)	25.62 (57.65)	.818	36.95 (60.63)[a]	24.89 (42.04)	.996	27.48 (54.69)	24.93 (59.05)	.837
Sleep disturbance (%)	53 (56.38)	58 (50.88)	.428	9 (47.37)	44 (58.67)	.375	15 (50.00)	43 (51.19)	.911
CGI-S score at baseline	5.12 (0.58)	4.96 (0.55)	.043*	5.42 (0.61)[a]	5.04 (0.56)	.008**	5.20 (0.48)	4.87 (0.55)	.005**
CGI-S score at 12-week	4.3 (0.77)	3.99 (0.83)	.007**	3.32 (0.58)[a]	4.55 (0.60)	<.001***	3.13 (0.57)	4.30 (0.67)	<.001***
BDI-II score	24.01 (12.34)	20.35 (10.94)	.025*	26.16 (16.17)	23.45 (11.21)	.398	16.87 (10.25)	21.60 (10.96)	.042*
STAI-State T score	62.7 (10.42)	58.27 (11.57)	.005**	64.47 (11.56)[a]	62.23 (10.13)	.264	55.63 (11.24)	59.23 (11.61)	.146
STAI-Trait T score	61.66 (11.55)	57.96 (12.25)	.029*	62.95 (13.23)[a]	61.33 (11.15)	.474	54.23 (10.88)	59.33 (12.49)	.051
STAXI-State score	17.01 (6.4)	15.07 (5.51)	.020*	17.68 (7.88)	16.84 (6.01)	.610	14.5 (5.05)	15.28 (5.69)	.511
STAXI-Trait score	19.52 (6.08)	19.69 (6.05)	.838	19.58 (6.64)[a]	19.50 (5.97)	.815	19.27 (5.67)	19.84 (6.20)	.656
PHQ-15 score	15.33 (6.01)	–	–	14.32 (7.37)	15.60 (5.63)	.411	–	–	–
APPQ-A score	–	23.56 (16.56)	–	–	–	–	21.19 (17.41)	24.75 (16.16)	.374
APPQ-S score	–	21.56 (18.80)	–	–	–	–	16.54 (18.30)	24.08 (18.71)	.095
APPQ-I score	–	20.58 (15.87)	–	–	–	–	19.58 (15.44)	21.08 (16.21)	.697
Dose of antidepressant (mg/d)	4.1 (4.04)	5.47 (4.87)	.030*	2.99 (2.78)[a]	4.38 (4.27)	.222	5.5 (5.3)	5.46 (4.74)	.970
Dose of benzodiazepine (mg/d)	0.38 (0.38)	0.42 (0.43)	.448	0.33 (0.35)[a]	0.39 (0.39)	.605	0.43 (0.44)	0.42 (0.43)	.911
Responder (%)	19 (20.21)	30 (26.32)	.302						

[a]Non-normally distributed variables.

Student's t-tests were performed for continuous variables that met the assumption of normality, while Mann-Whitney U tests were conducted for those that did not. Chi-squared tests were used for categorical variables. Continuous variables were presented as means with standard deviations, and categorical variables were described as frequencies with percentages.

Note: CGI-S, Clinical Global Impression-Severity; BDI-II, Beck Depression Inventory-II; STAI, State-Trait Anxiety Inventory; STAXI, State-Trait Anger Expression Inventory; PHQ-15, Patient Health Questionnaire-15; APPQ-A, Albany Panic and Phobia Questionnaire-Agoraphobia; APPQ-S, Albany Panic and Phobia Questionnaire-Social Phobia; APPQ-I, Albany Panic and Phobia Questionnaire-Interoceptive Fear.

Table 2. Stepwise multivariable logistic regression results for response to pharmacotherapy.

	Total ($n = 208$)				Somatic symptom disorder ($n = 94$)				Panic disorder ($n = 114$)			
	Forced entry		Stepwise forward		Forced entry		Stepwise forward		Forced entry		Stepwise forward	
Variables	OR (95% CI)	*p*	OR (95% CI)	*p*	OR (95% CI)	*p*[a]	OR (95% CI)	*p*[a]	OR (95% CI)	*p*[a]	OR (95% CI)	*p*[a]
Sex[b]	0.52 (0.25-1.1)	.088	–	–	0.47 (0.13-1.69)	.246	–	–	0.65 (0.22-1.91)	.432	–	–
Age	1 (0.97-1.03)	.987	–	–	0.98 (0.94-1.02)	.333	–	–	1.02 (0.99-1.06)	.224	–	–
Diagnosis[c]	0.57 (0.27-1.21)	.141	–	–	–	–	–	–	–	–	–	–
Duration of illness	1 (0.99-1.01)	.767	–	–	1 (0.98-1.01)	.852	–	–	1 (0.99-1.01)	.785	–	–
Sleep disturbance[d]	0.71 (0.34-1.48)	.363	–	–	0.56 (0.16-1.96)	.363	–	–	0.83 (0.3-2.3)	.722	–	–
CGI-S at baseline	5.35 (2.41-11.8)	<.001***	4.55 (2.17-9.54)	<.001***	6.78 (1.61-28.52)	.009**	3.56 (1.33-9.54)	.012*	6.58 (1.96-22.0)	.002**	6.09 (2.01-18.42)	.001**
BDI-II	0.98 (0.93-1.02)	.325	0.96 (0.93-0.99)	.015*	1.01 (0.93-1.1)	.774	–	–	0.95 (0.89-1.03)	.200	–	–
STAI-State	1 (0.95-1.05)	.916	–	–	1 (0.92-1.1)	.963	–	–	0.99 (0.92-1.06)	.765	–	–
STAI-Trait	0.98 (0.94-1.03)	.520	–	–	0.99 (0.91-1.08)	.904	–	–	1 (0.93-1.08)	.950	–	–
STAXI-State	1.01 (0.93-1.09)	.852	–	–	1.02 (0.9-1.15)	.742	–	–	1.04 (0.91-1.18)	.597	–	–
STAXI-Trait	1 (0.93-1.08)	.909	–	–	1.01 (0.89-1.15)	.871	–	–	0.98 (0.88-1.09)	.717	–	–
PHQ-15	–	–	–	–	0.9 (0.78-1.02)	.110	–	–	–	–	–	–
APPQ-A	–	–	–	–	–	–	–	–	1.01 (0.95-1.07)	.791	–	–
APPQ-S	–	–	–	–	–	–	–	–	0.95 (0.9-1)	.051	0.95 (0.92-0.99)	.010*
APPQ-I	–	–	–	–			–	–	1.02 (0.96-1.08)	.524	–	–
Dose of antidepressant	0.95 (0.86-1.04)	.277	–	–	0.8 (0.63-1.01)	.062	–	–	1.01 (0.89-1.13)	.933	–	–
Dose of benzodiazepine	0.91 (0.34-2.41)	.853	–	–	1.82 (0.29-11.31)	.518	–	–	0.99 (0.27-3.65)	.985	–	–

[a]Bonferroni-adjusted *p*-values with a threshold of $p < .025$.
[b]Reference = male.
[c]Reference = panic disorder.
[d]Reference = no.
Note: SSD, Somatic symptom disorder; PD, Panic disorder; OR, Odds ratio; CI, Confidence interval; CGI-S, Clinical Global Impression-Severity; BDI-II, Beck Depression Inventory-II; STAI, State-Trait Anxiety Inventory; STAXI, State-Trait Anger Expression Inventory; PHQ-15, Patient Health Questionnaire-15; APPQ-A, Albany Panic and Phobia Questionnaire-Agoraphobia; APPQ-S, Albany Panic and Phobia Questionnaire-Social Phobia; APPQ-I, Albany Panic and Phobia Questionnaire-Interoceptive Fear.

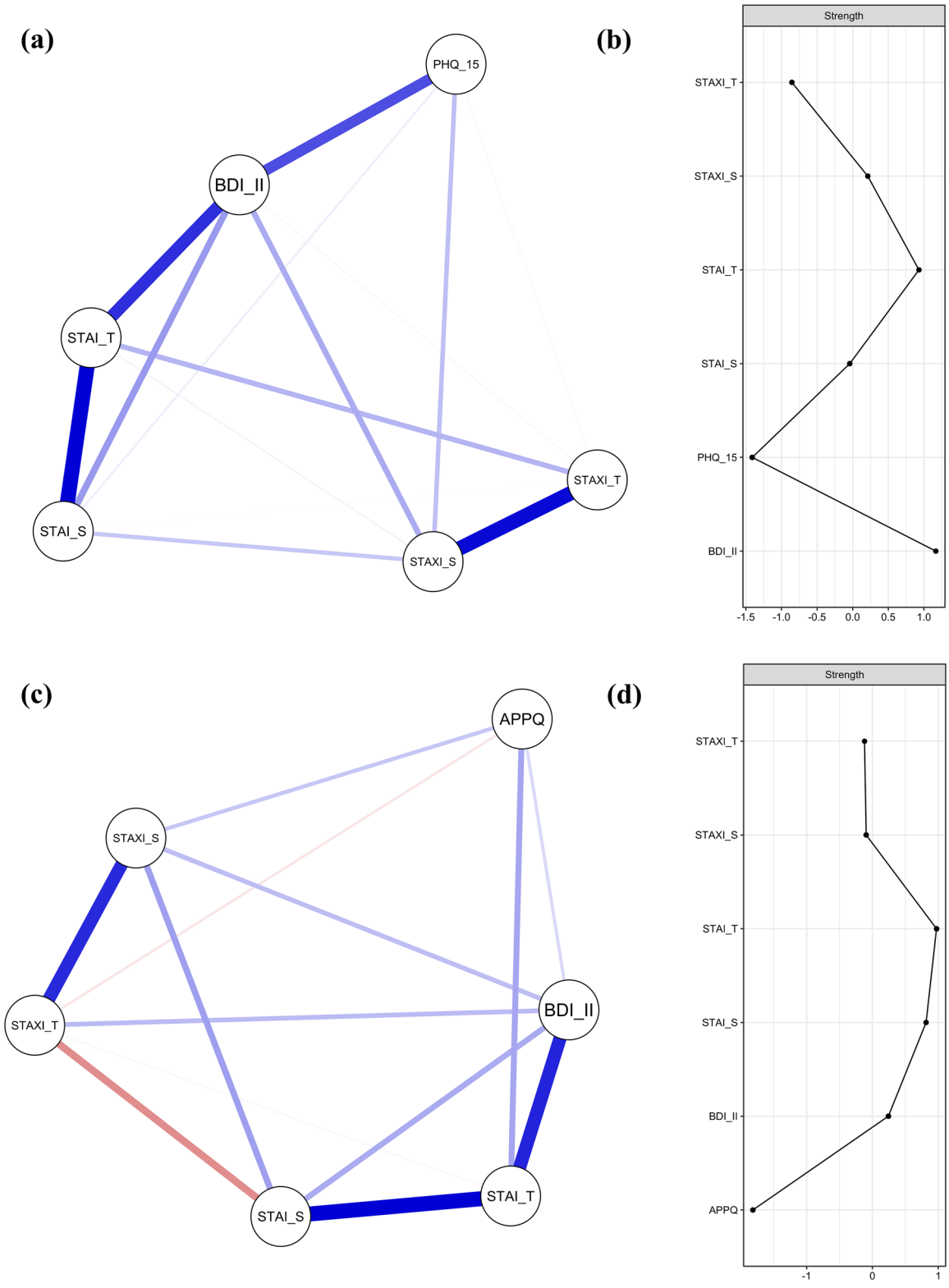

Figure 1. Network structure of emotional domain and node centrality indices of strength.

Note: Network structure of dimension of emotional domain in individuals with (a) somatic symptom disorder, (b) node centrality indices of strength in somatic symptom disorder (c) network structure in panic disorder, (d) node centrality indices of strength in panic disorder

Nodes are represented as circles, and edges are depicted as connecting lines, with thickness indicating the strength of association. Blue lines indicate positive associations between nodes, while red lines indicate negative associations. Centrality indices are shown as standardised z-scores.

BDI-II, Beck Depression Inventory-II; STAI-S, State-Trait Anxiety Inventory-State; STAI-T, State-Trait Anxiety Inventory-Trait; STAXI-S, State-Trait Anxiety Inventory-State; STAXI-T, State-Trait Anger Expression Inventory-Trait; PHQ-15, Patient Health Questionnaire-15; APPQ, Albany Panic and Phobia Questionnaire.

strongest connections were observed between STAI-S and STAI-T, BDI-II and STAI-T, STAXI-S and STAXI-T, and STAXI-T and STAI-S (Figure 1c and Supplementary Figure 2b). Especially, the connection between STAXI-T and STAI-S showed a negative relationship compared to other edges. In SSD patients, the nodes with the highest strength were BDI-II, followed by STAI-T, and then STAXI-S (Figure 1b). In PD patients, the nodes with the highest strength were STAI-T, STAI-S, and BDI-II in that order (Figure 1d).

Discussion

In this study, we examined the emotional symptom profiles and treatment responses of patients with SSD and PD, focusing on both shared and distinct features of these conditions. Both SSD and PD patients presented with overlapping physical symptoms, such as palpitations, dyspnoea, and dizziness. In addition to similar physical symptoms, both groups displayed several overlapping clinical characteristics. More than half of the patients in both groups reported sleep disturbances, and both groups, on average, showed moderate levels of depressive symptoms. Nonetheless, emotional symptoms – particularly depression, anxiety, and anger – were significantly more elevated in the SSD group. These findings suggest that while both SSD and PD patients frequently report similar autonomic arousal-related physical symptoms, the psychological mechanisms driving these symptoms likely differ (Schnabel et al. 2022; Kyriakoulis and Kyrios 2023). For instance, although depression was common in both disorders, anger was significantly associated with SSD, as indicated by higher STAXI scores. In contrast, PD patients did not show a direct association between anger and somatic symptoms. This may suggest that emotional dysregulation, particularly involving anger, plays a certain role in SSD, whereas anxiety and panic-related fears dominate the emotional landscape in PD. Previous research has indicated that affective dysregulation in SSD is associated with impaired emotional processing and heightened physiological responses, such as increased autonomic activity and muscle reactivity (Okur Güney et al. 2019; Park et al. 2024). Anger, in particular, has been linked to somatic symptoms in conditions such as functional gastrointestinal disorders (Alia-Klein et al. 2020) and fibromyalgia (Trucharte et al. 2020). Anger is also known to activate the noradrenergic system, increase catecholamine release, and cause hormonal imbalances, all of which can contribute to the development of somatic symptoms (Yadav et al. 2017). These findings suggest that therapeutic interventions focusing on anger and emotional regulation could be especially beneficial for SSD patients.

The network analysis further highlighted the emotional differences between SSD and PD. In SSD, depression was more closely linked to both somatic and emotional symptoms, while in PD, anxiety traits interacted more strongly with other symptoms. Prior research has indicated that anger suppression contributes to somatic symptoms through its interplay with depression and anxiety (Koh et al. 2008). Our study builds on this by demonstrating distinct central emotional drivers in SSD and PD, with depression being central in SSD and anxiety in PD, emphasising the need for emotion-focused, disorder-specific treatment approaches. Interestingly, the overall severity of emotional symptoms was lower in PD patients, and the network analysis revealed a negative correlation between trait anger and state anxiety. While network analysis provides valuable insights into the relationships between emotional symptoms, it is crucial to note that these findings are correlational and cannot establish causality. Therefore, further research utilising longitudinal or experimental designs is necessary to confirm these emotional associations and determine their causal pathways. Previous studies suggest that PD patients may focus predominantly on bodily sensations related to anxiety, often with reduced emotional awareness, making it harder for them to differentiate between fear and other emotions, which can result in a limited emotional experience (Oussi et al. 2023). When combined with the correlation analysis, these findings suggest that SSD may involve a broader range of emotional dysregulation, whereas PD appears more narrowly focused on anxiety and fear (Šago et al. 2020). While these results support the hypothesis that the emotional and cognitive processes underlying SSD and PD differ, further research is necessary to confirm these distinctions and explore their treatment implications.

One of the key focuses of this study was to identify predictors of pharmacotherapy response in SSD and PD. As hypothesised, baseline symptom severity, particularly as measured by the CGI-S scale, was a strong predictor of treatment response in both disorders. Patients with higher baseline CGI-S scores showed better overall response to treatment, which may indicate that those with more severe initial symptoms have more room for improvement when treated with appropriate medication. Interestingly, no significant association was found between the dose of antidepressants or benzodiazepines and the overall treatment response, although higher doses were correlated with specific symptoms such as sleep disturbance and state anxiety. This suggests that while medication dosage may be effective in managing particular symptoms, it does not necessarily translate to a more comprehensive

treatment outcome. Other factors, such as baseline severity of emotional symptoms, appear to play a more decisive role in predicting treatment response, indicating the complexity of treatment mechanisms in these disorders. We identified baseline emotional distress, particularly BDI-II scores, as a key predictor of treatment response in both disorders, highlighting the importance of addressing specific emotional symptoms to optimise treatment strategies. Our findings indicate that lower baseline BDI-II scores are associated with better treatment responses. This result may suggest that patients with milder depressive symptoms might have more targeted and treatable emotional concerns, such as somatic or anxiety-related symptoms, which respond more readily to therapeutic interventions. In contrast, patients with higher BDI-II scores may present with more pervasive and chronic depressive symptoms that require longer and more intensive treatment to achieve significant improvement (Roughan et al. 2021). In line with our second hypothesis, the factors predicting treatment response differed between SSD and PD. In PD patients, the baseline APPQ-S score, which measures panic-related social phobia, was associated with treatment response, whereas no such relationship was observed in SSD patients. Severe social phobia symptoms and distorted cognitive patterns can hinder therapeutic interactions and adherence, making symptom relief through pharmacotherapy alone challenging (Pelissolo et al. 2019). Therefore, for patients with pronounced social phobia symptoms, combining pharmacotherapy with CBT or other psychosocial interventions may be more effective in improving their symptoms (Mayo-Wilson et al. 2014). The lack of a specific treatment predictor in SSD may reflect the disorder's broader range of emotional dysregulation, suggesting that multiple pathways may contribute to treatment outcomes. This complexity underscores the need for individualised treatment strategies that consider the diverse emotional symptoms present in SSD.

The results of this study provide valuable insights into the differential treatment needs of SSD and PD patients. For clinicians, the findings underscore the importance of considering emotional symptoms beyond anxiety and depression when assessing and treating patients with SSD. In particular, the role of anger in SSD warrants further exploration, as it may represent a key therapeutic target that is currently under-recognised in clinical practice. Moreover, the distinct emotional networks observed in SSD and PD suggest that personalised treatment approaches based on emotional symptomatology could improve treatment outcomes. For PD patients, interventions that specifically address panic-related fears and anxiety may be most effective, whereas SSD patients may benefit from a broader approach that includes managing anger, depression, and somatic symptom-related distress. Future research should continue to explore the role of emotional symptoms in SSD and PD, particularly through longitudinal studies that can assess the stability of these emotional networks over time and their response to different therapeutic interventions. Additionally, further investigation into potential biomarkers or psychological characteristics that can distinguish these disorders will be crucial in refining treatment strategies and improving diagnostic accuracy.

This study has several limitations that should be acknowledged. First, the retrospective design and reliance on EMR data may introduce biases, particularly regarding the accuracy of symptom assessments and medication adherence. Additionally, while network analysis provides valuable insights into the relationships between emotional symptoms, it cannot establish causal relationships. Future studies using prospective designs and more comprehensive assessments of treatment adherence and symptom dynamics would help to address these limitations. Furthermore, while we identified key emotional symptoms associated with treatment response, other factors, such as psychosocial stressors, lifestyle factors, genetic predispositions, and temperamental traits, were not accounted for in this study. Temperamental traits, such as neuroticism, harm avoidance, and self-defeating characteristics, have been associated with symptom formation in SSD and PD in previous researches (Kampman et al. 2017; Macina et al. 2021). Incorporating these variables into future studies, along with their interplay with negative affectivity, could provide a more holistic understanding of the factors influencing treatment outcomes and contribute to developing more tailored therapeutic strategies for these disorders. In addition, the APPQ was assessed only for PD patients and the PHQ-15 only for SSD patients due to the clinical setting. Future studies could consider administering both measures to both groups to allow for more meaningful comparisons. Lastly, as this is a single-institution study, there are limitations in generalising the findings.

This study highlights the distinct emotional symptomatology and treatment response predictors in SSD and PD patients. While these disorders share overlapping physical symptoms, their emotional networks and treatment responses suggest different underlying mechanisms. Anger, in particular, emerged as a key emotional symptom in SSD, offering a potential target for more tailored therapeutic interventions. By further integrating emotional symptom profiles with biological,

psychological, and lifestyle factors, clinicians can develop more nuanced, personalised treatment strategies for patients with SSD and PD, ultimately enhancing patient outcomes and quality of life.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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Data availability statement

The dataset is available from the corresponding author upon reasonable request.

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