Body Mass Index and Somatic Symptom Severity in Patients with Somatic Symptom Disorder: The Mediating Role of Working Memory

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Objective: In somatic symptom disorder (SSD), cognitive dysfunction is related to perceptive distortion that excessively amplifies bodily sensations. The association between high body mass index (BMI) and cognitive dysfunction could be attributed to underlying systemic inflammation. We aimed to evaluate whether patients with SSD and high BMI exhibit increased somatic symptom severity and whether this is mediated by cognitive dysfunction.
Methods: This retrospective, cross-sectional study was conducted on 54 patients with SSD (20 males/34 females, mean age ± standard deviation: 40.65 ± 13.23 years). Participants' BMI, laboratory data including complete blood count and lipid profile, results from the Korean Wechsler Adult Intelligence Scale-IV, and scores of the Korean-Symptom Checklist 95-Somatization (SCL95-SOM) were analyzed. We performed a path analysis to evaluate BMI as a predictor of somatic symptoms.
Results: In a path analysis, the SCL95-SOM score was directly influenced by working memory (b = −0.326, p = 0.032), which was significantly influenced by BMI (b = −0.338, p = 0.009), although there was no direct effect of BMI on the SCL95-SOM score. The path analytic model showed a close fit to the data with the following values: χ^2 (df) = 0.918 (1), p = 0.338, root mean square error of approximation = 0.000 (< 0.001), and comparative fit index = 1.00.
Conclusion: Patients with SSD and high BMI may exhibit increased somatic symptom severity, and this is mediated by working memory dysfunction. Weight management may help improve symptoms in patients with SSD and high BMI.

KEY WORDS: Somatoform disorder; Body mass index; Working memory; Cognitive dysfunction.

INTRODUCTION

Patients with somatic symptom disorder (SSD) exhibit multiple somatic symptoms that are distressing, but not necessarily related to identified medical conditions [1]. They have excessive thoughts, feelings, and behaviors related to somatic symptoms and visit multiple doctors for similar symptoms, resulting in considerable healthcare burdens [1-3]. Although the etiology of SSD remains unclear [4], one explanation is a faulty cognitive schema, leading to patients focusing their attention on bodily sensations, misinterpreting them, and getting alarmed by them [5]. Thus, patients amplify their somatic sensations and have low tolerance for physical discomfort [5]. Deficits in several cognitive domains, such as working memory and attention, have been reported in SSD [6-13].

Obesity increases the risk of chronic medical illnesses including hypertension, diabetes mellitus, dyslipidemia, cardiovascular diseases, autoimmune diseases, and cancers [14-19]. Moreover, obesity is related to the major depressive and bipolar disorders [20-24], possibly through chronic low-grade systemic inflammation that affects brain function and causes emotional and cognitive changes [25-27].

Although several studies have focused on the association between increased body mass index (BMI) and mood

disorders, few have investigated the association between BMI and SSD. Considering the underlying mechanism that obesity causes mood disorders by dysfunctions in the top-down regulation of emotion through connections with several brain structures [28], it is also compelling to assume that dysregulation of top-down process over cognitive function would lead to another neuropsychiatric disorder such as SSD [7]. Working memory difficulties are often reported in patients with SSD [6,10]. Therefore, we aimed to investigate the association between BMI and somatic symptoms and possible mediators. We hypothesized that patients with SSD and high BMI exhibit increased somatic symptom severity, mediated by cognitive dysfunction, specifically working memory dysfunction.

METHODS

Study Participants and Data Collection

We retrospectively extracted data of participants who visited the Department of Psychiatry, Chung-Ang University Hospital in Seoul, Korea, between June 1, 2013 and April 30, 2019. We included (1) patients aged between 20 and 65 years and (2) those diagnosed with SSD based on the Structured Clinical Interview for DSM-5-Clinician Version [29]. We excluded the following patients: (1) those with past or current episodes of any other psychiatric diseases, including neurocognitive disorder and intellectual disability; (2) those with a past or current diagnosis and/or pharmacological treatment of chronic or severe medical illnesses including malignancy, hematologic disease, cardiovascular disease, stroke, and rheumatic disease; (3) those with a history of psychotropic medication use during the last month; and (4) those with a history of head trauma. The first data search included 64 patients with SSD. Ten patients were excluded because of incomplete data. Finally, 54 patients with SSD were included in the statistical analyses.

Measures and Procedure

Biomedical factors

Patients' weight and height were measured by nurses, and BMI was computed. Glucose, low-density lipoprotein cholesterol, triglyceride (TG), and total cholesterol levels and white blood cell and absolute neutrophil counts (ANCs) were measured using standardized procedures at the Depart-

ment of Diagnostic Tests, Chung-Ang University Hospital.

Neurocognitive factors

We used the Korean Wechsler Adult Intelligence Scale-IV, which is designed to assess one's intellectual abilities within four domains [30,31]: (1) Verbal comprehension is the capability to listen to information provided orally and then remember, understand, and use the information; (2) perceptual reasoning is the mental capacity to think and reason using visual information; (3) working memory is short-term memory with limited capacity to hold information temporarily and is important for learning, reasoning, and guidance for decision-making and action; and (4) processing speed is the speed at which cognitive processes and responses occur.

Somatic symptom severity

The Korean-Symptom Checklist 95-Somatization (SCL95-SOM) was used to evaluate the severity of distress arising from somatic symptoms [32]. The 12 items of the SCL95-SOM reflect the distress caused by perceptions of bodily dysfunction, including strong autonomic mediation and muscle pain. The items are scored using a 4-point Likert scale (1 ["not at all"] to 4 ["almost always"]); we used the T-scores of the sum of the 12 item scores.

Statistical Analyses

Descriptive statistics for each characteristic were calculated and tested for normality using the Shapiro−Wilk test. Because BMI, TG, ANC, and SCL95-SOM score distributions were positively skewed, these variables were log-transformed. Preliminary analyses were performed to characterize the nature of the cross-sectional association among the factors of interest. We performed bivariate (Pearson) correlations among BMI, TG, ANC, total intelligence quotient (IQ), verbal comprehension, perceptual reasoning, working memory, processing speed, and SCL95-SOM score. To evaluate BMI as a predictor of somatic symptoms, we performed a path analysis using IBM SPSS Amos version 24.0 (IBM Co., Armonk, NY, USA). Model fit was assessed by examining the chi-squared (χ^2) statistic ($p > 0.05$ was considered acceptable), root mean square error of approximation (RMSEA; < 0.08 was acceptable), and comparative fit index (CFI; values close to 1 indicated a good fit).

Ethics Statement

The research protocol was approved by the Chung-Ang University Hospital Institutional Review Board (no. 1908-003-16275). Informed consent from the participants was waived because the study was conducted retrospectively.

RESULTS

Characteristics of Study Participants

Demographic factors, somatic symptom severity, neurocognitive factors, biomedical factors, past medical history, and behavioral factors of the participants are described in Table 1.

Preliminary Analyses

Bivariate correlations performed to examine the cross-sectional associations among the factors of interest revealed that BMI correlated with TG (r = 0.387, p = 0.004), ANC (r = 0.393, p = 0.003), total IQ (r = −0.303, p = 0.026), verbal comprehension (r = −0.350, p = 0.009), and working memory (r = −0.302, p = 0.026). Additionally, the SCL95-SOM score correlated with working memory (r = −0.306, p = 0.025). For other factors, there were no significant correlations.
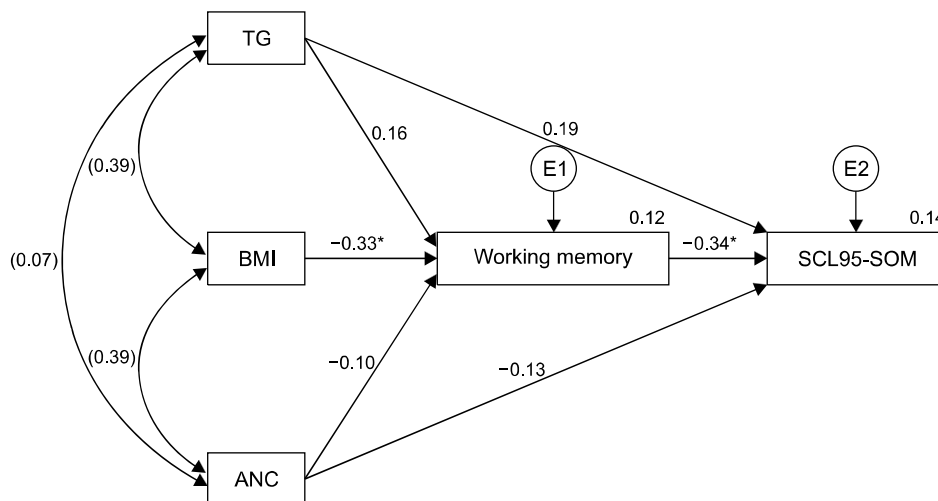
Path Analyses

We constructed a path analytic model to evaluate BMI as a predictor of somatic symptom severity and vice versa. The model showed a close fit to the data with χ^2 (df) = 0.918 (1), p = 0.338, RMSEA = 0.000 (< 0.001), and CFI = 1.00 (Fig. 1). The SCL95-SOM score was directly influ-

Table 1. Characteristics of study participants (n = 54)

Variable	Value
Demographic factors	
Age (yr)	40.65 ± 13.23
Sex, female	63.0
Education (yr)	13.57 ± 3.08
Marriage, married	44.4
Duration of SSD (mo)	40.25 ± 61.51
Somatic symptom severity	
SCL95-SOM score	68.59 ± 16.78
Neurocognitive factors	
Total IQ	94.80 ± 14.64
Verbal comprehension	97.67 ± 15.53
Perceptual reasoning	95.89 ± 16.21
Working memory	100.26 ± 11.81
Processing speed	90.93 ± 13.70
Biomedical factors	
Body mass index (kg/m^2)	22.25 ± 3.72
Random glucose (mg/dl)	110.52 ± 37.03
LDL cholesterol (mg/dl)	104.33 ± 70.04
Triglycerides (mg/dl)	99.71 ± 22.78
Total cholesterol (mg/dl)	171.68 ± 33.53
White blood cell count (×10^6/L)	6,473.47 ± 1,694.10
Absolute neutrophil count (×10^6/L)	3,645.92 ± 1,360.02
Past medical history	
Diabetes	11.1
Hypertension	14.8
Dyslipidemia	13.0
Thyroid disorder	1.9
Behavioral factors	
Smoking, current smoker	13.0
Alcohol use	31.5

Values are presented as mean ± standard deviation or %.
SSD, somatic symptom disorder; SCL95-SOM, symptom checklist 95-somatization; IQ, intelligence quotient; LDL, low-density lipoprotein.

Fig. 1. Results of path analysis to test the integrated model.
BMI, body mass index; TG, triglyceride; ANC, absolute neutrophil count; SCL95-SOM, symptom checklist 95-somatization; RMSEA, root mean square error of approximation; CFI, comparative fit index.
Statistical significance, *p < 0.05.
χ^2 (df) = 0.918 (1), p = 0.338, RMSEA = 0.000 (< 0.001), CFI = 1.00.

enced by working memory (b = −0.326, *p* = 0.032), which was significantly influenced by BMI (b = −0.338, *p* = 0.009). We found no direct effect of BMI on the SCL95-SOM score.

DISCUSSION

The positive correlation between BMI and TG and between BMI and ANC observed in this study is consistent with previous findings that obesity increases the risk of metabolic dysfunction [15-18] and systemic inflammation [25-27]. Previous studies reported a significant association between inflammation and psychiatric symptoms [33-35]. Increased pain sensitivity was shown to be associated with increased concentrations of tumor necrosis factor-α (TNF-α) [36]. Obesity-related inflammation originating from the adipose tissue spreads to the brain and causes changes in neural circuits, neurotransmitter activity, neuroendocrine system, and neurogenesis [37,38]. Increased BMI is associated with detrimental changes in the white matter microstructure that supports working memory [39,40].

However, we found no direct effect of BMI on somatic symptom severity. Instead, the positive association between BMI and somatic symptom severity was mediated by working memory, indicating that rather than directly affecting the autonomic nervous system, obesity causes systemic inflammation, affects brain regions associated with working memory, causes cognitive distortion [25-27], and worsens somatic symptoms. SSD patients showed reduced attention and increased functional connectivity between the sensorimotor and dorsal attention network [41]. Similarly, fibromyalgia patients showed reduced working memory, correlating with the frontal lobe and anterior cingulate gyrus volumes [42]. Working memory is the capability to keep information accessible over time and is closely related to attention as attention helps this process by preserving the information selectively [43]. Reduced working memory and attention suggests misunderstanding of external stimuli and failure to regulate functions aimed at interaction with external stimuli [41], which might lead to the development and worsening of somatic symptoms. Several studies reported co-occurrence of somatic symptoms and cognitive deficits [6-12], but the causality between them remained to be determined. However, the results of this study suggest

that working memory may precede exacerbation of somatic symptoms, considering that the path analytic model implies a possible causal association. Previous findings that nociception could be modulated by cognitive factors support this association [44,45].

Our study had some limitations. First, the sample size (n = 54) was relatively small. However, in path analysis, a sample size of 10 times the number of parameters is considered adequate [46]. In our study, the number of parameters was 5, indicating a minimal sample size of 50, which means that our sample size was appropriate. Second, we measured only ANC, not inflammation markers such as ESR, CRP, TNF-α, and interleukins because they are not included in routine laboratory tests in clinical settings. Future prospective studies should include measurement of various inflammatory markers representing chronic inflammatory states. Third, potential covariates such as medications, hormonal status, and physical activity were not completely controlled due to the retrospective study design, although we excluded patients having chronic or severe medical illnesses.

In conclusion, patients with SSD and high BMI may exhibit increased somatic symptom severity, mediated by working memory dysfunction. Weight management may help improve symptoms in these patients. Future prospective studies, with larger sample sizes, are required.

■ Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

■ Author Contributions

Conceptualization: Sun Mi Kim, Hye Ri Kim. Data acquisition: Hee Jin Kim, Hye Ri Kim, Jae-Chan Jin. Formal analysis: Sun Mi Kim, Jae-Chan Jin. Supervision: Doug Hyun Han. Writing−original draft: Hee Jin Kim, Sun Mi Kim. Writing−review and editing: Doug Hyun Han. Approval of final manuscript: all authors.

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