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Original Article

Somatoform disorders in post-stroke patients from Southern China

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Abstract: *Objective:* This study aimed to establish the frequency and characteristics of somatoform disorders in patients with acute stroke in the Neurology Department in Nanfang Hospital, Southern China. *Methods:* This cross-sectional study was initiated from October 2014 to February 2015 in the Neurology Department in Nanfang Hospital, Southern China. A total of 70 patients with acute stroke were evaluated to study the frequency and characteristics of somatoform disorders. The participants were interviewed by experienced neurologists and psychiatrists. Each patient was also assessed using Symptom Checklist 90 (SCL-90) somatic domain, Hamilton Anxiety Scale (HAMA), and Hamilton Depression Scale (HAMD). *Results:* Of the 70 patients recruited in the study, 33 patients (47%) were with somatoform disorders (assessed by SLC-90 somatoform domain) and 37 patients without somatoform disorders. Both HAMA and HAMD scores were significantly higher in patients with somatoform disorders. The morning serum cortisol level was significantly lower in the patients with somatoform disorders compared with the patients without somatoform disorders. *Conclusion:* Somatoform disorders are quite common in patients with acute stroke. Hence neurologists should pay attention to this problem when treating such patients.

Keywords: Cortisol, neurology, somatoform disorder, stroke

Introduction

Somatization is the expression of personal and social distress in an idiom of bodily complaints with medical help-seeking. It is a condition in which patients have psychological distress and complain of physical symptoms that cannot be explained by a medical or organic cause [1]. Over the past decades, the diagnostic criteria and classification of somatization have been extensively debated in the literature [1]. Diagnostic and Statistical Manual of Mental Disorders (DSM)-5 brings some substantial changes of this category and also suggests relabeling somatoform disorders as "somatic symptom disorder" (SSD) [2]. The new name is less prone to misunderstandings and might be better accepted in the medical community. The most significant change in the classification criteria is the complete abolishment of the distinction between medically explained and medically unexplained somatic complaints [2].

Stroke is a common disease reported in the neurology department, which causes significant disability and distress. Stroke produces a wide range of mental/emotional disorders, including post-stroke depression (PSD), mania, bipolar disorder, anxiety, and apathy without depression, psychotic disorder, pathological affect, and catastrophic reaction [3]. The comorbidity of stroke and PSD has been widely studied, while little is known about somatic symptoms with stroke. Somatic complaints of patients with stroke are commonly encountered in health care. When the patients have lots of physical complaints that cannot be explained medically, neurologists may think about the diagnosis of PSD or anxiety and consult the psychologists. Sometimes, the patients cannot be diagnosed with depression or anxiety, and somatoform disorders should be considered. Considerable research has been undertaken to study somatic symptom disorders in patients with gastric disease, urinary tract symptoms, or cancer, but rarely in patients with stroke. The underlying mechanisms of somatoform disorders are not clear. Cortisol levels were found to be changed, and the current reports showed controversial results with either elevated or reduced cortisol levels.

Table 1. Sociodemographic characteristics of patient groups

Variables	Somatoform disorders, *N* = 33	Nonsomatoform disorders, *N* = 37	*P* value
Gender			0.582
Male (*n*, %)	24 (72.7%)	29 (78.4%)	
Female (*n*, %)	9 (27.3%)	8 (21.6%)	
Age (year) (mean, SD)	58.5 (10.5)	57.4 (12.9)	0.671
Married (*n*, %)	32 (97.0%)	36 (97.3%)	0.935
Educational status (*n*, %)			0.429
Lower education	15 (45.5%)	12 (32.4%)	
Middle education	12 (36.4%)	19 (51.4%)	
Higher education	6 (18.2%)	6 (16.2%)	
Profession			0.687
Blue-collar worker	9 (27.3%)	8 (21.6%)	
White-collar worker	7 (21.2%)	11 (29.7%)	
Retired	17 (51.5%)	18 (48.6%)	
Financial situation[a]			0.168
Low	11 (33.3%)	7 (18.9%)	
Middle to high	22 (66.7%)	30 (81.1%)	

[a]Based on self-evaluation by the patients. SD, Standard deviation.

This study aimed to establish the frequency and characteristics of somatoform disorders in patients with acute stroke in the neurology department in Southern China.

Materials and methods

Design and participants

A cross-sectional study was initiated from October 2014 to February 2015 in the Neurology Department in Nanfang Hospital, Southern China. The sample consisted of 70 patients with acute stroke. The study was approved by the Hospital Ethics Committee. Informed consent was obtained from each participant. The confidentiality of the information obtained was assured.

The inclusion criteria were as follows: (1) aged more than 18 years and (2) diagnosed with a newly onset stroke, either ischemic or hemorrhagic. The patients were evaluated between the 7th and 14th days since the onset of the stroke.

The exclusion criteria were as follows: (1) severe aphasia or intellectual disability; (2) hearing difficulties; and (3) a history of psychiatric or other severe diseases (heart failure, respiratory failure, chronic kidney disease). Patients using illicit drugs or alcohol were also excluded.

Data collection

Each participant was assessed by an independent assessor (a well-trained neurologist or a psychiatrist). The general information, including name, age, sex, educational background, family financial situation, and medical history, was collected. Each patient was also assessed using Symptom Checklist 90 (SCL-90) somatic domain, Hamilton Anxiety Scale (HAMA), and Hamilton Depression Scale (HAMD). The somatization domain of the SCL-90 contained 12 items ranking from 1 to 5. The average value more than 2 was thought to be meaningful. In this study, the participant with a total score of the domain ≥24 was considered to have somatization.

Medical workups, including laboratory, cranial MRI, or other vascular evaluations such as transcranial Doppler (TCD) or ultrasound of cervical arteries, were performed. The hypothalamus-pituitary-adrenal (HPA) axis was also evaluated. According to the medical situations for different patients, autoimmune antibodies were ordered by their doctors. On average, a thorough data collection took about 2 h for each participant.

Statistical analyses

The data analyses were conducted using the SPSS 20.0 software (SPSS, IL, USA). The normally distributed continuous variables were summarized by mean and standard deviation (SD), non-normally distributed variables by median, and categorical variables by absolute frequencies and percentages. Chi-square tests were used to compare categorical variables, and Student t tests were used to compare continuous variables between the studied groups. The significance level for all bivariate and multiple analyses was set at $P < 0.05$.

Results

Sociodemographic characteristics of patient groups

The sociodemographic characteristics of the research patients are presented in **Table 1**. The

Table 2. Clinical profiles and laboratory tests of the research groups

Variables	Somatoform disorders		Nonsomatoform disorders		*P* value
	Mean ± SD	*n* (%)	Mean ± SD	*n* (%)	
Hypertension		26 (78.8%)		25 (67.6%)	0.292
Diabetes		12 (36.4%)		8 (21.6%)	0.173
NIHSS	3.52±2.44		3.05±2.24		0.412
HAMA	10.03±4.52		6.46±3.90		0.001
HAMD	13.64±7.06		9.16±5.61		0.004
Cholesterol (mmol/L)	4.34±1.07		4.67±1.20		0.224
LDL-C (mmol/L)	2.75±0.85		3.08±0.92		0.128
Uric acid (mmol/L)	351.84±138.11		366.22±114.17		0.635
FBG (mmol/L)	6.32±2.55		5.64±1.77		0.197
HAb1c (%)	7.16±2.36		6.82±1.98		0.516
HCY (mg/L)	13.37±3.76		11.89±2.63		0.058
CRP (mg/L)	10.60±15.14		13.63±34.19		0.641
TSH (mU/L)	1.35±1.09		1.11±0.56		0.232
FT3 (pmol/L)	3.00±0.50		2.84±0.53		0.201
FT4 (pmol/L)	1.25±0.20		1.22±0.17		0.550
ACTH (pmol/L)	25.43±13.44		36.63±19.16		0.007
CORT[a] (nmol/L)	14.64±4.71		17.60±5.66		0.021

Abbreviations: NIHSS, National Institutes of Health Stroke Scale; LDL-C, low-density lipoprotein cholesterol; FBG, fasting blood glucose; HAb1c, glycosylated hemoglobin; HCY, homocysteine; CRP, C-reactive protein; TSH, thyroid stimulating hormone; FT3, free triiodothyronine; FT4, free thyroxine; ACTH, adrenocorticotropic hormone; CORT, cortisol. [a]Cortisol at 8 a.m.; SD, standard deviation.

research recruited a total of 70 patients with stroke, including 33 patients (47%) with somatoform disorders (assessed by SLC-90 somatoform domain) and 37 patients without somatoform disorders. Most of the patients in both groups were males. The two groups did not differ significantly in terms of age, marital status, financial situation, profession, and educational status.

Clinical profiles and laboratory tests of patient groups

Table 2 shows the general clinical profiles of the patient groups, including the National Institutes of Health Stroke Scale (NIHSS) values, HAMA scores, HAMD scores, medical history of diseases, and laboratory test results. No group differences were found for NIHSS scores, hypertension, diabetes, thyroid hormones, and C-reactive protein (CRP) values. The patients with somatoform disorders had significantly higher HAMA and HAMD scores (P = 0.001 and P = 0.004, respectively). Significant differences were also found for adrenocorticotropic hormone (ACTH) and cortisol levels at 8 a.m. (P = 0.007 and P = 0.021, respectively). The patients with somatoform disorders showed dramatically lower ACTH and cortisol levels than the patients without somatoform disorders.

Discussion

Somatoform disorders and medically unexplained symptoms (MUS) are common in later life. A systemic review revealed that the prevalence rates for somatoform disorders in the general population were 11%-21% in younger, 10%-20% in the middle (50-60 years), and 1.5%-13% in the older age groups (≥ 65 years). The prevalence rates for MUS showed wider ranges, 1.6%-70%, 2.4%-87%, and 4.6%-18%, in the younger, middle, and older age groups, respectively [4].

During the last decades, some authors suggested that somatization could be better understood as a somatic equivalent of affective disorders. More studies revealed that somatization was strongly associated with anxiety disorders and subjective feelings of distress [5, 6]. Depression, anxiety, and feelings of distress seemed to be linked to alterations of the HPA axis. Patients with depression tended to have elevated cortisol scores and be nonsuppressors after taking dexamethasone [7].

Based on the research results, Rief and his colleagues postulated a hypothesis that somatization might be associated with elevated cortisol scores. They recruited 77 participants classified into 3 groups: somatization syndrome (at least 8 physical symptoms from the DSM-IV somatization disorder list), somatization syndrome combined with major depression, and healthy controls. The following data were collected including salivary cortisol at three time points (morning, afternoon, and evening): nighttime urinary cortisol, serum cortisol after the dexamethasone suppression test (DST), and psychological variables such as depression, anxiety, somatization, and hypochondriasis. However, the groups did not differ in terms of any of the cortisol variables [5].

Nowadays, many somatoform conditions seem to be associated with either average or reduced concentrations of cortisol and a facilitated suppression of the cortisol response after the intake of low-dose dexamethasone [8]. Clinical studies revealed that patients with a chronic widespread pain display altered HPA axis function. A large community-based study demonstrated that patients with widespread pain were associated with lower levels of salivary cortisol and higher levels of post-stressor serum cortisol [9]. The present data revealed that ACTH and morning cortisol levels decreased significantly in patients with somatization, which is in line with current reports [10]. In patients with somatoform disorders, both HAMA and HAMD scores were significantly higher, which meant that the higher the anxiety and depression scores, the lower the average morning cortisol level. The result was different from the published finding that patients with depression tend to have elevated cortisol levels [7]. The relationship between somatization, anxiety, depression, and cortisol levels is controversial and needs more investigation.

The underlying mechanisms of the abnormal HPA axis in somatoform disorders are not well elucidated. A possible explanation for the decreased cortisol level in somatization was HPA axis exhaustion. Pukhalsky and his colleagues, based on the integration of immunological and endocrinological approaches, suggested that permanent psychogenic stress coupled with high antigen load leads to gradual depletion of HPA axis, which is manifested by the decrease in the cortisol level [11].

The dysregulation of immune-to-brain communication is considered to play a role in the biopsychological process of somatization [12]. Dantzer defined somatization as the outward manifestation of sensitization of the brain cytokine system that mediates the subjective, behavioral, and physiological components of sickness. The dysregulation of the cytokine balance is considered as a potential biopsychological mechanism to explain the underlying concept of somatization [13]. Houtveen et al. compared patients with heterogeneous MUS with healthy controls and collected the blood samples from the patients for leukocyte subset cell count, *in vitro* T-cell mitogen-stimulated cytokine production (interleukin [IL]-2, IL-4, IL-5, IL-6, IL-10, tumor necrosis factor [TNF]-α, and interferon [IFN]-c), and *in vitro* monocyte cytokine release (IL-1b, IL-6, IL-8, IL-10, and TNF-a) in response to increasing concentrations of lipopolysaccharide. No significant group differences were found for any of the cytokines measured [12]. The proinflammatory process in somatic symptoms with depression was studied by investigators. Soluble P-selectin was found elevated in somatic symptom group, indicating that early microvascular changes occur subtly; this provides neurobiological evidence for somatic symptoms in depression [14]. Tak *et al.* tried to find a biological marker for somatization by studying a large population. They found that the level of high-sensitivity CRP was not significantly associated with somatization. This study found that CRP was not a biological marker for somatization.

The findings of this study should be interpreted in light of some limitations. First, patients with stroke having low NIHSS scores were recruited; patients with severe aphasia or altered mental status were excluded because the study needed patient cooperation. Second, to evaluate the HPA axis, this study only tested the morning cortisol levels of the participants and did not conduct DST.

Somatoform disorders are quite common in patients with stroke, accounting for 47% of the patients with acute stroke having these disorders. Hence neurologists should pay attention to this problem when treating such patients. Also, the underlying mechanisms of HPA axis abnormality in somatoform disorders are not clear and need to be elucidated further.

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Disclosure of conflict of interest

None.

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