Neuroscience Letters 543 (2013) 42–46

Contents lists available at SciVerse ScienceDirect

Neuroscience Letters

journal homepage: www.elsevier.com/locate/neulet

Severity of generalized social anxiety disorder correlates with low executive functioning

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HIGHLIGHTS

- We evaluate neurocognitive functions of patients with social anxiety disorder.
- The WCST performance was lower than that of healthy controls.
- Performance of the WCST correlates with the LSAS score.
- Social anxiety disorder has low executive function correlates with the severity.

ARTICLE INFO

Article history:
Received 20 November 2012
Received in revised form 14 February 2013
Accepted 25 February 2013

Keywords:
Liebowitz social anxiety scale
Major depressive disorder
Neurocognitive function
Neuropsychological assessment
Social phobia

ABSTRACT

To evaluate neurocognitive functions of patients with social anxiety disorder (SAD) without comorbidity using neuropsychological assessments and to investigate the relation between neurocognitive functions and clinical severity of SAD, this study assessed 30 SAD patients (10 female, 20 male) without comorbidity and 30 healthy subjects matched on gender, education level, and age. The neuropsychological assessment consisted of the Wisconsin card sorting test (WCST), the continuous performance test, the trail-making test, the word fluency test, and the auditory verbal learning test. On the WCST, patients showed lower performance than healthy controls did. The Liebowitz Social Anxiety Scale score correlated significantly with the numbers of perseverative errors of the WCST, although the State anxiety score of State-Trait Anxiety Inventory and the Beck Depression Inventory – Second Edition score showed no correlation with neuropsychological test scores. Results show that the executive functioning of patients with SAD was low and that the low functioning correlates with the SAD symptom severity.

1. Introduction

Social anxiety disorder (SAD) is characterized by a persistent fear of one or more social or performance situations. The prevalence of this disorder is high: approximately 1.4–12.1% of the population meets DSM-IV TR criteria [4] for SAD over their lifetime [1,19,20,27,30]. A person with SAD, although recognizing that the fear is unreasonable or excessive, usually cannot resolve it and deflect attention away from the fear.

According to the cognitive model of SAD, self-focused attention is an important factor in maintaining the illness. A person with SAD tends to increase access to self-referent negative thoughts and feelings that interfere with performance, thereby preventing the individual from observing external information that might disprove the thoughts and feelings of patients with SAD [11]. Presumably, this inflexibility of informational processes is based on neurocognitive dysfunction. The dysfunction of executive functioning, attention and memory is frequently reported in relation to anxiety disorders [10]. Executive functioning is defined as a set of general-purpose control mechanisms, often linked to the prefrontal cortex of the brain, that regulate the dynamics of human cognition and action [28]. Unlike psychotic or mood disorders, little is known about the neurocognitive impairment of anxiety disorders except for obsessive–compulsive disorder and posttraumatic stress disorder. The impairment of executive functioning, visual memory,

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http://dx.doi.org/10.1016/j.neulet.2013.02.059

attention and processing speed has been reported as associated with obsessive–compulsive disorder. The dysfunction of executive functioning, attention, verbal and visual memory has been reported as associated with posttraumatic stress disorder. However, few reports describe studies of SAD patients' neurocognitive functions [10,13].

Several studies have examined threat biases by which individuals with SAD devote selective attention to socially threatening situations [26,32,41,42]. Results do not necessarily support threat biases. Nevertheless, few studies have targeted cognitive functions independently of social context. Among such studies that have assessed potential cognitive impairment in SAD, one found that patients with SAD were more impaired than healthy controls in terms of verbal memory [3]. Another study revealed that patients with SAD had lower executive functioning and visual memory scores than healthy controls had [12]. Based on these studies, patients with SAD might show low performance in tasks of verbal memory, visual memory, and executive functioning. However, results of these studies were not congruent, perhaps because of comorbidities, especially major depressive disorder.

Generally speaking, SAD is frequently comorbid with major depressive disorder, with estimated frequency of 44–70% [9,23,33]. In connection with major depressive disorder, many reports have described that memory, learning, attention, motor function, and problem-solving might be affected [5,40]. Therefore, it is possible that depressive symptoms affect the neurocognitive functions of patients with SAD. Actually, one study demonstrated that verbal memory impairment is correlated with the Beck Depression Inventory score [7]. The impairment of verbal memory might be influenced by comorbid depression. Moreover, another study set their exclusion criterion as 16 points (and above) on the Hamilton rating scale for depression (HAM-D) [15], presented the possibility that some of their subjects had mild depression.

However, one report has described that comorbid depressed versus non-depressed SAD patients respond uniquely to stress in terms of their neuropsychological function [14]. We were unable to find a report in the relevant literature describing the evaluation of a relation between clinical severity and neurocognitive functions in patients with SAD who had few depressive symptoms, but who nevertheless had severe social anxiety symptoms for which they had sought clinical treatment.

For this study, we recruited outpatients without comorbidity to exclude effects of depressive symptoms. This study was conducted to evaluate neurocognitive functions in SAD patients without comorbidity, and to investigate the relation between clinical severity and neurocognitive functions.

2. Methods

2.1. Participants

From outpatients at the Department of Psychiatry, Hokkaido University Hospital, 30 patients (10 female, 20 male; mean age (S.D.), 23.9 (6.7) years) were recruited. Using the Mini-international neuropsychiatric interview [31,34], psychiatrists who had at least 10 years of clinical experience and who were blind to this study diagnosed all patients as having generalized SAD in DSM-IV TR [4]. At recruitment, the HAM-D and the global assessment of functioning (GAF) were checked. Patients were excluded from the study if they had organic brain disease or other Axis I disorder or a HAM-D score above 8 points. Patients were also excluded if they were under 16 or over 60 years old, or if they had had a previous neuropsychological assessment, or if they had received neurotropic medication aside from selective serotonin reuptake inhibitor (SSRI) antidepressants. Ten (33%) of the patients with SAD used SSRIs: paroxetine or fluvoxamine. During the study, no additional treatment was provided to the patients.

The control group consisted of 30 healthy volunteers (10 female, 20 male; mean age (S.D.), 25.6 (5.6) years) who had been recruited from the community via advertisement. They were also interviewed using the Mini-international neuropsychiatric interview administered by the psychiatrists. They had no Axis I psychiatric disorder. They were selected carefully to correspond to the gender, age (difference within five years), and education level (difference within two years) of SAD patients.

2.2. Materials

2.2.1. Neuropsychological assessment

The neuropsychological test battery administered at our hospital comprised five tests [39].

1) Wisconsin card sorting test (WCST): A computerized Japanese Keio University version [18,21] was used. The number of category achievements and perseverative errors of Milner were used as measures of executive functioning.
2) Continuous performance test (CPT): A computerized A-X CPT was administered for about 7 min. Several characters were presented at the center of a display. Subjects were instructed to respond as quickly as possible to "X", which appeared immediately after "A". The target stimulus was presented 70 times. Each stimulus was presented for 100 ms with an interstimulus interval that varied from 1500 ms to 2000 ms. The average reaction time was used as a measure of psychomotor speed. The total number of errors was used as a measure of sustained attention.
3) Trail making test (TMT) [17]: Both parts of the Trail Making Test consist of 25 circles distributed over a sheet of paper. In Part A, the circles are numbered 1–25. The participant must draw lines to connect the numbers in ascending order. In Part B, the circles include both numbers (1–13) and 12 hiragana (Japanese cursive syllabary) letters. The participant must draw lines with the added task of alternating between the numbers and letters. Part A measures the psychomotor speed and visual scanning, whereas Part B requires more mental flexibility, ability to shift attention, and strategy. The time to complete part B was used as a measure of executive function. The arrangements of characters of the Japanese version differ from those of the traditional version. Moreover, the times to complete part A and part B differ [38].
4) Word fluency test (WFT): Each participant had to say as many words as possible beginning with sound "Shi", "I" or "Re" in 60 s. The total number of words was used as a measure of verbal fluency.
5) Auditory Verbal Learning Test (AVLT), adopted from the RBANS Japanese version [43]: Each participant was required to learn a 10-item word list over four trials (trials 1–4) and was then assessed 30 min later (trial 5). The number of recall words of trial 1 was used as indicating the immediate recall. That of trial 5 indicated delayed recall.

2.2.2. Clinical measures

Before the neuropsychological assessment, each participant's age, sex, duration of illness, years of education, antidepressant dosage [8] and full scale IQ of Japanese Adult Reading Test [25] were noted. Furthermore, after the tests, they took the following symptom scales for clinical assessments: The Self Report version of the Liebowitz Social Anxiety Scale (LSAS) [2,24], the state anxiety part of the State-Trait Anxiety Inventory (STAI) [29,36], and the Beck Depression Inventory – Second Edition (BDI-II) [6,22].