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Size abnormalities of the superior parietal cortices are related to dissociation in borderline personality disorder

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Abstract

Recent evidence suggests that borderline personality disorder (BPD) is related to reduced size of the parietal lobe. Dissociative symptoms occur in the majority of individuals with BPD. Structural magnetic resonance imaging (3D-MRI) was used to assess volumes of the superior (precuneus, postcentral gyrus) and inferior parietal cortices in 30 young women with BPD who had been exposed to severe childhood sexual and physical abuse and 25 healthy control subjects. Compared with control subjects, BPD subjects had significantly smaller right-sided precuneus (−9%) volumes. The left postcentral gyrus of BPD subjects with the comorbid diagnosis of dissociative amnesia (DA) or dissociative identity disorder (DID) was significantly increased compared with controls (+13%) and compared with BPD subjects without these disorders (+11%). In BPD subjects, stronger depersonalization was significantly related to larger right precuneus size. Possibly, larger precuneus size in BPD is related to symptoms of depersonalization. Increased postcentral gyrus size in BPD may be related to the development of DA or DID in the presence of severe childhood abuse.

Keywords: Precuneus; Postcentral gyrus; Inferior parietal cortex asymmetry; Structural magnetic resonance imaging (3D-MRI); Trauma; Psychotic symptoms

1. Introduction

Borderline personality disorder (BPD) is defined as an intermediate level of personality organization that is considered to occupy a borderline area between neurosis and psychosis (Kernberg, 1967). Stress-related dissociative symptoms occur in about 75% of individuals with BPD (Skodol et al., 2002) and in about 40% of individuals with posttraumatic stress disorder (PTSD) (Bremner et al., 1992; David et al., 1999). Use of the *Structured Clinical Interview for DSM-IV Dissociative Disorders* has shown that dissociative amnesia is the area most strongly affected in persons who had been exposed to traumatic stress (Bremner et al., 1993). Childhood abuse, particularly chronic abuse beginning at early ages, was shown to be related to the development of high levels of dissociation, including dissociative amnesia (DA) and dissociative identity disorder (DID) (Boon and Draijer, 1993; Lewis et al., 1997; Chu et al., 1999).

Research so far has established size reduction and pronounced dysfunction of amygdala, hippocampus and prefrontal cortices in individuals with BPD (for review, see Zanarini, 2005). A recent study (Vermetten et al.,

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2006) demonstrated reduced amygdala and hippocampal size in subjects with DID and posttraumatic stress disorder (PTSD) who had been exposed to severe childhood abuse. Dissociative symptoms of survivors of childhood abuse with or without BPD have been repeatedly related to small hippocampal size (Bremner et al., 1997, 2003; Stein et al., 1997). Increased activity of the precuneus has been found in individuals with depersonalization disorder (Simeon et al., 2000) and in PTSD subjects with dissociative states (Lanius et al., 2002). BPD subjects were shown to have elevated pain thresholds that were correlated with dissociative symptoms (Ludäscher et al., 2007) and associated with reduced activity of the right-sided posterior parietal cortex (Schmahl et al., 2006).

Research on individuals with epilepsy of the temporal or parietal lobes has consistently demonstrated that abnormal EEG activity, seizures, and brain stimulation of the temporal or parietal cortices are associated with dissociative states (Halgren et al., 1978; Mesulam, 1981; Gloor et al., 1982; Salanova et al., 1995; Blanke et al., 2002). Stimulation of the parietal cortex typically leads to somatosensory aura or disturbed bodily perceptions (Salanova et al., 1995; Blanke et al., 2002). Lesion studies provide evidence that the superior parietal cortex is engaged in the generation and maintenance of an internal (sensorimotor) representation of the body (Sirigu et al., 1996; Berlucchi and Aglioti, 1997; Wolpert et al., 1998).

In previous investigations, we found reduced size of the parietal lobe (Irle et al., 2005) and reduced glucose metabolism of the precuneus (Lange et al., 2005) in a sample of women with BPD who had been exposed to severe childhood abuse. Subjects presented with pronounced dissociative and psychotic symptoms. In the present investigation, the size of specific parietal cortices of BPD subjects (who were also included in earlier reports: Irle et al., 2005; Lange et al., 2005) were compared with those of a healthy matched control group. To our knowledge, there is only one previous study on BPD that used an automated whole-brain investigation; it yielded negative results for all cerebral cortical areas (Rusch et al., 2003). The goals of our study were to investigate whether superior parietal cortices have abnormal size in BPD and whether superior parietal cortex size abnormalities are related to dissociative symptoms. Special emphasis was placed on the investigation of BPD subjects with the comorbid diagnosis of DA or DID. A further concern was to specify whether psychotic symptoms in BPD are related to size abnormalities of the inferior parietal cortex, as was previously suggested for subjects with schizophrenia (Frederikse et al., 2000; Niznikiewicz et al., 2000).

2. Methods

2.1. Subjects

The sample comprised 30 young female in-patients with the diagnosis of borderline personality disorder (BPD) according to the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV*) (American Psychiatric Association, 1994) consecutively admitted to the Psychiatric State Hospital of Lower Saxony, Göttingen, Germany. The hospital has a specialized therapeutic unit for women who have experienced severe childhood sexual and physical abuse. All subjects had been treated earlier in stationary units for BPD and/or chronic PTSD. Subjects with a history of neurological disease or psychotic disorders (*DSM-IV* axis I) were excluded. Eight subjects were on antidepressant medication (selective serotonin reuptake inhibitors). Six subjects were occasionally treated with sedatives (benzodiazepines: $n=5$, hypnotics: $n=6$). All subjects were included in a previous report on parietal cortex size in BPD (Irle et al., 2005).

BPD subjects were compared with 25 healthy female control subjects comparable with regard to age (31 ± 6 vs. 33 ± 7) and years of education (11 ± 2 vs. 11 ± 2). Control subjects were recruited for the study by an advertisement in a local newspaper and leaflets distributed in the Hospital of the University of Göttingen and in town. Only subjects without a history of neurological or psychiatric disorder were studied.

After a complete description of the study was given to the subjects, written informed consent was obtained. The study design was approved by the Ethical Committee of the Medical Faculty of the University of Göttingen.

2.2. Clinical assessment

All subjects were investigated with the *Structured Clinical Interview for DSM-IV* (*SCID-I* and *SCID-II*) (First et al., 1995, 1997; Wittchen et al., 1997) and the *Structured Clinical Interview for DSM-IV Dissociative Disorders* (*SCID-D*) (Steinberg, 1994; Gast et al., 2000). All subjects met *DSM-IV* criteria for BPD. Twenty-seven (90%) subjects met criteria for lifetime or current depersonalization disorder. Seven (23%) subjects met criteria for lifetime or current dissociative amnesia (DA), and four (13%) for current dissociative identity disorder (DID). Eleven BPD subjects (37%) met criteria for lifetime or current PTSD. Three (10%) subjects met criteria for lifetime panic disorder with agoraphobia, eight subjects (27%) for current panic