



Mild neurological impairment may indicate a psychomotor endophenotype in patients with borderline personality disorder



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ABSTRACT

The aim of the present study was to determine whether patients with borderline personality disorder (BPD) show any neurological soft signs compared to healthy controls. Furthermore we sought to examine the role of common symptoms related to BPD, such as depression, anxiety or impulsivity, in association with neurological soft signs. Thirty patients with borderline personality disorder and thirty hospital-based controls were examined for neurological soft signs. The total score of neurological soft signs in BPD was significantly higher than controls. In terms of subscales, patients had higher scores in Sensory Integration and Motor Coordination and other neurological soft signs compared to control group. Multiple regression analysis showed that the impulsivity score was the best significant predictor of neurological soft signs in BPD. The increase of neurological soft signs in patients with BPD may address a non-focal neurological dysfunction in borderline personality disorder.

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1. Introduction

Borderline personality disorder (BPD) is a prevalent disabling psychiatric disorder that affects 1–2 percents of the general population. Based on defining criteria, BPD is associated with unstable relationships, affective instability, disturbance in identity, extreme anger and impulsivity, frantic efforts to avoid abandonment and suicidal behavior (APA, 2000). BPD frequently occurs with other disorders of axis I as well as other personality disorders. Almost 40% of the BPDs meet the criteria for at least one mood disorder (31% major depression, 16% dysthymia, 9% bipolar I and 4% bipolar II) that is correlated with a poorer functional outcome (Skodol et al., 2002). Moreover, BPD is also related to the long term impairment of social functioning and extensive use of psychiatric and community services (Bender et al., 2001; Zanarini et al., 2010).

Comparatively little data exists on the neurological manifestations of BPD. Therefore studying the correlation of clinical or behavioral data with underlying structural and functional neurological findings in BPD has been recently the focus of interest (Laddis, 2015). For example neuroimaging data from BPD showed a reduced volume in the frontal lobe, orbitofrontal cortex, anterior

cingulate cortex, hippocampus, and amygdala. In addition, fMRI data indicated a hyperactivation of limbic structures in BPD patients. Findings showed that dysfunctional dorsolateral prefrontal and limbic systems are in line with the conceptualization of BPD as an emotion dysregulation disorder (Schulze et al., 2016).

To better understand the neurological correlates, researchers have investigated whether sensory or motor areas other than regulatory brain regions are involved in BPD. They found that BPD patients showed an increased cortical volume in the right cerebellum, the supplementary motor area, the left posterior insula, and the right middle frontal gyrus compared to healthy individuals. Simultaneous involvement of dorsolateral prefrontal cortex (dlPFC) and motor networks suggested an overlap between emotional regulation and motor function in BPD (Laddis, 2015; Schulze, et al., 2016).

However researchers have been unsure about the clinical importance of the above-mentioned neuroimaging findings. Notably while there was no focal neurological deficit identified in patients with BPD (De la fuente and Bobes, 2009), there was an increased observation of mild neurological soft signs (NSS) in BPD (De la Fuente et al., 2006). NSS indicate a nonspecific brain dysfunction related to a non focal neurological impairment that are classified into sensory integration, motor coordination, sequence of complex motor acts and primitive reflexes (Buchanan and Heinrichs, 1989; Neelam et al., 2011). NSS have been already described in many psychiatric disorders such as schizophrenia (Hirjak et al., 2013; Peralta et al., 2014), obsessive compulsive disorder (OCD) (Jaafari

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et al., 2013; Peng et al., 2012), and bipolar disorder (Goswami et al., 2007), as well as substance use (Dervaux et al., 2013). For example, existing research in schizophrenia have shown that NSS are associated with changes in cortical structure and function though it was not clear to what extent these findings reflect underlying pathophysiological processes (Chan et al., 2010a, 2010b; Sevincok et al., 2004). To further investigate clinical importance of NSS, they were also studied in healthy populations (Dazzan et al., 2006); neuroimaging data from healthy individuals indicated a relationship between NSS and structural cortical abnormalities. Furthermore, findings of an fMRI study in a healthy individual group showed negative correlations between NSS levels and functional connectivity of the right precuneus, right superior frontal areas, supplementary motor area, and left paracentral gyrus. In fact these data suggested that NSS may be associated with the neural activity in cortical motor networks rather than subcortical networks (i.e., striatal or thalamic) (Thomann et al., 2015). The idea of potential shared mechanisms of NSS and BPD was supported by findings from healthy population studies and raised a question on whether there is an association between BPD and development of NSS (e.g. impairments in movement planning, execution and control). Addressing therapeutic importance of these findings, some studies suggested that neurological abnormalities may be related to an incomplete response in BPD to psychopharmacotherapy (Laddis, 2015). Therefore we believed studying NSS in BPD extends our understanding of BPD and their underlying mechanisms and would also have therapeutic implications.

The aim of the present study was to determine whether patients with borderline personality disorder (BPD) show any neurological soft signs compared to healthy controls. Furthermore we sought to examine the role of common symptoms related to BPD (such as depression, anxiety or impulsivity) in association with neurological soft signs. We hypothesized that the patients with BPD showed a significantly higher rate of soft sign neurological abnormalities compared to the control participants. We further assumed that NSS were connected to psychological symptoms related to BPD.

2. Methods

2.1. Participants

Thirty patients with borderline personality disorder (mean age=32.80, S.D=4.52, 19 females) and thirty healthy control individuals with no acute psychiatric disorders (mean age=25.60, S. D=2.89, 14 females) participated in the study. Participants were selected from two clinics in a Psychiatry Hospital in Tehran. A trained psychiatrist evaluated the patients for borderline personality and other axis II disorders using the structured clinical interview (SCID-II) (First et al., 1997). Comorbidity or absence of axis I disorders were also assessed with the structured clinical interview for DSM-IV. To the objective of this study and to limit study bias, we excluded patients with acute or lifetime schizophrenia, major depressive disorder, and any substance abuse. Additional exclusion criteria included absence of any psychotropic medications for at least 6 weeks prior to the study. As controls those who accompanied the patients or who visited the Psychiatric hospital for mental health care but lacking lifetime Axis I or II disorders were asked to participate to the study. They were also required to use no psychotropic medications for at least 6 weeks prior to the study (For demographic characteristics see Table 1).

Table 1
Demographic characteristics of borderline personality disorder and control groups.

Variables	BPD		Control		p
	N=30		N=30		
	Mean	SD	Mean	SD	
Age	23.80	4.5	25.60	2.8	0.182
Gender	Frequency	%	Frequency	%	0.299
Female	19	63.3	14	46.7	
Male	11	35	16	53.3	
Level of education					0.095
Higher	17	56.7	24	80	
Lower	13	43.3	6	20	
Smoking					1.00
Positive	4	13.3	3	10	
Negative	26	86.7	27	90	
Family history					0.506
Positive	7	23.3	4	13.3	
Negative	23	76.7	26	86.7	

2.2. Measures

2.2.1. Borderline personality disorder SCID-II

SCID-II is a 119-item, yes-no format questionnaire assessing the DSM-IV diagnostic criteria for personality disorders (Huprich, 2003). Individuals with self-reported criteria for any personality disorder were interviewed and assessed by SCID-II. SCID-II has demonstrated a very high inter-rater reliability in diagnosing personality disorders (Lobbstaël et al., 2011). We used a 15-items version of SCID-II, known as SCID-II-BPD, to assess patients and control participants.

2.2.2. Beck depression inventory-second edition (BDI-II)

BDI-II is one of the most widely used instruments for examining depressive symptoms and assessing their severity. It is a self-rating scale based on the diagnostic criteria in the DSM-IV to assess depression severity in adults and adolescents older than 13 years. Twenty one items were summed to calculate a total BDI-II score ranging from 0 to 63 with higher scores showing more severe depression.

2.2.3. Beck anxiety inventory (BAI)

BAI was used to assess the severity of anxiety symptoms in individuals. BAI consists of 21 items evaluating the severity of anxiety in adults and adolescents during the past week. The total score ranges from 0 to 63 with higher scores indicating higher level of anxiety.

2.2.4. The Barratt impulsiveness scale-11 (BIS-11)

BIS-11 is a commonly used scale to measure the impulsivity (Steinberg et al., 2013). The BIS is a 30-items self-rating scale assessing different aspects of impulsivity. BIS presents three subscales: non-planning, motor impulsivity, and attentional impulsivity as well as a total score of impulsivity.

2.2.5. Neurological soft signs examination

The neurological soft sign examination method was adapted from Quitkin et al. (1976). We examined 29 neurological soft signs mainly categorized in the following functional areas: integrative sensory dysfunction, motor incoordination, and complex motor sequencing. For more details refer to Table 2.

2.3. Procedure

All participants received information about the study and completed the informed consent before enrollment in the

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