Social-emotion recognition in borderline personality disorder

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Abstract

Borderline personality disorder (BPD) is characterized by interpersonal disturbances, but the neurocognitive aspects of these symptoms are poorly understood. We hypothesized that patients with BPD have impaired perception of emotional expressions, which are related to symptoms of interpersonal dysfunction. To control potential confounding factors, this study excluded subjects with comorbid diagnoses known to be associated with impaired affect perception. We tested 43 outpatients with BPD and 26 healthy controls on emotion recognition tasks (facial, prosodic, and integrated facial/prosodic), nonemotional facial feature recognition, and interpersonal antagonism (Buss-Durkee Hostility Index). Patients with BPD showed normal ability to recognize isolated facial or prosodic emotions but had impaired recognition of emotions in integrated facial/prosodic stimuli, as well as impaired discrimination of nonemotional facial features. In patients with BPD, impaired recognition of integrated emotional stimuli was associated with interpersonal antagonism, particularly suspiciousness and assaultiveness. These results suggest that patients with BPD have deficits in higher order integration of social information, which may be related to some of the more serious symptoms of the disorder.

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1. Introduction

Borderline personality disorder (BPD) is a serious, chronic psychiatric disorder characterized by disruptions of mood, impulse control, and interpersonal relations. Although the first 2 problems have been extensively studied, the neurocognitive aspects of interpersonal disturbance in BPD remain poorly addressed. However, the emerging empirical literature and methodology of social cognitive neuroscience provide new constructs and tools to investigate these phenomena [1]. One fundamental neurocognitive function necessary to the establishment and maintenance of interpersonal relations is the ability to recognize social signals, such as emotional expressions. For instance, facial expressions serve as important signals that help regulate subjective emotional experience and behavior [2]. Vocal expressions are also important, such as prosody, which has been classically defined as the aspect of speech that communicates meaning by variation in stress and pitch, independent of lexical and syntactic content [3]. Similarly to facial expression, prosody has a feature of emotional expression that serves an important role in interpersonal communication.

The performance of psychiatric patients on tests on facial and prosodic emotion recognition has been increasingly investigated. For instance, patients with schizophrenia consistently exhibit deficits in the recognition of facial emotion (reviewed in Edwards et al [4]). Facial emotion recognition deficits are also observed in depression [5-8] and intermittent explosive disorder [9], and abnormal neural responses to facial emotion have been demonstrated in posttraumatic stress disorder (PTSD) [10,11]. Emotion recognition deficits have been associated with interpersonal disturbances in schizophrenia [12], alcoholism [13], and psychopathy [14].

Two studies on facial emotion recognition have been reported to date in BPD. In the first study [15], 30 outpatients with BPD showed significantly less accurate facial emotion recognition when tested using a self-paced, multiple-choice format. Emotion recognition performance was unrelated to self-reported affect intensity, level of emotion awareness, and ability to coordinate mixed emotions. In the second study [16], 21 women with BPD and histories of childhood sexual abuse were compared with 21 women without BPD who also reported childhood sexual abuse and a control group of 20 women with neither BPD
nor childhood abuse. Facial emotion recognition was tested using a self-paced, free-response format. In that study, the BPD group was more accurate than the other 2 groups in recognizing fearful facial expressions, which was related to a response bias toward fear. Results of a functional brain imaging study suggest that the negative attributional bias of patients with BPD may be related to heightened amygdala responsivity to facial emotion [17].

These studies suggest that patients with BPD exhibit impaired or altered facial emotion recognition. However, some questions remain. It is unclear whether social-emotion recognition deficits in BPD exist primarily in one or more sensory modes individually or alternatively in higher level heteromodal integration of emotional perceptions. In addition, the relative role of speed versus accuracy in emotion processing remains to be assessed. Patients with BPD may exhibit a lower accuracy compared with nonpsychiatric subjects on emotion recognition tests because of excessively rapid responding (which could be consistent with the prominent behavioral impulsivity exhibited by these patients). Conversely, to achieve accurate performance on these tests, subjects with BPD may need to take a relatively longer time to adequately process the stimuli. These alterations could confer a functional impairment if patients with BPD do not optimize speed and accuracy in processing emotional expressions as they unfold at the rapid pace that is typical in real-world interpersonal contexts.

The possible effects of common comorbidities with BPD also need to be considered. The disruptive effects of BPD often are associated with comorbid psychiatric disorders, such as major depression, PTSD, or substance dependence [18], which are also associated with impaired emotion perception (cited above). These disorders are therefore potential confounds when attempting to identify core characteristics of BPD. Although patients with BPD frequently present with such features that are important to address clinically, they are not essential to the diagnosis of BPD itself. Thus, a complete understanding of emotion perception in BPD requires that some studies be conducted excluding these comorbid disorders. The present study was designed with such exclusion criteria.

The relationship of social-emotional processing with interpersonal dysfunction in BPD also remains unclear. The interpersonal disturbances in BPD often consist of emotions, thoughts, and behaviors that are antagonistic in nature. For example, patients with BPD exhibit a significant degree of hostility [19], suspiciousness [20], and aggressive behavior [21,22], which are reliably assessed with the Buss-Durkee Hostility Inventory (BDHI) [23]. Although other forms of interpersonal dysfunction are also seen in BPD, we have chosen to focus on symptoms of antagonism because of the public health impact of these symptoms in the general population [24,25].

In the present study, we evaluated clinically stable outpatient adults with BPD on their ability to recognize isolated facial and prosodic emotions (in both speed and accuracy), as well as on a heteromodal emotion recognition task combining these 2 sensory features. A facial recognition task was also used to evaluate the perception of social but nonemotional facial stimuli. Finally, we addressed the relationship of social neurocognition with interpersonal dysfunction in BPD. We hypothesized that patients with BPD would exhibit multiple social-emotional processing deficits, which would be related to interpersonal antagonism.

2. Methods

2.1. Subjects

For this study, 43 adults with BPD and 26 control subjects were recruited from outpatient mental health clinics and the community. The patient recruitment included referrals made from the investigators’ outpatient clinician colleagues and recruitment from the community through advertisements placed on the Internet. This sample also participated in other studies reported elsewhere [26]. The BPD group was similar to the control group in age, sex ratio, ethnicity distribution, parental education, and employment status (Table 1). We excluded subjects younger than 18 or older than 60 years and those with a history of neurologic disease, schizophrenia, schizo-affective disorder, or bipolar disorder, as well as subjects with current PTSD, major depressive disorder, or substance dependence. We also excluded subjects with uncorrected impairments in visual or auditory acuity. Subjects with BPD were clinically stable during the study: none were hospitalized in the month before the study nor had psychotic or dissociative symptoms at the time of study. The mean Global Assessment of Function was 56 ± 9, and 77% were being treated with psychiatric medication (Table 1). Diagnostic evaluation included the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I), and the SCID-II screening questionnaire and interview for DSM-IV Axis II disorders [27,28]. Twenty-one (49%) of the BPD subjects were randomly chosen for videotaping of their diagnostic interview, which was reviewed by a second SCID-trained diagnostician (with a PhD in clinical psychology). Interrater agreement for BPD criteria was high (κ = .81). Comorbid Axis I diagnoses included dysthymic disorder.

Table 1 Demographic and clinical data

<table>
<thead>
<tr>
<th>Variable</th>
<th>BPD (n = 43)</th>
<th>Control (n = 26)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>35 ± 13</td>
<td>34 ± 9</td>
</tr>
<tr>
<td>Sex (% female)</td>
<td>88</td>
<td>89</td>
</tr>
<tr>
<td>Education (y)</td>
<td>14 ± 3*</td>
<td>16 ± 2</td>
</tr>
<tr>
<td>Parental education (y)</td>
<td>15 ± 3</td>
<td>15 ± 2</td>
</tr>
<tr>
<td>Ethnicity (W, B, L, As, Nat) (%)</td>
<td>77, 7, 5, 9, 2</td>
<td>77, 8, 4, 12, 0</td>
</tr>
<tr>
<td>On medications at study (%)</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Global Assessment of Function</td>
<td>56 ± 9</td>
<td>N/A</td>
</tr>
<tr>
<td>Age at symptom onset</td>
<td>12 ± 6</td>
<td>N/A</td>
</tr>
</tbody>
</table>

W indicates white; B, black; L, Latino; As, Asian; Nat, Native American.

Group totals for ethnicity greater than 100% because of rounding.

* P < .05.
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