

Why Psychiatrists Are Reluctant to Diagnose **Borderline Personality Disorder**

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ABSTRACT

Clinicians can be reluctant to make a diagnosis of borderline personality disorder (BPD). One reason is that BPD is a complex syndrome with symptoms that overlap many Axis I disorders. This paper will examine interfaces between BPD and depression, between BPD and bipolar disorder, and between BPD and psychoses. It will suggest that making a BPD diagnosis does more justice to patients than avoiding it.

WHAT IS BORDERLINE PERSONALITY DISORDER?

Borderline personality disorder (BPD) is a diagnosis with an unusual history. The idea that patients might fall on some sort of “borderline” between psychosis and neurosis dates back to 1937, at which time the syndrome was first described.¹ BPD patients do have quasipsychotic or micropsychotic symptoms, such as voices telling them to kill themselves, paranoid feelings, and depersonalization.² However these cognitive symptoms are not essential features of BPD. The core of the syndrome is a striking instability of mood, accompanied by a wide range of impulsive behaviors, particularly self-cutting and overdoses, and with intimate relationships that are impulsive, stormy, and chaotic.³



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Since BPD begins early in life and can continue over many years, it is classified as a personality disorder. However, BPD differs from other categories on Axis II in that it is associated with a wide range of active symptoms.⁴ Moreover, BPD is one of the most common clinical problems psychiatrists see in practice. One study found that half of all patients with repetitive suicide attempts in emergency rooms meet criteria for this diagnosis.⁵ Due to suicidal threats and actions, BPD patients are often admitted to hospital.⁶ BPD cases are also common in out-patient settings,⁷ where the pathology is often serious enough to use a large amount of clinical resources.

WHY CLINICIANS ARE RELUCTANT TO DIAGNOSE BPD

Structured interviews pick up many cases of BPD missed in ordinary practice.⁷ This finding shows that practitioners are not consistently making this diagnosis. There are a number of reasons why clinicians may be reluctant to recognize BPD.

First, Axis I diagnoses are more familiar to most professionals. Making an accurate Axis II diagnosis requires experience. Personality disorders often seem to lack precise symptomatic criteria, since many of their features describe problems in interpersonal functioning that require clinical judgment for accurate assessment.

Second, resistance to diagnosing patients with a personality disorder may be based on the idea that these conditions are untreatable,⁸ or at least not treatable using the pharmacological tools that have come to dominate the treatment of so many other disorders. While there is good evidence for the efficacy of psychotherapy in BPD,⁹ not every clinical setting has the resources to provide that form of treatment. Simpler constructs such as major depression lead to more familiar treatment options, particularly pharmacotherapy.

Third, clinicians may wish to avoid making diagnoses associated

with stigma. It is an unfortunate reality that a diagnosis of BPD can indeed lead to rejection by the mental health system. If BPD were to be reclassified as, for example, a mood disorder, patients would tend to be seen as having a biological illness instead of having a problematical personality. However, stigma cannot be removed by reclassification. Patients who are chronically suicidal and who do not form strong treatment alliances will continue to be just as difficult, even under a different diagnostic label.

COMORBIDITY AND DIAGNOSTIC PROBLEMS

Patients with BPD frequently meet criteria for multiple Axis I diagnoses.¹⁰ Considering that the disorder is associated with so many symptoms, this level of comorbidity should not be surprising. Changing the diagnosis of a patient with BPD, however, to one of these comorbid disorders focuses on only one aspect of the syndrome and fails to account for BPD's broad range of clinical phenomena (affective, impulsive, interpersonal, and cognitive).

While it is tempting to conclude that diagnoses such as major depression are the "real" problems in BPD, similar symptoms can derive from entirely different causes. Clinical phenomena, such as low mood or unstable mood, are no more specific than fever or inflammation. All that "comorbidity" says is that there are enough symptoms in one patient to meet criteria for more than one DSM category.¹¹ Moreover, overlap is common in the DSM system—major depression has at least as much comorbidity as any Axis II disorder.¹²

Another source of confusion is that the description of BPD in DSM-IV-TR is not specific.⁴ The definition introduced in DSM-III was an advance because it operationalized diagnosis using observable criteria. As with other disorders, DSM instructs the clinician to make a diagnosis when five out of nine criteria are met. The result is that many permutations lead to the same

category, producing a heterogeneous group.

It would be better to identify crucial features without which the diagnosis should not be made. One can group the current DSM criteria into affective, impulsive, interpersonal, and cognitive components. Patients should have most or all of these features to merit the diagnosis. That approach has been used in a research measure, the Diagnostic Interview for Borderlines (DIB),¹³ later revised as the DIB-R.¹⁴ This semistructured interview scores each of four areas of pathology on four sub-scales (0–2 for affective and cognitive symptoms and 0–3 for impulsive and interpersonal symptoms), using an overall cutoff point of eight out of 10 for positive diagnosis. Patients who meet DIB-R criteria are much more homogeneous, as shown by studies demonstrating that this instrument distinguishes them from patients with other Axis II disorders, and diagnosis achieves similar specificity if one requires six or seven criteria rather than five.¹⁵

BPD AND PSYCHOSIS

The original concept of BPD as lying on a border between neurosis and psychosis found a parallel in the diagnostic term *pseudoneurotic schizophrenia*.¹⁶ The concept was that patients with such a wide variety of neurotic symptoms could be latently psychotic. However, this diagnosis confused personality disorders primarily affecting mood and impulsivity (like BPD) with categories that primarily affect cognition, such as schizotypal personality.¹⁷ Neither family history studies nor biological markers support a link between BPD and schizophrenia.¹⁸ Nonetheless, some cases are challenging for differential diagnosis, since the cognitive symptoms of BPD can occasionally be florid. However, these phenomena are transient and stress-related, while insight is retained, as the following case example illustrates.

Case example—Patient 1. Bill was a 25-year-old man under

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treatment for chronic suicidality, unstable relationships, and mood instability. He also had paranoid ideas, sometimes thinking that neighbors were plotting against him. All these thoughts, however, were exaggerations of real situations and never had the bizarre quality of delusions. Bill also heard critical voices in his head when stressed, but knew that such experiences were imaginary. Bill received a DIB-R score of 9/10.

Nonetheless, Bill was initially diagnosed with schizophrenia and treated for psychosis for over five years with injectable antipsychotic medication. Bill actually liked attending this clinic and getting the injections, since it gave him a reason to come in every two weeks and to talk with a nurse. However, as his life stabilized, Bill's micropsychotic symptoms eventually remitted, along with his impulsive and affective symptoms. By age 30, Bill stopped taking neuroleptics and never had a relapse of paranoid ideas or hallucinations.

BPD, DEPRESSION, AND DYSTHYMIA

Depression is a common reason for clinical presentation in patients with BPD. It has been suggested that the BPD is an atypical form of unipolar depression.¹⁹ BPD is associated with chronic lowering of mood, particularly dysthymia with an early onset.²⁰ One argument in favor of BPD as a form of major depression was based on the frequency of family history of depression in BPD patients. However, impulsive disorders, such as substance abuse and antisocial personality, are actually more

common in families than mood disorders.¹⁸ Another argument was based on commonalities in biological markers, such as REM latency.¹⁹ However, it has never been shown that these markers are specific to DSM categories.

There is an important phenomenological distinction between temporal patterns of depressive symptoms in depression and BPD.²¹ In classical depression, mood is stable over weeks and is relatively unresponsive to the environment. In contrast, mood in BPD is highly mercurial. Moreover, mood can be strikingly unstable in the course of a single day, depending on life events. Patients have a mixture of affects—not only sadness or anxiety, but also anger, brief periods of elation, and feelings of numbness. On a more practical note, depression in BPD does not respond in the same way as classical depression to antidepressant drugs, as the following case illustrates.⁹

Case example—Patient 2.

Susan was a 24-year-old woman under treatment for chronic depression, with rapid shifts of mood, usually to anger and rageful outbursts. She also had a history of self-cutting and repetitive overdoses. Susan received a DIB-R score of 10/10.

Nonetheless, Susan was diagnosed with major depression and treated with a variety of antidepressants from several classes, none of which had any lasting effect. Each medication change led to short-term improvement for a few weeks followed by relapse to her previous state. Once Susan became engaged in psychotherapy, however, she improved to the point that

antidepressants were no longer considered necessary.

BPD AND THE BIPOLAR SPECTRUM

It has been proposed that borderline pathology falls within the spectrum of bipolar illness,²² based on a wish to expand the narrower diagnostic construct of bipolar disorder into a much broader range of conditions termed the *bipolar spectrum*. In this model, the range of bipolar spectrum disorders would be extended to include bipolar III (antidepressant-induced hypomania), as well as bipolar IV (ultra-rapid-cycling bipolar disorder). The last category, bipolar IV, describes mood swings typical of BPD (i.e., rapid shifts over hours). This expanded definition might include many, if not most, patients with BPD.

The issue is whether the lability of mood seen in BPD is identical to phenomena observed in mood disorders, such as bipolar II.²³ Hypomanic episodes have to last for at least four days, and this consistency of mood is rarely seen in BPD. Instead, affective instability (AI) is a characteristic feature that distinguishes BPD from classical bipolar disorder (as well as from other personality disorders). Emotion dysregulation is a similar concept.²⁴

Other lines of evidence have also failed to support the idea that BPD and bipolar disorder reflect the same underlying psychopathology.^{25,26} To summarize, there is no evidence for a common etiology, family prevalence data shows that impulsive disorders are more common than mood disorders in the

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first-degree relatives of patients with BPD, the longitudinal course of BPD rarely shows evolution into bipolar disorder, and treatment studies have failed to show that mood stabilizers have anywhere near the same efficacy in BPD as they do in bipolar disorder.

The following case demonstrates some of the problems in differential diagnosis between BPD and bipolar disorder.

Case example—Patient 3. Lisa had been self-cutting since age 16 and presented to a clinic with chronic suicidal ideation, irritability, and rages. Lisa received a DIB-R score of 9/10.

Nonetheless, bipolar II disorder was diagnosed on the basis of Lisa's mood swings, as well as repeated episodes in which she impulsively became involved with men—flying thousands of miles to meet them after an initial internet contact. At certain points of her illness, Lisa also showed quasipsychotic symptoms, such as an intense fantasy that she was Jesus's sister who had been sent to earth with a mission. Yet lithium, prescribed for a full year in adequate doses, had no effect on her symptoms. Instead, all of these problems came under control within weeks once Lisa entered psychotherapy and formed a solid therapeutic alliance.

BPD AND POSTTRAUMATIC STRESS DISORDER

The concept that BPD might be a “complex” form of posttraumatic stress disorder (PTSD) has been suggested by frequency of childhood abuse histories in these patients.²⁷

The problem is the assumption that trauma is the primary cause of BPD, rather than one among many risk factors. Research shows that biological, psychological, and social factors are all involved in the etiology of BPD, that severe trauma histories are only found in about a third of cases, and that most people exposed to child abuse in community samples have neither BPD nor any other diagnosable psychiatric disorder.²⁸

Case example—Patient 4. Lisa came for treatment of chronic suicidal ideation, multiple overdoses, and unstable intimate relationships. She also had transient episodes of depersonalization. Lisa's DIB-R score was 8/10.

A previous therapist had diagnosed Lisa with PTSD, and Lisa did have serious problems resulting from having been sexually abused by her stepfather between ages of 7 and 12. It was interesting, nonetheless, to note that her older sister, who was abused in precisely the same way, never experienced psychological problems to the extent that she ever sought treatment. While the issue of child abuse played an important role in her psychotherapy, Lisa's symptoms resolved gradually over time as she was able to find regular employment and become involved in more stable, less demanding relationships.

POSITIVE REASONS FOR DIAGNOSING BPD

What are the advantages in making the diagnosis of BPD? The first concerns the recognition of complex forms of psychopathology

with symptoms that do not occur in isolation. BPD is a construct that can account for the co-occurrence of a wide range of affective, impulsive, and cognitive symptoms in the same patient.⁴

The second advantage concerns prediction of outcome. BPD has a characteristic course over time, beginning in adolescence, with symptoms peaking in early adulthood, followed by gradual recovery in middle age.²⁹ This outcome pattern provides a useful frame for therapy.

The third value of diagnosing BPD lies in predicting response to treatment. Pharmacotherapy for depression is less effective in the presence of any personality disorder, and patients with BPD respond inconsistently to antidepressants.⁹ The problem is that drugs are not as effective in BPD as they are in the disorders for which they were originally developed. In several of the case examples presented above, the patients were treated with pharmacotherapy based on an Axis I diagnoses, without obvious benefit. Unfortunately, such results do not always lead physicians to reconsider diagnosis and therapy—all too often, patients are tried on a variety of medications or given nonevidence-based polypharmacy.

The fourth advantage, closely related to the last point, is the strong evidence that psychotherapy can be an effective form of treatment for BPD.⁹ We now know that several forms of cognitive and dynamic therapy are at least as effective, if not more effective, than drugs in relieving the symptoms of BPD. If

one does not make the diagnosis, patients may not be referred for these forms of psychotherapy.

The BPD diagnosis has its problems, but so do most of the disorders listed in DSM. Professionals treating patients meeting criteria for this disorder can benefit from the large empirical literature bearing on this complex clinical problem. The cases presented above are examples of how Axis I pathology can lead to mistaken expectations about course and treatment response. Finally, the the proper diagnosis of BPD can help us to inform and educate patients and their families.

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