Discussion about the ‘interface’ between borderline personality disorder (BPD) and bipolar disorder (BD) often resembles a ‘turf war’. Discussion largely focuses upon phenomenology and treatment, rather than aetiology or pathogenesis. One casualty of this turf war is clinician confidence, because focusing on the interface tends to overstate the problem. BPD and BD share some features but not...
“many” (1, p.2), unless one defines the bipolar phenotype so broadly as to render it meaningless. In day-to-day clinical practice, many (if not most) diagnoses of BPD or BD can be made with reasonable confidence. Bassett and colleagues point to the importance of the basic clinical principles of longitudinal and developmental assessment. However, in suggesting that these are more important for BPD than BD, they overlook what should be an explicit aim of any clinical assessment, distinguishing state from trait phenomena. Clearly, in a minority of presentations, this task can be challenging, especially in mixed affective states, and Bassett and colleagues, academic psychiatrists with clinical expertise in the management of mood disorders, offer guidance in the form of a consensus statement.

Although affective dysregulation is characteristic of both BD and BPD, Bassett and colleagues (1) point to useful clinical differences in terms of valence, amplitude, and time course. However, Figure 1a, showing the relative prominence of risk factors and features in BPD and BD, efficiently illustrates the fundamental clinical problem; viz. that these factors lack genuine specificity for either disorder. Although informative, it is difficult to see how Figure 1 might dissect the problem in such a way as to allow individual clinicians to draw conclusions in relation to individual patients in their consulting rooms. The dubious suggestion to rely upon the poorly defined constructs of “projective identification in relationships” and “noxious sense of self” seems to imply that these should usurp the application of recognised diagnostic criteria. Curiously, at no stage do the authors recommend resorting to the established diagnostic criteria for BPD, which (although imperfect) are more clearly defined and about which we have a wealth of published evidence in terms of their predictive value for a diagnosis of BPD.

The statement seems to travel well beyond the evidence in suggesting that the presence of sexual abuse or early trauma might be a useful feature for distinguishing BPD from BD. Both disorders share numerous distal risk factors and precursor signs and symptoms (2). These include childhood or familial attention deficit hyperactivity disorder, traumatic or stressful life events and childhood abuse, and substance abuse. Even certain personality traits, such as high harm avoidance and high novelty seeking, as well as impulsive aggression are also associated with later bipolar disorder.

Similarly, the authors’ assertions regarding psychosis are based on clinical folklore that has been refuted in the empirical literature (3). Auditory verbal hallucinations (AVH) occur in up to 50% of patients with BPD (4) and are commonly longstanding, severe, and qualitatively indistinguishable from AVH in other psychotic disorders (3, 4), suggesting that a categorical diagnostic approach, reliant solely on the presence of hallucinations, is unhelpful (3). In clinical practice, AVH in BPD
are also widely assumed to be unresponsive to treatment with antipsychotic medication, despite the fact that no controlled clinical trial has tested this assumption.

The authors are also more sanguine about the prognosis for BPD than seems justified from the major longitudinal studies of BPD. Although they point toward the psychopathological improvement of BPD over years and decades, functional outcomes, distress and life satisfaction tend to remain stably poor.

The statement is much more helpful in its recommendations regarding treatment, largely because some useful, evidence-based distinctions can be drawn between the disorders, as illustrated in Figure 1b. These markedly different treatment options underscore why accurate diagnosis is clinically important. The recommendations not only advise about which treatments to select but also which treatments should be avoided, such as pharmacotherapy as first-line treatment for BPD. They are also useful as a guide to re-evaluating (including re-diagnosing) patients in the event of non-response to a particular treatment, such as mood stabilising medication.

In closing, ‘permeability’ is an inevitable product of categorical diagnosis. The notion that BPD and BD are particularly noteworthy because of a “semi-permeable boundary” underemphasises the general problems of ‘comorbidity’ and syndromal overlap that are associated with our current system of categorical diagnoses, which do not reflect the hierarchical and dimensional structure of psychopathology (5).

References


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