GUIDELINE WATCH: PRACTICE GUIDELINE FOR THE TREATMENT OF PATIENTS WITH BORDERLINE PERSONALITY DISORDER

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Since the 2001 publication of APA's *Practice Guideline for the Treatment of Patients With Borderline Personality Disorder* (1), more studies have been published on borderline personality disorder (BPD) than on any other personality disorder (2, 3). New analyses of the validity of the DSM-IV-TR criteria–defined construct of BPD have been published, new data on the prevalence of BPD are available, risk factors for and biological characteristics of BPD are being elucidated, and new studies on the treatment of BPD have been carried out. This guideline watch highlights the most important of these developments.

DEFINITION, DIAGNOSTIC STABILITY, AND LONGITUDINAL COURSE

Definition

With an eye toward DSM-V, careful examination of axis II is occurring. This has, in part, included comparing the relative merits of categorical systems such as DSM-IV-TR and dimensional systems such as the five-factor model (4). This debate is as yet unresolved. However, recent studies have analyzed the nine DSM-IV criteria that define BPD and have concluded that, as defined, BPD appears to reflect a statistically coherent construct (5, 6). Utilization of a polythetic system requiring five out of nine criteria to make a categorical BPD diagnosis inevitably results in extensive heterogeneity within and between BPD populations being treated

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or studied. Increased attention is being focused on how to set the appropriate diagnostic "cutoff point" (i.e., the minimum number of criteria required to make the diagnosis). Also under discussion is whether some core criteria should be essential or whether all criteria should be weighted equally in establishing a diagnosis. New data also emphasize the importance of evaluating the degree of functional impairment in patients with BPD (7). A dimensional approach can be used to measure overall levels of dysfunction (8). Alternatively, the DSM criteria can be assessed in a dimensional fashion to capture functional impairment (9). However, even with proposed dimensional approaches, a discrete diagnostic threshold would need to be established in terms of illness severity and need for treatment (10).

A number of recent studies have examined the boundaries between BPD and selected axis I conditions. Since some patients with BPD may benefit from mood-stabilizing medications, the overlap between BPD and bipolar II disorder has been of particular interest (11–13). Although some individuals with BPD will also have a co-occurring bipolar disorder, the majority of the evidence to date supports BPD as an independent diagnosis rather than an attenuated form of a mood disorder (14–16). Similarly, though patients with BPD frequently have a history of trauma, the diagnosis is not considered a variant of posttraumatic stress disorder (PTSD) (17, 18). Of continuing interest is the identification of early precursors of BPD, such as a possible association between a history of childhood attention deficit hyperactivity disorder (ADHD) symptoms and diagnosis of BPD in adulthood (19).

Diagnostic stability and longitudinal course

New prospective longitudinal studies evaluating the long-term stability of BPD have been published. Results of the NIMH Collaborative Longitudinal Personality Disorders Study indicate that at 2 years after BPD diagnosis, only 44% of the patients retain the diagnosis (20). The McLean Hospital group has reported a 6-year follow-up of borderline patients in which 74% of the patients met criteria for remission over the entire follow-up period (21). Longer-term results have been published by Canadian researchers indicating that only 8% of the total borderline patient group still met diagnostic criteria at 27-year follow-up (22, 23). High rates of remission are speculated to relate to a number of factors, such as treatment effectiveness and/or fluctuating severity of comorbid axis I conditions. BPD has been shown to be significantly associated with major depressive disorder (MDD) and PTSD longitudinally (24). Somewhat counterintuitively, however, when BPD and MDD were followed over time, improvement in MDD was not followed by improvement in BPD; instead, improvement in MDD was more often predicted by prior improvement in BPD (25). Thus, the primary determinants of change in patients with BPD may not be related to comorbid axis I psychopathology (26). Other suggested contributions to high rates of remission of BPD over time include diminished impulsivity with increased age (27), social learning, and stress-reducing life adjustments such as avoidance of interpersonal intimacy (22). It should be noted that the concept of remission of BPD reflects dropping below the DSM-IV-TR threshold on the continuum of criteria defining the disorder, yet significant problems relating to the personality structure and organization can continue even if the diagnostic threshold has not been met.

EPIDEMIOLOGY, BIOLOGY, AND ETIOLOGY

To date, few population-based studies of personality disorders have been carried out. Torgersen (28) tabulated the results of eight epidemiological studies, including the large Norwegian study done by his own group; the mean prevalence of BPD across all studies was 1.2%. In Torgersen et al.'s study of 2,053 individuals in Norway, BPD was found to be equally prevalent among males and females (29). Although it is widely perceived that treatment populations of patients with BPD consist predominantly of females, published studies are in fact contradictory on this

point (30). Prevalence of BPD in treatment populations remains much higher than in the general population, and a recent study of a primary care population reported BPD to be fourfold higher than in the general population (31).

New studies are examining the multiple etiologies of BPD, based on the clear relevance of genetic, neurobiological, and psychosocial factors (32, 33). Although the magnitude of the heritability of BPD is thought to be significant (34), genetic risk factors specific to BPD have not been identified. One promising approach may be the study of underlying traits, such as novelty seeking (35) or impulsivity (36). Among the environmental contributions to the etiology of BPD, the role of childhood abuse remains prominent (37), particularly severe abuse (38) and sustained abuse (39).

Deficits in cognitive functioning, such as decision making (40), conflict resolution (41), and "effortful control" (41, 42), are being reported. These deficits are thought to be mediated in the frontal lobe, although not all studies have found frontal lobe impairment in cognitive functioning (43). A number of studies have suggested reduced central nervous system serotonin levels in borderline patients and other patients who demonstrate impulsive-aggressive behavior, but such behavioral disinhibition may reflect dysfunction in multiple monoaminergic systems and may not be specific to BPD (44).

Brain imaging studies of patients with BPD suggest irregularities in both structure and function (44–46). Limbic studies have found reduced volume in the amygdala and hippocampus (47, 48), altered amygdala activation (49, 50), and hippocampal hypometabolism (51). Frontal lobe studies suggest dysfunction of the dorsolateral and medial prefrontal cortex in connection with memories of abandonment (52), of the medial orbital frontal cortex in connection with diminished regulation of impulsive behavior (53), and of the prefrontal cortex under resting conditions (51). In addition, decreased binding of a serotonin precursor has been reported in the medial prefrontal cortex and anterior cingulate cortex in patients with BPD (54). Taken together, these studies suggest abnormalities in prefrontal, corticostriatal, and limbic networks perhaps related to lowered serotonin neurotransmission and behavioral disinhibition (55). Although findings from brain imaging studies are "not ready for prime time" to guide treatment planning for patients with BPD, these techniques may well have relevance in the future for individualized prediction of treatment outcome (46).

► TREATMENT

Evidence and opinion continue to support the recommendation of the 2001 guideline that psychotherapy represents the primary, or core, treatment for this disorder and that adjunctive, symptom-targeted pharmacotherapy can be helpful. For personality disorders in general, studies of psychotherapy report its effectiveness (56, 57). Persuasive data from randomized, controlled trials (RCTs) of BPD suggest that more than one type of psychotherapy is effective, and additional studies are under way. Dialectical behavior therapy (DBT) has been shown in an RCT to be effective for borderline symptoms in patients with comorbid BPD and substance abuse, though no improvement was shown for the substance abuse itself (58). In another RCT, in patients with BPD and comorbid opiate use, DBT was compared with comprehensive validation therapy (CVT). Both types of treatment were effective. There were fewer dropouts in the CVT group, and the maintenance of gains was greater in the DBT group (59). Studies of DBT by diverse research groups are being published, including a study of female veterans with BPD that compared 6 months of DBT with treatment as usual and reported improvement in the DBT group compared with the control group (60). Another study from the Netherlands that compared 12 months of outpatient DBT with treatment as usual showed better treatment retention, reduced self-mutilation, and reduced self-damaging impulsivity in the DBT group (61). An intriguing inpatient RCT that compared 3 months of inpatient DBT with treatment

as usual in the community demonstrated significant gains in the DBT group compared with the control group, including reduced self-injury, dissociation, depression, and anxiety, and improved interpersonal functioning and social adjustment (62).

Promising new psychotherapies for BPD are being piloted in open trials. These include interpersonal therapy (63); cognitive therapy (64); cognitive analytic therapy (CAT), a fusion of cognitive and psychodynamic therapy (65, 66); and systems training for emotional predictability and problem solving (STEPPS), a cognitive-behavioral systems-based form of time-limited group treatment for patients with BPD (67). In an ongoing study comparing transferencefocused psychotherapy (TFP) with DBT and supportive psychotherapy, TFP appeared to be beneficial (68), although the comparative analysis of the other two treatments has yet to be completed. The efficacy of TFP is also being assessed in a large multicenter study comparing TFP with schema therapy for patients with BPD (69).

A number of recent studies have focused on the benefits of pharmacotherapy for patients with BPD. An RCT that compared patients receiving fluvoxamine with a control group showed robust, long-lasting reduction in rapid mood shifts only in the treatment group (70). Another RCT that compared olanzapine with placebo in borderline patients showed improvement in global functioning in the medication group compared with the placebo group (71). Another RCT studied three groups of BPD patients—one group receiving fluoxetine, a second group receiving olanzapine, and a third receiving a combination of both; all three interventions led to substantial improvement, though a significantly greater rate of improvement in clinician-rated depression and impulsive aggression was seen in the olanzapine and the combination groups (72). Also, double-blind, placebo-controlled trials demonstrated benefit of divalproex sodium for patients with BPD (73) and for patients with cluster B personality disorders who demonstrate impulsive aggression (74). A number of case reports and noncontrolled medication trials have also been published (75–81).

All in all, the database is growing, and further evidence is accumulating that BPD is a condition that can be effectively treated by a combination of psychotherapy and symptom-targeted pharmacotherapy. Further research is needed to validate the approach taken by the 2001 guideline to select one of three different medication algorithms on the basis of the predominance of cognitive-perceptual symptoms, affective dysregulation symptoms, or impulse dyscontrol symptoms. One retrospective report from the NIMH Collaborative Longitudinal Personality Disorders Study produced mixed results on this question (82).

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