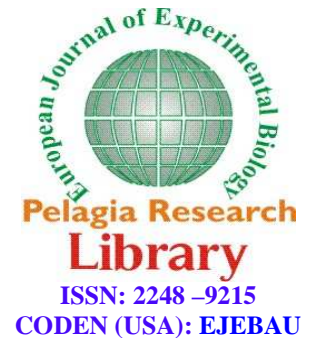




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Borderline personality disorder (BPD): An overview

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ABSTRACT

Borderline personality disorder (BPD) is a serious mental illness characterized by pervasive instability in moods, interpersonal relationships, self-image, and behavior. Clinical signs of the disorder include emotional dysregulation, impulsive aggression, repeated self-injury, and chronic suicidal tendencies, which make these patients frequent users of mental health resources. Borderline personality disorder is a chronic psychiatric disorder characterized by marked impulsivity, instability of mood and interpersonal relationships, and suicidal behaviour that can complicate medical care. Identifying this diagnosis is important for treatment planning. Although the cause of borderline personality disorder is uncertain, most patients improve with time. There is an evidence base for treatment using both psychotherapy and psychopharmacology. The clinical challenge centres on managing chronic suicidality. Causal factors are only partly known, but genetic factors and adverse events during childhood, such as physical and sexual abuse, contribute to the development of the disorder.

Key words: BPD, Clinical signs, Mental health

INTRODUCTION

Borderline personality disorder is a severe and chronic psychiatric condition, prevalent throughout health care settings. Borderline personality disorder is a common mental disorder associated with high rates of suicide, severe functional impairment, high rates of comorbid mental disorders, intensive use of treatment, and high costs to society. While a person with depression or bipolar disorder typically endures the same mood for weeks, a person with BPD may experience intense bouts of anger, depression and anxiety that may last only hours, or at most a day [7]. These may be associated with episodes of impulsive aggression, self-injury, and drug or alcohol abuse. Distortions in cognition and sense of self can lead to frequent changes in long-term goals, career plans, jobs, friendships, gender identity, and values. Sometimes people with BPD view themselves as fundamentally bad, or unworthy. They may feel unfairly misunderstood or mistreated, bored, empty, and have little idea who they are. Such symptoms are most acute when people with BPD feel isolated and lacking in social support, and may result in frantic efforts to avoid being alone. People with BPD often have highly unstable patterns of social relationships. While they can develop intense but stormy attachments, their attitudes towards family, friends, and loved ones may suddenly shift from idealization (great admiration and love) to devaluation (intense anger and dislike). Thus, they may form an immediate attachment and idealize the other person, but when a slight separation or conflict occurs, they switch unexpectedly to the other extreme and angrily accuse the other person of not caring for them at all. Even with family members, individuals with BPD are highly sensitive to rejection, reacting with anger and distress to such

mild separations as a vacation, a business trip, or a sudden change in plans. These fears of abandonment seem to be related to difficulties feeling emotionally connected to important persons when they are physically absent, leaving the individual with BPD feeling lost and perhaps worthless. Suicide threats and attempts may occur along with anger at perceived abandonment and disappointments. People with BPD exhibit other impulsive behaviors, such as excessive spending, binge eating and risky sex. BPD often occurs together with other psychiatric problems, particularly bipolar disorder, depression, anxiety disorders, substance abuse, and other personality disorders. Although the cause of BPD is unknown, both environmental and genetic factors are thought to play a role in predisposing patients to BPD symptoms and traits. Studies show that many, but not all individuals with BPD report a history of abuse, neglect, or separation as young children [1]. Forty to 71 percent of BPD patients report having been sexually abused, usually by a non-caregiver [3]. Researchers believe that BPD results from a combination of individual vulnerability to environmental stress, neglect or abuse as young children, and a series of events that trigger the onset of the disorder as young adults. Adults with BPD are also considerably more likely to be the victim of violence, including rape and other crimes. This may result from both harmful environments as well as impulsivity and poor judgement in choosing partners and lifestyles. NIMH-funded neuroscience research is revealing brain mechanisms underlying the impulsivity, mood instability, aggression, anger, and negative emotion seen in BPD. Studies suggest that people predisposed to impulsive aggression have impaired regulation of the neural circuits that modulate emotion [19]. The amygdala, a small almond-shaped structure deep inside the brain, is an important component of the circuit that regulates negative emotion. In response to signals from other brain centers indicating a perceived threat, it marshals fear and arousal. This might be more pronounced under the influence of drugs like alcohol, or stress. Areas in the front of the brain (pre-frontal area) act to dampen the activity of this circuit. Recent brain imaging studies show that individual differences in the ability to activate regions of the prefrontal cerebral cortex thought to be involved in inhibitory activity predict the ability to suppress negative emotion [17]. Serotonin, norepinephrine and acetylcholine are among the chemical messengers in these circuits that play a role in the regulation of emotions, including sadness, anger, anxiety and irritability. Drugs that enhance brain serotonin function may improve emotional symptoms in BPD. Likewise, mood-stabilizing drugs that are known to enhance the activity of GABA, the brain's major inhibitory neurotransmitter, may help people who experience BPD-like mood swings. Such brain-based vulnerabilities can be managed with help from behavioral interventions and medications, much like people manage susceptibility to diabetes or high blood pressure [4]. Borderline personality is a serious psychiatric disorder, with a prevalence of about 4% in the community, but as high as 20% in many clinical psychiatric populations, and significant morbidity. It is difficult to treat (both in the sense of responding poorly and as personally troubling to the therapist and the treatment team) and poorly understood. However, we have made tremendous strides in only a few decades, beginning with a theoretical concept in psychoanalysis that was ridiculed by most other psychiatrists, and progressing to a widely recognized clinical entity; from a pejorative label for disliked patients to a carefully defined diagnostic category; from the subject of almost no systematic study to one of the most intensively researched personality disorders in terms of diagnosis, epidemiology, genetics, developmental psychology, biological correlates, pathophysiology, and treatment and perhaps most important, from a hopeless prognosis to a hopeful one, and particularly one for which we have several evidence-based effective treatments.

DIAGNOSIS

According to the current psychiatric classification system in the fourth edition of the diagnostic and Statistical Manual of Mental Disorders (DSM-IV), borderline personality disorder is characterised by a pervasive pattern of instability in interpersonal relationships, identity and impulsivity [6]. For a diagnosis of borderline personality disorder, at least five of the nine criteria must be met. However, suicidal tendency or self-injury are the most useful indications for a correct diagnosis, whereas suicidal tendency or self-injury and unstable relationships have been the most predictive features in follow-up studies [9]. There are two quite different notions of the clinical meaning of the term borderline. The older one, that goes back to its earliest use in the psychoanalytic literature, refers to a broad category of patients whose underlying psychology does not have the chaos, disorganization, or defect in reality testing associated with psychotic patients, but also lacks the integration, stability of relationships, and regulation of affect associated with neurotic patients. This is, in terms of severity, a middle group between psychosis and neurosis, diagnostically linked to more severe personality disorders and shifting, unstable, or polysymptomatic presentations of axis I disorders. Importantly, it is defined by underlying psychologic structure, not by surface phenomenology. Accordingly, the rank ordering of criteria as most prototypical of this disorder in DSM-IV was not supported by the evidence. Further research is needed to establish whether some criteria should be given more emphasis than others [7]. The nine DSM-IV criteria of borderline personality disorder seem to indicate a statistically coherent construct. Because factor analyses have established both a one-factor model and a three factor model (disturbed relatedness, behavioural dysregulation, affective dysregulation), an underlying

multidimensional structure of borderline personality disorder consisting of three homogeneous components might exist [13]. The second meaning of borderline, “official” in current psychiatric nosology (DSM-III, DSM-III-R, and DSM-IV) is of a specific axis II cluster B disorder, one which encompasses many of the characteristics of the first meaning but particularly as they appear in histrionic personalities, and which is defined (as is standard in the DSM) by surface phenomenology rather than underlying psychological structure. Essentially all of the second type of borderline would be included in the first type. However, a number of the first type would be classified in the DSM axis II system as histrionic, narcissistic, antisocial, cluster A or C, or, quite often, not otherwise specified. Most American psychiatrists have a fuzzy notion, somewhere between the two, with the psychoanalytically oriented being closer to the first view and the others being closer to the second. A second theme in the literature on borderline personality concerns etiology. Once again there have been two distinct views. The first, popular among psychotherapists and many early psychoanalytic thinkers, emphasized early experience pre-oedipal and separation-individuation were common terms. Parental care had been unempathic, there had been traumatic experiences, the mother-child “match” was poor, etc. A second theme, popular among psychiatric researchers, emphasized constitutional factors genetic links to bipolar or affective disease, temperamental characteristics such as impulsivity or affective dysregulation, brain abnormalities, etc. Currently there have been several efforts to meld these two perspectives, as is occurring in other areas of psychiatry. Parents who may have little difficulty raising a temperamentally well-modulated infant may face major challenges with a dysregulated one, with unempathic and traumatic interactions resulting. Endogenous affective storms may interfere with the normal development of internalized object relations. In sum, development is complicated, always involves the interaction of nature and nurture, and although in extreme cases one or the other may predominate as the determinant of pathology, there is much more likely to be a complex interaction when the outcome is less extreme that is, borderline. Both the restricted, “surface” descriptive diagnosis of the borderline personality disorder, and the broader, “deep structure” psychodynamic concept present significant problems. The descriptive criteria of borderline personality disorder, in practice, present with a comorbidity with other severe personality disorders of approximately 60%, which points to underlying common personality features. The psychodynamic definition, originally based on hypotheses regarding common unconscious, early infancy- and childhood-derived conflicts, has defied efforts at precise clinical description, in addition to its lack of empirical research support. Clinically, it is undeniable that similar “surface” personality traits may correspond to different “deeper” psychological meanings: social timidity, for example, may be a reaction formation against exhibitionist trends, an expression of paranoid tendencies, or a schizoid symptom. The search for the relationship between surface features and underlying psychological or neurobiological structures lends itself to reductionist shortcuts that do not do justice to the complexity of psychopathological conditions. A major stumbling block for further progress in the construction of a borderline personality category is the temptation to consider personality disorders as reflecting either underlying neurobiological structures, or psychological structures disconnected from their neurobiological roots. In the case of borderline personality disorder, hypersensitivity to negative stimuli and excessive activation of negative affect, linked to hyperactivity of the amygdala and related structures of the limbic system, and, at the same time, a lack of the capacity for cognitive contextualization and affect control, linked to decreased functioning of the prefrontal and preorbital cortex and the anterior cingulate area, represent significant neurobiological correlates of this pathology. From this perspective, the descriptive symptoms of borderline personality disorder would express this pathology of brain systems and the consequential behavioral interactions with the environment under the influence of this pathology. From a psychodynamic viewpoint, the common features of borderline personality disorder, and of the severe personality disorders that are frequently comorbid with it, would be a lack of integration of the concept of self, caused by the lack of integration of self-representations and of object-representations under contradictory loving and hateful affect states. The patient’s subjective life, therefore, remains contradictory and chaotic, with severe identity problems, and a closely related incapacity to integrate the perception of significant others, thus motivating discontinuous, chaotic, contradictory social behavior. Both of these neurobiological and psychological structural assumptions correspond to clinical and empirical research data, but we still have to clarify how neurobiological disposition and structures relate to psychological development and its derived structures.

TREATMENT

Treatments for BPD have improved in recent years. Group and individual psychotherapy are at least partially effective for many patients. Within the past 15 years, a new psychosocial treatment termed dialectical behavior therapy (DBT) was developed specifically to treat BPD, and this technique has looked promising in treatment studies [16]. Pharmacological treatments are often prescribed based on specific target symptoms shown by the individual patient. Antidepressant drugs and mood stabilizers may be helpful for depressed and/or labile mood. Antipsychotic drugs may also be used when there are distortions in thinking [4]. The concept of borderline was

initially developed to explain a group of patients who had at first been seen as appropriate candidates for psychodynamic psychotherapy—troubled but not psychotic, and having a wide range of strong affects and intense relationships. However, they got worse rather than better in the unstructured settings of such therapy. This led to attempts to develop strategies for identifying such patients before they had entered psychotherapy psychological tests, structural interviews, and diagnostic criteria. These were accompanied by strategies for modifying “classical” psychoanalysis or psychoanalytic psychotherapy so that it might be more useful for these patients, including more active therapists, a greater focus on the patient-therapist relationship in the “here and now,” the utilization of countertransference responses to explore the relationship, educating patients to recognize their affective reactions and what triggers them, to connect actions with thoughts and feelings, both their own and others, and to regard behavior as motivated, reflecting intentions and desires. This has led not only to modifications in the treatment of borderline patients, but to a reconsideration of our technique of therapy with other patients as well. The polarity of neurobiological and psychodynamic viewpoints also permeates questions regarding alternative treatment strategies with borderline patients. The development of our knowledge regarding genetic and constitutional predisposition to excessive activation of negative affects, temperamental dispositions that influence early object relations, and the lack of adequate contextualization and control of primitive affects derived from inadequate prefrontal cortical functioning has stimulated the search for biological treatments directly influencing the activation and intensity of affect. The frequent development in borderline patients of characterologically based depression, rage attacks and affect storms in general, pervasive anxiety, and dissociative symptoms has stimulated the utilization of anxiolytic, antidepressant, and mood stabilizing drugs, and, more recently, the use of low-dose atypical neuroleptics. The most important finding, perhaps, has been that some borderline patients respond to one or another of a broad spectrum of medications, although only approximately 30% of these patients respond satisfactorily over an extended period of time. After many months of treatment, many patients who initially responded favorably to medications tend to experience a loss of the effectiveness of drugs, and the underlying structural predisposition to their affective symptomatology seems to override the effects of medication. It would seem that, at this time, the major role of medication is that of an auxiliary treatment tool in the context of a psychotherapeutic treatment. Recently we have seen the emergence of systematic studies of the efficacy and mechanism of action of several different psychotherapies with these patients [10]. These efforts are in their infancy, but it is already apparent that this kind of research is possible and that it has much to offer. Several of the treatments are effective and, interestingly, their patterns of specific effects may differ. This could lead to a rational strategy for prescribing optimal treatment for specific patients and to the development of new and improved treatments. It also serves as a model for psychotherapy research in general. Dialectic behavior therapy, a specific cognitive behavior therapy, has proven effective, and constitutes a major practical approach to the treatment of borderline patients, perhaps particularly those with prevalent suicidal and parasuicidal symptoms and affect dysregulation. Several psychodynamic psychotherapies also have been demonstrated to be effective, including transference-focused psychotherapy and mentalization-based psychotherapy. Early evidence indicates that they may operate by specific mechanisms that differ from each other. Biological and psychotherapeutic approaches probably affect different points in the chain of events that characterizes the psychopathology of borderline patients. A major shortcoming of present day research in the treatment of borderline personality is the limited time span of randomized, controlled, clinical trials, contrasting with the widespread clinical impression that long-term treatments are essential for these patients. A gradually emerging finding is that while the major symptoms that define borderline personality disorder in the description of the DSM or ICD respond relatively quickly to well-structured specific forms of cognitive behavior or psychodynamic psychotherapy, basic underlying chronic personality dispositions may remain unchanged. Borderline patients, 20 or 30 years after completion of treatment, still show impoverishment of their personality: a lack of effectiveness and satisfaction in their lives, in their work and professions, and a lack of stability in intimate love and sexual relationships, in establishing families, and difficulty overcoming social isolation. The focus on the long-range course of borderline psychopathology and the effect of interventions on modifying it constitute a major challenge for future research. The study of the effectiveness of treatment so far has focused mostly on the descriptive symptoms of the DSM and ICD classifications of borderline personality disorders, and much less on the subtle and permanent features of their difficulties in work, love, social life, and creativity. The present-day prevalent instruments for evaluating degrees of psychopathology and symptomatic change have not yet been geared to those fundamental aspects of personality functioning that determine the long-term satisfaction and effectiveness of a person’s life project. This is a major area, we believe, for future research. Finally, the relationships between clinical symptoms, deeper psychological structures, and underlying neurobiological systems are, as yet, to be explored. More subtle and precise relationships, for example, between affect processing by different brain systems and the development of psychological defensive operations to deal with conflictual affects will require the development of new research methods. As one illustration of these relationships that calls for further exploration, it appears that

there is no capacity in the amygdala to combine positive and negative affects, while at the level of the limbic-cortical brain area, the possibility of such integration and mutual toning down of contrasting affects in the context of cognitive integration exists. At the same time a key mechanism of change in psychodynamic psychotherapies may be related to the cognitive integration of mutually split-off internalized, affectively invested object relations. This cognitive integration, however, may only be effective in the context of affectively invested relations in the patient-therapist interaction. These two, psychological and neurofunctional processes of affect activation and modification, are presumably related. How to understand this relationship illustrates one of the many research questions in the present challenge to link neurobiological and psychodynamic research. Borderline patients have long been to psychiatry what psychiatry has been to medicine a subject of public health significance that is underrecognized, undertreated, underfunded and stigmatized by the larger discipline. As with psychiatry and medicine, this is changing [3]. New knowledge, new attitudes, and new resources promise new hope for persons with borderline personality. Although much has been learned about borderline personality disorder in recent years, several questions remain. Despite conceptual coherence, borderline personality disorder seems to be a heterogeneous diagnostic category that is less stable and distinct over time than expected. These findings raise questions of both how to conceptualise this disorder and how to implement it in future versions of DSM as a form of personality pathology that is both enduring and distinct from other personality disorders [5]. Furthermore, the discussion on whether a categorical or a dimensional model best suits personality disorders is ongoing [7]. The results of the Collaborative Longitudinal Personality Disorders Study (CLPS) suggest reconceptualising personality disorders as hybrids of stable personality traits and as intermittently expressed symptomatic behaviours that are attempts to cope with or defend against or compensate for these pathological traits (eg, self-harm to reduce affective tension). Further research is needed on the association between personality traits and personality disorder psychopathological changes as well as on the relation between personality disorders and personality functioning [21]. Personality might function differently at different ages and in response to different needs [18]. Future research on the causes of this disorder should investigate how genetic and psychosocial factors interact with neurotransmitter function to lead to cognitive and emotional regulations and specific traits [9].

REFERENCES

- [1] Bateman A, Fonagy P. *Am J Psychiatry*, **2001**, 158: 36-42.
- [2] Coccaro EF, Siever LJ. *Neurobiology*. American Psychiatric Publishing, **2005**, 155-69.
- [3] Davidson RJ, Jackson DC, Kalin NH. *Psychological Bulletin*, **2000**, 126 (6): 873-89.
- [4] Dulit, R. A., & Frances, A. J. *American Journal of Psychiatry*, **1990**, 147: 1002-1007.
- [5] Gardner DL, Cowdry RW. *Psychiatric Clinics of North America*, **1985**, 8(2): 389-403.
- [6] Grilo CM, Sanislow CA, Skodol AE, *Can J Psychiatry*, **2007**, 52: 357-62.
- [7] Hollander E, Tracy KA, Swann AC. *Neuropsychopharmacology*, **2003**, 28: 1186-97.
- [8] Koenigsberg HW, Harvey PD, Mitropoulou V. *Am J Psychiatry*, **2002**, 159: 784-88.
- [9] Koerner K, Linehan MM, *Psychiatric Clinics of North America*, **2000**, 23(1): 151-67.
- [10] Rinne T, van den Brink W, van Dyck R. *Am J Psychiatry*, **2002**, 159: 2048-54.
- [11] Sanislow CA, Little TD, Ansell EB. *J Abnorm Psychol*, **2009**, 118: 507-19.
- [12] Schmahl CG, Elzinga BM, Bremner JD. *Biol Psychiatry*, **2003**, 54: 142-51.
- [13] Siever LJ, Koenigsberg HW. *The Dana Forum on Brain Science*, **2000**, 2(4).
- [14] Skodol AE, Oldham JM, Bender DS, *Am J Psychiatry*, **2005**, 162: 1919-25.
- [15] Stiglmayr CE, Shapiro DA, Bohus M. *J Psychiatr Res*, **2001**, 35: 111-18.
- [16] Tebartz van Elst L, Hesslinger B, Thiel T. *Biol Psychiatry*, **2003**, 54: 163-71.
- [17] Tyrer P, Coombs N, Ibrahimi F, *Br J Psychiatry Suppl*, **2007**, 49: s51-59.
- [18] Warner MB, Morey LC, Finch JF, *J Abnorm Psychol*, **2004**, 113: 217-27.
- [19] Widiger TA. *Psychiatric Annals*, **2007**, 32: 93-99.
- [20] Zanarini MC, Frankenburg FRSilk KR. *Am J Psychiatry*, **2003**, 160: 274-83.
- [21] Zanarini MC. *Psychiatric Clinics of North America*, **2000**, 23(1): 89-101.