#### review article

## Overview on Borderline Personality Disorder

### **Abstract:**

Borderline Personality Disorder (BPD) is a psychiatric disease marked by unstable interpersonal relationships, fear of abandonment, difficulty regulating emotions, feelings of emptiness, persistent dysphoria or sadness, impulsivity, and increased risk-taking behaviors. The prevalence of borderline personality disorder has been reported at 11 percent in the psychiatric outpatient community and as high as 20% in the psychiatric inpatient population. Patients with BPD have a high rate of morbidity, which makes medical treatment more difficult. Although the role of genetics in BPD is unclear, inheritance of BPD appears to be considerable. Life events are also found to play a role in the development of BPD. The most important risk factor for the development of BP is childhood trauma. Symptoms that have been present since adolescence or early adulthood and manifest in a variety of settings are used to make the diagnosis. There are no lab or imaging tests available to aid in the diagnosis. Patients with borderline personality disorder benefit from three evidence-based treatments. Mentalizing-based therapy (MBT). Dialectical behavior therapy (DBT) and transference-focused psychotherapy (TFP). In this review we will be discussing epidemiology, etiology, clinical features diagnosis and treatment of BPD

### **Introduction:**

Borderline Personality Disorder (BPD) is a psychiatric disease marked by unstable interpersonal relationships, fear of abandonment, difficulty regulating emotions, feelings of emptiness, persistent dysphoria or sadness, impulsivity, and increased risk-taking behaviours. Dissociative experiences and paranoid ideation are also transitory symptoms of the condition. Furthermore, many BPD patients engage in self-injurious or suicidal behaviours on a regular basis. BPD affects around 6% of people at some point in their lives. BPD is significantly more frequent in professional settings, making it extremely important for health care workers and the general population. [1]

Borderline personality disorder is frequently diagnosed as comorbid with depression and anxiety, eating disorders such as bulimia, post-traumatic stress disorder (PTSD), substance misuse disorders, and bipolar disorder, either as a result of its location on the 'border' of other conditions or as a result of conceptual confusion (with which it is also sometimes clinically confused). There can be a lot of overlap with psychotic illnesses. People can have both visual and auditory hallucinations, as well as obvious delusions, in extreme situations, although these are generally short and connected to periods of high emotional instability. As a result, it may be differentiated from the fundamental symptoms of schizophrenia and other illnesses. [2]

Patients with BPD have a high rate of morbidity, which makes medical treatment more difficult. BPD was first described in 1978, and the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III) was published in 1980, followed by the International Classification of Diseases (ICD-10) ten years later. Emotionally unstable personality disorder has become a diagnosis based on the systematic identification of clinical characteristics. Affective instability was recognised as an important criteria for BPD in both the DSM-5 and the ICD-10.

# **Etiology And Pathogenesis:**

The development of "mistrustful inner working models" based on insecure attachment, according to etiological theories of BPD, predisposes to viewing others as untrustworthy and rejecting. Childhood trauma, such as emotional neglect or physical and sexual abuse, is a causal element in this development, however linking BPD with traumatic experiences alone is an oversimplification. Although the role of genetics in BPD is unclear, inheritance of BPD appears to be

considerable. When early adversity is combined with other factors, such as the emotional unresponsiveness of attachment figures, trauma, or abuse, an individual's expectations for future resource availability, including the quality of interpersonal relationships in terms of others' reliability and trustworthiness, are formed. [1,4-11]

Despite the fact that studies are few and results vary, there is at least considerable evidence for the genetic transmission and heredity of BPD. The concordance rate for BPD was shown to be greater in monozygotic twins than in dizygotic twins in two investigations (36 and 35 percent versus 19 and 7 percent). However, a third twin research found that, when compared to environmental factors, a shared genetic effect had minimal impact on the development of BPD (42 percent versus 58 percent). In summary, BPD is caused by a genetic tendency to emotional dysregulation combined with a non-supportive environment. Future research should concentrate on the connections between specific endophenotypes and environmental variables.[3]

Personality disorders are caused by temperamental (inborn or heritable) qualities that manifest in maturity as stable personality traits: mind, emotions, and behaviour patterns that distinguish individuals and remain stable over time. In nearly every characteristic investigated, heritable variables account for roughly half of the variation. Emotive instability and impulsivity, in particular, have a significant heritable component, and research involving twins have shown that BPD has a comparable genetic effect. in addition, family history studies have revealed that impulsive disorders such as antisocial personality disorder and drug misuse are more frequent in first-degree relatives of BPD patients. Impulsive characteristics, a prominent component of BPD, have been linked to abnormalities in central serotonergic functioning, according to studies of core neurotransmitter activity. The biochemical connections of emotional instability, on the other hand, remain unclear, and no particular indicators for the condition have been found. [12-18]

Life events are also found to play a role in the development of BPD. The most important risk factor for the development of BP is childhood trauma. This link between childhood trauma and BPD is not obvious since childhood trauma is not always present in BPD and patients who have experienced trauma do not always acquire BPD. Childhood trauma does not appear to be a necessary step in the development of BPD. Sexual abuse, physical abuse and neglect, verbal abuse, and early parental separation or bereavement have all been linked to childhood trauma

in BPD patients in prospective studies. More physically abused and/or neglected children matched the criteria for BPD as adults, according to a prospective study of 500 people. Surprisingly, a history of sexual abuse is not proven to be a risk factor for BPD. Having a parent with alcohol or substance abuse issues, as well as a diagnosis of drug misuse, major depressive disorder, and post-traumatic stress disorder, have all been linked to the development of BPD, although these are all non-specific variables. Another prospective, longitudinal study including 639 children found that childhood abuse/neglect was linked to BPD in adulthood. Meta-analyses have also discovered that the connection between the development of BPD and childhood abuse has very modest impact effects. While no one cause can explain the development of BPD, like with other mental illnesses, several variables can assist in understanding the disorder's development. Although other studies have found that childhood trauma does not play a substantial role in the development of BPD, it is still a significant risk factor for BPD, and further research is needed to fully understand this connection. [3,19-25]

## **Epidemiology:**

The lifetime prevalence of BPD is estimated to be 5.9%, with a point prevalence of 1.6 percent. Although BPD is not as common as other personality disorders in the general community, it is common in treatment settings; studies in clinical settings found that BPD was present in 6.4 percent of primary care visits, 9.3 percent of psychiatric outpatients, and 20% of psychiatric inpatients. In the clinical community, however, the ratio of girls to males with the illness is also higher. In clinical settings described in the DSM-5, the ratio is 3:1. In contrast to the clinical setting ratio, the lifetime prevalence of BPD was found to be comparable in males and females in two epidemiologic studies of the general population in the United States. This finding suggests that women with BPD are more likely than males to seek therapy. Women make up around 80% of patients treated for BPD, according to reports. [3,26-30]

The prevalence of borderline personality disorder has been reported at 11 percent in the psychiatric outpatient community and as high as 20% in the psychiatric inpatient population. Several research looking at the link between ethnicity and borderline personality disorder have come up with conflicting conclusions. [31]

## **Clinical Features and diagnosis:**

Symptoms that have been present since adolescence or early adulthood and manifest in a variety of settings are used to make the diagnosis. There are no lab or

imaging tests available to aid in the diagnosis. A variety of organised and semistructured interviews can help in diagnosis, but they frequently need specific training to conduct. The Diagnostic Interview for Borderlines – Revised is a widely used and validated tool that is often referred to as the "gold standard," but it can take 30–60 minutes to administer. In the last decade, several self-report measures have been created, although they are rarely utilised in clinical practise. The Mood Disorder Questionnaire, a widely used self-report questionnaire for mood disorders, frequently misdiagnoses borderline personality disorder as bipolar disorder.

Borderline personality disorder diagnostic criteria: [32]

A chronic pattern of interpersonal connection instability, self-image and affective instability, and significant impulsivity that begins in early adulthood and manifests itself in a variety of situations, as indicated by 5 (or more) of the following:

- frantic attempts to prevent desertion, real or imagined Note: Suicidal or self-mutilating behaviour is not covered by criteria.
- A pattern of insecure and intense interpersonal connections marked by fluctuations between idealisation and depreciation.
- Identity disruption is defined as a self-image or feeling of self that is significantly and consistently unsettled.
- At least two categories of impulsivity that are potentially self-destructive (e.g., spending, sex, substance abuse, reckless driving, binge eating). Note: Suicidal or self-mutilating behaviour is not covered by criteria 5.
- Suicidal behaviour, gestures or threats, or self-mutilation on a regular basis.
- Affective instability caused by a high level of mood reactivity (e.g., intense episodic dysphoria, irritability or anxiety usually lasting a few hours and only rarely more than a few days).
- Feelings of emptiness on a regular basis.
- Anger that is inappropriate, strong, or difficult to manage (e.g., frequent displays of temper, constant anger, recurrent physical fights).
- Paranoid ideation or severe dissociation symptoms caused by stress.

**Neuropsychology:** One of the signatures of BPD is patients' inability to regulate their emotions appropriately, which may explain a variety of symptoms such as idealisation and demonization of others, impulsivity, and risk-taking behaviour. These symptoms and indications might be thought of as behavioral manifestations of high stress reactivity. According to the Adaptive Calibration Model, high stress

sensitivity encourages a quick LHS in risky and uncertain situations by increasing threat alertness and down-regulating social feedback sensitivity. Several studies have found abnormalities in the hypothalamic–pituitary–adrenal stress axis in BPD, which correspond with symptom severity and a history of childhood trauma, supporting this theory. Early trauma has been linked to long-term variations in stress reactivity, potentially through epigenetic processes. In a manner similar, research into emotion perception shows that individuals with BPD had increased awareness or avoided unpleasant feelings like fear and rage. [1]

Patients with BPD are frequently 'alexithymic,' that is, they have difficulty reflecting on their own and others' feelings, and alexithymia has been linked to stress intolerance and impulsivity in BPD. However, given LHS's early difficulties, this apparent "empathy contradiction" is logical. Patients with BPD, according to Linehan, may be hypersensitive to emotional signals that might suggest rejection or abandonment. When overactivation of the attachment system interacts with problems in emotion control, such altered emotion perception has an influence on social interaction. Partly as a way of self-protection against continued traumatization by an abusive caregiver, overactivation of the attachment system leads to a functional down-regulation of mentalizing capacities. As a result, hypersensitivity to unpleasant emotions may contribute to skewed opinions of others, leading to a general perception of people as untrustworthy. Seeing others as untrustworthy and uncooperative, on the other hand, may boost one's own (unconscious) opportunistic attitude toward short-term resource exploitation. [1]

The most specific and sensitive criterion for BPD has been shown to be affective instability. Patients with BPD are emotionally unstable, respond quickly, and experience dysphoric feelings including sadness, anxiety, and irritability. A research that looked at the connection between age and emotional instability in BPD patients found an inverse relationship between age and affective instability in BPD patients. BPD patients have tumultuous and complicated relationships. They have a tendency to see others as both good and terrible, which is referred to as "splitting." They can quickly become reliant on others, yet their attitudes toward others can also change dramatically. A meta-analysis found that BPD patients performed worse on measures of attention, cognitive flexibility, planning, learning, and memory. [3]

**NEUROIMAGING:** There is a lot of evidence that childhood abuse is linked to reduced volume in the limbic system and the corpus callosum, and that impulsivity

in BPD is linked to changes in blood flow in frontal cortical regions. While this review cannot cover all relevant neuroimaging results in BPD, one essential point to consider when interpreting neuroimaging data is the belief that changes in brain metabolism or structure are not always indicative of poor functioning. According to Teicher et al., early environmental stress, such as that experienced as a result of childhood neglect or abuse, is not only harmful to the brain, but also to the body. As a result, (normal) brain growth is inhibited. Instead, 'significant stressors during a vulnerable developmental period cause the brain to develop along a stressresponsive pathway,' resulting in 'a cascade of stress responses that organises the brain to develop along a specific pathway selected to facilitate reproductive success and survival in a world of deprivation and strife.' This fundamentally different interpretation of structural and functional brain imaging findings is fully consistent with the Adaptive Calibration Model, which states that early experiences not only shape the psychological development of inner working models and how individuals adapt their LHS to their predictions of future resource availability, but also that early experiences leave a mark on how individuals adapt their LHS to their predictions of future resource availability (i.e. inner working model). This implies that in the case of BPD, changes in limbic anatomy may actually favour a rapid LHS. [1]

Bilateral decreases in the hippocampus, amygdala, and medial temporal lobe have been observed in neuroimaging studies comparing BPD patients to healthy controls. The top-down control delivered by the orbitofrontal cortex and anterior cingulate cortex, as well as the bottom-up control drives created in the limbic system, such as the amygdala, hippocampus, and insular cortex, are anomalies in the neurobiology of BPD. Cognitive control regions are delivered by top-down control, whereas salience detection is delivered by bottom-up control. [3]

## Management

The range of pharmacologic therapy for BPD remains restricted. The end outcome is a moderate degree of symptom alleviation for the most part. Low-dose atypical neuroleptics, selective serotonin reuptake inhibitors, and mood stabilisers are all effective in treating impulsive symptoms. Antidepressants, on the other hand, are considerably less helpful in treating mood symptoms in BPD patients than in individuals who do not have a personality disorder. Benzodiazepines are ineffective in treating BPD and are subject to misuse. Psychotherapy remains the cornerstone of BPD treatment. Dialectical behaviour therapy is a type of cognitive

behavioural therapy that focuses on emotional instability and impulsivity, teaching patients how to control their emotions via group and individual sessions. Within a year, this type of behaviour treatment has been proven to be successful in reducing suicide behaviour. However, it is uncertain if this approach will be beneficial in the long run. A randomized controlled study found that using a modified version of psychoanalytic therapy in a day-treatment environment that also incorporates cognitive methods is effective. [12]

Patients with borderline personality disorder benefit from three evidence-based treatments. First, mentalizing-based therapy (MBT) helps patients control emotion dysregulation by making them feel understood, which allows them to be more inquiring and make fewer assumptions about the intentions of others. Dialectical behaviour therapy (DBT), a second treatment option, combines mindfulness practises with concrete interpersonal and emotion regulation skills. Third, transference-focused psychotherapy (TFP) focuses on developing the patient's awareness of problematic interpersonal relationships through the patient-therapist interaction. Individual and group treatment are combined in MBT and DBT over the course of 12 to 18 months. Family therapy can sometimes, but not always, be a good replacement for group therapy for teenagers. [31]

Borderline personality disorder patients have unique therapy problems due to self-injurious conduct, boundary concerns, and frequent suicide threats. Treatment for borderline personality disorder patients may be complicated by high rates of chronic drug misuse. Patients with borderline personality disorder do not usually need to be admitted to a hospital; but, in some cases, inpatient care may be necessary, such as: [31]

- Due to overt suicide thoughts or impulsivity, a significant risk of high lethality behaviours is imminent.
- Severe social stresses that result in strong negative thoughts or a brief psychotic episode
- The intensity of self-injurious conduct that is rapidly increasing.
- Comorbid psychiatric diagnosis or severe substance abuse decompensation

### **Conclusion:**

Borderline Personality Disorder (BPD) is a serious psychiatric disease, with high prevalence in both inpatient and outpatient psychiatric clinics. Diagnosis of the disease depends on Borderline personality disorder diagnostic criteria rather than any imaging or laboratory testing method. Treatment of the disease is also largely psychological with pharmacological treatment has moderate to little effect.



### **References:**

- 1. Brüne M. Borderline Personality Disorder: Why 'fast and furious'? Evol Med Public Health. 2016 Feb 28;2016(1):52-66. doi: 10.1093/emph/eow002. PMID: 26929090; PMCID: PMC4782519.
- National Collaborating Centre for Mental Health (UK). Borderline Personality Disorder: Treatment and Management. Leicester (UK): British Psychological Society; 2009. (NICE Clinical Guidelines, No. 78.) 2, BORDERLINE PERSONALITY DISORDER. Available from: https://www.ncbi.nlm.nih.gov/books/NBK55415/
- 3. Kulacaoglu F, Kose S. Borderline Personality Disorder (BPD): In the Midst of Vulnerability, Chaos, and Awe. Brain Sci. 2018 Nov 18;8(11):201. doi: 10.3390/brainsci8110201. PMID: 30453675; PMCID: PMC6266914.
- 4. Bowlby J. Attachment and Loss, Vol. 1. Attachment. New York: Basic Books.
- 5. Agrawal HR, Gunderson J, Holmes B. et al. Attachment studies with borderline patients: a review. Harv Rev Psychiatry 2004;12:94–104.
- 6. Fonagy P, Target M, Gergely G. Attachment and borderline personality disorder. A theory and some evidence. Psychiatr Clin North Am 2000;23:103.
- 7. Zweig-Frank H, Paris J. Parents' emotional neglect and overprotection according to the recollections of patients with borderline personality disorder. Am J Psychiatry 1991;148:648–51.
- 8. Bierer LM, Yehuda R, Schmeidler J. et al. Abuse and neglect in childhood: relationship to personality disorder diagnoses. CNS Spectrum 2003;8:737–54.
- 9. Paris J, Zweig-Frank H. A critical review of the role of childhood sexual abuse in the etiology of borderline personality disorder. Can J Psychiatry 1992:37:125–8.
- 10.Amad A, Ramoz N, Jardri R. et al. Genetics of borderline personality disorder: systematic review and proposal of an integrative model. Neurosci Biobehav Rev 2014;40:6–19.
- 11.Few LR, Miller JD, Grand JD. et al. Trait-based assessment of borderline personality disorder using the NEO five-factor inventory: phenotypic and genetic support. Psychol Assess 2015;28:39–50.
- 12.Paris J. Borderline personality disorder. CMAJ. 2005 Jun 7;172(12):1579-83. doi: 10.1503/cmaj.045281. PMID: 15939918; PMCID: PMC558173.

- 13.Rutter M. Temperament, personality, and personality development. Br J Psychiatry 1987;150:443-8.
- 14.Livesley WJ, Jang KL, Vernon PA. Phenotypic and genetic structure of traits delineating personality disorder. Arch Gen Psychiatry 1998;55:941-94.
- 15. Hinshaw SP. Impulsivity, emotion regulation, and developmental psychopathology: specificity versus generality of linkages. Ann N Y Acad Sci 2003;1008:149-59.
- 16. Torgersen S, Lygren S, Oien PA, Skre I, Onstad S, Edvardsen J, et al. A twin study of personality disorders. Compr Psychiatry 2000;41:416-25.
- 17. White CN, Gunderson JG, Zanarini MC, Hudson JI. Family studies of borderline personality disorder: a review. Harv Rev Psychiatry 2003;11:8-19.
- 18.Gurvits IG, Koenigsberg HW, Siever LJ. Neurotransmitter dysfunction in patients with borderline personality disorder. Psychiatr Clin North Am 2000;23:27-40.
- 19. Zanarini M.C., Williams A.A., Lewis R.E., Reich R.B., Vera S.C., Marino M.F., Levin A., Yong L., Frankenburg F.R. Reported pathological childhood experiences associated with the development of borderline personality disorder. Am. J. Psychiatry. 1997;154:1101–1106.
- 20.Hengartner M.P., Ajdacic-Gross V., Rodgers S., Muller M., Rossler W. Childhood adversity in association with personality disorder dimensions: New findings in an old debate. Eur. Psychiatry. 2013;28:476–482. doi: 10.1016/j.eurpsy.2013.04.004.
- 21. Johnson J.G., Cohen P., Gould M.S., Kasen S., Brown J., Brook J.S. Childhood adversities, interpersonal difficulties, and risk for suicide attempts during late adolescence and early adulthood. Arch. Gen. Psychiatry. 2002;59:741–749. doi: 10.1001/archpsyc.59.8.741.
- 22. Widom C.S., Czaja S.J., Bentley T., Johnson M.S. A prospective investigation of physical health outcomes in abused and neglected children: New findings from a 30-year follow-up. Am. J. Public Health. 2012;102:1135–1144. doi: 10.2105/AJPH.2011.300636.
- 23. Johnson J.G., Cohen P., Brown J., Smailes E.M., Bernstein D.P. Childhood maltreatment increases risk for personality disorders during early adulthood. Arch. Gen. Psychiatry. 1999;56:600–606. doi: 10.1001/archpsyc.56.7.600.
- 24. Fossati A., Madeddu F., Maffei C. Borderline Personality Disorder and childhood sexual abuse: A meta-analytic study. J. Pers. Disord. 1999;13:268–280. doi: 10.1521/pedi.1999.13.3.268.

- 25. Paris J. Childhood trauma as an etiological factor in the personality disorders. J. Pers. Disord. 1997;11:34–49. doi: 10.1521/pedi.1997.11.1.34.
- 26. American Psychiatric Association . Diagnostic and Statistical Manuel of Mental Disorders. 5th ed. American Psychiatric Association; Arlington, VA, USA: 2013.
- 27.Lenzenweger M.F., Lane M.C., Loranger A.W., Kessler R.C. DSM-IV personality disorders in the National Comorbidity Survey Replication. Biol. Psychiatry. 2007;62:553–564. doi: 10.1016/j.biopsych.2006.09.019.
- 28.Grant B.F., Chou S.P., Goldstein R.B., Huang B., Stinson F.S., Saha T.D., Smith S.M., Dawson D.A., Pulay A.J., Pickering R.P., et al. Prevalence, correlates, disability, and comorbidity of DSM-IV borderline personality disorder: Results from the Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions. J. Clin. Psychiatry. 2008;69:533–545. doi: 10.4088/JCP.v69n0404.
- 29.Gross R., Olfson M., Gameroff M., Shea S., Feder A., Fuentes M., Lantigua R., Weissman M.M. Borderline personality disorder in primary care. Arch. Intern. Med. 2002;162:53–60. doi: 10.1001/archinte.162.1.53.
- 30.Zimmerman M., Rothschild L., Chelminski I. The prevalence of DSM-IV personality disorders in psychiatric outpatients. Am. J. Psychiatry. 2005;162:1911–1918. doi: 10.1176/appi.ajp.162.10.1911.
- 31. Chapman J, Jamil RT, Fleisher C. Borderline Personality Disorder. [Updated 2021 Aug 1]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan-. Available from: <a href="https://www.ncbi.nlm.nih.gov/books/NBK430883/">https://www.ncbi.nlm.nih.gov/books/NBK430883/</a>
- 32.Biskin RS, Paris J. Diagnosing borderline personality disorder. CMAJ. 2012 Nov 6;184(16):1789-94. doi: 10.1503/cmaj.090618. Epub 2012 Sep 17. PMID: 22988153; PMCID: PMC3494330.