IJHSR
ijhighschoolresearch.org
REVIEW ARTICLE

Neurological and Cognitive Impacts of Cluster B Personality Disorders

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ABSTRACT: Some emerging evidence suggests that the instability and impulsive behaviors seen in Cluster B personality disorders may not only stem from structural abnormalities in the brain but also from underlying neurochemical and metabolic dysfunctions. Malfunctions in glucose metabolism and disruptions of the serotonin system have also been observed in certain regions that are critical for control over emotions. Some examples are the amygdala and orbitofrontal cortex, highlighting the biological basis for disturbances in emotional control and impulsive decision-making. This literature review explores how metabolic and neurochemical dysfunctions in affective brain regions contribute to emotional dysregulation and impulsivity in individuals with Cluster B personality disorders. Based on the previous literature, Cluster B personality disorders are characterized by consistent metabolic and functional abnormalities, specifically in regions responsible for emotional regulation, such as the amygdala and orbitofrontal cortex. The dysfunctions are responsible for the key symptoms such as emotional instability and impulsivity, and their identification through the studies of images provides a means to justify more targeted and biologically informed psychiatric treatment in the future. Understanding the relationship between the metabolic and neurofunctional bases of Cluster B personality disorders could not only provide the possibility of more efficient treatment in the future, but could also provide possible prevention by treating the issue during development and the early stages.

KEYWORDS: Cluster B Personality, Metabolism, Impulse, Orbitofrontal Cortex, Amygdala, PET. fMRI, Dysfunction, Psychiatric.

Introduction

Personality disorders (PD) are often misunderstood as irrational behavior, a bad attitude, or anger management issues. Especially for adolescents, parents often do not understand the core problem behind certain types of misbehavior. New data has shown that irrational behavior and emotional instability may arise from measurable biological factors. [1]

Cluster B personality disorders are often described as showcasing dramatic and erratic behavior. This includes borderline personality disorder (BPD), antisocial personality disorder (ASPD), narcissistic personality disorder (NPD), and histrionic personality disorder (HPD), which are known for emotional outbursts, impulsive decision-making, irrational anger, and unstable relationships. While these symptoms have often been attributed solely to upbringing or trauma, modern neuroimaging and psychiatric research suggest there are also significant biological and neurological contributions.[2] Silbersweig *et al.* conducted a brain scan and have proven that patients with Cluster B PDs commonly have unusual brain activity in areas that control emotions, such as the amygdala and prefrontal cortex.[3]

The main methods in this field of research are positron emission tomography (PET) and functional magnetic resonance imaging (fMRI). They are used to examine brain metabolism and activity patterns in individuals with Cluster B personality disorders. PET scans have revealed abnormalities in glucose metabolism in the orbitofrontal cortex and amygdala, which are two key regions in emotional regulation and impulse control. On the other hand, fMRI studies demonstrated functional connectivity in the front-limbic circuit.

The research paper demonstrates the findings from imaging studies from PET and fMRI, together with neurochemical and psychiatric findings, to identify the metabolic and functional patterns and consistency underlying the affective symptoms within Cluster B personality disorders. Previous studies have suggested that Cluster B personality disorders are defined by metabolic and functional abnormalities specifically in regions responsible for emotional regulation, such as the amygdala and orbitofrontal cortex. Understanding the neural mechanisms behind Cluster B personality disorders could help with early onset treatment and diagnosis that could stabilize or prevent the symptoms from emerging entirely. This literature review investigates cognitive and affective dysregulation of cluster B personality disorders, the neurobiology and metabolism of BPD, as well as the treatment evolution and efficacy over time.

Discussion

Cognitive and affective dysregulation in Cluster B personality disorders:

Patients with the most common Cluster B disorder, Borderline personality disorder (BPD), often exhibit alterations in brain structure. This helps explain their emotional instability and impulsive behavior. For example, a study conducted with high-resolution MRI scans showed that individuals with BPD have increased gray matter concentration in the amygdala, which is the part of the brain responsible for strong emotion-

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DOI: 10.36838/IJHSR89.169

al responses.[4, 5] At the same time, patients also present with signs of decreased gray matter in the anterior cingulate cortex (ACC), which is the part of the brain that helps to regulate control over emotions and stress.[2] In simpler terms, their emotional "accelerator" (the amygdala) may be overactive, while their "brakes" (the ACC) are underactive. The imbalance contributes to the intense bipolar mood swings and rapid emotional reactions, and the difficulty for patients to calm down.

Another study, which had ten unmedicated BPD subjects and twenty healthy controls, found that BPD subjects had comparably smaller left and right hippocampal volumes. Early childhood trauma contributes to the atrophy of the hippocampal area, while substance abuse may enlarge the putamen.[24] The implication of a hippocampal volume reduction is unclear. It may cause short-term memory loss and neurocognitive deficits, perhaps including the ability to process the memory of the childhood trauma.[24] The differences in the structures help us to understand that emotional dysregulation for these disorders is not only psychological but also has a biological basis in the brain. [6] Therefore, it is crucial to explore neurobiology and the metabolism linked to irregular brain activity within patients with Cluster B personality disorders.

Neurobiology and metabolism:

Several clinical studies have discovered that people with BPD often have abnormal brain metabolism patterns in areas that control emotion and behavior. During a PET study comparing depressed individuals with and without BPD, researchers found that individuals with BPD showed reduced glucose metabolism at rest in regions responsible for emotional regulation and impulse control.[7]

The levels of impulsive emotions were linked to increased glucose utilization in the frontal cortex.[8] Review studies have revealed that many people with BPD show symptoms of reduced glucose activity called hypometabolism in the prefrontal cortex and ACC, alongside hypermetabolism, an increase in glucose activity in regions like the insula, cerebellum, hypothalamus, and motor cortex.[9] These glucose metabolism patterns suggest that emotional instability and impulsive behavior displayed in Cluster B PDs could possibly be fueled by an irregular use of brain energy. It has been shown that there is reduced activity in control regions and increased activity in areas that generate emotional or automatic responses.

This connection does not function properly after emotionally taxing tasks. This might explain why patients have problems trying to calm down or think rationally during emotional situations. Other neuroimaging research has also shown that individuals with BPD often exhibit increased connectivity between the insula and self-referential brain networks. while displaying weaker connections in regions related to attention and memory. These patterns have been linked to impulsivity, emotional confusion, and episodes of dissociation.[10]

In addition to these connectivity differences, studies have highlighted the important neurochemical imbalances that affect brain activity. Reduced serotonin activity has been discovered to be associated with impulsive behavior and aggression,[11] while altered dopamine signaling may increase emotional sensitivity and cause paranoia.[12] Overall, these findings demonstrate that both disrupted neural connectivity and glucose, dopamine, and serotonin imbalances play a central role in the emotional dysregulation characteristic of Cluster B personality disorders. Understanding the mechanisms behind a patient's actions will further help to develop advanced treatment.

Treatment evolution and efficacy over time:

There are several distinct phases for the treatment of Cluster B personality disorders. Each level of understanding about the conditions at the time yields its own best choice of treatment. In the early 20th century, the mainstream approach was psychoanalysis and long-term institutional care. Psychoanalytic treatment aimed to uncover unconscious conflicts through extended talk therapy. Meanwhile, institutionalization was also commonly used to keep patients who are in severely depressed or hypomanic states, to keep them from harming themselves or others. Overall, the previous methods listed were ineffective in addressing the severe emotional instability and impulsivity that are presented as symptoms of the disorder. Furthermore, treatment from the past may also be inhumane, reinforcing stigma and sometimes causing further trauma for patients.[13]

Medication became the most important part by the second half of the 20th century. Drugs such as antidepressants, mood stabilizers, and antipsychotics were widely prescribed.[14] The medication could reduce certain symptoms such as mood swings, anxiety, and aggression, improving the patient's quality of life. However, the medication does not directly address the deep-seated emotional dysregulation issues. As a result, while symptoms became less severe, the core difficulties often persisted.[14] Nowadays. Medication is still mainly used as a supportive measure rather than a cure. In the past few decades, more research has led to the development of evidence-based psychotherapies that more directly target the symptoms and behavior of Cluster B disorders.

For borderline personality disorder (BPD) in particular, there are three main empirically supported psychotherapies. Dialectical Behavioral Therapy (DBT) combines cognitive-behavioral techniques with mindfulness training. It teaches emotional regulation, distress tolerance, and interpersonal effectiveness, showing strong results in reducing self-harm and emotional instability.[15] Mentalization-Based Therapy (MBT) helps patients improve awareness of their own mental states and those of others, fostering empathy and more stable relationships.[16] Transference-Focused Psychotherapy (TFP) focuses on identifying and restructuring dysfunctional relationship patterns within the therapeutic setting, promoting healthier interpersonal functioning.[17]

These modern therapies have been consistently shown to be more effective than earlier psychoanalytic or purely pharmacological approaches, and they represent a significant step forward in addressing the core issue of Cluster B personality disorders. Especially for BPD, Mentalization-based (MBT) has been shown to decrease suicidality and be one of the most effective therapies listed.[22] A trial compared treatment as usual to DBT, and it had reduced the rates of overdosing and hospi-

talization.[20,21] The efficacy of TFP is roughly equivalent to that of DBT.[23] These three modern behavioral therapy approaches have been shown to be more effective than treatment as usual. Knowing which brain regions and chemical systems are involved during the emotional situation can help doctors and therapists design improvised therapies based on each symptom, which could be used to design more targeted therapies, making treatment more efficient and individualized.[16]

Conclusion

The findings from imaging studies from PET and fMRI, together with neurochemical and psychiatric findings, demonstrated the metabolic and functional patterns and consistency underlying the affective symptoms within Cluster B personality disorders. Cluster B personality disorders are characterized by consistent metabolic and functional abnormalities, specifically in regions responsible for emotional regulation, such as the amygdala and orbitofrontal cortex. The dysfunctions are responsible for the key symptoms such as emotional instability and impulsivity, and their identification through the studies of images provides a means to justify more targeted and biologically informed psychiatric treatment in the future. Understanding the relationship between the metabolic and neurofunctional basis of Cluster B personality disorders could not only provide the possibility of more efficient treatment in the future, but could also provide possible prevention by treating the issue during development and the early stages. This could stabilize or prevent the symptom from showing up, or decrease the severity at a later age.

Across multiple studies, there is a strong agreement that Cluster B personality disorders, especially in the case of BPD, involve both structural and functional brain differences from those of regular brains. Many neuroimaging studies have reported reduced activity and structural alterations in the prefrontal cortex, a region critical for impulse control and decision-making[3,4] The researches also consistently shows evidence of altered connectivity between these regions, supporting the idea that emotional instability is linked to "emotional brakes" and "emotional accelerators" not functioning properly and coordinately.1 However, there are also disagreements in the discussions of the studies. Some report hyperactivity in certain brain regions, while others find hypoactivity. It depends on the type of method used or type of emotional state being studied.[2] Difference in research details, such as whether participants were on medication, the type of imaging techniques used, and different specific subgroups of patients, may explain the inconsistencies between the studies and results. There are various research findings on which neurotransmitter systems, such as serotonin or dopamine, play the most central role in the symptoms.

While current research has made progress in identifying biological patterns in Cluster B personality disorders, there are still important gaps in the literature. First, most studies rely on small sample sizes, limiting the generalizability of their findings.[1] Second, there is also a lack of long-term longitudinal research tracking how brain structure and function change over time in response to treatment. Most imaging studies provide only snapshots rather than developmental trajectories, making it difficult to determine whether observed abnormalities are causes, consequences, or adaptations to symptoms.[18] Another gap is the limited amount of research comparing different subtypes of Cluster B disorders. Much of the literature focuses on borderline personality disorder, while narcissistic, antisocial, and histrionic personality disorders are studied far less frequently. Direct comparisons within the same study could clarify both the shared and unique neurobiological features of these disorders.[16]

Clinical research into the neurobiology of Cluster B personality disorders plays an important role in improving treatment. In the future, research that could identify reduced prefrontal activity may inspire new interventions that aim to enhance decision-making and impulse control. This literature review supports the idea that PDs can be diagnosed as a biological condition rather than being treated solely as a result of early childhood trauma or a psychological issue. This discovery can shift the focus from the "bad temperament" to understanding what happens on a neurobiological level when the outbursts happen.[19]

From an ethical and social perspective, this research is important for showing biological evidence for these disorders. It's possible to reduce the stigma that surrounds personality disorders, and that is a big step towards awareness for those struggling with all these symptoms. The presented literature helps the public understand that PDs involve measurable change in the brain, allowing the disorder to be treated as a biologically rooted condition, and makes it easier to explain the condition and advocate for proper care.[13] In the future, the more study and research that goes into this field, the more we can demonstrate better knowledge of it, making way for better education and less judgment for those affected by PDs.

Acknowledgments

I would like to express my sincere gratitude to my advisor, Fiona De Visser.

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