Pseudobulbar Affect
An Overview
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ABSTRACT
Pseudobulbar affect (PBA) is a disorder that develops in the context of a brain injury or underlying neurological dysfunction. It is characterized as an affective disorder of emotional expression. PBA manifests as frequent uncontrollable outbursts of laughing or crying, incongruent with the individual’s internal emotional state. It can be challenging for clinicians to differentiate PBA from mood disorders, contributing to its high rate of misdiagnosis. This lack of recognition leads to ineffective and insufficient treatment, impacting patients’ quality of life. The current article provides an overview of PBA, including its history, prevalence, pathophysiology, diagnostic criteria, clinical implications, and treatment. [Journal of Psychosocial Nursing and Mental Health Services, 58(9), 19–24.]

Pseudobulbar affect (PBA), a neurological syndrome primarily affecting individuals with neurological disease, can be socially debilitating. Characterized by affective lability, emotional deregulation, and sudden involuntary emotional expression, episodes comprise involuntary, uncontrollable, inappropriate, and exaggerated laughing or crying incongruent with the individuals’ emotional state (King & Reiss, 2013; Sauvé, 2016). PBA develops in the context of a brain injury or underlying neurological dysfunction and stems from damage to neural circuits in the brainstem responsible for emotional expression (Hammond et al., 2016). Conditions associated with PBA include traumatic brain injury, stroke, multiple sclerosis, amyotrophic lateral sclerosis (ALS; Lou Gehrig’s disease), Parkinson’s disease, and other neurodegenerative disorders including Alzheimer’s disease (Girotra et al., 2018; Luhoway et al., 2019).
Due to PBA’s core neuropsychopathy, differentiating PBA from other common mood disorders, such as depression, anxiety, and bipolar disorder, can be challenging for clinicians, contributing to its high rate of misdiagnosis and leading to ineffective and insufficient treatment. The social impact of emotional outbursts associated with PBA can be distressing and can degrade quality of life significantly; therefore, it is important for clinicians to identify PBA and prescribe appropriate treatment. The aim of the current article is to provide an overview of PBA, including its prevalence, neurobiology and psychopathology, diagnostic criteria, and current treatment strategies.

HISTORY OF PSEUDOBULBAR AFFECT TERMINOLOGY

Naturalist Charles Darwin referred to involuntary emotional expression more than a century ago in his 1872 book, *The Expression of the Emotions in Man and Animals*, writing, “certain brain diseases, such as hemiplegia, brain-wasting, and senile decay, have a special tendency to induce weeping” (Darwin, 1872, p. 156, as cited in Lochhead et al., 2018, para. 2; Pinkowish, 2020, para. 1). In 1911, Oppenheim coined the term *pseudo-bulbar affect* to describe “spasmodic explosive bursts of laughter or weeping,” and various other terms have been used to describe its symptoms (Brooks et al., 2013, para. 6).

The terminology used to characterize PBA includes: pathological crying and laughing, pseudobulbar affect, emotional lability, involuntary emotional expression disorder, emotional incontinence, emotionalism, excessive emotionality, and others (Brooks et al., 2013; Luhoway et al., 2019). Currently, PBA is the unifying and medically accurate term worldwide. Throughout the years, the array of nomenclature used to describe PBA has led to confusion, a paucity of diagnostic standards, and lack of clinical guidelines.

The exact cause of PBA is unknown; its name derives from two ancient Greek terms. *Pseude*, meaning “false,” describes the incongruence between the patient’s affect and internal emotional states. *Bulbar* refers to the brain stem because PBA is associated with symptoms similar to those caused by lesions on the medulla oblongata or a loss of certain brain regions, such as the brain stem andpons. However, insult does not need to occur in the brain stem. Due to the observable emotional changes associated with PBA, it is most often characterized as a psychiatric disorder; however, due to the overlap of damaged neurological circuitry, PBA is best classified as a neuropsychiatric syndrome (Ahmed & Simmons, 2013; King & Reiss, 2013). Essentially, PBA is a neurological brain disease accompanied by disordered emotional expression (Brooks et al., 2013; Luhoway et al., 2019).

CHARACTERISTICS OF PSEUDOBULBAR AFFECT

Because the most common symptom of PBA is mood-incongruent behavior, it is important to carefully distinguish between mood and affect to best understand the core psychopathology of PBA. Mood is the subjective experience of an emotion, an underlying feeling or internal state (Barrett et al., 2007). Affect is the visible display of emotion observable by others (Barrett et al., 2007; Sauvé, 2016). Affect is considered to be incongruous when it is discordant with the individual’s emotional state. Notably, PBA is characterized as a disorder of mood affect and not of mood itself (Sauvé, 2016). Individuals with PBA have unpredictable, sudden, involuntary, and exaggerated affective displays, often crying or laughter, lasting seconds to minutes that are incongruent with their internal emotional state. For example, an individual may display a sudden, exaggerated, brief episode of intense crying without experiencing internal sadness. Due to the loss of affective control, and unawareness of their inappropriate responses, individuals with PBA are at increased risk of depression, anxiety, impaired social interaction, decreased quality of life, and impaired overall personal functioning (Hammond et al., 2016).

PREVALENCE

The incidence of PBA is unclear and remains difficult to estimate due to several variables in diagnostic criteria, screening methodology, causative neurological disorders, and patient populations (Brooks et al., 2013). PBA’s long history of confusing terminology, high rate of misdiagnosis, and long-standing history of underreporting has complicated assessments of its prevalence rate. In addition, PBA is easily underlaid by comorbid neurodegenerative processes that help make it intrinsically difficult to study. Because PBA occurs secondary to other neurological conditions, the estimated U.S. prevalence is approximately 2 million individuals with underrecognized and undertreated PBA (Brooks et al., 2013). Approximately 1.8 to 7.1 million U.S. individuals are affected with symptoms suggestive of PBA (Foley et al., 2016), including 10% of individuals with posttraumatic brain injury and 9.1% of nursing home residents with documented crying and tearfulness (King & Reiss, 2013; Perotti et al., 2016). Estimates of prevalence in a study considering six distinct neurological conditions revealed that PBA prevalence in neurodegenerative disorders may range from 2% to 60% in ALS, 5% to 17% in Parkinson’s disease, 7% to 29% in multiple sclerosis, and 10% to 74% in Alzheimer’s dementia (King & Reiss, 2013). Other researchers found a 10% to 46% prevalence in individuals with multiple sclerosis (Luhoway et al., 2019).

PATHOPHYSIOLOGY

Voluntary motor and involuntary emotional centers in the brain influence emotional expression (Domino et al., 2019; Finegan et al., 2019). PBA is seen in individuals with various brain and motor-neuron diseases and is a syndrome caused by defective brain circuitry or faulty neurochemistry in areas of the brain involved in the inhibition of emotional expression (King & Reiss, 2013). Due to the complexities of neurological pathways involved in voluntary and involuntary control, the exact neural pathways are not fully un-
understood, and medical researchers lack a clear neurophysiological explanation (Brooks et al., 2013; Lapchak, 2015).

One hypothesis is that the root cause of PBA stems from a malfunction of neural networks including the frontal lobe, limbic system, brainstem, cerebellum, and connecting white-matter tracks. The cerebellum plays a large role in PBA (Ahmed & Simmons, 2013; Lapchak, 2015), as its main function is to modulate emotional responses based on input received from the cerebral cortex and frontal and temporal lobes (Lapchak, 2015). Disrupted communication in the emotional motor pathway, the cortico–pontine–cerebellar circuit, has been theorized as the root cause of PBA (Domino et al., 2019). This circuit includes motor, limbic, and other associated cortices—as well as descending neural pathways located at the basis pontis that converge to the cerebellum—and is primarily responsible for emotional modulation.

PBA leads to altered inhibition from sensory cortices to motor or limbic cortices and consequently results in disinhibition of cerebellar gate control. Researchers have reported correlations between PBA and lesions found in the brain’s frontal lobes (Lapchak, 2015). It is the alteration in these pathways descending to the brain stem that is responsible for the impaired cerebellum neurotransmission or compromised cerebellar control of emotional responses.

Although not fully understood, the neurotransmitters norepinephrine, dopamine, serotonin, glutamate, and acetylcholine contribute to PBA. The role of neurotransmitters in PBA is mainly associated with the impact and importance these neurotransmitters have on PBA-impacted circuitry. Effective medications include antigu- ratamate, serotonergic, and norepinephrine reuptake blockers (Domino et al., 2019; Girotra et al., 2018; Stahl, 2013). Brain-mapping studies have suggested that reduced serotonin and dopamine transmission and enhanced glutamate transmission are essential components of PBA’s emotional dysregulation (Fonda et al., 2015; Lapchak, 2015). At the cellular level, cerebellar Golgi cells are thought to play an important role in emotional gate control. Golgi cells, when activated from various peripheral inputs, show decreased firing rates that are responsible for reducing inhibition of granule cells (Finegan et al., 2019).

DIAGNOSTIC CRITERIA

Fundamental features of PBA include brief, spontaneous, and exaggerated crying or laughing and visible affect incongruent with mood. Diagnosis of PBA rests on patient- or caregiver-reported clinical history and is confirmed based on physical examination.

A work-up should include a thorough medical history, physical examination, assessment of underlying neurological or psychological disorders, and evaluation of functional impairment, psychosocial impact, and comorbidities (King & Reiss, 2013).

PBA is often an incidental finding, diagnosis is subjective, and to date, no definitive diagnostic test exists; therefore, diagnostic criteria have been established to delineate the syndrome more effectively (Ahmed & Simmons, 2013). Poeck (1969) identified four criteria for PBA (Table 1). These criteria focus on episodes that are situa-

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Note: Adapted from Brooks et al. (2013) and Poeck (1969).

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Note: Adapted from Cummings et al. (2006).
tion inappropriate or incongruent with the patient’s internal emotional state (Finegan et al., 2019). Cummings et al. (2006) later revised Poeck’s (1969) criteria, emphasizing PBA’s involuntary emotional expressions and responses (Table 2) (Ahmed & Simmons, 2013; Finegan et al., 2019).

The Center for Neurologic Study-Lability Scale (CNS-LS) was the first validated clinical tool used to measure self-reported symptoms of PBA (Brooks et al., 2013; Chang et al., 2016; Moore et al., 1997). The CNS-LS is a 7-item self-report measure of affective lability, with four items relating to labile laughing and three to crying. The scale evaluates the subjective burden of symptoms during the previous 1 week. Responses are measured on a scale from 1 (never) to 5 (most of the time), for a maximum score of 35 and a minimum score of 7 (Brooks et al., 2013; Lapchak, 2015; Moore et al., 1997). A score ≥13 suggests possible PBA and scores ≥21 have shown sensitivity (74%) and specificity (80%) for the presence of PBA (Brooks et al., 2013; Finegan et al., 2019; Fonda et al., 2013; Girotra et al., 2018; Moore et al., 1997; Smith et al., 2004). According to Moore et al. (1997) for PBA in ALS, CNS-LS scores ≥13 showed a test–retest reliability of 0.88, an incremental validity of 0.31 to 0.34, a negative predictive value of 0.84 to 0.92, and high internal consistency (Cronbach’s alpha coefficient = 0.87; Colamonico et al., 2012).

TREATMENT

Currently, empirical evidence for the treatment of PBA is limited. For high value, collaboration can provide patient-centered care, lower health care costs, and enhance quality and health outcomes. An interprofessional collaborative approach is recommended and treatment aims to diminish the frequency and severity of involuntary emotional outbursts. Although not fully understood, numerous neurotransmitter disruptions are thought to occur in PBA. Therefore, treatment foci include norepinephrine, serotonin, and glutamate, using tricyclic antidepressants, selective serotonin reuptake inhibitors (SSRIs), and the cough suppressant dextromethorphan (DM), combined with quinidine sulfate (Ahmed & Simmons, 2013; Roman, 2015). Despite lack of substantial clinical evidence supporting their use for this indication, the serotonergic actions of SSRIs and tricyclic antidepressants seem to have reduced the frequency of PBA episodes by increasing the availability of serotonin at the synapses in corticobulbar and cerebellar pathways (Stahl, 2013). Typically, antidepressants are prescribed in lower doses for PBA and the onset of medication action is days rather than weeks (Ahmed & Simmons, 2013; Stahl, 2013).

In 2010, the U.S. Food and Drug Administration approved the first and only medication indicated for PBA, dextromethorphan hydrobromide and quinidine sulfate (DM/Q; Lochhead et al., 2018). Although DM/Q’s mechanism of action in treating PBA is not known, the basis for using DM/Q for the treatment of PBA hypothetically relates to its actions on sigma-1 receptor agonism and antiglutaminergic action through N-Methyl-d-aspartate (NMDA) receptor antagonism and serotonin and norepinephrine reuptake inhibition (Girotra et al., 2018; Lochhead et al., 2018; Roman, 2015). In administering DM/Q as monotherapy, the liver rapidly converts DM/Q to a compound unable to cross the blood-brain barrier, leading to low DM/Q plasma concentrations (Girotra et al., 2018). The addition of quinidine sulfate at a subtherapeutic concentration is for the sole purpose of inhibiting or blocking the otherwise-rapid hepatic CYP 450 2D6 metabolism of DM/Q. The quinidine sulfate leads to higher and sustained plasma concentrations of DM/Q, increasing its bioavailability (Ahmed & Simmons, 2013; Girotra et al., 2018; Lochhead et al., 2018; Stahl, 2013). It is important to reassess patients periodically for spontaneous improvement and reassess their treatment recommendations (Cruz, 2013). In addition, it is important to routinely monitor for adverse medication reactions when using DM/Q (McGrane et al., 2017).

CLINICAL IMPLICATIONS

PBA needs to be distinguished from other mood disorders and can be challenging to diagnose. Researchers found that 41% of individuals with PBA symptoms who discussed their inability to control emotional responses during clinical visits were diagnosed and only 52% of those received treatment (Ahmed & Simmons, 2013; Work et al., 2011). Clinicians can overlook inappropriate and unexpected emotional responses or identify them as poor coping, depression, or other mood disorders (Chang et al., 2016; Lochhead et al., 2018); therefore, lack of clinician awareness on criteria for diagnosing PBA may lead to its high rate of misdiagnosis (Brooks et al., 2013).

PBA is often overlooked because it can be easily confused with depression, high emotionality, or other mood disorders. Diagnosis can be particularly complicated for patients who may not be able to accurately communicate their feelings (Finegan et al., 2019). PBA is an affective illness: an observable, exaggerated, and unprovoked emotional response with subjective mood incongruence. The first step in diagnosis is to distinguish crying due to PBA from depressed mood. Notable distinctions include: (a) PBA symptoms may wax and wane, but do not tend to get progressively worse, as can depression; (b) individuals with PBA exhibit sudden, abrupt, and short crying or laughing episodes multiple times per day, lasting seconds to minutes rather than weeks or months; (c) crying attacks are incongruent with subjective mood; (d) patients experience no reported ongoing sadness, anhedonia, hopelessness, guilt, insomnia, or fatigue; and (e) responses to pharmacotherapy including serotonergic medications are quick (within days) compared to weeks, as is often the case with depression. It is important for clinicians to be mindful that up to one third of individuals with PBA experience comorbid depression (Chang et al., 2016; Finegan et al.,
Individuals with PBA more commonly report crying and tearfulness than laughter (Ahmed & Simmons, 2013). PBA can exist with depressive symptoms, which can complicate the clinical picture for clinicians (Finegan et al., 2019). In cases where PBA coexists with depressive symptomatology, emerging evidence suggests that PBA may respond to SSRIs within a few days, compared to depression, which typically responds to medications after several weeks.

Clinicians may find it challenging to differentiate PBA from bipolar disorders, especially in individuals who experience rapid cycling or mixed-mood episodes (Ahmed & Simmons, 2013). PBA can be confused with mania. Notably, in PBA, crying or laughing episodes will be brief (seconds to minutes) and present without any observable mood disturbances between episodes; overall cognition and behavior will be steady (King & Reiss, 2013). In contrast, bipolar disorders may present with some affective lability, but overall affect is congruent with mood and often lasts for days (King & Reiss, 2013). Semi-structured interviews and the Diagnostic and Statistical Manual of Mental Disorders (5th ed.) criteria for the diagnosis of mood disorders are useful in differentiating PBA from other neurological and psychiatric disorders and drug-induced effects (Ahmed & Simmons, 2013).

CONCLUSION

PBA is often unrecognized and misdiagnosed. Therefore, it is vital for clinicians to differentiate PBA from other conditions to improve prognosis and treatment outcomes. Heightened clinician awareness promotes early recognition, improved diagnostic accuracy, optimized treatment recommendations, and enhanced interprofessional collaboration among members of the health care community. Living with affective lability that manifests as spontaneous or exaggerated inappropriate laughing or crying is consistently associated with depression, social isolation, and overall negative quality of life (Brooks et al., 2013). PBA can be a burden to patients and caregivers, causing embarrassment that leads to social isolation and impaired overall functioning. It is imperative to raise awareness of PBA and its debilitating impact on individuals. Moreover, individuals with PBA should be referred to neuropsychologists and mental health care providers who specialize in affective disorders.

REFERENCES

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