Pseudobulbar affect: No laughing matter

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Pathological laughter and crying—pseudobulbar affect (PBA)—is a disorder of emotional expression characterized by uncontrollable outbursts of laughter or crying without an environmental trigger. Persons with PBA are at an increased risk of depressive and anxiety symptoms associated with an inappropriate outburst of emotion[1]; such emotional acts might be incongruent with their underlying emotional state.

When should you consider PBA?

Consider PBA in patients with new-onset emotional lability in the presence of certain neurologic conditions. PBA is most common in patients with amyotrophic lateral sclerosis and stroke, in which an incidence of >50% has been estimated.[2] Other conditions associated with PBA include Parkinson's disease, multiple sclerosis, frontotemporal dementia, traumatic brain injury, Alzheimer's disease, epilepsy, normal pressure hydrocephalus, progressive supranuclear palsy, Wilson disease, and neurosyphilis.[3]

Avoid PBA misdiagnosis

Depression is the most common PBA misdiagnosis (***Table***). However, many clinical features distinguish PBA episodes from depressive symptoms; the most prominent difference is duration. Depressive symptoms, including depressed mood, typically last weeks to months, but a PBA episode lasts seconds or minutes. In addition, crying, as a symptom of PBA, might be unrelated or exaggerated relative to the patient's mood, but crying is congruent with subjective mood in depression. Other symptoms of depression—fatigue, anorexia, insomnia, anhedonia, and feelings of hopelessness and guilt—are not associated with pseudobulbar affect.

Table

Differential diagnosis of pseudobulbar affect

Acute psychosis
Bipolar disorders with rapid cycling or mixed mood episodes
Borderline personality disorder
Conversion disorder
Major depression
Malingering
Substance-induced mood disorder

PBA also can be differentiated from bipolar disorder (BD) with rapid cycling or mixed mood episodes because of PBA's relatively brief duration of laughing or crying episodes—with no mood disturbance between episodes—compared with the sustained changes in mood, cognition, and behavior seen in BD.

Options for treating PBA

Serotonergic therapies, such as amitriptyline and fluoxetine, may exert effects by increasing serotonin in the synapse; dextromethorphan may act via antiglutamatergic effects at *N*-methyl-D-aspartate receptors and sigma-1 receptors.[4] Dextromethorphan binding is most prominent in the brainstem and cerebellum, brain areas known to be rich in sigma-1 receptors and key sites implicated in the pathophysiology of PBA. Although the precise mechanisms by which dextromethorphan ameliorates PBA are unknown, modulation of excessive glutamatergic transmission within corticopontine-cerebellar circuits may contribute to its benefits.

References

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