Whiplash presents as Pseudobulbar Affect: The challenges we face
Through the eyes of Psychosomatic Medicine
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BACKGROUND

• Pseudobulbar affect (PBA) is a syndrome of affect disinheritment, characterized by involuntary and inappropriate outbursts of laughing, crying and at times episodic irritability, agitation and anger [1-2].

• PBA is commonly associated with Cerebrovascular Accident (CVA). Amyotrophic Lateral Sclerosis, Alzheimer’s disease/dementia, Multiple Sclerosis, Parkinson’s disease and Traumatic Brain Injury.

• The pathophysiology of PBA is not completely understood, but thought to be caused by disruption to microcircuitry of cerebro-ponto-cerebellar pathways.

• Currently, there is a lack of consensus on the nomenclature and diagnostic criteria of PBA [5]. A recently proposed set of diagnostic criteria has been proposed which emphasizes that PBA is a change from the patient’s previous emotional responses and some specific characteristics [7].

• PBA is often undiagnosed, and untreated by neurologists and psychiatrists and many cases go unrecognized or misdiagnosed [6].

OBJECTIVES

1. To bring awareness how symptoms of PBA can be confused with psychiatric illnesses such as bipolar disorder, mood disorder, schizophrenia and other psychotic disorders.

2. To highlight how “only” Whiplash injury can cause the PBA.

3. To underline that patients with PBA may present as a seizure-like activity because of sudden and unpredictable onset of emotional outbursts, therefore PBA can be confused with epilepsy particularly gelastic and staring seizures. [5, 6-9]

4. Pre-existing PBA can be exacerbated by another neurological insult such as CVA.

5. PBA can be exacerbated and worsened by medication like Levetiracetam “Keppra.”

CASE STUDY

• This is a 71-year-old male with H/O Hypertension, unknown prior Psychiatric history who sustained a traumatic brain injury 6 months prior to his admission.

• Patient reported that his vehicle was struck by another one, and although there was no direct blow to the head, he developed whiplash injury due to acceleration -deceleration, coup and countercoup forces.

• In the ED, a Head CT showed no acute findings, he was medically cleared on same day and discharged home.

• A few weeks after the accident, he developed episodic angry outbursts and aggression towards his wife. These episodes were not reported by the family to any physician.

• Later he developed generalized body shaking, which prompted a visit to a Neurologist, who prescribed Valproic acid 500mg BID. No EEG was done at that time.

• Five months after the car accident, he developed a severe frontal headache, with left upper and lower extremity weakness, and diagnosed with a massive Cerebrovascular Accident [Exhibit 1]. He was admitted to medicine where his angry outbursts and seizure-like activity worsened.

• Neurology was consulted and diagnosed him with seizure disorder Vs. Post CVA epilepsy. No EEG was reported from the previous hospital. He was started on Levetiracetam 1000mg oral daily, which further worsened the agitation and aggression. Levetiracetam was stopped and changed to Valproic acid 500mg orally BID.

• Clinical Psychology was consulted for Neuropsychiatric Inventory (NPI) to rule out “Sundowning.” The diagnosis of PBA was made and they recommended a trial of Nuedexta 1 tab daily. [dextromethorphan hydrobromide/quinidine sulfate].

• Patient was discharged from another facility and transferred to our hospital for Post-CVA Rehabilitation. During the transfer, he became acutely agitated and jumped out of the ambulance but had no injuries.

• On the 2nd day of his hospitalization, a psychiatry consult was called because the patient was seen laughing inappropriately and later attacked a nurse.

• Psychiatry Consultation and Liaison team evaluated the patient and titrated Valproic acid to a total of 1500mg daily and Nuedexta to a total of 2 tabs daily. After this titration, his overall behavior improved and his aggression and agitation subsided in the next 2 days.

SUPPORTING EVIDENCE

• Mental Status Exam: Patient was dressed in hospital scrub, speech was non fluent with slow rate and rhythm, mood was euthymic, affect labile, incongruent with mood. Patient was noticed to be smiling inappropriately, then he suddenly became mute. Thought process was non linear, thought content showed no overt delusions, hallucinations, phobias, no S/H ideations, intent or plan. Insight, judgment and impulse control were poor. There was a mild decline in memory and cognition.

• Diagnostic Criteria Used: • Episodes of involuntary or exaggerated emotional expression that result from a brain disorder; including episodes of laughing, crying, or related emotional displays. • Episodes represent a change from the person’s usual emotional reactivity. • Episodes may be incongruent with the person’s mood or in excess of the corresponding mood state. • Episodes are independent or in excess of any provoking stimulus. Adapted Cummings et al., 2006 [7]

• Images results: CT Head 8/9/2017 - Acute hemorrhage is approximately 3.0 x 1.9 cm [Exhibit 01]

CHALLENGES

1. Incident #01: When the patient had a car accident, he developed PBA within the next few weeks as manifested by a new onset of emotional and angry outbursts. This was overlooked and unreported.

2. Incident #02: Subsequently, when he developed seizure-like activity, his actual diagnosis was missed and treated for a seizure disorder. It is important to remember that Epilepsy is an important differential diagnosis of PBA [3].

3. Incident #03: Five months after the car accident, the patient had a massive CVA. He continued to exhibit angry outbursts and seizure-like activity; his diagnosis was missed again. He was suspected to have a primary seizure disorder vs. Post-CVA epilepsy and treated with Levetiracetam.

4. Incident #04: One of the most important clinical aspects of this case study: when the patient was started on Levetiracetam, the symptoms of PBA (agitation, aggression) worsened. It is important to recall that Levetiracetam is well known for its side effects of irritability, anger, agitation [5].

5. Incident #05: When the patient’s LAV was changed to Valproic acid, and he was started on Nuedexta, his regimen was sub-optimized, which caused him to remain symptomatic. Clinicians should be liberal in optimizing medications. Untreated or under medicated PBA can cause severe distress, embarrassment, and social dysfunction.

RECOMMENDATIONS

1. Generally people do not see the need to report a multitude of head injuries if they were “only” dazed briefly, or in a motor vehicle accident where they suffered “only” whiplash [10]. This case is an evidence that “only” whiplash can cause PBA.

2. 6% to 52% of CVA survivors suffer from PBA [2, 5, 6, 11]. Though this patient had pre-existing PBA, his symptoms worsened after CVA. This suggests that pre-existing PBA can be worsened by another neurological insult such as CVA.

3. Patients with PBA may or may not present with classic symptoms, however clinicians should consider PBA in the differential diagnosis of any patient exhibiting agitation and emotional lability, especially after TBI.

4. In patients with PBA, Valproic acid should be considered as a better alternative.

5. To the best of our knowledge, this is the first case report of worsening of PBA after use of Levetiracetam. It is best to avoid any medication with the potential of worsening PBA.

REFERENCES

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