Case Report

Pathological Laughter in a Female with Multiple Episodes of Stroke and Subdural Hematoma

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ABSTRACT

Various brain areas in both cortical as well as subcortical locations are involved in pathological laughter. Pathological laughter may be seen as a prodromal symptom or acute manifestation or late sequel of stroke. Various other neuropsychiatric conditions attribute to stroke. It is often difficult to ascertain the cause of pathological laughter in the presence of multiple brain pathologies. Here, we highlight a case of a 55-year-old female, who had multiple episodes of stroke and subdural hematoma, presented with pathological laughter and other behavioral abnormalities.

Key words: *Pathological laughter, stroke, subdural hematoma*

INTRODUCTION

Pathological laughter occurs in various neuro-psychiatric conditions, of which stroke is an important cause. It results from the impairment in the connectivity between frontal cortex, temporal cortex, hypothalamus, amygdala, and cerebellum, which are involved in various steps of modulation of laughter phenomenon.[1] It was argued that dominant hemisphere involvement in a vascular event (left middle cerebral artery stroke) might be responsible for pathological laughter as the motor speech area has a key role in the processing of laughter.[2] In addition, evidences suggest that the involvement of brainstem structures (midbrain and pons) in stroke may manifest as pathological laughter as the ascending cortical projections from these areas process laughter.[3,4] Sometimes, pathological laughter may present as the prodromal symptom of transient ischemic attacks.[5] The phenomenon of pathological laughter or pseudobulbar affect is reported in one-fourth to half of the patients following stroke.[6]

Medications such as serotonergic and noradrenergic antidepressants, lamotrigine, dextromethorphan and quinidine combination, and levodopa have been used with variable success in the management of pathological laughter.[1]

CASE REPORT

A 55-year-old, nondiabetic and nonhypertensive female from a rural background was brought for psychiatric consultation for her abnormal behavior, increased

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irritability, and disturbed sleep for the past 2 years. Three years back, she had an episode of unconsciousness lasting for few hours, which was of sudden onset and associated with weakness of the left upper and lower limbs and deviation of mouth to the right side. She was hospitalized in a private nursing home and diagnosed with ischemic stroke of the right middle cerebral artery territory. She was managed conservatively and discharged after 2 weeks with improvement. Over the next 3 to 4 months, she regained power in her limbs and was able to do her activities of daily living without assistance. However, over the next 6 months, she had multiple episodes of unconsciousness without any motor weakness, which resolved on her own in few hours. Two years back, she had again developed sudden onset unconsciousness with weakness of the right upper and lower limbs, for which she was again hospitalized and managed conservatively. Family members reported that the patient was not able to speak and producing some incomprehensible sound when she regained her consciousness. With treatment, she had shown improvement; power in both the right upper and lower limbs improved to a greater extent that she was able to resume her daily activities. However, her speech remained incomprehensible. In addition, there was an increased irritability. Frequently, she would run away from home overnight. She would roam around her village and come back. Family members also reported that she would laugh loudly clapping her hands for several minutes without any obvious reason. Such behavior was reported 2 to 3 times in a day, which was increased to several times (10–20 times) a day for the last 6 months. In the past 6 months, she would have frequent anger outburst in which she would become assaultive, throw things, shout or run away from home. It caused significant embarrassment for the family members. However, she had never complained of headache, vomiting, or visual impairment. There was no history of head injury in the recent past. Her past and family histories were uneventful. Premorbidly, she was an uneducated homemaker who was capable of doing the regular household chores as well as farming-related work.

On general physical examination, no abnormality was detected. Her respiratory and cardiac examination did not reveal any abnormality. Neurological examination revealed upper motor neuron type of facial weakness of the left side.

She was prescribed quetiapine 50 mg per day in divided doses, which was later increased up to 100 mg per day. With quetiapine, her sleep cycle became normal. Her frequency of anger episodes and running away from home was decreased. However, inappropriate laughter episodes persisted as before. Hence, she was hospitalized. Her vital parameters (pulse, blood pressure, and temperature) remained within normal limit. Her hematological investigations, including coagulation profile, blood sugar, and lipid profile, were unremarkable.

Neuroimaging was advised in view of residual weakness. Computed tomography of the brain [Figure 1a and b] showed right dorsolateral frontal, temporal, and posterior parietal gliotic changes. There were also left posterior frontal and parietal gliotic areas. In addition, there was diffuse cerebral atrophy and left hemispheric chronic subdural hematoma. Magnetic resonance imaging [Figure 1c and d] confirmed the same findings. Her clinical features were more suggestive of repeated attacks of stroke, and chronic subdural hematoma *per se* was not producing any mass effect, the decision was taken to manage her conservatively. Escitalopram was added at a dose of 5 mg/day for her pathological laughter. She had shown improvement in her symptoms and became noncompliant in few days resulting in a relapse of symptoms. Restarting escitalopram improved her symptoms. The patient did not turn in follow-up after first follow-up visit (4 weeks following discharge) [Figure 1].

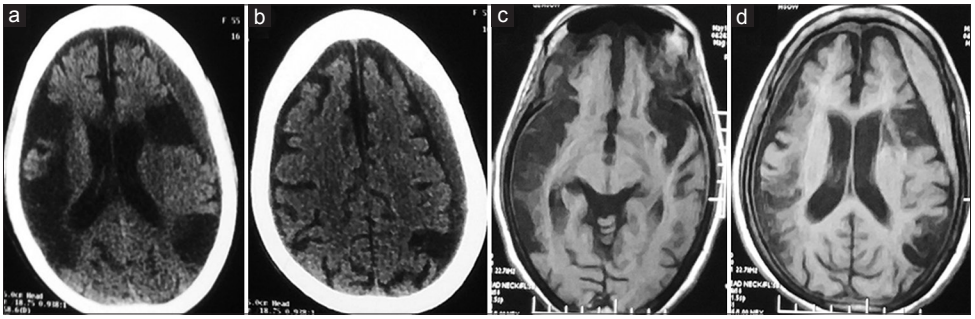

Figure 1: (a and b) Noncontrast computed tomography scan showing left posterior frontal, temporo-parietal, and right posterior frontal and posterior parietal gliotic changes with diffuse cerebral atrophy. Note the chronic subdural hematoma on the left fronto-temporo-parietal region. (c and d) T1-weighted magnetic resonance imaging showing the same gliotic changes with the left hemispheric chronic subdural hematoma without any midline shift. The sulci and gyri on the left side are not compromised due to the hematoma

DISCUSSION

Selective serotonin reuptake inhibitors such as fluoxetine, sertraline, and citalopram have been successfully used in the management of poststroke pathological laughter and are recommended as the first-line treatment.[7] Pathological laughter following stroke was successfully managed with various psychotropic medications such as quetiapine, lamotrigine, and mirtazapine, as reported in several case studies.[8-10]

Quetiapine was prescribed to our patient during the initial visits. Pathological laughter persisted, though there was an improvement in her impulsive behavior and irritable mood. Hence, the patient was hospitalized for a detailed evaluation. Addition of escitalopram resulted in an improvement in pathological laughter.

The patient had extensive gliosis in bilateral cerebral hemispheres following brain ischemia. She had multiple episodes of stroke resulting in ischemic demyelination. It also attributed to the cerebral atrophy, which was evident in the neuroimaging. Extensive lesion in the bifrontal region and subcortical regions might be responsible for the pathological laughter and impulsive behavior.

Detection of subdural hematoma was an incidental finding. There was no mass effect as the sulci–gyri architecture was intact and there was no midline shift. Potential free space between the cranium and brain surface facilitated the accommodation of hematoma without pressure effect. Hence, the patient had no features of raised intracranial tension, despite having a hematoma in the subdural space.

At times, it may misguide the clinician. The clinician may misattribute the patient's change of behavior to the subdural hematoma, warranting urgent surgical intervention for the evacuation of hematoma. This case gives the message that the subdural hematoma may exist silently without any attribution to psychopathology of the patient. Silent subdural hematoma may not require any active intervention. Low-dose escitalopram may be effective in the management of poststroke emotional disturbances such as pathological laughter.

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Conflicts of interest
There are no conflicts of interest.

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