Catatonic stupor after off-pump coronary artery bypass grafting

The Editor,

Neurocognitive dysfunctions are common after cardiac surgeries and are associated with significant postoperative complications. Association of catatonia following cardiac surgery has never been described earlier. We report a rare case of postoperative catatonic stupor following off-pump coronary artery bypass grafting, highlighting some of its key features and management options.

A 75-year-old male, with no history of any medical or neuropsychiatric disease, was admitted with acute coronary syndrome. He was investigated and found to have non-ST-elevation myocardial infarction, with normal left ventricular ejection fraction of 60% and mild mitral regurgitation. The patient was stabilized with antianginal medications. Elective coronary angiogram revealed 60% left main coronary artery stenosis with significant triple vessels disease. Off-pump coronary artery bypass grafting was performed with anastomosis of the left internal mammary artery to left anterior descending artery, saphenous vein grafts to posterior descending artery, obtuse marginal and diagonal artery. The surgery was uneventful, and the patient was transferred to the Intensive Care Unit (ICU) in stable condition with minimal dose of inotropic supports. The patient was shifted to the ICU late in the evening; hence, after overnight ventilation, the patient was extubated in the morning. On the second postoperative day (POD), the patient was found to be drowsy and gradually became stuporous. The patient was hemodynamically stable with no signs of low cardiac output or infection. Blood gases, serum electrolytes, blood sugar, liver function tests, and transthoracic echocardiography were within normal limits. Neurological examination revealed the muscular rigidity of both upper and lower limbs. On passive movement, there was waxy flexibility and the patient was found to be maintaining the rigid posture for a considerable period [Figure 1]. Computerized tomography scan of the brain and electroencephalography were found to be normal. The patient was diagnosed to have catatonic stupor. He was given injection lorazepam 2 mg intramuscularly as per neurologist’s advice. Within 30 min, the patient was able to follow commands, and the muscular rigidity decreased. Thereafter, tablet lorazepam 1 mg was continued per oral twice daily for 5 days, and the patient was discharged on the 7th POD in stable neurological condition.

DISCUSSION

Karl Ludwig Kahlbaum in 1874 first described catatonia as a state of neurogenic motor immobility and behavioral abnormality. Catatonic patients often manifest as extreme loss of motor skill, hyperactive motor activity, echolalia, echopraxia, or catalepsy. The best-known sign of catatonic stupor is waxy flexibility, in which a rigid position of the body is maintained for a considerable period.

Although catatonia is one of the important manifestations of schizophrenia, other disease

Figure 1: Catatonia with rigid position of the body
states can mimic catatonia and should be considered in the differential diagnosis. These conditions include Kahlbaum syndrome, stiff-person syndrome, akinetic Parkinson's disease, neuroleptic malignant syndrome, selective mutism, conversion disorder, and other hyperkinetic and hypokinetic states. About 75% of patients with catatonia are not associated with any psychiatric disorder. Catatonia may occur due to disorders of the thyroid or adrenal glands, injury to the central nervous system due to stroke, trauma, vasculitis, tumor, or anoxia. A catatonic-like state may also be caused by epilepsy, and even as an adverse effect of anticonvulsants, neuroleptics, dopamine-blocking antiemetics, and corticosteroids.

Pathogenesis of catatonia involves a complex interplay of dysregulated neurotransmitters such as γ-aminobutyric acid-A, glutamate, dopamine, and N-methyl-D-aspartate (NMDA) receptors.

Benzodiazepines are the first-line of treatment. A dose of 1–2 mg of parenteral lorazepam will often result in marked improvement within an hour. When the first-line treatment fails, NMDA antagonists such as amantadine or memantine can be used. Memantine is more specific for the glutamate system with reduced incidence of psychosis and may therefore be preferred over amantadine. Topiramate is another therapeutic option for resistant catatonia; it produces its effects by glutamate antagonism via modulation of α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptors. Electroconvulsive therapy is also an effective treatment for catatonia. Antipsychotics should be used with caution as they can worsen catatonia and may cause neuroleptic malignant syndrome, a dangerous condition that can mimic catatonia and requires prompt discontinuation of the antipsychotic.

In conclusion, our patient did not have any neurological disorder and was recovering well; postoperatively, he developed acute catatonic stupor which got relieved by lorazepam.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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