Takotsubo cardiomyopathy in aneurysmal subarachnoid haemorrhage

Sir,

Reversible cardiac dysfunction is a well-known

complication of sub arachnoid haemorrhage (SAH).[1] We report a case of an Anterior communicating (Acom) artery aneurysm with SAH who manifested with takotsubo cardiomyopathy (TTC) peri-operatively. The compromised cardiological status owing to the regional wall motion abnormalities due to TTC superimposed additional challenges on the hemodynamic management in terms of optimal blood pressure and cerebral perfusion during perioperative management of clipping of the aneurysm. This clinical scenario demands a well-tailored and judicious anaesthetic management in neurosurgical intervention.

A 60-year-old male farmer presented with a history of sudden onset of headache and multiple episodes of vomiting followed by the loss of consciousness of 2-3 h. He was diagnosed as a case of SAH based on non-contrast computed tomography (NCCT) brain findings and came to neurosurgical attention on 8th day post ictus. During computed tomography angiography his Glasgow Coma Scale (GCS) deteriorated (E2 V1 M3) and had to be mechanically ventilated. A diagnosis of ruptured Acom artery aneurysm was made and an emergency left pterional craniotomy and clipping of Acom artery aneurysm was undertaken.

On pre-anaesthetic evaluation, all his systemic parameters including cardiac enzymes were unremarkable. Electrocardiogram (ECG) depicted V2-V6 ST elevation with biphasic T waves. Pre-operative two-dimensional echocardiography revealed ballooning of apical segment with left ventricular (LV) dysfunction [Figure 1] and ejection fraction (EF) of 38%. Patient was accepted in American Society of Anaesthesia grade 4 E. On account of pre-operative deterioration in GCS, patient was put on elective mechanical ventilation post-operatively. Post-surgery NCCT brain revealed an infarction in Bilateral Anterior Cerebral Artery territory with intraventricular haemorrhage and an oedematous brain.

Subsequent two-dimensional echocardiography [Figure 2] and NCCT brain findings on the 3rd post-operative day revealed no marked improvement. Tracheostomy was performed on the 3rd post-operative day and patient weaned off to T piece ventilation with oxygen support with minimal improvement in EF 45%. Patient rapidly deteriorated and succumbed to his neurological and cardiovascular injuries on the 6th day post-operatively.

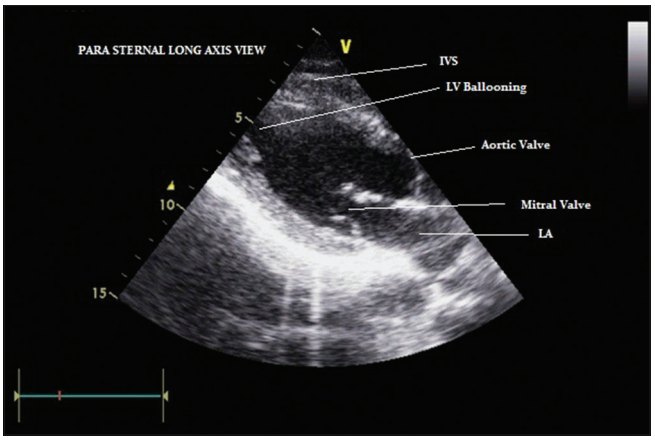

Figure 1: Parasternal long axis view showing apical ballooning

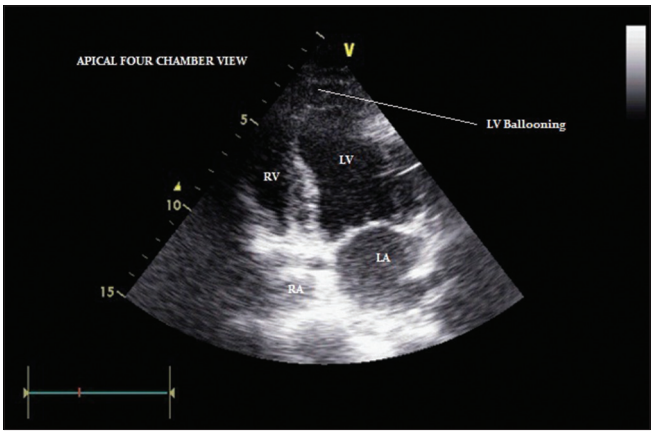

Figure 2: Apical four chamber view showing apical ballooning

The prevalence of TTC is 0.02% among all hospitalised patients[2] and 0.8% among all patients of aneurysmal SAH. TTC consists of cardiomyopathy with transient apical ballooning and systolic dysfunction of apical and mid segments of the left ventricle in the absence of atheromatous disease of the coronary arteries, accompanied by ECG changes together with the minimal release of cardiac enzymes appearing in a context of emotional or physical stress.[3] There is compensatory hyperkinesis of basal walls, producing characteristic ballooning of apex with systole. The proposed pathogenesis are multivessel coronary vasospasm, abnormalities in coronary microvascular function, catecholamine cardiotoxicity and adrenocepter hyperreactivity.

Approximately, 20-30% of patients with SAH manifest a secondary cardiomyopathy and/or regional wall motion abnormality, referred as neurogenic stress cardiomyopathy or neurogenic stunned myocardium.[4] Despite the severity of the acute illness, TTC is a transient disorder managed with supportive therapy and is noted as a cause

of sudden cardiac disease in individuals with no cardiac disease. A high suspicion index for TTC has to be maintained if sudden hemodynamic deterioration occurs in patients with SAH and the definitive diagnosis can only be achieved with help of coronary angiography.

Conservative treatment for LV systolic dysfunction, angiotensin converting enzyme inhibitors, beta blockers, and diuretics with resolution of the physical or emotional stress usually results in rapid resolution of symptoms. Although TTC carries a favourable prognosis in general population,[5] this pattern of cardiac dysfunction in population with SAH may be associated with pulmonary oedema, prolonged intubation, and cerebral vasospasm. Additional studies are required to elucidate the spectrum of cardiac abnormalities arising as a result of catecholamine excess.

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