Editorial

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Quick response code

'Broken-heart syndrome'... Be aware..

Takotsubo cardiomyopathy (TCM), also known by various other names such as broken-heart syndrome, stress-induced cardiomyopathy and apical ballooning syndrome, is an acute, reversible, transient left ventricular dysfunction that was first described by a Japanese cardiologist in 22 patients in the year 1990. The Japanese word 'takotsubo' means an octopus trap, used to describe the shape of the left ventricle in this condition during systole. Since its original publication, there have been several case reports describing its occurrence in patients in the periprocedural and perioperative periods, underscoring the importance of the need for awareness about this rare syndrome among anaesthesiologists. There are several case reports published from India too. In this issue of the Indian Journal of Anaesthesia, a case of this entity is reported after thyroidectomy and also one describing its occurrence in a young patient who developed anaphylaxis to cephalosporin intraoperatively during limb salvage surgery for osteosarcoma of the tibia.[1,2]

TCM is more common in elderly postmenopausal women, although presentations have been reported in young men and women also. A systematic review of this syndrome has recently been published by Ono and Falcão.[3] The triggering factor for this cardiomyopathy seems to be the elevated levels of plasma catecholamines due to physical or emotional stress, which leads to coronary spasm, direct myocardial toxicity or microvascular impairment. Iatrogenic administration of catecholamines can also be a trigger of this syndrome.[4] Lyon *et al*. have described a novel pathophysiological hypothesis to explain the myocardial stunning due to catecholamine excess.[5] It has been proposed that apical ballooning could be due to the abundance of beta receptors in this region that can lead to negative inotropy due to change in the intracellular signal trafficking. The Heart Failure Association of the European Society of Cardiology has recently published a position statement on takotsubo syndrome that includes a diagnostic algorithm comprising seven criteria including anatomic features, electrocardiogram (ECG) changes, cardiac biomarkers and reversibility of the ventricular dysfunction.[6]

WHAT IS THE DILEMMA: STEPS IN DIAGNOSIS

The dilemma posed to an anaesthesiologist with the presentation of this syndrome is that it closely resembles acute coronary syndrome (ACS). Patients may present with symptoms of chest pain, breathlessness, syncope or with complications such as cardiogenic shock, ventricular arrhythmias, pulmonary oedema, left ventricular outflow tract obstruction and cardiac arrest. The similarities of this syndrome with ACS are marked in terms of the clinical presentation, ECG changes and elevation in biomarker profiles. However, the crucial difference is that the epicardial coronaries are normal and the wall motion abnormalities are not restricted to a single coronary vascular bed.

Looi *et al*.[7] in a recent study from Australia observed the evolution of ECG changes in TCM compared to myocardial infarction (MI). They found that though ST elevation occurs in TCM, it is less prominent and is not accompanied by reciprocal ST depression or pathological Q waves. Deep T-wave inversion and significant QT prolongation are seen after 24–48 h that can lead to torsades de pointes. The biomarker elevation is also only moderate in TCM, as compared to ACS. B-type natriuretic peptide (BNP) levels are much more elevated in TCM, compared to acute MI (AMI), and Scantlebury and Prasad[8] have showed that the ratio of BNP to Trop T could help in distinguishing between the two conditions. Wittstein *et al*.[9] have shown that serum catecholamine levels

are much more exaggerated in TCM, compared with MI. It may not be possible to differentiate TCM from AMI based on systolic and diastolic function of the heart, although on echocardiography (ECHO), basal segments are usually spared in TCM. Coronary angiography is essential to distinguish the two. Pathak *et al*. have also reported only a mild elevation in biomarkers, CP-MB and Trop T, although there is no mention of BNP levels. Demonstration of elevated levels of BNP would have made their diagnosis of TCM more conclusive along with absent occlusive findings on a coronary angiogram. Their recommendation to use intraoperative transoesophageal ECHO for the diagnosis is a very useful suggestion.

HOW DO WE MANAGE? WHAT ARE THE CONTROVERSIES?

Prognosis in this syndrome is favourable, with the mortality ranging from 0 to 10%, which emphasises the importance of timely diagnosis and appropriate management. The Heart Failure Association of the European Society of Cardiology stratifies patients into high-risk and low-risk groups based on major and minor risk factors. The aim of treatment should be supportive to maintain cardiac function and prevent complications. Although in the case report by Pathak *et al*., ephedrine and adrenaline were used to manage hypotension, suggested acute therapy involves the management of myocardial ischaemia with oxygen, intravenous heparin, antiplatelet agents and β blockers along with cessation of any sympathomimetics. The role of β blockers has been a controversy with some concerns that their use can lead to cardiogenic shock. A recent retrospective cohort study in Japan by Isogai *et al*.[10] found no significant association between the use of β blockers early in the course of the syndrome and in-hospital mortality. Anticoagulants should be initiated early, provided there is no contraindication to reduce the risk of thromboembolism from the apical thrombosis. Cardiogenic shock should be managed with mechanical supports such as left ventricular assist devices or extracorporeal membrane oxygenation or with levosimendan, and the use of inotropes is regarded as a contraindication. Continuous monitoring of ECG, haemodynamics and regular ECHO is essential. A team approach is important with the involvement of the cardiologist, the anaesthesiologist and the primary physician when this condition presents perioperatively.

In summary, although many case reports have been published in the literature, several questions remain unanswered regarding the exact pathophysiology of the syndrome; it is much important for all the perioperative physicians to be aware of the existence of this rare syndrome and lay focus on its timely diagnosis and appropriate management to prevent mortality.

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Conflicts of interest

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