Guest Editorial

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Systolic anterior motion of the mitral valve in hypovolemia and hyper-adrenergic states

In this issue of the Journal, Reddy and Ueda[1] present an interesting case of refractory hypotension resulting from unidentified hypovolaemia, which was aggravated by administration of inotropes. The authors describe the use of trans-oesophageal echocardiography (TEE) as the rescue tool, which helped diagnose hypovolaemia and correct the line of treatment.

Perioperative hypotension is often seen on administration of anaesthesia. Anaesthesia-mediated venodilation and hypovolaemia are the most common causes of hypotension. Hypotension occurs most particularly in patients with acute cardiac failure and those suffering from systemic inflammation or sepsis. Venodilating medications, anaesthetic agents and neuroaxial anaesthesia cause reductions in left ventricular (LV) preload and peripheral vascular resistance perioperatively. Hypotension may contribute to adverse post-operative outcomes such as renal dysfunction and myocardial infarction and significantly increase morbidity/mortality.

In the case described, hypotension occurred soon after induction of anaesthesia possibly due to sedation, venodilation by drugs and right heart compression by extra-cardiac cysts described. Although the authors do not mention that the patient had a pre-surgery haemodialysis, over-zealous dialysis also leaves patients hypovolaemic. Hypovolaemia worsened on application of peak-end-expiratory-pressure (8 cm). Hypotension further worsened on drainage of ascites. The misleading high levels of central venous pressure (CVP) (16 mmHg) seen could be partly explained by the transmission to the chest of abdominal hypertension, compression of the right side of the heart by the cysts or the reduced cardiac compliance associated with tachycardia.[2] Tachycardia and reduced cardiac compliance were worsened by the effects of the inotrope on a hyper-contractile and under-filled LV. It is well-established that CVP cannot be used to assess the need for volume expansion.[3]

Geometric changes resulting from either absolute or relative decreases in preload in an under-filled, hyper-contractile LV displaces parts of the mitral valve (MV) apparatus into the LV outflow tract. This is seen as systolic anterior motion (SAM) of one or both MV leaflets and it leads to LV outflow obstruction, MV insufficiency and severe cardiovascular destabilization.[4] Left bundle-branch block or ischaemia (the patient reported also had septal ischaemia) could cause mitral insufficiency. SAM is a less-recognised cause of acute severe perioperative hypotension in non-cardiac surgery.

Death from SAM has been reported in severely ill-patients even without pre-existent cardiac disease[5] and SAM has even been hypothesized to be the terminal pathway of lethal hypovolaemia.[4] "Functional" SAM has also been described in association with pheochromocytoma,[6] induction of general anaesthesia[4] and in association with catecholamine infusion[7] and hypovolaemia[8] in critical patients. SAM has also been described in hypertensive heart disease, diabetes mellitus, acute myocardial infarction, after mitral valve repair and even in asymptomatic patients during pharmacologic stress with dobutamine.[9]

The pressure differential between the left atrium and LV outflow tract is thought to lead to deviation of MV towards the septum. Rapid velocities in the outflow tract were thought to lift MV leaflets towards the septum, to cause SAM, by Venturi effect. Current studies indicate that anterior MV leaflet

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is swept towards the septum by a drag and as the leaflet approaches the septum, the gradient across LV outflow tract rises and it becomes the major hydraulic force, encouraging further excursion towards the septum.

Visualisation of SAM of one or both MV leaflets by echocardiography is the perioperative diagnostic approach of choice.[10] Although TEE is superior for diagnosing LV outflow obstruction,[11] transthoracic echocardiography is a reasonable alternative, if access to the anterior chest wall is available. Echocardiography has been recommended on critically ill-patients with unexplained hypotension that is poorly responsive to conventional resuscitative measures.[12]

Medical therapy is based on prompt restoration of normal blood volume, reduction of hyper-contractile state (beta-blockers, calcium channel blockers) and withdrawal of inotropes and vasodilators. Treatment of SAM in patients presenting with absolute reductions in LV preload includes volume substitution and vasopressor infusion. In case of relative reductions in LV preload, treatment may include beta-adrenergic blockade. Although infusion of beta-blockers in a hypovolaemic patient can severely aggravate cardiovascular failure, their cautious administration may reduce both force of contraction and heart rate, increasing LV filling time and volume in patients with hyper-contractile ventricles.[11]

Mukul Chandra Kapoor

Department of Anesthesia, Saket City Hospital, New Delhi, India
E-mail: mukulanjali@gmail.com

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