

Cerebral aneurysm with valvular heart disease: Anaesthetic management and challenges

Sir,

Aneurysmal subarachnoid hemorrhage (a-SAH) and severe valvular heart disease (VHD) is rarely encountered. The a-SAH is frequently associated with systemic manifestations include: Neurogenic pulmonary edema, cardiac dysfunction, hepatic injury, renal impairment, and electrolyte abnormalities. There is a wide range of cardiac complications described in SAH, which range from innocuous electrocardiographic (ECG) abnormalities to even sudden cardiac arrest.^[1] If such a condition co-exists with underlying VHD and effects of general anesthesia, you have a recipe for cardiac disaster and a neuroanesthetist worst nightmare. Here, we describe the anesthetic management of a patient with ruptured anterior communicating aneurysm (ACOM) having severe mitral stenosis (MS) posted for clipping.

A 48-year-old hypertensive female, known case of rheumatic heart disease (RHD) with MS was referred to our hospital with a history of sudden onset of severe headache. On examination she was conscious, disoriented with Glasgow coma scale of 13 (E3 V4 M6) and had stable vitals. The patient had history of edema on both lower limbs 2 months back. She was on oral amlodipine 5 mg and atenolol 25 mg combination along with tablet digoxin 0.25 mg once a day. Noncontrast computed tomography of head showed SAH in anterior inter-hemispheric fissure and left Sylvian fissure. Computed tomography angiogram confirmed ACOM aneurysm that was 10 mm × 12 mm and neck width of 6 mm. The ECG demonstrated sinus rhythm, bifid P waves in lead II. Two-dimensional echocardiography revealed moderately severe MS (mitral valve area 1.0 cm²), mild tricuspid regurgitation, moderate pulmonary artery hypertension (PAH), and left ventricular (LV) ejection fraction 55%. Chest X-ray showed marked pulmonary vascular markings, blunting of both costophrenic angles and cardiomegaly. Currently, there were no signs and symptoms of cardiac failure.

We planned to take the patient for left pterional craniotomy and clipping of aneurysm neck. After

attaching routine monitors, invasive lines including central venous catheter and arterial line were secured. Anesthesia was maintained as per institutional protocol. Fluid and blood transfusion management were done according to central venous pressure (CVP) to avoid hypovolemia or hypervolemia. Infusion of mannitol 1 g/kg was given prior to opening of duramater. The surgery lasted for about 4 h and aneurysm was clipped. At time of extubation, tachycardia was controlled using the intravenous esmolol. Chest auscultation revealed basal crepitations, which was managed by 20 mg bolus dose of intravenous furosemide. The patient was extubated after surgery and shifted to Intensive Care Unit (ICU) for postoperative care. The patient was discharged successfully from ICU on 15th postoperative day.

The ruptured cerebral aneurysm leading to SAH has both neurological and nonneurological manifestations.^[1,2] Neurogenic cardiac manifestations include ECG abnormalities, arrhythmias, LV dysfunction, myocardial infarction, and stunned myocardium. All these cardiac effects lead to increase in mortality and morbidity. The various mechanisms described for these cardiac abnormalities are: Release of catecholamines, activation of sympathetic response, and systemic inflammatory response syndromes such as response to cerebral injury.^[2] RHD manifests with MS or combined with mitral regurgitation in approximately 25-40% of the patients.^[3] The pathophysiological changes in MS include left atrial (LA) outflow obstruction leading to increased LA pressure, resulting in increased pulmonary capillary wedge pressure (PCWP) and PAH. In longstanding cases severity of PAH is increased, leading to function tricuspid regurgitation and finally right ventricular failure.^[4] Increased LA pressure causes its enlargement and in severe cases atrial fibrillation (AF). The anesthetic challenges for managing a patient with severe MS for noncardiac surgery includes, preoperative rhythm disturbances, low fixed cardiac output state, dependence of LV filling on atrial kick, congestive heart failure, and increased risk of pulmonary edema.

Various anesthetic challenges of combined effects neurogenic cardiac dysfunction and preexisting cardiac problems are described here. As there is elevated LA pressure due to LA outflow narrowing, CVP or PCWP do not correlate with LV end diastolic pressure. Hence, these monitors may not be helpful in accurately guiding the fluid therapy. Increased LA pressure causes lung congestion due to backpressure

rendering the patients with severe MS, as poor candidates for instituting triple-H therapy.^[5] In the presence of congestive heart failure, use of mannitol to relax brain becomes a challenging task and using diuretics causes hypokalemia keeping these patients at risk of digitalis toxicity. In the patients with severe MS, LV filling is dependent on atrial kick. Progressive LA enlargement precipitates AF causing drastic fall in cardiac output, and increases the chances of cerebral ischemia. Beside this, SAH produces hypercoagulable states that increase the chances of atrial thrombi manifolds in the presence of AF. Above all, if patients with SAH had a history of prosthetic valve replacement and receiving anticoagulants, it becomes a challenge to make a balance between prevention of valve thrombus formation and further hemorrhage. Sometimes, we need to institute special techniques to facilitate surgery such as hyperventilation, cerebral dehydration, and deliberate hypertension/hypotension. In patients with cardiac disease, osmotherapy or hyperventilation may compromise cardiac function.^[6] The hypoxia, hypercarbia, hypothermia, and acidosis must be avoided as these factors increases pulmonary artery pressure. The supine position with head elevated by 30° should be considered to promote surgical exposure. Another major consideration for aneurysm surgery is the requirement for temporary clipping during dissection, and that period requires induced hypertension to maintain cerebral blood flow through collaterals. Hypotension is best treated with direct α-adrenergic agonist, as β-adrenergic agonists cause tachycardia and vasodilation, which are undesirable effects in patients with MS. Thus, phenylephrine is preferred agent over ephedrine, dopamine, dobutamine, and epinephrine. Judicious use of fluids in combination with vasopressors and maintaining cardiac grid helps in maintaining blood pressure.

The risk of cardiac disease can be minimized by maintaining the cardiac grid in these patient, including low normal heart rate, sinus rhythm, maintaining systemic vascular resistance as well as contractility, and decreasing pulmonary vascular resistance. In these patients, we should consider appropriate positioning for surgery, judicious use of osmotherapy using vasopressor rather than inotropes, prompt control of heart rate, and invasive monitoring. Brain directed therapies should be used with caution.

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Conflicts of interest

There are no conflicts of interest.

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