CASE REPORT

Anesthesia Management of a Case of Subdural Hematoma in a Patient of Severe Mitral Stenosis with Warfarin Toxicity: A Case Report

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Abstract

Valvular heart diseases have a mixed presentation in healthcare settings all over the world. Rheumatic heart disease still forms the major cause of these valvular lesions. Mitral and aortic valvular diseases top the list of valvular pathology.[1] Thereby, a thorough understanding of the pathophysiology of valvular heart disease is essential in planning anesthesia and perioperative care of such patients. Judicious use of fluids, close monitoring of the changing hemodynamics, and avoiding major reduction of cardiac output and fluid shifts are mandatory to achieve a good clinical outcome. These patients are at risk of the thromboembolic episode for which they are on long-term anticoagulation.[2] So timely monitoring of the coagulation profile is needed. The risk of bleeding increases if international normalized ratio (INR) >4.5.[3] We hereby present a case of subdural hematoma in a case of severe mitral stenosis with warfarin toxicity posted for burr hole surgery.

Keywords: Mitral stenosis, Pulmonary hypertension, Rheumatic heart disease, Subdural hematoma, Warfarin toxicity.

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Introduction

The incidence of heart disease ranges from 0.7% in the 18–44 years age group up to 13.3% in individuals who are 75 years or older.[4] Rheumatic fever is by far the largest cause of mitral stenosis (MS) in developing countries like India. Other causes of MS include congenital heart disease, infiltrating diseases, and diseases affecting multiple systems like sarcoidosis.[5] A thorough understanding of the pathophysiology of valvular heart disease is essential while planning anesthesia and perioperative care for such patients. Prolonged anticoagulation increases the risk of bleeding and so the anesthesia management of these patients becomes challenging in view of deranged coagulation, hemodynamic instability, and poor Glasgow Coma Scale (GCS). The key to anesthesia management of such patients is an urgent reversal of anticoagulants, maintenance of baseline parameters, and close hemodynamic monitoring.

Case Description

A 39-year-old female patient, a known case of rheumatic heart disease (RHD), came to our hospital with complaints of multiple episodes of vomiting associated with one episode of convulsion and left-sided weakness. There was no history of trauma or head injury. She was a diagnosed case of rheumatic MS with pulmonary hypertension on tablet warfarin 5 mg once a day for 1 year. She was also a known case of hypothyroidism on tablet thyronorm 25 µg a day for 2 years. The patient had a history of cerebral vascular accident 2 years ago with right hemiparesis that had completely recovered. She was recently diagnosed as hypertensive and was started on antihypertensive tab metoprolol 25 mg once a day (OD) and tab lasilactone 20 mg twice a day (BD). On admission, she presented with left-sided weakness. She was conscious but a bit disoriented, hemodynamically stable, and was started on inj. mannitol 100 mg thrice a day (TDS) and inj. levetiracetam 500 mg BD. Blood investigations showed Hb—9.8 with a normal platelet count of 2.25 lakhs. The patient had raised prothrombin time (PT)/international normalized ratio (INR) 78.8/6.13 and her thyroid function test showed a raised thyroid stimulating hormone (TSH) of 10.36. The patient was diagnosed to have warfarin toxicity and tab. warfarin was immediately withheld. Four units of fresh frozen plasma (FFP) were transfused to the patient. Chest X-ray showed cardiomegaly (Fig. 1) and electrocardiogram (ECG) had right ventricular hypertrophy (RVH) with p-pulmonale and p-mitrale with ST-segment depression in chest lead V2–V6, limb leads L2, L3, and augmented vector left (AVF). 2D echocardiography (2D ECHO) of the patient showed severe MS with a valvular area of 0.8 to 0.9 sq/cm associated with severe pulmonary hypertension with dilated right and left atrium (LA). On computed tomography (CT) brain, the patient had a subdural hematoma (SDH) in the right fronto-temporo-parietal region with a midline shift of 4 mm.

She was posted for emergency burr hole surgery for SDH under general anesthesia. On examination, she gave a history of poor effort tolerance with a history of chronic cough. Her heart rate was

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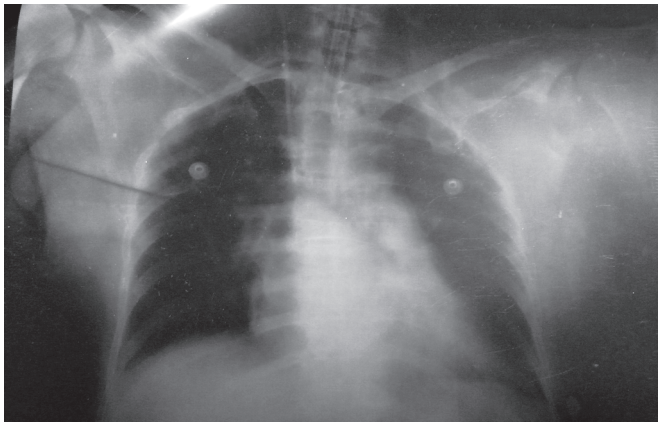

Fig. 1: Chest X-ray shows cardiomegaly

70 beats/minute, regular rhythm, and blood pressure of 110/70. Diastolic murmur was present in the mitral area.

High-risk consent was taken from relatives about perioperative cardiac events like cardiac arrhythmias, heart failure, and the need for postoperative ventilatory support. All American Society of Anesthesiologists (ASA) standard monitors like non-invasive blood pressure (NIBP), 5-lead ECG, pulse oximeter, and capnograph after intubation were attached and baseline vitals were recorded. The patient was preoxygenated with 100% oxygen following which she was premedicated with inj. midazolam 2 mg. The patient was induced with 100 µg of inj. fentanyl and 6 mg of inj. etomidate. The airway was secured using a 7.0 mm cuffed endotracheal tube after complete relaxation with inj. vecuronium 1 mg/kg. The right internal jugular vein was cannulated and the arterial line in the left radial artery was taken after induction. Central venous pressure (CVP)-guided fluids were given and intraoperatively beat-to-beat blood pressure was recorded. Anesthesia was maintained with air, oxygen, and sevoflurane mixture and intermittent boluses of fentanyl and vecuronium. Intraoperative nitroglycerin (NTG) infusion was started to reduce pressure response and as a pulmonary vasodilator. The patient tolerated the procedure well and was not reversed nor extubated. After surgery, the patient was shifted to anesthesia intensive care unit (ICU) on ventilatory support in view of high pulmonary pressures and poor general condition. In ICU, she was kept sedated and paralyzed overnight. Meanwhile, NTG infusion was continued in ICU with intermittent boluses of inj. frusemide was given. The patient was weaned off and extubated the next postoperative day. The patient was stable and shifted to the general ward on the second postoperative day and later discharged 10 days after the procedure. She was well oriented on discharge with full recovery of her power.

Discussion

Group A streptococcal infection is responsible for causing acute rheumatic fever. If it remains untreated, it can cause chronic granulomatous inflammation in heart valves leading to the development of RHD. At least 60% of untreated patients develop RHD.[6] Mitral and aortic valves are most commonly involved in RHD. The persistent inflammatory and hemodynamic valvular injury is responsible for the progression of the disease process. In a normal adult, the mitral valve has an area of 4 to 6 cm^2. In MS, when the mitral valve area is reduced to <2.0 cm^2, a pressure gradient develops across the mitral valve (Table 1). The magnitude of this gradient depends on stenosis, severity, and the amount of blood flow across the valve. Mitral stenosis is classified based on valve area and pressure gradients across the valve.

Table 1: Criteria for echocardiographic diagnosis of MS severity

Mitral stenosis	*Mild*	*Moderate*	*Severe*
Mean pressure decrease	<5 mm Hg	5–10 mm Hg	>10 mm Hg
Pressure half-time	<139 ms	140–219 ms	>220 ms
Valve area	1.6 cm^2	1.5–1.0 cm^2	<1.0 cm^2

An increase in left atrial pressure is reflected in the pulmonary venous circulation and increases the risk of pulmonary edema. This results in pulmonary arterial hypertension that eventually leads to an increase in right ventricular pressures and possibly, to right ventricular failure. As the severity of stenosis increases, patients develop decreased exercise tolerance, orthopnea, cough, paroxysmal nocturnal dyspnea, and pulmonary edema. Clinical presentation of rheumatic MS is usually associated with symptoms of congestive heart failure but patients may also present with hemoptysis and chest pain from pulmonary hypertension. Hypoxia, hypercarbia, acidosis, lung hyper expansion, and nitrous oxide are to be avoided to avoid an increase in pulmonary artery pressure.

They develop a large-sized LA to pump against a stenotic valve which gives rise to atrial fibrillation on ECG. A slower heart rate is therefore needed to pump blood effectively from the LA to fill the left ventricle (LV). Any fast-atrial rate or arrhythmias such as atrial fibrillation diminishes the LV filling and thus the cardiac output. The large-sized atrium serves as a nidus for clot formation and so these patients are put-on long-term anticoagulation with drugs like warfarin. This reduces the risk of LA clot or any thromboembolic phenomenon. But at the same time, the dosing of warfarin is to be monitored by regular measurement of INR. The guidelines for target INR for such patients are 3 (2.5–3.5). On the contrary, it increases the risk of bleeding if the INR becomes >4.5.[3] The reported risk of intracranial hemorrhage with anticoagulation is 0.3–1.0% every year and the associated mortality is about 60%. The reported mortality of anticoagulant-related SDH is about 13–20%.[2]

Warfarin is a drug belonging to coumarin derivative which inhibits the carboxylation of vitamin K-dependent clotting factors (factor II, VII, IX, X) in the liver. It has a half-life of 36–48 hours and a duration of action of 4–6 days. Clinically its action is monitored by measuring PT and INR which gets prolonged and increase the risk of spontaneous bleeding if INR is >4.5. Development of SDH is not only grievous but catastrophic if the patient is on anticoagulants.[7] The guidelines for reversal of the anticoagulant effect of warfarin mention the transfusion of FFP and normalization of INR to ≤1.4. This allows the patient to be safely taken for any surgical procedure. Fresh frozen plasma takes 6–8 hours to reverse the effect of warfarin. Besides FFP, prothrombin complex concentrate (PCC) (normalize INR in 1 hour), recombinant factor VII (rapid and complete INR correction but costly), and vitamin K (10 mg I/V in 3 doses take 24 hours) are used.

Anesthetic management of a case of MS on anticoagulation with intracranial bleed having elevated INR poses a challenge for the anesthesiologist. Rapid reversal of the anticoagulant effect of warfarin to normalize INR and reduce the risk of further bleeding *vs* the need for blood transfusion and maintaining the fluid balance to avoid overload, maintain the heart rate and blood pressure

is a tough job to be done. In our patient, four units of FFP were given a night before surgery and repeat INR was found to be 1.33 and then the patient was taken for burr hole. Cardiac induction was done and intraoperatively hemodynamic parameters were maintained with the help of NTG infusion at the rate of 5–10 µg/minute for pulmonary hypertension. We used NTG infusion as we do not have inj. milrinone/inj. amrinone at our center which is used for the management of high pulmonary atrial pressures. We were able to manage patients' hemodynamic parameters with inj. NTG infusion. Postoperatively, the patient was not reversed nor extubated in view of her high pulmonary pressures, risk of pulmonary edema, and risk of re-bleeding. We kept the patient sedated and paralyzed overnight and after confirming her repeat CT brain the next morning, and normal INR report, she was extubated. She was discharged on tenth postoperative day without any neurological deficit.

Conclusion

We conclude that a patient on anticoagulation can anytime present with spontaneous or traumatic bleed but timely management of airway, ventilation, and hemodynamic stability along with a multidisciplinary approach from medicine, cardiologist, neurosurgeon, hematologist, and anesthesiologist together aids to the comprehensive care of such patients.

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