Case Report

Severe aortic stenosis and subarachnoid hemorrhage: Anesthetic management of lethal combination

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Abstract

Despite advances in various modalities of management, subarachnoid hemorrhage (SAH) continues to be associated with high mortality, which is further increased by associated comorbidities. Aortic stenosis (AS) is one such disease which can further complicate the course of SAH. We recently managed a known patient of severe AS, who presented with aneurysmal SAH. Patient was planned for neurovascular intervention. With proper assessment and planning, patient was managed with favorable outcome despite the restrictions faced in the neurovascular intervention laboratory.

Key words: Neurovascular intervention, severe aortic stenosis, subarachnoid hemorrhage

Introduction

Despite many advances in anesthetic techniques, severe aortic stenosis (AS) continues to be associated with high mortality and morbidity. The scenario is further complicated when it coexists with another equally life-threatening illness. We are reporting the management of an unusual case of severe AS in New York Heart Association functional class III, who presented with subarachnoid hemorrhage (SAH).

Case Report

A 50-year-old female patient, a known case of severe AS, presented to us with complaints of acute onset severe headache with nausea and vomiting for 2 days. She had history of marked limitation of physical activities and shortness of breath even on mild routine activity. Clinically, she had no neurological deficit. Heart rate was 70/min and noninvasive blood pressure was 168/70 mmHg. On auscultation, she had a pansystolic murmur in aortic area. Computed tomography (CT) scan [Figure 1] and CT angiography [Figure 2] of head showed SAH (Fisher grade 2) with lobulated, saccular anterior communicating artery aneurysm. Electrocardiogram suggested left ventricular (LV) hypertrophy. Transthoracic echocardiography revealed stenotic, calcified, bicuspid aortic valve with area of 0.7 cm^2, increased pressure gradient (PG) across aortic valve [Figure 3] (mean PG 45 mmHg and peak PG 74 mmHg), concentric LV hypertrophy and good LV systolic function. Complete blood count, liver function, renal function, and coagulation profile tests were normal except hypokalemia (2.9 mEq/L). In that clinical scenario, patient was planned for emergency neurovascular intervention. Patient was classified as American Society of Anesthesiologists IIIE and accepted for anesthesia and

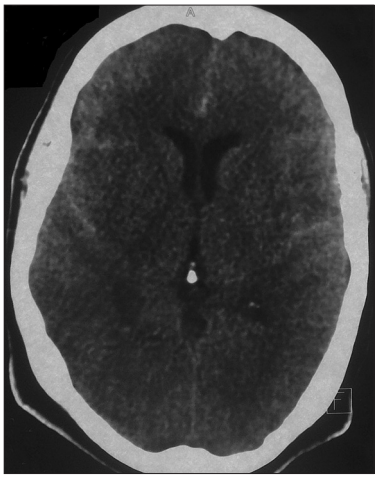

Figure 1: Noncontrast CT head showing subarachnoid hemorrhage (Fisher grade 2)

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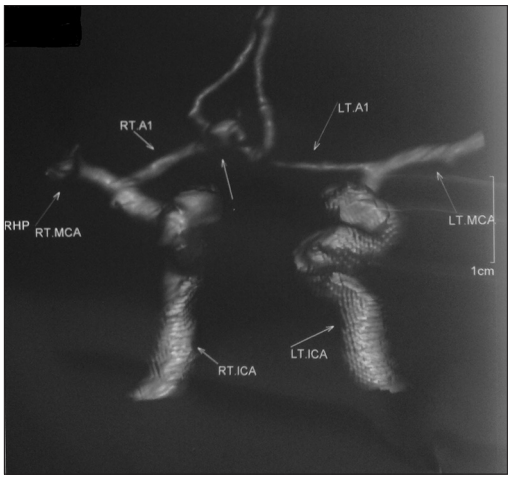

Figure 2: Cerebral CT angiography showing anterior communicating artery aneurysm

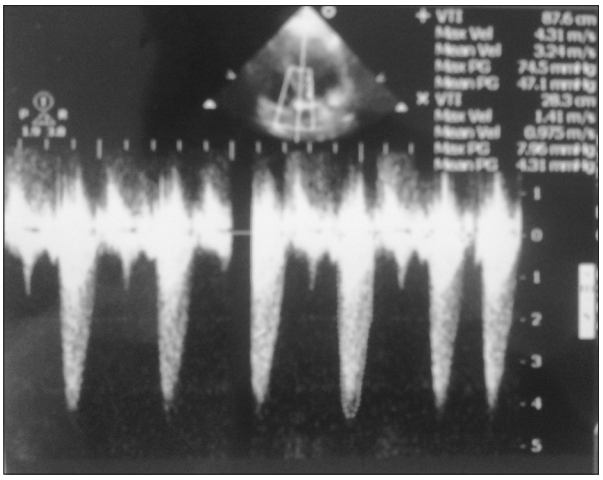

Figure 3: Transthoracic echocardiography with pulse wave Doppler showing high pressure gradient across aortic valve

procedure with high-risk informed consent from the patient and relatives.

After all the preparations, patient was shifted to cerebral digital subtraction angiography (DSA) lab. An intravenous line with normal saline was secured through 14G cannula under local anesthesia (LA). A total of 1.2 g of amoxicillin clavulanate was given intravenously for prophylaxis against infective endocarditis (IE). Monitoring with noninvasive blood pressure, continuous electrocardiography, and pulse oximetry was started. Left radial artery was cannulated with 20G arterial cannula under LA. Central venous pressure (CVP) line was placed with ultrasonography guidance in right internal jugular vein under LA. Continuous invasive hemodynamic monitoring was started. Baseline CVP was 5 mm Hg. Intermittent pneumatic pressure above knee stockings were applied over lower limbs. Esmolol and norepinephrine infusions were kept ready.

Patient was adequately preoxygenated with 100% oxygen. Anesthesia was induced with intravenous etomidate (10 mg) and fentanyl (50 + 25 mcg), followed by vecuronium (6 mg) and lignocaine hydrochloride 2% (4 mL). Gentle direct laryngoscopy and endotracheal intubation was done with endotracheal tube of internal diameter 7.5 mm. Patient was put on volume- controlled ventilation and minute ventilation was adjusted for normocapnia. Anesthesia was maintained with sevoflurane, oxygen in air, and vecuronium. Fentanyl was administered in boluses and titrated according to hemodynamic response. Baseline activated clotting time (ACT) was 159 s. Total of 6000 units of heparin was used for a target ACT of 250-300 s. Potassium chloride was supplemented at 20 mEq/h through central venous line. Intravenous esmolol infusion was started after bolus injection of 25 mg and titrated for heart rate and mean arterial blood pressure. DSA and anterior communicating artery aneurysm coiling lasted for 3 h. Esmolol infusion was titrated from 150-300 mg/h for heart rate from 68 to 77/min and mean arterial pressure from 74 to 80 mmHg. A total of 1 g of intravenous paracetamol was administered toward the completion of procedure. Heparin was neutralized with 60 mg of protamine. At the end of the procedure, patient was shifted to the intensive care unit (ICU) for further management. Neuromuscular blockade was reversed with neostigmine and glycopyrrolate and trachea was smoothly extubated. For the next 4 days, patient was kept in the ICU for cardiac and neurological monitoring. She was discharged from the hospital on 12th day of admission with advice for early aortic valve replacement (AVR).

Discussion

SAH continues to be associated with high mortality and survivors are often left with severe disabilities. Intracranial aneurysm is the most common cause of SAH.[1] AS is the most common valvular heart disease, and its incidence increases with age. In a patient with normal LV systolic function, severity of AS can be classified according to mean Aortic Valve PG, AV area,[2] aortic jet velocity[3] [Tables 1 and 2].

Adverse perioperative risk in patients with AS depends on the interaction of factors such as the severity of valve disease, concomitant coronary artery disease (CAD), and the severity and/or urgency of the surgical procedures.[4] A thorough understanding of AS and its course are necessary for the anesthesiologist, for a good perioperative outcome.

The patient was referred to our institute with 2-day history of severe headache. She had earlier been diagnosed with bicuspid, calcified aortic valve with severe AS. Clinically, she belonged to the NYHA functional class III.[5] Although prior AVR is recommended for patients with severe AS, who are awaiting

Table 1: Severity of aortic stenosis[2]

Measurement	Diagnostic method		Severity of stenosis		
	Cardiac cath	Echo	Mild	Moderate	Severe
Mean pressure gradient	Direct transaortic measurement	Bernoulli equation	<25 mm Hg	25-50 mm Hg	>50 mm Hg
Aortic valve area	Gorlins formula	Planimetry; continuity equation	>1 cm^2	0.76-1 cm^2	<0.75 cm^2

Table 2: Severity of AS (EHJ 2010)[3]

Parameters	Aortic sclerosis	Mild AS	Moderate AS	Severe AS
Aortic jet velocity (m/s)	<2.6	2.6-3.0	3.0-4.0	>4.0
AV area (cm^2)	-	>1.5	1.0-1.5	<1.0
Mean pressure gradient (mmHg)	-	<30	30-50	>50

AS=Aortic stenosis, AV=Aortic valve

urgent high-risk surgical procedure,[3] this was not possible for our patient with ruptured intracranial aneurysm. Consequently, we decided to secure the aneurysm by neurovascular intervention.

Chronic obstruction to LV ejection due to AS, results in concentric LV hypertrophy rendering the heart susceptible to myocardial ischemia, even without CAD. So, large drop in systemic vascular resistance (SVR) should be avoided in these patients.[6] The goal of perioperative hemodynamic management should be to maintain sinus rhythm, myocardial contractility, preload, and SVR. Tachycardia, hypertension, and hypotension increase the risk of myocardial ischemia, so should be rapidly corrected.

For hemodynamic monitoring, pulmonary artery catheter can be inserted in this situation, but it has its own complications. Transesophageal echocardiography is one of the options but is very difficult to use in cerebral DSA lab due to poor patient access. So, we planned for invasive hemodynamic monitoring. Patients with stenotic valvular lesion are considered at high risk for development of IE,[7] so antibiotic prophylaxis was administered with amoxicillin clavulanate.

Lignocaine hydrochloride was administered intravenously 1 min before laryngoscopy, in order to avoid an increase in systemic and pulmonary artery pressures. Propofol is twice as likely as etomidate to evoke hypotension in patients with severe AS.[8] Hence etomidate was used as induction agent. Adequate levels of analgesia and anesthesia were maintained, throughout the procedure in order to avoid tachycardia and hypertension.

Esmolol is a short acting β-adrenoceptor antagonist with half-life of only 9 min and has no sympathomimetic activity.[9] Thus esmolol was administered by intravenous infusion during the whole procedure and titrated for HR and MAP. Hypokalemia has been frequently described in association with SAH and female gender is a risk factor for hypokalemia after SAH.[10]

In conclusion, patients with severe AS require careful preoperative, multidisciplinary assessment, and anesthetic planning, before and during neurointervention to optimize cardiac function and outcome in SAH.

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