Emergency Neurosurgery in a Patient with a Large Thoracic Aortic Aneurysm: Sitting on a Bomb

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Introduction

Large thoracic aortic aneurysms (TAAs) are associated with the fatal complication of rupture, hence are usually repaired by surgical or endovascular modalities promptly.1 When an emergency neurosurgical intervention is required in these patients before thoracic aneurysmal repair, safe conduct of the procedure without causing rupture can be extremely challenging, with little literature to advise safe conduct of anesthesia. We report a case scenario wherein a patient with an unruptured TAA (6.2 cm) underwent an emergency neurosurgical intervention for a subdural hematoma (SDH) prior to aneurysmal repair.

Case Report

An 80-year-old gentleman presented to our hospital with a new-onset headache and two episodes of seizures. He presented with a Glasgow Coma Scale of 14/15 (E4V4M6) with bilateral pupils normal in size and reaction. There were no other significant focal neurological deficits. Computed tomography imaging of the brain showed a large left-sided chronic SDH of > 5 mm thickness. He was planned for evacuation of hematoma, and a 6.2-cm large TAA with intramural thrombus was diagnosed during his preoperative workup (►Fig. 1). The cardiothoracic surgery team was consulted, and it was decided to perform SDH evacuation and middle meningeal artery ablation with subsequent frozen elephant trunk surgery combined with endovascular aneurysmal repair in a separate sitting.

Preanesthetic evaluation was done. The patient presented with new-onset hypertension, and was started on metoprolol 25 mg. He had no other premorbid conditions. His functional capacity was good. He was not taking any medications prior to the episode of seizure. After the seizure, levetiracetam (500 mg)
was started twice a day. There was no history of drug allergies, past surgeries, or anesthesia exposure. After due discussion with the patient about the need for SDH evacuation (drowsiness, seizures), risks of nonevacuation (the concerns of hematoma expansion due to systemic heparinization during aneurysm repair), and the high probability of complications associated with the coexisting unrepaired AA (mortality with aneurysmal rupture, embolic complications like stroke and myocardial infarction), informed consent was obtained for the neurosurgical intervention under high risk.

Anesthetic plan was to conduct the evacuation under monitored anesthesia care (MAC). His hematological and biochemical investigations were unremarkable. The cardiothoracic surgical team and cardiopulmonary bypass were standing by to take over in the event of inadvertent aneurysmal rupture during the procedure. Patient was shifted to the operating room (OR), monitors were applied, and baseline vital parameters were recorded. Fifty micrograms of fentanyl, 4 mg of ondansetron, and 1000 mg of levetiracetam were administered intravenously. The radial artery was cannulated under local anesthesia for invasive blood pressure monitoring. After starting an infusion of dexmedetomidine (0.2–0.7 mcg/kg/h titrated to effect), scalp block was given with 20 mL of 0.5% ropivacaine. Glyceryl trinitrate infusion was also administered titrated to maintain systolic pressures less than 130 mm Hg, with a view to avoid increases in transmural pressures. Surgery commenced after the confirming the adequacy of block. Minicraniotomy was done, and local anesthetic-soaked pledges were applied over dura before incision. Dexmedetomidine was titrated according to the sedation requirements. Patient’s resting heart rate was 61 beats/min. Throughout the procedure, heart rate ranged between 60 and 70 beats/min. Duration of surgery was 105 minutes. Procedure was uneventful with minimal blood loss (50 mL). Patient was neurologically stable postsurgery and was shifted to the intensive care unit for close monitoring (►Fig. 2). There were no new ischemic changes in electrocardiogram in the intraoperative or postoperative period. He now is being planned for aneurysmal repair.

**Discussion**

TAAs are rare, often diagnosed incidentally (as in our patient), and they can be asymptomatic. Dreaded complications due to large unrepaired TAA are aortic dissection and rupture (referred to as acute aortic syndromes) as they are associated with high mortality rates (> 50% without emergency intervention and 7–25% with emergency intervention). The risk of rupture increases multifold once the size of TAA crosses 6 cm. Hence, TAA > 5.5 cm in ascending aorta and > 6 cm in elderly patients with multiple comorbidities need to be surgically corrected and any other noncardiac procedure should be deferred until after the aneurysm surgery. If the noncardiac surgery is lifesaving, they are to be scheduled concomitant with aneurysm repair when the aneurysm is larger than 5 cm in diameter. However, if the noncardiac condition necessitates acute intervention, and nontreatment can potentially hinder the conduct of the aneurysm repair, there is a difficult decision to make. With the concerns of hematoma expansion during the aneurysm repair and repeated seizures necessitated SDH evacuation along with middle meningeal artery ablation in our patient.

Our challenge was conducting an emergency neurosurgery (SDH evacuation) in an 80-year-old hypertensive patient with a coexisting large unrepaired TAA. The major anesthetic concerns in this patient were the occurrence of acute aortic syndromes, acute coronary events, and concerns pertaining to the compression of surrounding thoracic structures.

![Computed tomography (CT) angiography showing presence of large thoracic aortic aneurysm with intramural thrombus.](image-url)
The term acute aortic syndromes refer to aortic dissection and aortic rupture. Dissection is more common than rupture in TAA, and both need immediate surgical intervention. Both can present as sudden severe pain in the chest/back, hypotension (rupture into pleura or mediastinum), massive hemoptysis (tracheobronchial rupture), or hematemesis (esophageal rupture). Factors contributing to rupture are mainly the size of the aneurysm, associated medical conditions and the sudden blood pressure fluctuations. Of these, the one factor that can be directly influenced by anesthesiologist in the OR is the patient's blood pressure. Sudden fluctuations of blood pressure increase the pulsatile load or the stress on the aortic wall (dp/dt) leading to aneurysmal expansion, dissection, or rupture. The safe blood pressure thresholds advocated in various guidelines range between 130/80 (American) and 140/90 (European) mm Hg. In case of a dissection, acute reduction of systolic blood pressure (SBP) < 120 mm Hg within 20 minutes is recommended to prevent progression or rupture.

Apart from risk of rupture, TAAs carry the additional risk of symptoms due to compression of surrounding structures leading to chronic cough, wheeze, dyspnea, VQ mismatch and hypoxia, recurrent pneumonia, hoarseness, hemoptysis, dysphagia, or rarely severe hemodynamic collapse. Chest pain or back ache can be seen in TAA even without dissection due to probable bony compression or erosion. The severity of these symptoms preclude surgery under MAC. Other concerns in our patient were rebleed, intraoperative seizures, and stroke.

Anesthetic technique and management in our patient targeted meticulous blood pressure management, avoidance of coughing/bucking, vigilance, and preparedness for early detection and management of complications. MAC with scalp block was chosen as the anesthetic technique with an intention to avoid coughing/bucking, as intermittent glottic obstruction in these patients can present an afterload challenge to the left ventricle, distend the aorta, and lead to dissection or rupture. Despite the size of aneurysm, our patient did not have any compressive symptoms and was able to lie comfortably for the duration of the procedure and tolerate neurosurgery under MAC.

Adequate analgesia and sedation were ensured all through the duration of surgery and acute blood pressure surges were managed with vasoactive agents, targeting SBP between 120 and 130 mm Hg. While under MAC, sudden and severe chest and/or back pain with or without hemodynamic collapse could point to the occurrence of acute coronary syndrome or aortic syndrome. In an anesthetized patient, electrocardiographic changes and transesophageal echo should be utilized for prompt diagnosis. Middle meningeal artery was ablated to prevent recurrence of bleed. Hemodynamic monitoring and analgesia were continued in the postoperative period. Beta blockers showed mortality benefit and reduced expansion rates and are routinely prescribed in patients with TAA. Statins, though lacking direct benefit in TAA, are still prescribed in patients with concurrent atherosclerotic disease. Both were continued in our patient.

Reports exist in the literature wherein nonaneurysmal surgery was conducted in patients with untreated aortic aneurysms. Cheng et al reported an orthotic liver transplantation, and Baptista et al reported an orthopaedic surgery, both in patients with unrepaired aortic aneurysms. According to both the reports, the prime strategy was meticulous blood pressure control and vigilant monitoring.

Conducting a neurosurgical procedure in a patient with unrepaired TAA is associated with high mortality and morbidity mainly due to the risk of acute aortic syndromes and embolic complications. Avoiding acute blood pressure fluctuations intraoperatively and close vigilance to detect complications are the prerequisites for successful anesthetic management.

Conflict of Interest
None declared.
References

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