Surgical evacuation of subdural hematoma in a patient with transplanted heart under anesthesia

Sir,

It is not very common scenario where a patient with transplanted heart undergoes noncardiac surgery. However, anesthetic management of such patients in the context of neurosurgical emergencies has never been described. A 42 kg 19-year-old male presented to our hospital with a headache and vomiting for 2 days. A noncontrast computed tomographic (CT) scan of head showed subacute subdural hematoma (SDH) in the right frontotemporal region with midline shift and mass effect [Figure 1]. He was a known case of restrictive cardiomyopathy and underwent heart transplantation 42 days back. His cardiac evaluation revealed a left ventricular ejection fraction of 25% and was on immunosuppressant therapy with cyclosporine, caspofungin, and methylprednisolone. On examination, he was malnourished with no neurological deficit. He had severe ascites but without pallor, icterus, or pedal edema. His laboratory investigation including renal, hepatic, and coagulation parameter was within normal limit. The baseline heart rate and blood pressure were 81/min and 112/56 mmHg, respectively, and electrocardiography showed normal sinus rhythm. An emergency burr-hole evacuation under anesthesia was planned. He was put on intravenous mannitol 100 mg every 8 h and furosemide 40 mg twice daily. In the operating room, the routine monitors were applied and an arterial line was placed; the patient was induced with etomidate 12 mg and fentanyl 75 µg, and endotracheal intubation was facilitated with atracurium. Sevoflurane was used for maintenance of anesthesia with oxygen and air (1:1), and the patient was put on mechanical ventilation with a target end-tidal CO₂ of was 35 ± 2 mmHg. He remained hemodynamically stable throughout the surgery. A volume of 1500 ml of normal saline was infused over 90 min periods. At the end, the neuromuscular blockade was reversed; however, the trachea was not extubated, as patient not able to follow commands and was shifted to Intensive Care Unit (ICU) for further management. Six hours later, the patient suffered an episode of generalized tonic–clonic seizure for 30 s which was managed with midazolam 2 mg followed by phenytoin infusion. Trachea was extubated 18 h later, and the patient was kept in ICU for the next 24 h before being shifted to the ward.

Acute SDH is associated with high mortality and an unfavorable long-term outcome.[1] In our patient, no noticeable risk factors other than ongoing immunosuppressant therapy could be identified which would have led to the occurrence of SDH. A clinical picture as described above requires appropriate interpretation to avoid potential problems. Surgical intervention has to be done as soon as possible to avoid ischemic and hypoxic brain damage owing to herniation. With the increased frequency of heart transplantation, this rare complication of SDH should be kept in mind. Patients who present with a headache and vomiting with nonspecific etiology must be followed up after transplantation. The rejection of transplant heart is common within the first 3 months[2] and may be the reason for high mortality if the patient undergoes noncardiac surgery.[3] Our patient reported on 42nd day, hence, an extensive preoperative workup was done on an emergency basis which included consideration for risk of infection, drug interactions, impact of immunosuppressant therapy, and graft viability apart from the functional status of the heart. More than half of these recipients present with dysrhythmias due to increase catecholamine or a lack of vagal tone.[3] Sound knowledge of the physiological changes due to denervated heart is essential for appropriate anesthetic management. As such patients are on corticosteroid therapy, an additional dose of glucocorticoids before induction may be preferred.[4]

![Figure 1: Computed tomographic scan of head showing axial view showing subdural hematoma on the right frontotemporal region with midline shift and mass effect](image1.jpg)
In view of raised intracranial pressure (ICP), the induction should be smooth; avoid any increase in ICP during laryngoscopy which can be blunted by administration of lidocaine, fentanyl, or esmolol. The goal is to avoid intraoperative hypotension, hypoxia, and hypo/hypercarbia. Hypovolemia, hypotension, and blood loss should be optimized urgently as denervated heart lack the ability to respond as compare to normal one. Recipient heart is “preload dependent” as stroke volume is dependent on venous return. Etomidate is effective in maintaining hemodynamic stability during induction and intraoperative hypotension can be managed with optimal fluid bolus and direct acting inotrope, for example, isoproterenol.

To conclude, we successfully managed a case of transplanted heart with subacute SDH who underwent uneventful burr-hole evacuation under anesthesia. It is emphasized that the anesthesiologist should understand the pathophysiology of denervated heart, implications of immunosuppressive medication, risk of infection apart from the neuroanesthetic concerns during perioperative management of such patients.

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Conflicts of interest
There are no conflicts of interest.

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Necrotizing fasciitis following spinal anesthesia: A rare and devastating complication
Sir,
A 23-year-old female was admitted to the surgery ward with the chief complaint of swelling over the left side of the back, with pus discharge for the last 16 days. Pain and swelling were accompanied by fever and chills. She underwent cesarean section delivery 20 days back at a local nursing home where spinal anesthesia was administered. She developed pain and redness over injection site on the 4th postoperative day. She consulted operating surgeon, and she was advised to consult a general surgeon. She came on the 20th postoperative day. On examination, she was found drowsy, dehydrated, and febrile (temperature 40.5°C). She had tachycardia and hypotension. Blood chemistry revealed hemoglobin of 8 g/dl, total white blood cell count of 26,000/cumm with 15% band cells, serum creatinine of 2.0, and serum glutamic-pyruvic transaminase of 100 IU/L. Arterial blood gas analysis revealed mild metabolic acidosis. Intravenous antibiotics and vasopressors