A stitch in time saves life: Massive tumor embolism

Madam,
We operate 180–200 renal cell carcinomas (RCCs) annually with 10–15 having vena caval extension. Literature reports embolism in 1.5% RCC with mortality as high as 75%.[1,2] We describe a case of tumor embolism in which a catastrophic event was averted by the prompt and coordinated anesthetic and surgical intervention.

A 59 American Society of Anesthesiologist II female with a diagnosis of RCC was posted for radical nephrectomy. Magnetic resonance imaging (MRI) showed left renal mass measuring 5.2 cm × 3.7 cm abutting the inferior vena cava (IVC). Her preanesthetic evaluation was remarkable with no comorbidities. She was planned for general anesthesia with epidural analgesia and induced with intravenous propofol and morphine. The tumor was vascular, extending 3–4 cm into the IVC. The dissection went uneventful. Vascular clamps were applied on suprahilar IVC, right renal vein along with infrahilar IVC, and radical nephrectomy was performed. Left renal along with the...
thrombus in situ was secured in the clamp. The thrombus was retrieved from the left renal vein after releasing its clamp. After ensuring complete thrombus retrieval (by palpation of the IVC), the clamps were released. As soon as the clamp was released, there was sudden hypotension and the blood pressure dropped to 60/30 mmHg. The central venous pressure rose to 25 cm H$_2$O. On suspicion of tumor embolism, 5000 units heparin intravenous (IV) was given. Two units of packed cell blood were transfused, and IV infusion of noradrenaline at 0.2 mcg/kg was started. The blood pressure rose to 128/75 mmHg, and a mean arterial pressure of 75 mmHg and above was maintained throughout the surgery. The arterial blood gas showed pH - 7.339, PaO$_2$ - 200, PCO$_2$ - 40.1, HCO$_3$ - 21.1, and Base excess (BE) - -4.3. The patient was extubated after complete reversal of neuromuscular blockade. Five minutes following extubation, the patient became unresponsive. There was an episode of ventricular tachycardia with hemodynamic instability which was reverted on cardioversion. The patient was reintubated, and the hemodynamics was maintained on noradrenaline 0.2 mcg/kg/min, adrenaline 0.1 mcg/kg/min, and dobutamine 2 mcg/kg/min. The computed tomography pulmonary angiography confirmed the diagnosis of pulmonary thromboembolism [Figure 1]. The right side cardiac chambers were dilated with reflux in inferior vena cava. The patient was shifted for embolectomy. Multiple fresh and organized thrombi were removed from both right pulmonary artery, left pulmonary artery and its 2nd generation branches. The patient was extubated on day 2 and discharged on day 10 from the hospital without any sequelae.

RCC with intravascular tumor thrombus is seen in 10% cases and is associated with severe cardiopulmonary morbidity and mortality. [3]

Preoperative characterization of tumor thrombus (Level 0 thrombus limited to the renal vein, Level I <2 cm into the IVC, Level II >2 cm in the IVC below hepatic veins, Level III extends above hepatic veins but below the diaphragm, Level IV above the diaphragm, including atrium) through an MRI is required to plan intraoperative management. [4]

Vascular bypass is classically indicated in Level III or IV tumor thrombus to facilitate safe resection. [3] In our case, it was Level II tumor extension into the IVC which demands complete caval isolation with control of the infrarenal and suprarenal IVC, contralateral renal vein, and lumbar veins. As the clamp was placed on the IVC, there could have been a possibility of thrombus discharge from the lumbar veins. Aggressive surgical technique or prolonged cavotomy can also add to the risk of thrombus discharge.

Through this case report, we aim to highlight that in a patient with suspected intraoperative embolism, prompt and coordinated multidisciplinary approach can revert the catastrophic event.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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Epiglottic cyst in Von Hippel-Lindau syndrome: Shared pathology or a separate entity?

Madam,

Von Hippel-Lindau (VHL) syndrome is a genetic disorder characterized by hemangioblastomas involving central nervous system and cysts in the liver, kidney, and pancreas. We encountered a cyst in the upper airway of a patient with VHL syndrome that led to difficulty in airway management.

A 43-year-old man with VHL syndrome was planned for excision of a recurrent cerebellar hemangioblastoma. Ultrasound of the abdomen revealed multiple cysts involving pancreas and kidneys. He gave no history of voice change, snoring, difficulty in swallowing or breathing. He had limited neck movements, which we attributed to previous two midline suboccipital craniotomies. Previous anesthesia records revealed no difficulty in airway management. The patient was induced with intravenous propofol, fentanyl, and vecuronium. Laryngeal structures could not be visualized during initial intubation attempt with Macintosh size 4 blade. Hence, mask ventilation was continued and intubation attempted with C-Mac Video Laryngoscope (Karl Storz, Tuttlingen, Germany). It revealed a pedunculated, 2.0 cm × 2.0 cm, smooth surfaced lesion in the vallecula obstructing the view of glottis [Figure 1]. With gentle manipulation of the scope, posterior part of the glottis could be visualized. Using a gum-elastic bougie, trachea was intubated avoiding injury to the lesion. Extubation and postoperative period were uneventful.

The pathogenesis of VHL syndrome involves mutation in chromosome 3p. This leads to abnormalities in tumor suppression, microtubule stability, cilia formation, and assembly of collagen (Type IV) and fibronectin in extracellular matrix, resulting in visceral cysts. Although not described previously, there is theoretical possibility that the pathology can involve airway mucosa as it also consists of various types of epithelial cells, cilia, Type IV collagen, and fibronectin.

Asymptomatic airway cysts can cause difficulty in mask ventilation by ball valve mechanism, difficulty in endotracheal intubation by obscuring glottic view, and difficulty in supraglottic airway placement by altering the anatomy of laryngeal inlet. Aggressive airway manipulation can cause rupture, and spillage of its contents bleeding from the cyst can cause aspiration and may even precipitate a “cannot ventilate – cannot intubate” scenario. In such scenarios, the attending anesthesiologist should be prepared to maintain ventilation and oxygenation of the patients. The alternative plans for securing the airway after induction of anesthesia will depend on the degree of difficulty, anesthetic agents used, availability of alternative airway gadgets, and the experience of the anesthesiologist in using them. The epiglottic cyst found in our case could be a casual coexistence or part of the syndrome itself. Preoperative endoscopic airway examination (PEAE) has shown to affect the clinical examination-based airway management plan in patients presented with airway pathology. However, the advantage of routine preoperative use of PEAE in asymptomatic patients is questionable considering the rare incidence of unanticipated difficult airway. Since VHL syndrome is associated with cysts involving various organs such as the pancreas, liver, and kidney, it would be wise on the part of the anesthetist to expect cysts in the airway as well. Obtaining careful history related to airway symptoms and using indirect laryngoscopy or any other endoscopic or...