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General Anesthesia on A Patient with Suspected Superior Vena Cava Syndrome in Cardiac Tamponade for Subxiphoid Tube Pericardiostomy: A Case Report

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Abstract:
Induction of general anesthesia in patients with cardiac tamponade can cause significant risks. All general anesthetics are myocardial depressants, and most are peripheral vasodilators. In this report, we discussed a 61-year-old woman with pericardial effusion and other signs suggestive of cardiac tamponade, together with facial edema and engorgement of neck veins, scheduled for subxiphoid tube pericardiostomy and pericardial biopsy under general endotracheal anesthesia. The patient was intubated successfully with no noted events on induction and during the perioperative period and immediately postoperatively. To conclude, general anesthesia may be safely performed in patients with cardiac tamponade but must be tailored according to tamponade physiology and their presenting signs and symptoms.

Introduction:
Cardiac tamponade is a medical emergency which occurs when excessive fluid accumulates in the pericardial sac, resulting in compression of the cardiac chambers and restricting filling causing hypotension and possibly cardiac arrest (Adoniadis et al, 2023). The anesthetic management of patients undergoing pericardial drainage should be tailored specifically to the etiology, acuity and presence of signs and symptoms, as well as the surgical approach to be done. Reported here is a case of a patient presenting with cardiac tamponade, with symptoms of facial edema and neck vein engorgement suggestive of superior vena cava syndrome, who underwent subxiphoid tube pericardiostomy and pericardial biopsy under general anesthesia.

Case Report:
A 61-year-old, 50 kg woman with a history of stage II hypertension and dyslipidemia was admitted to the emergency department due to a 1-month history of gradually enlarging right breast with associated facial edema and exertional dyspnea. Her laboratory and physical examination were within normal limits except for bilaterally engorged neck veins, prominent superficial veins. muffled heart sounds on auscultation and an enlarged right breast. The signs and symptoms were suggestive of superior vena cava syndrome, although no chest imaging was done at this point due to urgency of procedure. Point-of-care ultrasound

done by Cardiovascular Services revealed a large pericardial effusion with swinging heart, along with shuddering of the right ventricular output tract during diastole which is a sign of beginning tamponade. Because of this the patient was scheduled for emergency subxiphoid tube pericardiostomy and pericardial biopsy.

Patient was evaluated at the emergency department wheelchair borne. Able to speak in full sentences with no chest pain or dyspnea. Initial vital signs taken were a blood pressure of 120/80 mmHg, heart rate of 98/minute, respiratory rate of 22/minute and oxygen saturation of 98% at room air. On airway evaluation, the patient had a Mallampati score of 2, with mouth opening of at least 5 cm and thyromental distance of at least 7.5cm although edentulous. Her consciousness level was at GCS 15. General anesthesia was the chosen technique because of possible collapse of airway collapse due to the presence of facial edema and probable edema of the upper airway due to venous congestion. Prior to induction, ASA standard monitors were applied such as 5-lead ECG, pulse oximetry and non-invasive blood pressure cuff at the right arm. Two large-bore IV access were inserted in the great saphenous veins of both feet. An invasive arterial blood pressure monitor was also inserted in the patient's left radial artery. The patient had a blood pressure reading of 180/111mmHg, heart rate of 111 bpm and pulse oximetry of 99% at room air. After preoxygenation, the patient was induced using midazolam at 3mg (0.06mg/kg) IV, ketamine at 50mg (1mg/kg IV) and neuromuscular blockade with rocuronium at 50mg (1mg/kg) IV. After adequate depth of anesthesia, indirect visualization of the glottic opening was performed with video laryngoscopy and successful atraumatic intubation was done using a 7.5mm endotracheal tube on the first attempt. Maintenance of anesthetic consisted of volume-controlled mechanical ventilation, (50% oxygen, 50% medical air-delete) with following ventilatory setting: 60% fraction of inspired oxygen, tidal volume of 350 mL. Sevoflurane 2-3% was maintained MAC 0.8(?) was maintained for this procedure without the need for additional neuromuscular blocker other than rocuronium at induction. Intraoperative blood pressure and heart rate were maintained within 110-130/60-70mmHg using norepinephrine infusion titrated between 0.05 - 0.1 mcg/kg/min, while oxygen saturation was maintained at 98-99%, end-tidal carbon dioxide between 30–35 mmHg, and airway pressures less than 25 cm H2O. Baseline point-of-care testing was done prior to surgical incision showing compensated respiratory alkalosis, hypokalemia and hypocalcemia with a base deficit of -5.3. Electrolyte correction was done using hydration with plain lactated ringer's solution and potassium chloride.

Local infiltration with 3 mL of lidocaine 1% was applied over the surgical site prior to incision. Subxiphoid tube pericardiostomy with pericardial biopsy was performed without complications, intraoperative findings included a non-thickened pericardium together with drainage of approximately 800cc of serosanguinous fluid. The procedure lasted for 1 hour. Neuromuscular blockade reversal was done using sugammadex at 4mg/kg. The patient was extubated once she was fully awake and followed commands. The patient was observed with improvement of facial edema and orthopnea and was discharged from the hospital at the 10th post-operative day.

Discussion:

Cardiac tamponade and superior vena cava syndrome can present with very similar clinical features, including cough, dyspnea, orthopnea, jugular venous distention, and pulsus paradoxus (Webster et al, 2003). The hemodynamic goals pertinent in the physiology of cardiac tamponade relate to maintaining adequate preload, avoiding bradycardia, maintaining high systemic vascular resistance and conserving optimal contractility by avoiding myocardial depression (Madhivathanan et al, 2020). Surgical drainage

of pericardial effusion is usually done under general anesthesia. In these patients, acute decompensation may occur abruptly due to a combination of myocardial depression, vasodilation and decreased preload. During drainage of pericardial fluid, administration of intravenous fluids is important to restore preload an maintain adequate hemodynamic perfusion. The use of vasopressors such as norepinephrine to maintain systemic vascular resistance, and dobutamine to sustain cardiac contractility may also be utilized. Successful volume expansion primarily depends on the outcome measures used to define it such as cardiac index, end organ perfusion, or patient symptom relief, the type of tamponade, and the patient's overall fluid status. Hypovolemia, especially in the context of tamponade, has particularly pronounced effects. A single fluid challenge is likely beneficial, especially in cases of hypotension (<100mmHg). However, excess fluid administration risks worsening ventricular interdependence and decreasing cardiac output. Fluid management as a bridging measure is crucial for patients with poor preload, and a single fluid challenge is unlikely to cause harm. Subsequent fluid boluses should be carefully assessed, as they are unlikely to provide additional benefits.

In patients who are severely hemodynamically decompensated, pericardiocentesis via the subxiphoid incision may also be performed under local anesthesia, but this may be more difficult for certain patients where impending cardiovascular collapse is expected and immediate airway support is required (Yoshida et al, 2021). Providing positive pressure ventilation, as observed in mechanically ventilated patients undergoing general endotracheal anesthesia, produces characteristic hemodynamic changes which are opposite to that on spontaneous breathing: An inspiratory reduction in right ventricular preload due to a reduction in venous return and increase in afterload.

Anesthesia can be maintained using a balanced technique involving volatile inhalation agents, intravenous opioids, ketamine, and short- or intermediate-acting neuromuscular blocking agents. If intraoperative toe (IOT) is available, it can be particularly useful for monitoring and guiding hemodynamic management, especially after cardiac surgery. IOT also helps ensure adequate drainage of the effusion. After chest opening and surgical evacuation, there may be a rebound hypertension, during which anesthesia is deepened, and doses of vasopressor agents are reduced. To optimize coagulation management, point-of-care coagulation monitors (e.g., thromboelastographic) can be used if ongoing bleeding is a concern. Dramatic improvements in the hemodynamic state typically occur after fluid drainage in patients with tamponade. Postoperative pain relief can be achieved with longer-acting opioids (e.g., morphine, oxycodone). Depending on the surgical approach, regional anesthetic blocks such as intercostal nerve blocks may be appropriate. Postoperative care is dictated by the need for ongoing organ support and continuous monitoring. Usually, a period of observation in a critical care environment is required after drainage of an acute effusion.

Conclusion:

In patients with cardiac tamponade undergoing subxiphoid tube pericardiostomy, careful attention to hemodynamic goals, avoidance of SVR-reducing anesthetic agents, administering vasopressors to maintain SVR are the desired optimum management tools if maintenance of spontaneous ventilation are not feasible.

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