Superior Vena Cava Clamping during Thoracic Surgery: Implications for the Anesthesiologist

Abstract

Resection and reconstruction of the SVC is a challenging Intraoperative situation owing to the potential complications after clamping a patent vessel. Hemodynamic imbalance and neurological effects of SVC clamping can be life threatening. These complications can be prevented by careful intraoperative monitoring and management. Anaesthesiologist must be aware of different options to manage such challenging situations.

Keywords: Neuroprotective, superior vena cava, thoracic surgery

Introduction

Mediastinal mass (benign and malignant) can involve superior vena cava (SVC) mandating its surgery also. Complication of SVC injury caused by thoracic trauma or some diagnostic and therapeutic procedures such as mediastinoscopy may also lead to need for surgery on SVC. Patients with long-term central venous catheters or permanent pacing electrodes may develop stenosis and obstruction of the SVC requiring its repair.[1] Such patients needing SVC reconstruction may have the presence or absence of SVC syndrome. Significant SVC compression along with collateral vein networks formation is indicated by clinical findings such as swelling of the head, neck and upper limbs, profound dilated veins over the upper half of the body, and cyanosis. However, the absence of these features of chronic SVC compression suggests lack of adequate collateral venous networks. This also means venous return from the head will be severely hindered when SVC is clamped acutely.[1] It is difficult to manage the hemodynamic consequences of sudden clamping of SVC. Unanticipated clinical situation may land up patient in poor postoperative outcomes.

Case Report

A 45-year-old male patient presented with shortness of breath on mild exertion since 1 month. His chest X-ray - posterioranterior view [Figure 1] and computed tomography

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scan revealed large soft tissue density mass lesion (12.5 cm \times 12 cm) with areas of necrosis and internal calcification in the right side of mediastinum [Figure 2]. The mass was impinging on right atrium and SVC. There was absence of left SVC. Histopathological examination of the mass was suggestive of malignant leiomyosarcoma. The patient was scheduled for surgical removal of tumor. After smooth anesthesia induction, the patient was intubated with left-sided double-lumen tube considering the need for split-lung ventilation. Femoral venous sheath 6 Fr. was inserted for fluid resuscitation. Intraoperative transesophageal echocardiography examination revealed mass around right atrium and SVC [Figures 3 and 4]. After midline sternotomy, anterior mediastinal was resected. Considering the malignant potential of the tumor, it was decided to remove the encased portion of SVC during the surgery. Preparations for cardiopulmonary bypass institution were kept in standby. SVC was clamped and transected proximal to mass, and prosthetic graft (polytetrafluoroethylene PTFE-Gore-Tex 18 mm) was anastomized after adequate heparinization. Nasopharyngeal temperature maintained at 34°C with the help of cooling blanket and cooling ambient operating room temperature. Intravenous thiopentone 1 g was given. Total SVC clamping time was 55 min. Low-dose vasopressornoradrenaline 0.03 µg/kg/min was started

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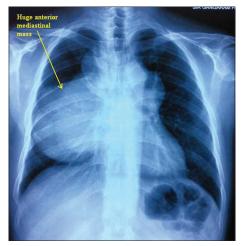


Figure 1: Chest X-ray showing anterior mediastinal mass

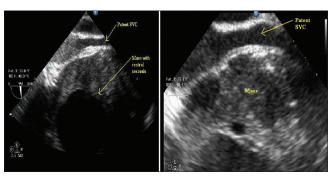


Figure 3: Transesophageal echocardiography image with bicaval view (zoom view-right panel) showing mediastinal mass lateral to superior vena cava but without any infiltration or compression

to maintain hemodynamics. Mean arterial pressure was maintained between 80 and 85 mm Hg. After completion of anastomosis, free flow of SVC was verified on transesophageal echocardiographically [Figure 5]. Intraoperatively and postoperatively, the patient was given reverse Trendelenburg position. The patient was extubated after 6 h of surgery. Neurological functions were normal postoperatively.

Discussion

SVC clamping in conditions such as invasive tumor-like thymoma or lung carcinoma with obstructed SVC would not cause hemodynamic effects due to well-developed venous collaterals. [2] In patients with unobstructed SVC, its clamping can cause significant hemodynamic effects due to the absence of alternate venous channels. Venous return from SVC contributes to 50% of cardiac output in children and 35% in adults. [3] SVC clamping would cause decreased cardiac output and mean arterial pressure on one side and raised cerebral venous hypertension on other side. Resultant effect is fall in cerebral perfusion pressure with potential to cause irreversible brain damage. [4] Experimental studies in animals anesthetized with barbiturates have shown safe duration of SVC to be around 1 h. [5] Measures to reduce central venous pressure and cerebral metabolic rate (CMRO2)

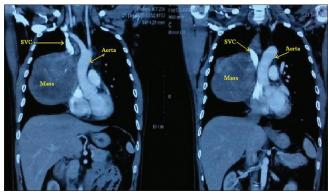


Figure 2: Computed tomography showing mediastinal mass encasing superior vena cava

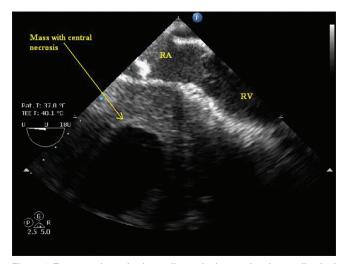


Figure 4: Transesophageal echocardiography image showing mediastinal mass lateral to right atrium and right ventricle

can effectively allow to increased SVC clamping time. [4,5] However, fluid infusion through cannula placed in upper limbs would cause further rise in cerebral venous pressure. Femoral venous or right atrial cannula would be effective in fluid resuscitation. Neuroprotective methods such as use of thiopentone and mild hypothermia can be used. Cerebral oximetry using near-infrared spectroscopy technology should be considered during such surgery.

Use of extracorporeal circulation electively or urgently during the resection of locally advanced thoracic malignancies has been described. [6] Cardiopulmonary bypass provides safety net in situations of vascular damage during mass resection. SVC decompression can be done by creating temporary venoatrial shunt or cavopulmonary anastomosis for drainage of the SVC. [2] Perentes *et al.* described a new temporary SVC bypass technique - preoperative jugulo-femoral bypass or intraoperative innominate-femoral bypass using standard perfusion tubing. SVC clamping induced jugulofemoral pressure gradient drives blood from jugular to femoral vein without any need of a pumping device or additional heparinization beyond that required for SVC reconstruction. [7]



Figure 5: Transesophageal echocardiography bicaval view showing free flow of superior vena cava with no turbulence on color Doppler after excision of the tumor

Alternatively, a temporary bypass between right atrium and innominate vein^[8,9] or axillosaphenous,^[10] axillofemoral,^[11,12] venovenous bypass using perfusion tubing and cavopulmonary anastomosis^[2] for SVC decompression, before SVC clamping, has also been reported. Advantages of such system are simplicity, no need of heart–lung machine and full heparinization, low costs, and reduced inflammatory response. However, it would always be imperative to keep preparations for cardiopulmonary bypass intraoperatively in case of hemodynamic instability or significant bleeding, and it would also permit the application of hypothermia.

Hemodynamic imbalance and neurological effects of SVC clamping can be life-threatening. Non-programmed clamping of SVC can be the anesthesiologist's Achilles' heel. Anesthesiologist must be aware of different options to manage such challenging situations.

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.

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Nil

Conflicts of interest

There are no conflicts of interest.

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