

Superior Vena Cava Syndrome and Intensive Care Process Following Mitral Valve Surgery

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Introduction

Superior vena cava syndrome (SVCS) results from the impairment of blood flow through the superior vena cava (SVC) to the right atrium. This blockage which may be caused by a mediastinal mass enlargement or thrombosis of the lumen also obstructs pulmonary parenchyma and cause respiratory problems [1]. Thrombosis of the SVC can be produced by the presence of catheters and pacemaker leads or by ruptures or lacerations during surgery and can lead to acute clinical manifestations [2]. In this case report, we aimed to evaluate a patient with SVCS in the early postoperative period after cardiac surgery.

Case Presentation

A 65-year-old woman with mitral insufficiency underwent surgery. She was transferred to the intensive care unit (ICU) in the postoperative period and ventilated with 50% oxygen using pressure-controlled ventilation. Four hours after surgery, the patient was reevaluated due to respiratory deterioration with lower tidal volume. There was no obstruction or plug in the respiratory tract and no evidence of pneumothorax or hemothorax in the chest X-ray. During this period, she was hemodynamically stable. Transthoracic echocardiography (TTE) was performed urgently after the appropriate tidal volume and lung protective ventilation values were maintained. TTE showed that blood flow decreased through SVC due to thrombosis and heparin infusion was started immediately. Two hours later the patient showed major hemodynamic deterioration, edema, and cyanosis of the head, chest, and upper limbs as in classical SVCS (Figure 1) so emergent redo surgery was performed. SVC was bypassed using an endovascular prosthesis (10cm ring PTFE graft) to provide adequate flow. Repeated TTE demonstrated patent SVC. The patient showed clinical improvement within 24 hours and was extubated on the postoperative second day.



Figure 1: The color change observed in the upper half of the neck due to SVCS.

Discussion

Malignant diseases are the most common cause of SVCS. Iatrogenic causes are the second most frequently reported SVCS cause, especially in association with intravascular devices like central venous catheters or pacemaker leads. Although extremely rare, ruptures or lacerations can also occur during surgery of the upper mediastinum, as in the present case. SVCS is a rare but highly mortal complication that may be encountered after cardiac surgery. The rapidity of symptom onset mainly depends on the speed of the obstruction, therefore in the present case, SVC obstruction occurs before the development of any collaterals, and symptoms are acute and more severe. Airway obstruction or mental dysfunction because of cerebral edema and/or cerebral venous hypertension are life-threatening complications hence correct and rapid diagnosis and treatment are of vital importance [3]. According to

the etiology of the SVCS and the severity of symptoms, therapeutic options include a surgical bypass, angioplasty, thrombolytic therapy, and endovascular metallic stent placement. Eventually, the most important parameter of the intervention to be performed in the postoperative period is the early diagnosis.

In acute-onset SVCS, anticoagulation and/or thrombolysis can be considered if the diagnostic tests show only venous thrombosis. If the thrombosis is associated with a stenosis, thrombolytic therapy is ineffective and other treatment options are required [4]. Thrombolytic treatment was contraindicated in the presented patient because the SVCS occurred within a few hours after the surgery. Percutaneous stenting did not appear to be a valid approach because of hemodynamical instability and the safety of the procedure was not established [5]. The surgical approach seemed to be reasonable and the patient showed clinical improvement within 24 hours. This case has been instructive in our

intensive care practice because respiratory deterioration alerted us before classical symptoms of the SVCS came into view.

References

1. Rice TW, Rodriguez RM, Light RW (2006) The superior vena cava syndrome: clinical characteristics and evolving etiology. *Medicine* (Baltimore) 85(1): 37-42.
2. Seligson MT, Surowiec SM (2017) Superior Vena Cava Syndrome.
3. Straka C, Ying J, Kong FM C (2016) Review of evolving etiologies, implications and treatment strategies for the superior vena cava syndrome. *SpringerPlus* 5(1): 229.
4. Jan MR, Jan AV, Wim JM (2012) Surgical management of superior vena cava syndrome after failed endovascular stenting. *Interact CardioVasc Thorac Surg* 15(5): 915-917.
5. Delgado M, Sanchez N, Colmenero M, Juan Lara-Torrano, Sergio Moyano-Calvente, et al. (2007) Superior Vena Cava Syndrome After Cardiac Surgery: Early Treatment by Percutaneous Stenting. *Journal of Cardiothoracic and Vascular Anesthesia* 21(3): 417-419.



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