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Case Report

Successful Anesthetic Management of Superior Vena Cava Syndrome in a Patient With a History of Hemodialysis: A Case Study

Amirnasseraldin Jadbabaei[1], MD; Robab Azizi[1], MD; Abdolreza Dayani[1], MD; Ziae Totonchi[1], MD; Maryam Ghanbari garekani[1*], MD

ABSTRACT

Superior vena cava (SVC) syndrome is a set of clinical indications and symptoms that occur due to the partial or complete blockage of blood flow through this vein. This rare complication, which leads to vein occlusion, will be more complicated if there is a mediastinal mass. Although the incidence of SVC syndrome has decreased significantly in recent years, the anesthetic management of such patients remains challenging due to life-threatening side effects, such as airway obstruction and cardiovascular collapse. We report the successful management of a 56-year-old woman with a history of hemodialysis who was a candidate for cardiac pulmonary bypass and clot removal under local and general anesthesia. ***(Iranian Heart Journal 2024; 25(2): 102-106)***

KEYWORDS: Superior vena cava syndrome, Local anesthesia, Cardiac pulmonary bypass

[1] Rajaie Cardiovascular Medical and Research Center, Iran University of Medical Sciences, Tehran, IR Iran.

*** Corresponding Author:** Maryam Ghanbari Garekani, MD; Rajaie Cardiovascular Medical and Research Center, Iran University of Medical Sciences, Tehran, IR Iran.
Email: dr.maryamGhanbari@rhc.ac.ir **Tel:** +989122500934

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Superior vena cava (SVC) syndrome was first described in 1757 by William Hunter. [1] SVC syndrome results from the partial or complete obstruction of the blood vein due to clot formation or tumor infiltration into the blood vessels. The SVC is constructed from the brachiocephalic vein and circulates from the head, neck, torso, and upper extremities to the heart. [2] In 60% of cases, SVC syndrome may not be diagnosed before the presence of a tumor or as a consequence of a previous malignancy. This syndrome is regarded as a secondary indication of cancer, particularly lung cancer and non-Hodgkin lymphoma, accounting for 85%–90% of SVC syndrome cases associated with malignancy. The most common symptoms of this condition include swelling in the face, neck, and upper arm regions, coughing, difficulty breathing, and enlargement of chest veins. [3, 4] Although the incidence of SVC syndrome has decreased in recent years, it is observed frequently as a secondary manifestation of bronchogenic tumors, metastatic tumors, and non-Hodgkin lymphoma. However, approximately 40% of SVC syndrome cases are idiopathic and induced by medical interventions, such as the administration of pacemaker wires and intravascular catheters applied temporarily for hemodialysis, as well as the long-term administration of antibiotics or chemotherapy. [5, 6] It is estimated that 1 in 650 to 1 in 3100 patients suffer from SVC syndrome, according

to different reports. [7] The frequency of occurrence in adults aged between 40 and 60 years with a history of malignancies, while it is more frequent in young adults between 30 and 40 years of age with benign tumors. Men suffer SVC syndrome more frequently owing to the high incidence of lung cancer in their sex; nonetheless, in benign tumors, there is no difference in terms of sex vis-à-vis the occurrence of SVC syndrome. [7] The common procedure for treating SVC syndrome is intravenous stent insertion and the administration of corticosteroids, thrombolytics, anticoagulants, and diuretics. In cancer-related SVC syndrome, radiation and chemotherapy reduce the tumor size, contributing to vein obstruction. [8] Following intravenous stent insertion and clot removal surgery, general anesthesia is challenging since even death is a possibility. The anesthetic management in patients with SVC syndrome should be considered because of airway obstruction and unexpected complications during surgery. The prognosis of the disease depends on etiology. In patients with malignancy, death is likely. Laryngeal and cerebral edema might prove life-threatening. Thus, it is vital to consider SVC syndrome in patients with hemodialysis. [7] Managing hemolytic and fluid therapy is complex owing to edema in the head and face, as well as the compromised airway. The gold direct therapy method is necessary to guide fluid therapy in these patients. [9]

The case presented in the current study indicates that indwelling pacemakers and catheters applied on the central venous system may contribute to silent SVC syndrome with slow progression. Our patient developed SVC syndrome following hemodialysis for chronic kidney disease. Additionally, the induction of anesthesia for clot removal and cardiopulmonary bypass (CPB) surgery is challenging and life-threatening, given the likelihood of airway obstruction and heart collapse.

We, herein, report a successful anesthesia management strategy for a 58-year-old woman with SVC syndrome.

Case Presentation

A 58-year-old woman with a history of hypertension and chronic kidney disease was hospitalized with pyrexia and rigors following hemodialysis with a catheter. Her blood pressure had decreased during hospitalization, and she was diagnosed with pericardial effusion and tamponade. The patient was referred to Rajaie Cardiovascular Medical and Research Center, affiliated with Iran University of Medical Sciences, in Tehran, Iran, for cardiac tamponade evacuation. On admission, she had a blood pressure of 68/47 mm Hg, a heart rate of 88 beats per minute, a respiratory rate of 19 breaths per minute, and an O_2 saturation level of 89%. The initial examination showed that the conjunctiva was pale, and the upper extremities were swollen. The patient had a history of hemodialysis 3 times a week, and her last hemodialysis was 2 weeks before referral. An informed consent form was obtained before the procedure and intervention. Under local anesthesia, fluoroscopy was applied via the subxiphoid approach to the pericardial cavity to insert a 0.35-inch guidewire into a 9 cm needle. Then, a 9F pigtail pericardiocentesis catheter was inserted into the pericardial space. Next, 400 mL of serosa fluid was drained following catheterization. During the hospitalization, the patient started experiencing swelling in her head, neck, and upper limbs (the edematous phenomenon). We ordered a venography computed tomography of the subclavian vein and the SCV. It demonstrated normal brachiocephalic veins and distal vasoconstriction of the SVC. The right jugular was occluded, while the left jugular vein and the inferior vena cava were normal. The patient became a candidate for surgery for clot removal.

In the operating room, disseminated edema was observed in the patient's face, neck, upper limbs, and upper chest. Moreover, she had dyspnea, exacerbated in the supine position.

Table 1: Patient's vital signs and laboratory criteria

Indication	Range
Blood pressure	110/73 mm Hg
Heart rate	107 bpm
O_2 saturation	93%
White blood cell	730 mm^3
Hemoglobin	9 g/L
Hematocrit	29/7 %
Platelet	131000 mm^3
Sodium	133
Potassium	4/1
Blood urea nitrogen	29
Creatinine	5

The radiography results showed a left ventricular ejection fraction of 55%, without signs of hypertrophy or clots in the left ventricle; a regular left ventricular diastolic pressure; a regular left ventricular systolic pressure; a normal mitral valve; mild-to-moderate mitral regulation; a tricuspid aortic valve with mild aortic insufficiency; a normal-sized ascending aorta (no indications of the coarctation of the aorta; mild pulmonary stenosis and insufficiency; a normal tricuspid valve with mild insufficiency (pulmonary pressure =37 mm Hg); a dilated inferior vena cava with a normal respiratory collapse; and mild pericardial effusion without tamponade. In addition, the dialysis catheter in the SVC was observed with a semi-mobile mass (3.17×0.78 cm) in the junction between the SVC and the right atrium, indicating thrombosis.

All these criteria made the patient a candidate for clot removal from the SVC. Nevertheless, since SVC syndrome exerts pressure on the airway system, and given the hemodynamic complications resulting from the venous return, it was not advisable to administer sedatives and muscle relaxants as a common anesthesia procedure.

A suitable vein path was placed in the upper limb, and an arterial line catheter was inserted into the radial artery. Then, with local anesthesia, a central vein catheter was also inserted into the left subclavian with the aid of mild sedation (midazolam [2 mg twice] and sufentanil [0.5 μg]). The local anesthesia, along with heparin administration, made it possible to insert arterial and venial centers into the femoral vein for CPB. Afterward, anesthesia was induced with rocuronium bromide (100 mg), sufentanil (40 mg), and midazolam (6 mg). Next, laryngoscopy and intubation were performed with a tracheal tube (No. 7.5), and fixation was done with a tracheal tube (No. 22). The clot was at the junction of the SVC to the right atrium on a permacath and stretched into the SVC. It was resected and replaced with a Shaldon percutaneous catheter in the left femoral artery. Due to blood oozing from the mediastinal tissues, the operation site was packed with gas and left open while the patient was transferred to the ICU. The surgery lasted for approximately 6 hours, with 10 minutes of CPB.

The next day, the patient was retransferred to the operating room for sternum closure. She had a blood pressure of 110/78 mm Hg, a heart rate of 100 beats per minute, and an SPO_2 level of 100%. Before the closure of the sternum, norepinephrine (0.04 mg/kg/min) and epinephrine (0.06 mg/kg/min) were prescribed. Afterward, anesthesia was performed with midazolam (2 mg), cisatracurium besylate (20 mg), and sufentanil (10 mg). The procedure was performed, and the patient was sent to the ICU for recovery. The following day, she was extubated, her hemodynamic status became normal, and she exhibited no signs of dyspnea or distress. Three days after the sternum closure, the patient was transferred to the post-cardiac surgery ward, where she had normal clinical status and no swelling in the upper limbs, right chest, neck, and face.

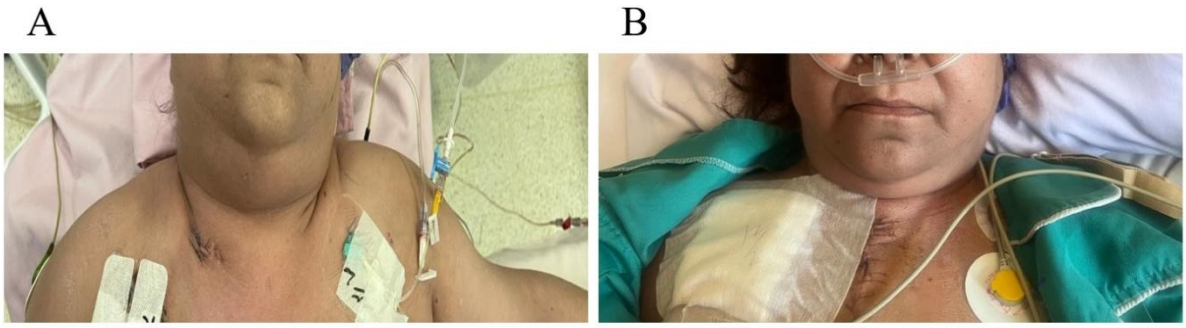

Figure 1: The images show the patient before (A) and after (B) the operation.

DISCUSSION

SVC syndrome is the most common complication following hemodialysis and contributes to clot formation and blockage in the veins. The diagnosis is primarily based on physical examinations and history taking, and determining the exact location of the stenosis or occlusion is vital. [10] The most frequent features of SVC syndrome include edema in the upper limbs, dyspnea, cough, and swelling in the neck, head, and chest wall. [11] The preferred methods for diagnosing SVC syndrome involve a chest computed tomography scan and magnetic resonance imaging, which can distinguish tumors from thrombosis and locate the place and extent of clot formation along the SVC. [12] The common strategy for SVC syndrome resulting from catheter-induced thrombosis is thrombolytic therapy and plasminogen activating factor, which should be initiated within 1 week after symptom manifestation. [12] Still, the management of SVC syndrome is closely related to the severity of the disease, the underlying etiology (eg, malignancy), radiation therapy, surgical approaches, and endovascular interventions. [13] In our current case, having monitored her vital signs and detected the place of the clot via fluoroscopy, we made her a candidate for surgery on CPB. Anesthesia strategies for patients with SVC syndrome are challenging and life-threatening. General anesthesia for patients with this condition increases the rate of morbidity. Further, the intubation of patients with airway obstruction and vocal cord paralysis is not feasible without general anesthesia. What might render anesthesia even more challenging is the presence of a mediastinal mass and instability in hemodynamic status, which could be exacerbated with ventilation pressure through intrathoracic pressure and venous return decrement. [14] The lengthy duration of surgery and bleeding risks compel most surgeons to opt for general rather than local anesthesia. However, to our knowledge, the literature features no reports on the application of local along with general anesthesia to achieve a more desirable outcome in patients with SVC syndrome. Of note, many reports have underscored the high mortality rate associated with general anesthesia in this group of patients. Spinal anesthesia causes spontaneous sympathectomy and worsens hemodynamic status as a result of the venous return from the upper and lower extremities if there is a mediastinal mass or a gravid uterus. [15]

Chan et al [15] employed general anesthesia to manage SVC syndrome in a woman who had a mediastinal tumor and gave birth to a 34-week neonate. They inducted general anesthesia rapidly to the femoral region for CPB and managed intravenous and intra-arterial catheters strictly for blood pressure changes.

In summary, our case report demonstrates the prompt identification of SVC syndrome

via an integrated team approach in a middle-aged woman undergoing hemodialysis. We, herein, highlighted the significance of immediate preoperative clinical assessment and successful anesthesia team management during surgery on CPB under both local and general anesthesia for clot removal. This uncommon occurrence, with a frequency of 1 case in approximately 50,000 operations per year, necessitates intricate medical care. This report provides a detailed description of the complex care as a practical approach.

Conflict of Interest

The authors have no conflicts of interest to declare.

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