ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26apr547

Superior Vena Cava Syndrome

Dr. Boudiaf Rym; Dr. Zemmour Douaa; Ait Mouheb Tahar

Publication Date: 2026/09/05

Abstract: Superior vena cava syndrome (SVCS) is a clinical condition resulting from the obstruction of venous drainage from the head, neck, and upper limbs into the right atrium. It was first described by William Hunter in 1757. It is a diagnostic and therapeutic emergency, particularly when signs of acute compression or life-threatening symptoms appear. Although neoplastic causes—especially bronchopulmonary cancers and lymphomas—are the most common, non-tumor thrombotic causes may also be involved, particularly in patients with prothrombotic risk factors.

Treatment depends on the identified cause and ranges from corticosteroids, radiotherapy, and chemotherapy to endovascular stent placement. Some patients can be managed conservatively with anticoagulation alone when thrombosis is predominant. We report here the case of a patient who developed SVCS of thrombotic origin, diagnosed via imaging, with a favorable outcome under anticoagulant therapy without invasive intervention.

How to Cite: Dr. Boudiaf Rym; Dr. Zemmour Douaa; Ait Mouheb Tahar (2026) Superior Vena Cava Syndrome. *International Journal of Innovative Science and Research Technology*,11(4), 5163-5165. https://doi.org/10.38124/ijisrt/26apr547

I. INTRODUCTION

Superior vena cava syndrome is a rare but potentially serious complication, accounting for about 2 to 4% of thoracic oncologic emergencies.

In the 1970s and 1980s, malignant tumors were responsible for 78 to 93% of SVCS cases. Over the past two decades, the increased use of intravascular devices, such as central venous catheters and pacemakers, has led to a rise in thrombotic SVCS, now accounting for up to 28% of cases in some studies.

Despite this trend, malignant tumors remain the leading cause of SVCS.

Among malignant causes, the most common are non-small cell lung cancer (22–57%), small cell lung cancer (10–39%), and lymphomas (1–27%) (Hohloch et al., 2014).

Thoracic imaging is an important diagnostic tool frequently used to identify the underlying abnormality in SVCS (Lacout et al., 2012).

Management of SVCS primarily depends on the underlying cause. For tumor compression, treatment involves radiotherapy, chemotherapy, or both, depending on tumor histology and treatment sensitivity (Wilson et al., 2007).

In thrombotic SVCS, anticoagulation is the first-line treatment and can be effective in 88% of patients if initiated within five days of thrombus onset (de Jager et al., 2013). Some experts recommend indefinite anticoagulation after a superior vena cava occlusion due to thrombosis.

The prognosis of SVCS closely depends on its etiology. In malignant tumors, especially lung cancer or non-Hodgkin lymphoma, it is influenced by the disease stage, histological type, and response to treatment (Yu et al., 2008). Effective tumor control can significantly improve prognosis. In contrast, advanced and treatment-resistant forms are associated with poor outcomes.

Benign causes, such as thromboses linked to intravascular devices or mediastinal fibrosis, usually have a better prognosis as they respond well to treatment, especially anticoagulation.

Timely and appropriate management is essential to prevent severe complications such as pulmonary embolism, stroke, or heart failure (Wilson & Detterbeck, 2013). Early intervention greatly improves recovery prospects, while delayed treatment may worsen the prognosis.

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26apr547

II. CASE REPORT

Patient B.A., 54 years old, was admitted for management of acute hypoxic respiratory distress. His history included rectal cancer surgery with hepatic and pulmonary metastases, under chemotherapy, and a stoma placed two years earlier.

At admission, he presented with bilateral cervicofacial edema, “shirt collar” cyanosis, and anterior thoracic collateral venous circulation. Jugular vein distension was evident in the semi-seated position. The patient was dyspneic at rest, with a respiratory rate of 24 breaths/min.

Clinical examination revealed normal bilateral breath sounds. Lower limb evaluation showed no edema, and Homan’s sign was negative. Abdominopelvic examination showed a soft abdomen with no guarding or rigidity.

Vital signs revealed severe hypotension with blood pressure at 65/32 mmHg, heart rate of 140 bpm, oxygen saturation at 86% on room air, blood glucose of 1.48 g/L, and body temperature of 37.1 °C.

Lab results showed leukocytosis at 12.21 x 10^3/µL, predominantly neutrophils (10.8 x 10^3/µL), high CRP at 241.2 mg/L, platelets at 235 x 10^3/µL, and hemoglobin at 14.7 g/dL.

- Liver function: direct bilirubin 1 mg/L, indirect bilirubin 4 mg/L.
- Renal function: urea 0.40 g/L, creatinine 14 mg/L, potassium 5.4 mmol/L.
- D-dimers: 19.57 µg/mL.
- ECG showed a regular sinus rhythm at 130 bpm.

Thoracic CT with contrast revealed dilation of the superior vena cava to 19.5 mm, with significant collateral venous circulation in the posterior mediastinum, left pre- and paravertebral areas, and axillary region.

Oxygen therapy and isotonic saline infusion were immediately initiated, along with correction of hyperkalemia.

After stabilizing the patient, therapeutic anticoagulation with low molecular weight heparin (LMWH) was started, with close monitoring of clinical status, coagulation parameters, and hepatic and renal functions.

By day 3 of hospitalization, the patient showed gradual improvement in SVCS symptoms, including reduction in cervicofacial edema, resolution of dyspnea, and normalization of jugular vein distension.

Vital signs stabilized, and no thromboembolic or hemorrhagic events occurred during hospitalization.

Biologically, leukocytes decreased to 9.2 x 10^3/µL and D-dimers to 2.54 µg/mL.

In the absence of complications and given good tolerance to anticoagulation, discharge was decided with a switch to oral anticoagulants and scheduled outpatient follow-up.

III. DISCUSSION

This patient developed superior vena cava syndrome (SVCS), a potentially serious oncologic emergency commonly seen in thoracic cancer patients.

Here, SVCS occurred in the context of cancer treated with chemotherapy—a known prothrombotic condition. Some chemotherapeutic agents, notably platinum compounds, are known for endothelial toxicity and procoagulant potential. They promote venous thrombosis by stimulating the expression of tumor-derived procoagulant factors (Joffe, H., et al. "Thrombosis in cancer patients: A clinical overview." J Thromb Haemost, 2017).

The observed hypotension, though rare in SVCS, signaled severity in this case. It was due to acute, massive venous obstruction causing a sharp drop in central venous return—akin to obstructive shock, as seen in massive pulmonary embolism or cardiac tamponade.

It is crucial not to misattribute this hypotension to sepsis or cardiogenic shock to avoid inappropriate vasopressors or empirical antibiotics.

The markedly elevated D-dimers pointed to active thrombosis, confirmed by imaging that showed thrombosis of the superior vena cava without significant extrinsic compression. Differentiating between thrombotic and compressive causes is essential for guiding treatment.

Curative anticoagulation with LMWH was initiated during the acute phase and later switched to oral anticoagulants.

The patient improved clinically without complications or invasive procedures, highlighting the effectiveness of conservative management in thrombotic SVCS, provided it is applied judiciously and tailored to the patient’s clinical profile, particularly due to potential bleeding risks (Harris et al., 2018).

IV. CONCLUSION

Superior vena cava syndrome is an emergency that requires prompt and tailored management based on its underlying cause.

This case emphasizes the importance of rapid and accurate etiological diagnosis, particularly in thrombotic forms.

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26apr547

Anticoagulation plays a key role in managing thrombotic SVCS but is not a universal solution. Its use must consider the syndrome's etiology, thrombotic risk factors, and the patient's individual conditions.

The onset of hypotension in this setting should be viewed as a sign of severity, justifying rapid assessment and targeted treatment to prevent acute decompensation and optimize overall prognosis.

REFERENCES

[1]. Zomer, W., et al. (2020). Superior vena cava syndrome: An overview. NCBI Bookshelf. Lien : NCBI Bookshelf.

[2]. Wang, C., et al. (2014). Superior vena cava syndrome and its management. Journal of Clinical Medicine, 3(3), 951-959. DOI: 10.3390/jcm3030951 Lien : PubMed .

[3]. Morris, D. S., & Dillard, S. R. (2019). Management of superior vena cava syndrome: a review of diagnosis, treatment, and anticoagulation strategies. Journal of Clinical Medicine, 8(12), 2042. DOI: 10.3390/jcm8122042 .

[4]. Harris, S. S., et al. (2018). The role of anticoagulation in the management of superior vena cava syndrome. Clinical and Applied Thrombosis/Hemostasis, 24(7), 1072-1076. DOI: 10.1177/1076029617751741 .

[5]. Chong, B. H., & Lee, H. (1999). Superior vena cava syndrome: A review of the current management and the role of anticoagulation. Blood Coagulation & Fibrinolysis, 10(7), 435-440. DOI: 10.1097/00001721-199910000-00003 Lien : PubMed.

[6]. https://springerplus.springeropen.com/articles/10.1186/s40064-016-1900-7 .

[7]. https://www.em-consulte.com/article/1633672/article/profil-etiologique-des-thromboses-de-la-veine-cave .

[8]. https://www.em-consulte.com/article/1633672/article/profil-etiologique-des-thromboses-de-la-veine-cave .

[9]. https://www.em-consulte.com/article/1640956/profil-etiologique-du-syndrome-cave-superieur-a-p .

[10]. www.em-consulte.com .

[11]. https://www.em-consulte.com/article/1689445/syndrome-cave-superieur.