# When arrhythmia hides a tumor: a case of atrial fibrillation unmasking superior vena cava syndrome

Sara Iommi-Diez,[1] Jorge Campos Bas,[1] Santiago Almanzo[2]

[1]Department of Family and Community Medicine, Hospital Universitari i Politècnic La Fe, Valencia; [2]Department of Surgery, Faculty of Medicine and Dentistry, University of Valencia, Valencia, Spain

Correspondence: Santiago Almanzo, Department of Surgery, Faculty of Medicine and Dentistry, University of Valencia, Avinguda Blasco Ibáñez 13, 46010, Valencia, Spain.
Tel.: +34.652.598 008
E-mail: almanzo@alumni.uv.es

Key words: superior vena cava syndrome, atrial fibrillation, venous congestion signs, small-cell lung carcinoma.

Contributions: SID contributed to the writing of the manuscript, data collection, patient care, and clinical analysis; JCB participated in data collection and patient care; SA was responsible for the conception of the work, clinical analysis, manuscript drafting, and final revision. All authors approved the final version and agree to be accountable for all aspects of the work.

Conflict of interest: the authors declare that they have no competing interests.

Funding: this research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Ethics approval: ethical review and approval were waived by the local ethics committee of our institution due to the retrospective nature of the study and the fact that all procedures were part of routine clinical care.

Informed consent: informed consent was obtained from the patient for publication of this case report and the accompanying images.

Availability of data and material: all data generated or analyzed during this study are included in this published article. Additional details are available from the corresponding author upon reasonable request.

Acknowledgments: the author would like to thank the staff of the Emergency Department and Radiology Unit at Hospital Universitari I Politècnic La Fe for their collaboration during the diagnostic process.

Received: 8 May 2025.
Accepted: 26 August 2025.
Early view: 24 September 2025.


*Emergency Care Journal 2025; 21:13961*
*doi:10.4081/ecj.2025.13961*



## Abstract

Superior Vena Cava Syndrome (SVCS) is typically caused by thoracic malignancies and may present with a wide range of symptoms, from mild facial edema to severe respiratory compromise. Atrial Fibrillation (AF), although common, is rarely the initial manifestation of SVCS. We report the case of a 65-year-old woman who presented to the emergency department with new-onset AF, and in whom physical examination revealed signs of venous congestion suggestive of SVCS. Imaging studies confirmed a right hilar mass with vascular invasion, thrombosis of the internal jugular vein, and complete obstruction of the superior vena cava. Histological analysis confirmed small-cell lung carcinoma. The patient underwent endovascular stenting and chemoradiotherapy. This case highlights the importance of thorough physical examination in emergency settings and suggests that new-onset AF in the presence of venous congestion may be an early clue to an underlying structural pathology such as SVCS.

## Introduction

Superior Vena Cava Syndrome (SVCS) is an uncommon clinical entity defined as a set of signs and symptoms resulting from partial or complete obstruction of the superior vena cava, leading to increased venous pressure in the upper body.[1] The majority of cases are secondary to malignant diseases, primarily lung cancer, although benign causes such as intravascular devices or fibrosing mediastinitis are increasingly recognized.[2] Clinical presentation ranges from asymptomatic cases to severe emergencies, with typical symptoms including dyspnea, facial and neck edema, cyanosis, and collateral venous circulation.[1] Diagnosis relies on imaging studies, and histological confirmation is necessary when malignancy is suspected.[1] Although once considered an oncological emergency, SVCS is now managed as a subacute condition requiring rapid diagnostic evaluation to guide timely treatment.[3]

Interventional techniques such as intravascular stenting have gained relevance in symptomatic patients, often combined with supportive therapy including corticosteroids, diuretics, and anticoagulation.[1,2] Moreover, the superior vena cava has been recognized as a possible source of Atrial Fibrillation (AF) triggers due to the presence of myocardial sleeves extending from the right atrium.[4] Structural changes and dilation in the SVC can facilitate ectopic electrical activity, making it a potential arrhythmogenic site.[5] Thus, new-onset AF may, in some cases, represent an early manifestation of SVCS, underscoring the need to investigate structural causes in such patients.[4] We report the case of a patient who presented with dyspnea, cough, and facial swelling, and was found to have new-onset atrial fibrillation on triage electrocardiogram. Subsequent imaging and diagnostic workup uncovered the underlying cause. We also discuss the relationship between SVCS and atrial fibrillation as a potential presenting feature.

## Case Report

A 65-year-old Ukrainian woman, living in Spain, with a history of smoking (10 pack-years) and no other known medical conditions, presented with a one-month history of facial and neck swelling accompanied by a dry, irritating cough. She reported exertional dyspnea (NYHA class II) in the preceding week. She denied chest pain, syncope, dizziness, headache, or palpitations.

Initial triage assessment revealed new-onset atrial fibrillation with a ventricular response of 123 bpm. The remaining vital signs were within normal range (axillary temperature: 36.2°C; BP: 129/75 mmHg; $SpO_2$: 96% on room air).

Physical examination revealed facial plethora and cervicothoracic edema ("collar of Stokes"), along with visible collateral venous circulation in the upper thorax and upper limbs (Figure 1A). A clear asymmetry in arm swelling was also noted, with the right upper limb more edematous than the left, consistent with impaired venous drainage (Figure 1B).

Cardiopulmonary auscultation revealed an irregular rhythm without murmurs, diminished breath sounds, and inspiratory crackles at the right lower lung field.

Initial workup included ECG, chest radiography, and blood tests. The ECG confirmed atrial fibrillation at 123 bpm with an S1Q3T3 pattern. Chest X-ray showed a right hilar mass and a mild right-sided pleural effusion (Figure 2). Blood tests revealed elevated D-dimer (751 ng/mL; normal range: 0-500 ng/mL), NT-proBNP (1,288 pg/mL; normal <125 pg/mL), and high-sensitivity troponin T (9.77 ng/L; normal <14 ng/L), with otherwise normal hematologic, renal, electrolyte, and coagulation profiles. Thyroid function tests and other systemic screening were unremarkable, ruling out common metabolic or endocrine triggers of atrial fibrillation.

Computed Tomography (CT) pulmonary angiography demonstrated a right hilar polylobulated mass or conglomerate lymphadenopathy extending into the mediastinum, with signs of bronchovascular invasion and carcinomatous lymphangitis, suggestive of lung neoplasm (Figure 3A-B). There was occlusion of the superior vena cava and azygos arch, with collateral venous formation in the anterior abdominal wall—findings consistent with SVCS. Thrombosis of the right internal jugular vein extending to the brachiocephalic trunk was also present, likely secondary to tumor compression or invasion.

The patient was admitted, and venography-guided placement of two overlapping self-expanding stents in the SVC was performed. She received dexamethasone, furosemide, and therapeutic-dose enoxaparin.

Bronchoscopic biopsy confirmed the diagnosis of small-cell lung carcinoma. Staging studies – including thoracoabdominopelvic CT, brain MRI, and PET-CT – identified a hypermetabolic nodule in the sigmoid colon (compatible with synchronous or metastatic disease) and an indeterminate enhancement in hepatic segment IVb. The patient declined further MRI liver imaging. No brain metastases were found.

Colonoscopy with polypectomy revealed a tubulovillous adenoma with high-grade dysplasia. The patient underwent concurrent chemoradiotherapy with curative intent using cisplatin-etoposide and thoracic radiotherapy (50.4Gy in 28 fractions). She tolerated treatment well but declined prophylactic cranial irradiation.

On follow-up, two brain metastases were detected. Despite this progression, the patient opted for palliative care only, declining further active treatment.

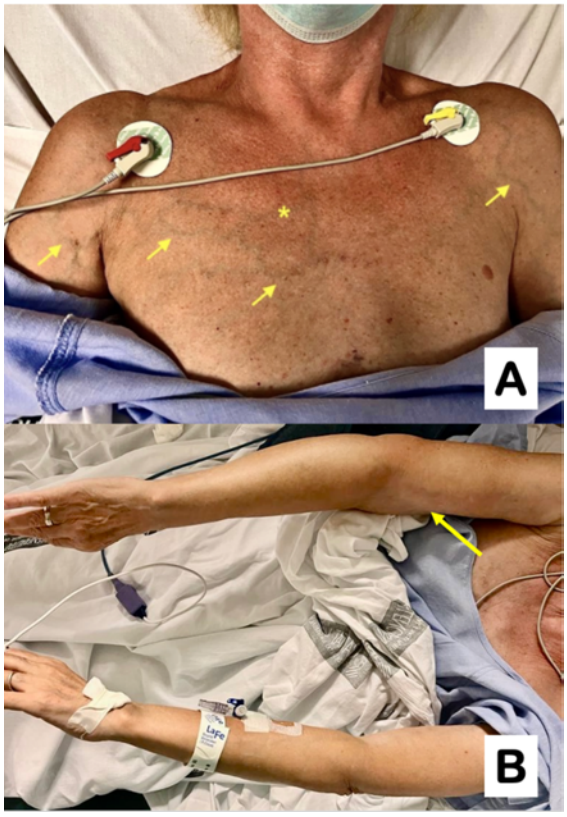


**Figure 1.** Physical findings consistent with superior vena cava syndrome. **A**) Erythema of the upper chest and neck (asterisk), with visible venous congestion (arrows) over the base of the neck ("Stokes' collar"), pectoral, and clavicular regions. **B**) Asymmetric right upper limb edema (arrow), more prominent than the left, suggestive of impaired venous return through the right-sided thoracic venous system.

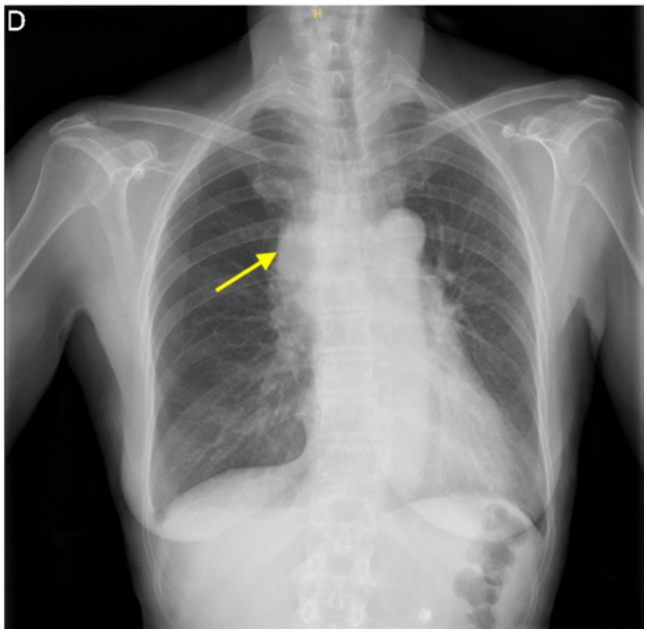


**Figure 2.** Posteroanterior chest radiograph showing a right hilar mass (arrow), partially obscuring the right cardiac border and causing mild right-sided pleural effusion. These findings raised initial suspicion of an underlying pulmonary neoplasm.

## Discussion

SVCS is most frequently associated with malignant tumors, particularly lung cancer and non-Hodgkin lymphoma, though benign causes—such as thrombosis from intravascular devices or fibrosing mediastinitis—are increasingly observed.[1,2] Management depends on symptom severity and underlying etiology. In symptomatic patients, endovascular stenting is the preferred therapeutic approach, supported by corticosteroids, diuretics, and anticoagulation when thrombosis is present.[1,2]

The superior vena cava contains myocardial extensions that can serve as arrhythmogenic foci. These anatomical features, combined with structural alterations (e.g., compression, dilation), may facilitate the initiation of atrial fibrillation.[4] Multiple studies support the SVC's role as a frequent source of non-pulmonary vein triggers in AF, particularly in patients with recurrent or unexplained arrhythmia.[4,5] Ablation of the SVC region, though not routinely indicated, has shown benefit in selected patients with arrhythmogenic foci in case reports.[6-8]

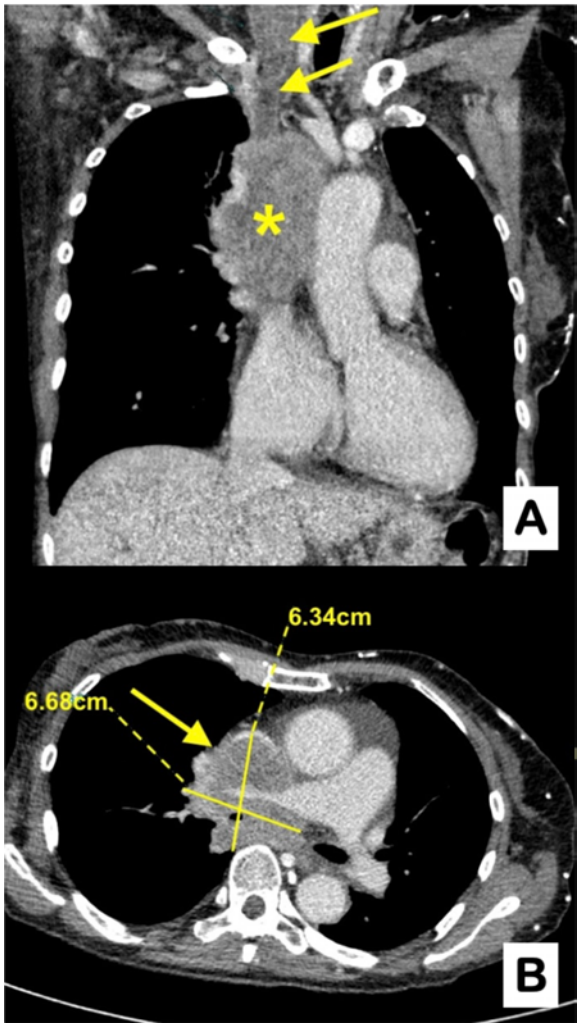


**Figure 3.** Contrast-enhanced computed tomography of the chest. **A**) Coronal view showing a mediastinal mass compressing the superior vena cava (asterisk), with associated vascular congestion (arrows) extending into the neck and upper thorax. **B**) Axial view at the level of the hilum demonstrating the tumor (arrow), measuring 6.34 × 6.68 cm, with evidence of bronchovascular invasion.

In this case, new-onset atrial fibrillation was the first clinical clue that led to the diagnosis of SVCS. The association is biologically plausible and supported by anatomical and electrophysiological findings.[4,5] The appearance of AF in a patient with facial swelling, dyspnea, or signs of venous congestion should prompt consideration of SVCS in the differential diagnosis.

## Conclusions

New-onset atrial fibrillation can be the initial manifestation of superior vena cava syndrome. This case underscores the importance of a thorough physical examination, which in our patient revealed clinical signs of upper body venous congestion that were essential for suspecting and confirming the diagnosis. In an era increasingly reliant on diagnostic imaging, this case highlights the enduring value of bedside assessment in guiding clinical reasoning and decision-making.

## References

1. Lepper PM, Ott SR, Hoppe H, et al. Superior vena cava syndrome in thoracic malignancies. Respir Care 2011;56:653-66.
2. Shaheen K, Alraies MC. Superior vena cava syndrome. Cleve Clin J Med 2012;79:410-2.
3. Yu JB, Wilson LD, Detterbeck FC. Superior vena cava syndrome--a proposed classification system and algorithm for management. J Thorac Oncol 2008;3:811-4.
4. Higuchi K, Yamauchi Y, Hirao K, et al. Superior vena cava as initiator of atrial fibrillation: factors related to its arrhythmogenicity. Heart Rhythm 2010;7:1186-91.
5. Ahmed A, Lakshman H, Coutteau S, Shah D. An uncommon focus of a common phenomenon: superior vena cava triggering atrial fibrillation. J Innov Card Rhythm Manag 2023;14:5654-6.
6. Sancho Zamora MA, Pacheco Galván A, Sánchez Corral J, Olavarría Delgado A. Varón de 76 años con disnea, edema facial y engrosamiento cervical: caso clínico. Med Paliat 2013;20:115–7.
7. Sharma SP, Sangha RS, Dahal K, Krishnamoorthy P. The role of empiric superior vena cava isolation in atrial fibrillation: a systematic review and meta-analysis of randomized controlled trials. J Interv Card Electrophysiol 2017;48:61-7.
8. Tao Y, Zhou Y, Sun X, et al. Pulsed field ablation of superior vena cava in paroxysmal atrial fibrillation: a case report. Front Cardiovasc Med 2023;10:1211674.