

Infiltrative Mediastino- Hilopulmonary Tumor Causing Cardiac Tamponade and SVC Syndrome: A Dramatic Presentation.

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Infiltrative Mediastino-Hilopulmonary Tumor Causing Cardiac Tamponade and SVC Syndrome: A Dramatic Presentation.

I. Abstract

Mediastinal tumors pose significant diagnostic and therapeutic challenges due to their heterogeneous nature and often insidious progression. We report the case of a 52-year-old male professional varnisher with chronic exposure to isocyanates and a history of heavy smoking, who rapidly developed acute cardiac tamponade and superior vena cava (SVC) syndrome secondary to an extensively infiltrative mediastino-hilopulmonary tumor. Clinical presentation included severe respiratory distress, hemodynamic instability, and a tumor syndrome characterized by bilateral cervical masses and an epigastric mass suggestive of metastatic spread. Imaging revealed a large mediastinal mass invading vital thoracic structures, accompanied by massive pericardial effusion and vascular thrombosis. Echocardiography confirmed tamponade physiology with hallmark features consistent with current guidelines. Critical hemodynamic instability precluded invasive histological diagnosis, and despite intensive supportive measures, the patient died shortly after admission.

This case highlights the diagnostic delays and therapeutic limitations in managing advanced mediastinal malignancies complicated by life-threatening cardiopulmonary sequelae. It also underscores the importance of early detection, particularly in occupationally exposed populations, and the need for a multidisciplinary approach to optimize outcomes in high-risk presentations.

II. Introduction

Mediastinal tumors represent a diagnostic challenge due to their diverse causes and often insidious progression (1). They may remain asymptomatic until invade vital structures such as the heart, large vessels, or the pericardium, leading to cardiac tamponade or superior vena cava obstruction (2). These compressive syndromes constitute medical emergencies with high mortality if not recognized promptly (3). The risk increased in individuals chronically exposed to toxic solvents and tobacco, which are involved in the development of respiratory and neoplastic diseases (4).

We report a dramatic case of an infiltrating mediastino-hilopulmonary tumor, discovered at the stage of acute cardiac tamponade and superior vena cava syndrome, illustrating the devastating consequences of delayed diagnosis and highlighting the importance of early screening in at-risk populations.

III. Case Presentation

A 52-year-old man, a professional varnisher chronically exposed to isocyanates and a former heavy smoker (20 pack-years, ceased two months prior), was admitted to the emergency room for severe respiratory distress and hemodynamic instability. Two months prior to admission, he had progressive dyspnea initially classified as mMRC stage I, evolving to resting dyspnea ten days before hospitalization. This was accompanied by productive greenish cough, dysphonia, dysphagia, headaches, and cervical and epigastric swelling, all in an afebrile context with major general condition deterioration (20 kg weight loss over two months, profound asthenia), without pruritus.

In the 24 hours preceding admission, dyspnea abruptly worsened (mMRC stage 4) with the onset of severe orthopnea, prompting consultation with a general practitioner. A thoracic CT scan performed two days prior, initially prescribed by this practitioner, revealed a large mediastino-hilopulmonary mass, leading to an emergency referral to our specialized unit.

Clinical Examination on Admission :

The patient was agitated, orthopneic, and cyanotic, conscious but confused. Vital signs included a heart rate of 128 bpm, blood pressure 85/55 mmHg, temperature 37.8 °C, respiratory rate 30 breaths/min, and oxygen saturation 85% in room air.

Physical examination revealed bilateral lower limb edema (pitting, soft, pale), labored breathing with suprasternal and intercostal retractions, thoraco-abdominal rocking, absent rales or stridor, muffled heart sounds, paradoxical pulse, and weak peripheral pulses.

A tumor syndrome was noted, associating firm bilateral cervical swelling (jugulocarotid and tracheal), epigastric mass, hepatomegaly, and left jugular vein distension, without palpable supraclavicular or axillary lymphadenopathies.

A superior vena cava syndrome was present, characterized by cape edema, conjunctival edema, headache, confusion, and agitation (WHO performance status 3).



Figure 1 : Bilateral firm jugulocarotid and pretracheal cervical masses with left jugular vein distension .

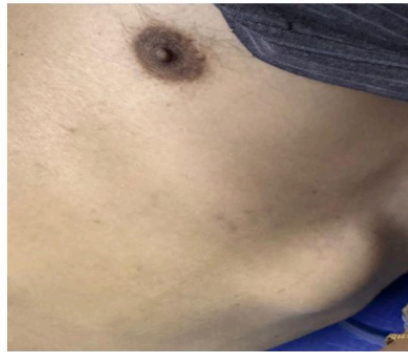


Figure 2 : Fixed, soft, and painless epigastric mass .

➤ This clinical presentation suggested cardiac tamponade associated with superior vena cava syndrome.

- Transthoracic echocardiography revealed a large circumferential pericardial effusion measuring 35 mm anteriorly and 32 mm posteriorly with complete diastolic collapse of the right atrium and right ventricle indicating critically elevated intrapericardial pressure. Marked respiratory variations of transvalvular Doppler flows including mitral inflow with inspiratory E-wave reduction of 55% exceeding the pathological threshold of 40% according to ESC and tricuspid inflow with inspiratory E-wave increase of 85% compatible with tamponade. Inspiratory decrease of aortic flow velocities by 30% and dilated inferior vena cava measuring 28 mm with less than 5% inspiratory collapse reflecting elevated central venous pressure. Paradoxical interventricular septal motion with right ventricular diastolic compression and preserved left ventricular systolic function with an ejection fraction of 58%.

These findings strongly suggested severe cardiac tamponade with major hemodynamic compromise.

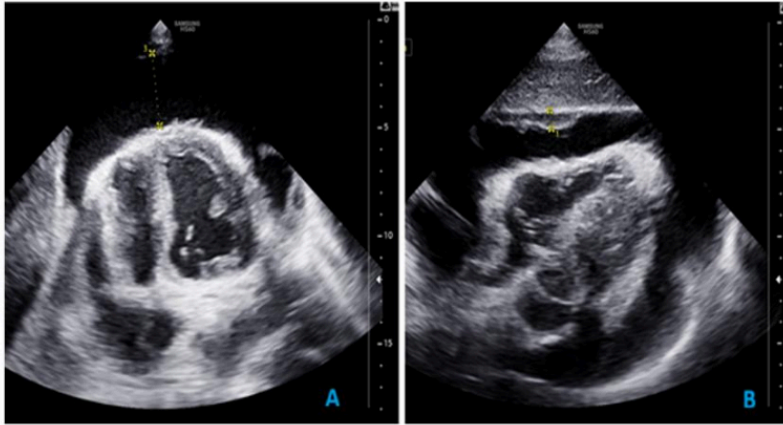


Figure 2 : Transthoracic echocardiographic views demonstrating pericardial effusion.

(A) Apical four chamber view showing a large pericardial effusion with swinging heart motion .

(B) Subcostal view showing a large circumferential pericardial effusion with probable pericardial metastasis.

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70 - The thoracic CT scan showed a large infiltrating mediastino-hilopulmonary mass invading mediastinal vessels
71 and the pericardium, with a large pericardial effusion and features suggestive of an aggressive malignant process
72 (Figure 3 and Figure 4).



Figure 3: Coronal CT scan of the thorax

A large tissular mass forming a mediastino-hilopulmonary ganglio-tissular complex, with heterogeneous enhancement, irregular and partly lobulated contours, and internal calcifications. The lesion measures approximately 120 x 127 x 103 mm (height x anteroposterior x transverse).

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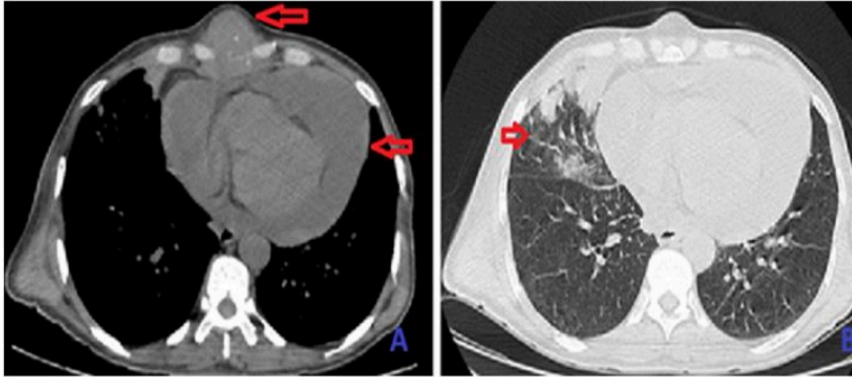


Figure 4: Sagittal CT scan of the thorax

(A) A large right mediastino-hilopulmonary tumor infiltrating the mediastinal vascular structures, including the pulmonary arteries and superior vena cava, was identified. This mass was associated with a massive pericardial effusion measuring up to 43 mm in maximal thickness, with suspicious nodular thickening of the parietal pericardium. An anterior chest wall mass with irregular, lobulated contours and heterogeneous enhancement, invading the xiphoid process of the sternum, measured approximately 58 × 53 × 52 mm (H × AP × T).

(B) Multiple irregular areas of parenchymal consolidation without air bronchogram, associated with septal thickening within the middle lobe, and scattered micronodules throughout both lung fields.

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76 - While an urgent pericardial drainage was being prepared, the patient experienced abrupt
77 hemodynamic decompensation with profound hypotension, bradycardia, and then pulseless electrical activity.
78 Death was declared 30 minutes after cardiac arrest despite intensive resuscitation maneuvers.

79 **IV. Discussion**

80 Mediastinal tumors represent a heterogeneous group of neoplasms characterized by diverse histological types and
81 a complex anatomic-clinical presentation given the tight anatomical confines involving vital structures such as the
82 heart, great vessels, thymus, esophagus, lymph nodes, and surrounding connective tissues (5).
83 These tumors often remain clinically silent until reaching advanced stages characterized by life-threatening
84 complications like cardiac tamponade and superior vena cava syndrome, which significantly worsen the
85 patient's prognosis if diagnosis is delayed (6).

86 In this case, the patient exhibited a voluminous mediastino-hilopulmonary mass directly invading the superior
87 vena cava with intraluminal thrombosis and occlusion, pericardial infiltration with nodular thickening, and
88 compression of the right pulmonary arteries, which is consistent with imaging profiles of
89 aggressive mediastinal malignancies described in recent literature (7). The tumor's metastatic nature was highlighted
90 by the presence of large necrotic mediastinal lymphadenopathies and a right adrenal metastasis, patterns
91 commonly observed in retrospective analyses where advanced disease frequently presents with such dissemination
92 and systemic spread (8).

93 Echocardiographic findings revealed a large circumferential pericardial effusion and hallmarks of
94 cardiac tamponade, including complete diastolic collapse of the right atrium and right ventricle, respiratory
95 variations in mitral and tricuspid Doppler flows beyond pathological thresholds, and paradoxical interventricular
96 septal motion, all aligning with the 2024 ESC guidelines and recent case series describing tumor-
97 induced pericardial tamponade (2). The preservation of left ventricular systolic function underscores the
98 typical hemodynamic impact of tamponade in this context before eventual decompensation (9).

Notably, the patient also presented with an epigastric mass, which likely reflected either direct tumor extension or a metastatic site, supporting the aggressive and advanced stage of the disease (10). Although biopsy of this mass could have yielded definitive histological confirmation, the patient's critical hemodynamic instability rendered invasive diagnostic procedures contraindicated at that time. This clinical scenario reflects the significant challenge in managing unstable patients with mediastinal tumors complicated by tamponade and SVC syndrome, where urgent stabilization takes priority over invasive diagnostics, a principle emphasized in current multidisciplinary management recommendations (11).

Therapeutic options in such advanced and life-threatening presentations remain limited (7). Current 2025 guidelines emphasize the role of surgery, chemotherapy, and radiotherapy tailored to histological subtype and disease stage, with surgery preferred for resectable tumors and systemic therapies for lymphomas or unresectable masses to improve survival (12). However, in cases complicated by tamponade and SVC syndrome, high early mortality persists, highlighting the vital need for earlier detection and intervention, especially among at-risk populations with chronic solvent exposure and heavy smoking histories, in whom preventive occupational health measures and surveillance could improve outcomes (4).

V. Conclusion

This case exemplifies the rapid progression and fatal potential of mediastinal tumors causing critical cardiopulmonary complications. It emphasizes diagnostic challenges, especially when invasive procedures are precluded by instability, and reinforces the importance of coordinated multidisciplinary care, alongside proactive prevention and early diagnosis strategies based on up-to-date evidence.

VI. Patient consent

I confirm in my own words that there is no legal conflict, the consent was obtained and declare that the family was informed of all the written information related to the patient's medical case, and accept it to be published.

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