

From Fluid to Air: Tamponade Due to Malignant Pneumopericardium

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Abstract

Pneumopericardium is a rare finding in malignant pericarditis, usually related to fistulous communication with adjacent structures. The coexistence of air and large pericardial effusion is uncommon and may create significant diagnostic and therapeutic dilemmas. We present a case of a 67-year-old man with pneumothorax and cardiac tamponade due to a large pericardial effusion with associated pneumopericardium in the setting of metastatic lung carcinoma. The pneumothorax was managed with thoracic drainage using a water-seal chest drainage system, but the patient deteriorated hemodynamically with tamponade physiology. Echocardiography-guided pericardiocentesis resulted in a transient clinical improvement, followed by recurrent hemodynamic deterioration. Malignant pericardial effusion is a well-recognized complication in advanced cancer, but the coexistence with pneumopericardium remains extremely uncommon, with only isolated cases reported in the literature. Presenting this case adds to the scarce global evidence and provides valuable insights for the scientific community.

Kew Words: malignant pericarditis; pneumopericardium; pericardial tamponade; spodick sign; pericardiocentesis

Introduction

Pericardial effusion is a frequent manifestation of advanced lung cancer [1]. Pneumopericardium, however, is rare and usually results from trauma, infection, or direct tumor invasion with pleuropericardial fistulization [2-4]. The accumulation of air or fluid in the pericardial cavity can be life-threatening when it leads to tamponade physiology [5,6]. In our case we present a 67-year-old man with pneumothorax and cardiac tamponade due to a large pericardial effusion with associated pneumopericardium in the setting of metastatic lung carcinoma. Malignant pericardial effusion is a well-recognized complication in advanced cancer, but the coexistence with pneumopericardium remains extremely uncommon, with only isolated cases reported in the literature [7-10].

The aim of this report is to present a rare case of malignant pneumopericardium with recurrent cardiac tamponade physiology, highlighting the diagnostic role of multimodal imaging and the challenges of clinical management.

Case Presentation

A 67-year-old man with hypothyroidism and end-stage metastatic lung carcinoma with poor prognosis presented with rapidly worsening

dyspnea. The initial electrocardiogram (ECG) showed sinus rhythm with ST-segment elevation in leads II, III, aVF, I, V5, and V6, and reciprocal ST depression in V1 and aVR. Careful review of the baseline tracing demonstrated a Spodick sign. **(Figure 1)** Transthoracic echocardiography (TTE) revealed a large circumferential pericardial effusion up to 3.5 cm. Emergency computed tomography angiography (CTA) of the thoracic and abdominal aorta was performed to exclude acute aortic dissection at the level of the right coronary sinus of Valsalva. No evidence of dissection was found. CTA demonstrated a large pericardial effusion with associated pneumopericardium, a right-sided pneumothorax with partial collapse of the upper lobe, pneumomediastinum, and pneumoperitoneum. The largest lesion was located in the right upper lung field and was considered a probable site of air entry into the pericardial sac through communication with the mediastinum. In addition, pathological mediastinal and hilar lymphadenopathy, as well as hepatic and osseous metastases, were noted. **(Figure 2)**

The right pneumothorax was managed with thoracic drainage using a water-seal chest drainage system. Despite this, the patient's hemodynamics deteriorated, and he developed supraventricular tachyarrhythmia evolving into atrial fibrillation. Invasive arterial

monitoring showed pulsus paradoxus. Under echocardiographic guidance, a subxiphoid pericardiocentesis was performed, draining 520 mL of serohemorrhagic fluid. The patient improved transiently after drainage, but air aspiration suggested ongoing communication, and soon the echocardiographic image became obscured due to progressive pneumopericardium. An urgent chest X-ray confirmed extensive air in the

pericardial sac. **(Figure 3)** Clinically, the patient showed transient deterioration due to air accumulation. Therefore, negative-pressure drainage of the pericardial sac using a water-seal chest drainage system was initiated in the absence of a Heimlich valve. Unfortunately, the patient died after 6 hours due to cardiogenic shock and tamponade physiology.

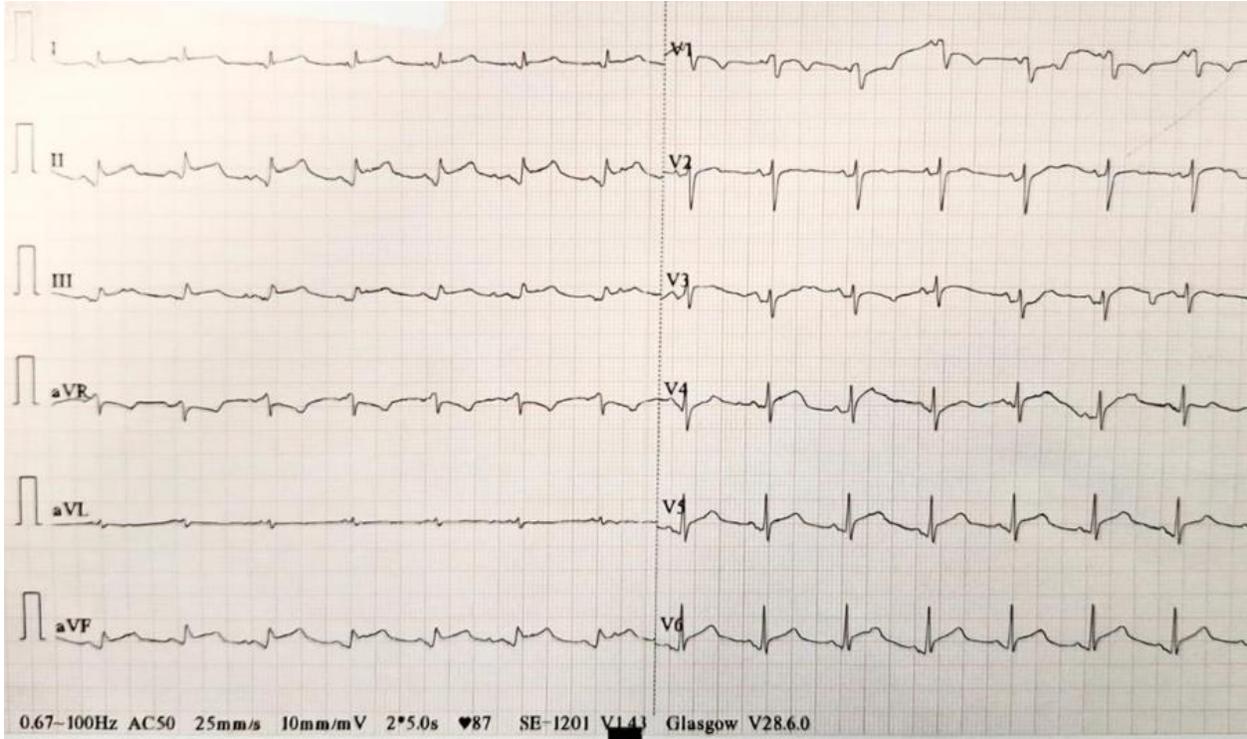


Figure 1: Initial ECG showing sinus rhythm with ST-segment elevation in leads II, III, aVF, I, V5, and V6, with reciprocal ST-segment depression in V1 and aVR.

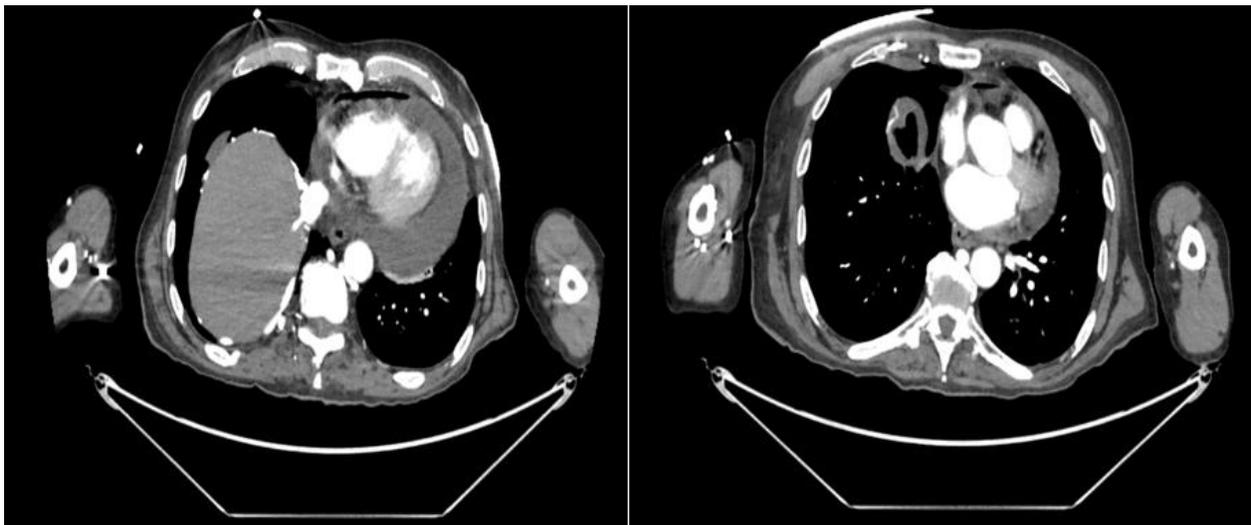


Figure 2: CTA showing large pericardial effusion and pneumopericardium.

Spodick sign present.

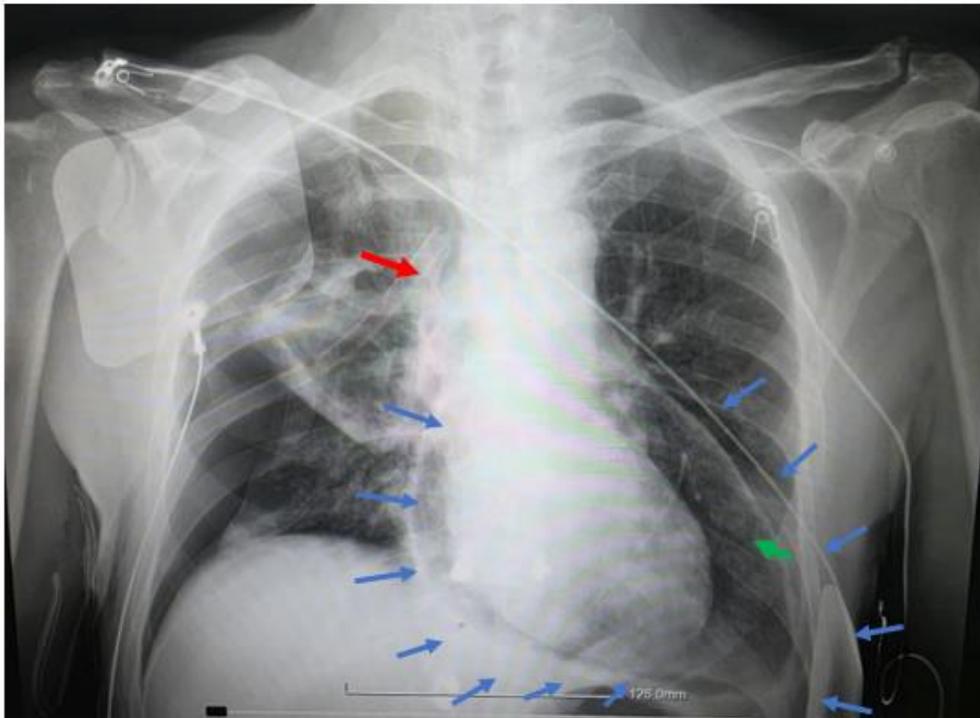


Figure 3: Chest X-ray showing pneumopericardium with concurrent right pneumothorax. Blue arrows define the borders of pericardium. A right pleural drainage tube (red arrow) and a pericardial drainage tube (green arrow) are visible.

Discussion

Large pericardial effusion is a relatively frequent finding in malignancy, but the coexistence with pneumopericardium is rare [1,11]. Electrocardiography remains essential in differentiating pericarditis from acute coronary syndromes [12,13]. The presence of the Spodick sign, defined as a downward-sloping TP segment most prominent in lead II, is characteristic of pericarditis and may raise suspicion for the diagnosis [14,15]. It has been reported in approximately 29% of pericarditis cases compared with only 5% of STEMI cases in a controlled study. However, it is not entirely specific and can be seen in a minority of STEMI cases [15]. In our case, recognition of the Spodick sign and the contribution of multimodal imaging guided the diagnosis and management. Emergency CTA was performed to exclude a type A aortic dissection at the level of the right coronary sinus of Valsalva, a lesion that in rare cases can present with hemopericardium and inferior STEMI due to right coronary artery compromise [16]. CTA was crucial for ruling out this life-threatening condition and for confirming the coexistence of pericardial effusion, pneumopericardium, pneumothorax, and pneumoperitoneum.

In our patient, the large pericardial effusion may have initially acted as a barrier to air entry into the pericardial space. Pericardiocentesis provided only transient improvement, as air re-accumulation caused recurrent hemodynamic compromise. Although this mechanism cannot be directly proven, similar cases of iatrogenic pneumopericardium after pericardiocentesis have been described, sometimes requiring repeat drainage when hemodynamic compromise occurs [4,17]. Continuous drainage systems and surgical pericardial window have been described as potential management strategies. However, advanced malignancy often limits the feasibility of such interventions [18-20].

Clinical management considerations

From a clinical perspective, the coexistence of pneumopericardium and large malignant pericardial effusion requires rapid recognition and careful hemodynamic monitoring, as both air and fluid accumulation may contribute to tamponade physiology [21]. Multimodal imaging, including echocardiography, chest X-ray, and computed tomography (CT), plays a crucial role in the diagnosis and assessment of the underlying mechanism [22]. Echocardiography remains essential for initial evaluation, guidance of pericardiocentesis, and follow-up assessment to detect recurrence of pericardial effusion [23]. In selected cases, continuous pericardial drainage or surgical pericardial window may be considered [24]. However, therapeutic decisions must take into account the overall prognosis and the stage of the underlying malignancy.

Limitations:

This report has several limitations inherent to single case reports. The exact mechanism leading to pneumopericardium could not be definitively confirmed, although imaging findings suggested a possible communication between the pulmonary lesion and the mediastinum. In addition, some management options were not available in the acute setting, including the use of a Heimlich valve.

Conclusion

Large pericardial effusion is a relatively frequent finding in malignancy, but the coexistence with pneumopericardium is rare [1,11]. In our case, recognition of the Spodick sign and the contribution of multimodal imaging guided the diagnosis. Pericardiocentesis provided only transient improvement, as air re-accumulation caused recurrent hemodynamic compromise. Continuous drainage systems and surgical pericardial window have been described in the literature as possible management strategies, but advanced malignancy often limits the feasibility of such interventions [18-20]. By documenting this rare presentation, we aim to

enrich the literature on malignant pericarditis complicated by pneumopericardium and provide additional insights for clinical practice.

Conflict of Interest

The authors declare no conflict of interest.

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