

Spontaneous pneumomediastinum mimicking acute pericarditis

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June 27, 2021

Abstract

ST-segment changes may provide a clue to the presence of pneumopericardium accompanying SPM. These EKG changes associated with SPM are rare. The management in SPM with concurrent pneumopericardium is mainly supportive. We describe two SP cases with concomitant pneumopericardium that presented with a deceptive clinical spectrum, closely mimicking acute pericarditis.

Abbreviations:

1. electrocardiogram (EKG)
2. spontaneous pneumomediastinum (SPM)
3. Computed tomography (CT)

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Keywords: Pneumomediastinum, EKG, ST-T wave changes, acute coronary syndrome, acute pericarditis

Key Clinical Message:

- The atypical clinical presentation of pneumomediastinum may produce EKG changes.
- The EKG changes of pneumomediastinum can mimic acute pericarditis.
- Early recognition and appropriate treatment can lead to improved clinical outcomes.

Introduction:

ST elevations on EKG have a broad differential diagnosis that can vary from benign to more ominous pathologies. These include early repolarization, coronary vasospasm, acute pericarditis, ST-elevation myocardial infarction, ventricular aneurysms, and dissecting aneurysm of the aorta reaching the pericardium. (1-5) The presentation of these diseases can closely resemble SPM, in which patients often present with sudden onset chest pain and shortness of breath. Furthermore, the clinical scenario in patients with underlying SPM can pose a diagnostic challenge due to ST-T changes on EKG that may mimic acute coronary syndrome. (6-8) These EKG changes associated with SPM have seldom been reported in the literature. We describe two SP cases with concomitant pneumopericardium that closely mimics acute pericarditis with a deceptive clinical spectrum.

Case presentation:

Patient characteristics are summarized in Table 1

Case 1.

A 19-year-old gentleman with a history of occasional marijuana use presented with acute onset progressive central and sharp chest pain for several hours, followed by a brief syncopal episode. The pain was pleuritic and exacerbated on lying flat. He denied any trauma, fever, cough, retching, or vomiting. He did not have any previous history of hospitalizations. On presentation, the blood pressure was 117/79 mmHg with a pulse rate of 76 beats/min, temperature 37.7 °C (99.9 °F), and a respiratory rate of 22 breaths/min with an oxygen saturation of 96 % on ambient air. He did not have orthostasis. There was crepitus around the left lower neck, appreciated on palpation, and precordial auscultation was significant for a Hamman's crunch best heard in the 4th left intercostal space suggestive of subcutaneous emphysema. Complete blood counts (CBC) revealed a white cell count (WBC) of 5.4×10^3 , Hb 15 g/dl, platelets 241×10^3 per microlitre. EKG showed ST-segment elevations in the precordial leads (**Figure 1a**). Serial troponins were negative. Chest X-ray revealed a continuous diaphragm sign suggestive of pneumomediastinum, which was confirmed by computed tomography (CT) of the chest showing evidence of spontaneous pneumomediastinum along with pneumopericardium (**Figure 2a**). The echocardiogram was unremarkable. An esophagram was not performed given the low suspicion for any esophageal rupture. The patient was successfully managed with mild analgesia, oxygen therapy, and clinical observation, with gradual resolution of EKG changes as the pneumopericardium resolved.

Case 2.

A 19-year-old gentleman with a history of smoking and marijuana use presented with left-sided chest pain, preceded by intractable nausea and vomiting for two weeks. The pain radiated to the neck, upper back, and shoulders base and was not associated with shortness of breath. He did not report hematemesis or melena. He denied upper respiratory tract symptoms. On presentation, his blood pressure was 162/91 mmHg, pulse rate of 82/min, temperature 36.7 °C (98.1 °F), and oxygen saturation of 98 % on ambient air. He had supraclavicular crepitus but otherwise normal systemic examination. CBC showed WBC count of 7.10×10^3 per microlitre., 13.8 g/dl, 214×10^3 per microlitre. EKG showed diffuse ST-segment elevation, PR segment depression, and evidence of left ventricular hypertrophy, with no reciprocal changes suggestive of acute pericarditis(**Figure 1b**). Chest X-ray showed pneumomediastinum. A subsequent CT chest showed air in the anterior mediastinum, pneumopericardium, and subcutaneous emphysema (**Figure 2b**).Esophagram did not show any evidence of a leak. The patient was initially started on ibuprofen and colchicine given the EKG changes highly suggestive of acute pericarditis, but later therapy was tailored to mild analgesia with clinical observation. EKG changes subsided upon resolution of pneumo-pericardium.

Discussion:

Pneumomediastinum relates to the accumulation of air in the mediastinal structures. It may result from blunt trauma leading to thoracic injury, esophageal perforation, or hollow viscus rupture. It can also occur spontaneously without any structural lung or mediastinal abnormalities in the absence of trauma, called SPM. In general, SPM can be caused by intrathoracic (involving trachea and major bronchial airways, esophagus, lung, and pleural cavity) or extra-thoracic (head, neck, or peritoneum) processes. SPM is a non-traumatic entity that occurs due to increased intrathoracic pressure leading to alveolar rupture. As the mediastinal pressure is more negative than the pulmonary parenchyma, the air enters the pulmonary interstitium. It dissects along the perivascular sheets to reach the hilum from where it spreads to the mediastinum. This phenomenon is described as the Macklin effect.(9) Occasionally, pneumopericardium can also occur with leakage of air into the pericardial space. (10) It is worth emphasizing, spontaneous alveolar rupture can occur even with no prior history of pulmonary disease or esophageal perforation and is usually precipitated by cough, emesis, physical exercise, labor, or upper airway infection.

The exact mechanism of ST-segment changes secondary to SPM remains unclear. The most common EKG

findings associated with SPM are the loss of R wave in the precordial leads and diminution of QRS voltages or ST-segment elevation in inferior leads (**11-15**). Previously described mechanisms propose cardiac rotation, right ventricular dilatation, and insulation effect caused by air accumulation between the cardiac structures and the chest wall as the cause of such changes. Secondly, SPM may lead to myocardial stretch leading to stretching and narrowing of the coronary arteries, which may masquerade as ST-elevation myocardial infarction with a troponin leak (**15**). We further postulate the EKG changes associated with pneumopericardium in our cases could be related to the direct inflammatory effect from air leakage between the pericardium and the chest wall leading to a presentation similar to acute pericarditis. Such ST-T changes may give an initial diagnostic clue of the presence of pneumopericardium accompanying SPM. The concurrent pneumopericardium diagnosis may be relevant if caused by fistula formation from the adjacent intra-abdominal structures as mortality rates can be as high as 50-70% in such cases. (**16, 17**) Given the lack of abdominal symptoms and CT findings not suggestive of an intra-abdominal or esophageal source in Case 1, an esophagram was not performed. The esophagram in the second described case was performed as the patient presented with hyperemesis, leading to Boerhaave syndrome suspicion. These patients appear sicker on presentation with hypotension and shock due to chemical mediastinitis, and a pleural effusion often accompanies findings of SPM. (**18**)

Management of SPM is mainly conservative with avoidance of trigger factors, analgesia, bed rest, and oxygen therapy in the absence of complications such as hemodynamic instability, pneumothorax, suspected chemical mediastinitis, or tamponade effect by the coexisting pneumopericardium.

Conclusion:

Our cases highlight the association of ST-segment elevation with SPM that may mimic acute pericarditis. This can pose a diagnostic challenge for clinicians and lead to unnecessary investigations and pharmacotherapy. Although the presentation can be dramatic, aggressive management is seldom required in these patients, even in the presence of a concurrent pneumopericardium without tamponade physiology.

Tables and figures:

Table 1. Patient Characteristics (EKG-electrocardiogram; CT- Computed tomography)

Patient Characteristics	Case 1	Case 2
Age	19 years	19 years
Sex	Male	Male
Risk Factors	Occasional marijuana use, Smoking	Marijuana use, Vaping
Symptoms	Chest pain, shortness of breath, syncope	Hyperemesis, Chest pain
CRP	0.11 (<1.00mg/dl)	0.50 (<1.00 mg/dl)
Troponin	0.03(<0.06ng/ml) x 2	0.03 (0.06ng/ml) x2
EKG changes	ST-T elevations V3-V4 with PR depression in AVR	Diffuse ST-T elevations with PR depression in AVR with evidence of LVH
Chest Xray	Subcutaneous emphysema, continuous diaphragm sign	The air along the mediastinum and subcutaneous emphysema
CT Findings	Subcutaneous emphysema, moderate pneumomediastinum with pneumopericardium, and a collapsed esophagus	Subcutaneous emphysema, moderate pneumomediastinum, pneumopericardium, esophagus not well visualized.

Patient Characteristics	Case 1	Case 2
Echocardiogram	Normal function, no evidence of pericardial effusion.	Mild concentric left ventricular hypertrophy normal chamber size and hyperdynamic systolic function with no apparent regional wall motion abnormalities.
Esophagram	Not performed	No evidence of an esophageal leak

Figure 1. EKG demonstrating (a) ST-T elevations V3-V4 with PR depression in AVR (b) Diffuse ST-T elevations with PR depression in AVR with evidence of LVH

Figure 2 . CT Thorax (Coronal View) with (a) Subcutaneous emphysema, Moderate pneumomediastinum with pneumopericardium and a collapsed esophagus (b) Subcutaneous emphysema, Moderate pneumomediastinum, pneumopericardium, esophagus not well visualized.

Acknowledgements:

Qatar National Library will fund the open access Article processing charges for the publication.

Author contributions:

Conceptualization: HC, KS

Patient consent: HC

Literature review: HC, KS, ZY

Manuscript writing: HC, KS

Data collection: HC, KS

Radiology part in writing and images: KS

Critical review and modifications: HC, KS, ZY

Final review and approval: HC, KS, UN, TW, ZY.

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