Received: 13 September 2021 | Accepted: 8 October 2021

DOI: 10.1111/anec.12907

CASE REPORT

WILEY

Reversible, regional ST-segment elevation due to chylothorax

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Abstract

Chylothorax is an uncommon complication of thoracic surgery and, to our knowledge, has never been documented as a cause of dynamic ST-segment elevation (STE). A 63-year-old woman with history of right pneumonectomy presented with chest pain and regional STE on 12-lead electrocardiogram (ECG). Normal troponin-I and a computed tomography (CT) scan showing a large right hemithoracic fluid collection indicated the unique cause of STE, which resolved after thoracentesis, was pericardial inflammation and cardiac compression from chylothorax. This case emphasizes nuances of ECG interpretation in the context of regional STE and explores the pathophysiology that links chylothorax with acute pericarditis.

KEYWORDS
chylothorax, pericarditis, ST-segment elevation

1 | INTRODUCTION

The differential diagnosis of ST-segment elevation (STE) on 12-lead electrocardiogram (ECG) spans emergencies such as acute myocardial infarction (MI) to benign entities such as early repolarization (Coppola et al., 2013). STE can be challenging to interpret, and alternative causes should be considered while emergent causes are ruled out. We describe a patient with a massive right chylothorax that caused regional, upsloping STE on 12-lead ECG suggestive of pericarditis that ultimately resolved upon thoracentesis. We explore the interpretation of STE in the context of this unique case and the most likely pathophysiology of the ECG findings: regional irritation of the pericardium from chylothorax and external cardiac compression.

2 | CASE PRESENTATION

A 63-year-old woman presented to the emergency department with acute dyspnea associated with chest tightness, non-radiating, non-pleuritic, "sharp" progressive right-sided chest pain, diaphoresis, and nausea. Two weeks previously, she underwent a right extrapericardial pneumonectomy at another hospital for T4N2 non-small-cell lung cancer with no known metastases. Her medical history included type 2 diabetes mellitus without diagnosed cardiovascular disease. Despite her dyspnea, the oxygen saturation was >90% on two liters of oxygen. The respiratory rate was 20 breaths/min, with heart rate of 105 beats/min and blood pressure of 123/70. There were absent breath sounds in the right hemithorax and coarse breath sounds on the left. The cardiovascular examination showed normal jugular venous pressure, no murmur, rub or gallop, and no edema. The remainder of the physical examination was unremarkable. A 12-lead ECG showed sinus tachycardia, poor precordial R-wave progression, plus J-point elevation with upsloping STE in leads I, II, aVL, and especially leads V4-6. There was PR-segment depression most notable in lead V6 and no reciprocal ST depression (Figure 1).

Although these findings raised concern for a STEMI in the territory of the left coronary artery, a chest X-ray showed a large right-sided effusion with mediastinal shift. The first troponin-I was <0.01 ng/ml, further suggesting an alternative diagnosis.

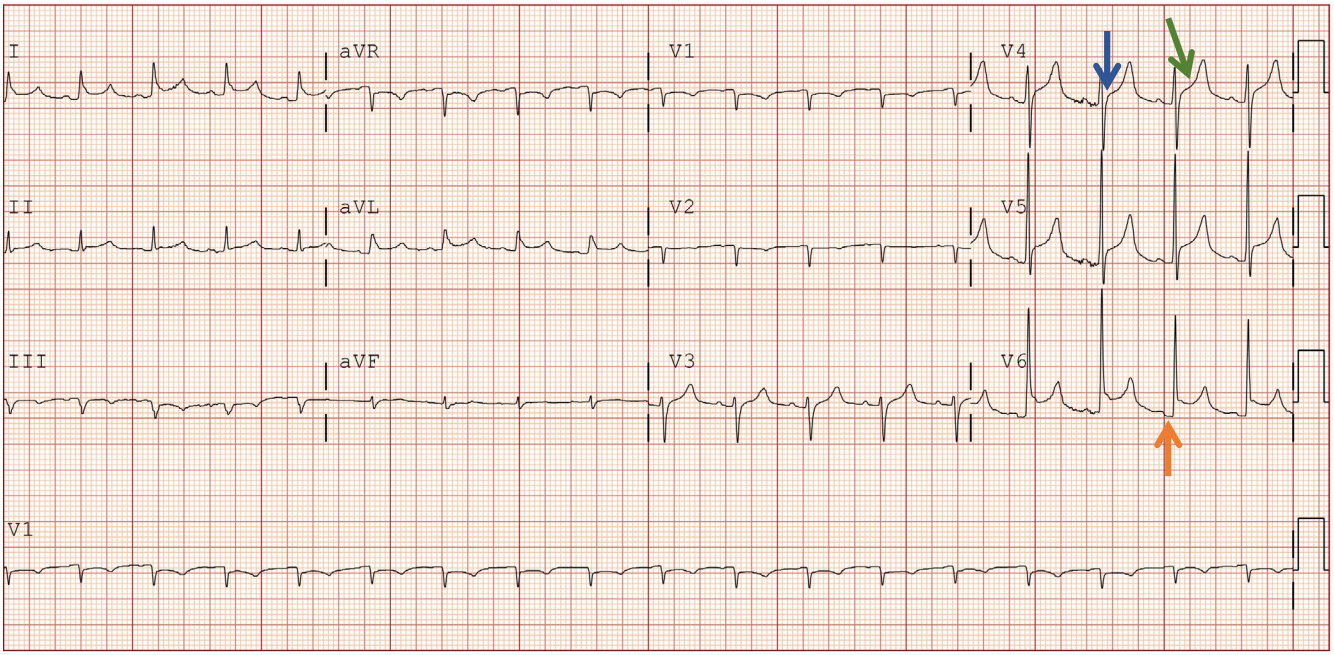

FIGURE 1 The 12-lead electrocardiogram on admission showing ST-segment elevation in leads I, II, aVL, and V4-6 consistent with pericarditis. Note J-point elevation (blue arrow), upsloping ST-segment (green arrow), and PR-segment depression (orange arrow)

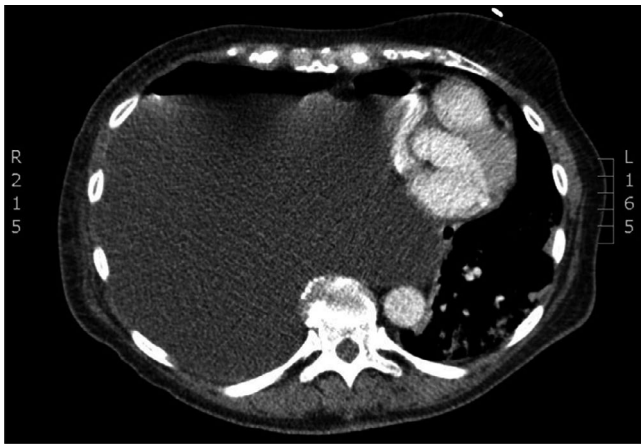

FIGURE 2 Chest CT with contrast performed after admission showing fluid accumulation in the right hemithorax and mediastinal shift

Computed tomography (CT) showed fluid and air in the right post-pneumonectomy space, a significant leftward mediastinal shift, possible compression of the right atrium, and no pericardial effusion (Figure 2). Three liters of chylous fluid (644 mg/dl triglycerides) was drained from the chest, and a repeat ECG showed a marked reduction in the initial abnormalities (Figure 3).

A post-procedural transthoracic echocardiogram showed a moderate pericardial effusion adjacent to the right ventricular free wall with no chamber compression. This effusion was not seen on the original CT scan but was likely present before the procedure because echocardiography has greater sensitivity than CT for pericardial effusion (Kolski et al., 2008). The ST-segment changes normalized completely by the next day and serial troponin-I remained negative. Subsequently, the thoracic duct was embolized by a radiological procedure. The chest tube was removed, and the patient gradually improved. A repeat CT scan several days later showed a residual effusion with improved but persistent mediastinal shift.

3 | DISCUSSION

Advanced age, female gender, or diabetic neuropathy could have caused atypical presentation of STEMI in this patient yet stable, normal troponin-I effectively ruled out infarction (Antman et al., 2004). Two mechanisms described in previous reports are considered as causes for the regional, reversible STE elevation: transient ischemia and pericardial irritation.

There are reports of reversible STE associated with cardiac compression. It is thought that such compression disrupts coronary artery perfusion or restricts the right ventricular outflow tract, leading to ischemia that is reversible with decompression (Alzghoul et al., 2017; Slay et al., 1979; Asteriou et al., 2013; Tarin et al., 1999). In this case, while regional STE suggested epicardial injury current, closer inspection of the ECG showed no reciprocal ST-segment depression. The more subtle findings suggestive of pericarditis included PR-segment depression in V6 and STE in lead II (Lee et al., 2015). The latter suggested a separate vascular distribution than seen with anterolateral ischemia or infarction. Persistent hemodynamic stability and normal cardiac biomarkers also reduced the probability of a prolonged ischemic event.

Pericarditis has been documented after extensive pneumonectomy that violates the pericardium (Tamarappoo and

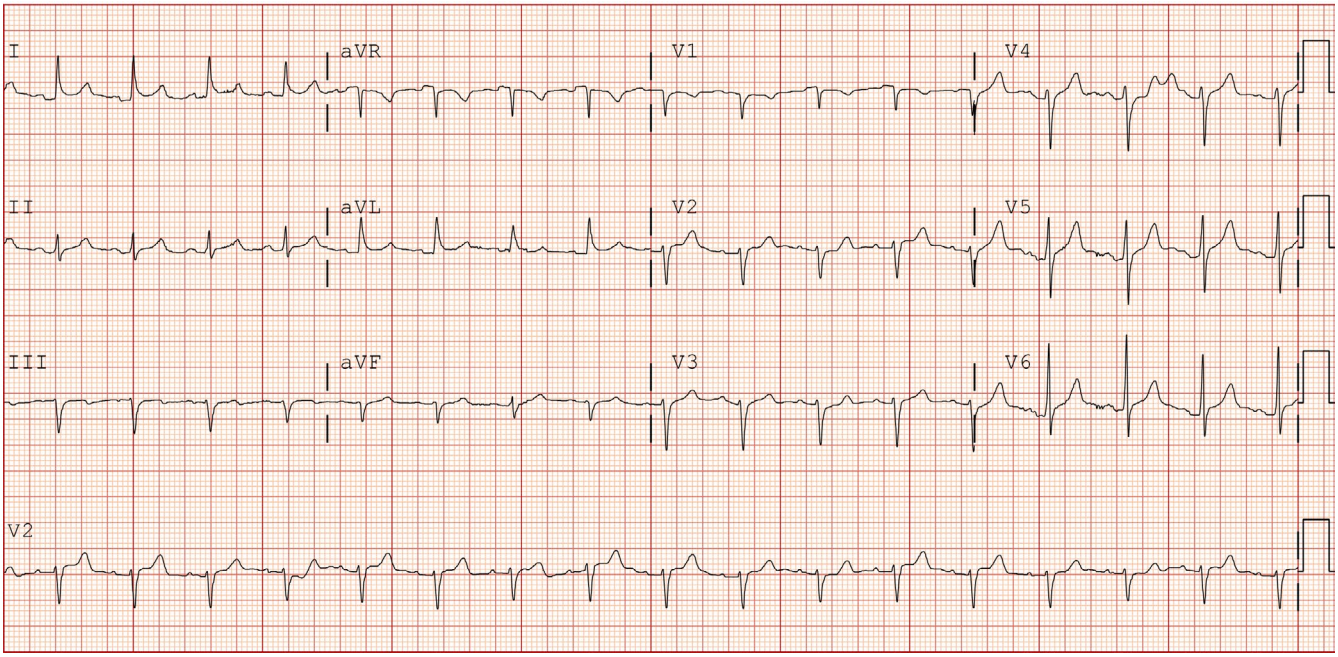

FIGURE 3 The 12-lead electrocardiogram after drainage of three liters of chylous fluid from the patient's right postpneumonectomy space, showing partial resolution of the previous ST-segment elevation

Klein, 2016; Vasic et al., 2017). However, this patient underwent an extrapericardial pneumonectomy, which indicates an alternative mechanism of pericardial inflammation. Massive hiatal hernias that caused cardiac compression have been reported to cause reversible STE consistent with pericarditis (Kaplan et al., 1995; Gard et al., 2011). The postpneumonectomy intrathoracic pressure required to cause mediastinal shift is significant and could plausibly compress the heart as well as irritate the pericardium (Hokamaki et al., 2005). Chylopericardium itself has not been reported to cause STE (Yu et al., 2017). We postulate that the small right ventricular free wall pericardial effusion on echocardiogram indicates an inflammatory reaction to cardiac compression from the large chylothorax.

This case emphasizes the importance of interpreting STE on ECG in the context of the patient's medical history as well as nuances in the interpretation of STE that indicate ischemia versus pericarditis.

ACKNOWLEDGEMENT

The research and publication of this case report was performed as part of the employment of the authors at Vanderbilt University Medical Center and Vanderbilt University School of Medicine.

CONFLICT OF INTEREST

The authors have no conflicts of interest to report.

AUTHOR CONTRIBUTIONS

Sarah Brown: Conceptualization (lead), Investigation (equal), Writing – Original Draft Preparation (lead), Writing – Review & Editing. **Michael Neuss:** Conceptualization (support), Investigation (equal), Writing – Original Draft Preparation (support), Writing – Review & Editing. **J. Brett Heimlich:** Conceptualization (support), Investigation (equal), Writing – Review & Editing. **Marvin Kronenberg:** Conceptualization (support), Investigation (equal), Writing – Review & Editing.

ETHICAL APPROVAL

Written informed consent was obtained from the featured patient in this case report.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

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How to cite this article: Brown, S. H., Neuss, M. J., Heimlich, J. B., & Kronenberg, M. W. (2022). Reversible, regional ST-segment elevation due to chylothorax. *Annals of Noninvasive Electrocardiology*, 27, e12907. https://doi.org/10.1111/anec.12907