A 56-year-old woman (height 165 cm, weight 65 kg) was admitted to the hospital because of a 2-day history of epigastric pain and emesis. Two weeks prior to admission, she had a Billroth II with Roux-en-Y procedure for stomach stricture and biliary reflux. Her medical history was significant for 1 episode of atrial fibrillation many years previously, osteoporosis, bilateral hip replacements, and gastric ulcer surgery. Drug therapy at the time of admission included simethicone 80 mg/d, rabeprazole 10 mg/d, and as-needed doses of clonazepam for anxiety and of acetaminophen.

At admission, the patient’s blood pressure was 120/70 mm Hg and her heart rate was 90/min and regular. The physical examination was notable for facial and conjunctival pallor. Mucous membranes were dry. The patient’s abdomen was distended and tender to palpation in the upper quadrants. Bowel sounds were decreased. Her extremities were cool and her peripheral pulses were weak. The abdominal surgical wound was healing. The patient denied any cardiopulmonary symptoms and the results of her neurological examinations were normal.

The patient’s white blood cell count was slightly elevated at 10.9 x 10⁹/L, and her hemoglobin level was 112 g/L. Her serum level of creatinine was elevated at 110 μmol/L (normal 50-100 μmol/L). International normalized ratio, partial thromboplastin time, and serum levels of electrolytes, amylase, and troponin T were all normal. Results of a urine screen indicated no signs of infection. The serum level of myoglobin was elevated at 240 μg/L (normal 24-58 μg/L). A 12-lead electrocardiogram (ECG) obtained with the patient supine showed normal sinus rhythm (Figure 1).

The patient was moderately dehydrated, and intravenous fluid replacement was deemed necessary. Efforts to obtain peripheral venous access were unsuccessful. Air was aspirated during an attempt to cannulate the left subclavian vein for central venous access. Subsequently, a catheter was inserted in the left internal jugular vein. Ten minutes after insertion, the patient began experiencing left-sided, pleuritic chest pain with no radiation or concomitant nausea, diaphoresis, or palpitations. Vital signs, including oxygen saturation during delivery of oxygen at a rate of 3 L/min via nasal prongs, were stable except for tachycardia of 126/min. Lack of air entry and hyperresonance were detected over the left side of the thorax.

A 12-lead ECG obtained with the patient supine showed ST-segment elevation in leads II, III, aVF, V₅, and V₆; increased R wave in leads III and aVF; and inversion of the QRS complex in aVL (Figure 1). Findings in modified right-sided leads were normal. Because the findings were suggestive of acute myocardial infarction, aspirin 160 mg and metoprolol 25 mg were administered. Pain was treated with morphine 2.5 mg intravenously. Metoclopramide 10 mg was given intravenously along with morphine to prevent nausea.

Chest views were added to the abdominal computed tomography that previously had been ordered to detect any intra-abdominal abnormalities. The images of the chest confirmed a 70% left-sided pneumothorax, with the heart in anatomical position (Figure 2). An obstruction of the small bowel also was present. A chest tube with a one-way valve was placed to relieve the pneumothorax. Immediately after decompression of the pneumothorax, the patient’s pleuritic chest pain resolved. Chest radiographs and a 12-lead ECG obtained 1 hour after decompression revealed a completely reexpanded lung and resolution of ECG abnormalities (Figure 1), with the central venous catheter in good position in the superior vena cava. Serial measurements...
Figure 1  Electrocardiographic recordings from before (left), during (middle), and after (right) pneumothorax. Note ST-segment elevation in leads II, III, aVF, V5, and V6; increased R waves in leads III and aVF; and reversed polarity of QRS in aVL. T-wave abnormalities in leads V1 to V3 that were present before and during the pneumothorax resolved after decompression.
of troponin T were normal. The patient was in stable condition, and general surgery was consulted about the small-bowel obstruction.

Discussion

Left-sided pneumothoraces greater than 30% are often associated with recognizable findings on 12-lead ECGs. The most common findings are right-axis deviation, diminution of the QRS complex, T-wave inversion, and loss of R waves in the precordial leads.1 Early studies suggested that the absence of ST-segment elevation could be used to differentiate between pneumothorax and myocardial infarction; however, ST-segment elevation and pneumothorax are not mutually exclusive. Two cases of ST-segment elevation with pneumothorax have been reported,2,3 though in both cases the ST-segment elevation was associated with right-sided tension pneumothoraces. The authors2,3 suggested that ST-segment and T-wave changes were most likely due to transient hypoxia.
from impaired coronary blood flow caused by marked mediastinal displacement.

Unlike previously described cases of ST-segment elevation, in this case the patient did not have any mediastinal shift. Even in the absence of mediastinal shift, it is likely that the amount of air in the patient’s pleural cavity (70% pneumothorax) was sufficient for an adverse effect on cardiovascular physiology. Possibly, the intrapleural air was enough to restrict cardiac contractility and/or compress coronary vessels supplying inferolateral areas of the heart (most likely the left circumflex and right coronary arteries). With increased intrapleural pressures and reduced venous return, cardiac preload would decline, leading to decreased stroke volume. Tachycardia would then ensue to maintain cardiac output. The patient’s tachycardia would further increase myocardial oxygen demand, possibly leading to ischemia from underlying vessel disease. This may have been the case with this patient, especially with the increased physiological stress of her recent surgery and her present illness.

In the absence of mediastinal shift or elevation of cardiac enzyme levels, air acting as an insulator between the conducting electrode and the heart may have caused the 12-lead ECG findings in this patient, without the mechanism of myocardial ischemia described earlier. Decreased QRS voltage in pneumothorax can be simulated by the insertion of rubber dams beneath the precordial electrodes. Furthermore, 12-lead ECG changes associated with pneumothorax can normalize when the ECG is obtained with the patient in an upright position. This normalization is thought to be due to the majority of air moving to the apical aspects of the lung, relieving the pressure effects on the heart. Use of echocardiography to assess abnormalities in wall motion and flow dynamics during pneumothorax would have allowed a better assessment of cardiovascular physiology during this time. Cardiac stress testing after the patient had recovered also would have been beneficial to identify any underlying cardiac disease.

Although still controversial, several hypotheses have been proposed to explain 12-lead ECG changes during pneumothorax. Air in the pleural cavity exerts pressure on the heart, causing the heart to change location within the mediastinum. Clockwise rotation about the longitudinal axis (as viewed from the apex) and posterior shifting of the heart have been suggested as possible reasons for such 12-lead ECG changes. Marked rotation or translocation of the heart causes deviation of the mean QRS vector, altering such variables as axis and QRS amplitude. During the cardiac cycle, pendular motion of the heart within intrathoracic air alters the distance between the heart and the chest wall. This movement portends changes in the ECG baseline voltage leading to ST-segment shifts. Interposed air between the heart and the conducting electrode and right ventricular dilatation from increased pulmonary vascular resistance also may alter voltage recordings of electrodes. Because 12-lead ECG findings associated with pneumothorax are highly variable, the relative contribution of each of these factors may be crucial to the 12-lead ECG abnormalities observed.

Unlike the classic findings described earlier, the most apparent 12-lead ECG finding in this patient was ST-segment elevation in leads II, III, aVF, V5, and V6. A suggestion of decreased left-sided forces was observed with inversion of the QRS complex in aVL; however, the amplitude of the R wave in the precordial leads remained unchanged. Inferior forces also may have been augmented during the pneumothorax, because increased R waves were noted in leads III and aVF.

Although ECG is more than 100 years old, it remains a steadfast tool in the diagnosis of myocardial injury. The initial change with myocardial ischemia is a loss of resting membrane potential, causing depression of the TQ segment. In transmural myocardial infarction, myocytes surrounding the infarct zone lose membrane stability and are in a state of “diastolic leak.” This partial depolarization decreases the amplitude of myocyte action potential and directs force vectors away from the injured myocardium and recording electrode. The result is a downward shift of the 12-lead ECG baseline and the appearance of ST-segment elevation as the heart repolarizes.

Intrapleural air, either by having a deleterious effect on preexisting, fragile coronary vessels leading to compression and transient ischemia or by causing atypical electrode recordings (or a combination of both), most likely was responsible for the novel 12-lead ECG changes in this case.

A STAT ECG is often the first test ordered for patients experiencing chest pain. A fundamental history and physical examination are essential to avoid acting prematurely on the basis of changes on 12-lead ECGs. Astute clinicians should recognize that the underlying reasons for ST-segment elevation can be extensive and may include such diagnoses as pancreatitis and subarachnoid hemorrhage. In the appropriate clinical setting, pneumothorax should be ruled out.

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