

Case Letter

Stress-induced cardiomyopathy with electrocardiographic ST-segment elevation in a patient with pneumothorax

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Dear editor,

In many cases, ST-segment changes on electrocardiogram (ECG) are suggestive of acute coronary syndrome, but there may be other causes,^[1,2] such as stress-induced cardiomyopathy. However, reports of such changes in patients with pneumothorax are very rare.^[3–11] Here, we report a case of ST-segment elevation due to stress-induced cardiomyopathy in a patient with pneumothorax.

CASE

An 81-year-old man was brought to the emergency department (ED) with dyspnea lasting for several hours. He had complained of chest discomfort 2 days before and had a history of hypertension and chronic obstructive pulmonary disease (COPD). Two years prior, he had developed iatrogenic left pneumothorax from an acupuncture needle and had undergone closed thoracostomy. Additionally, he had undergone surgery for gastric cancer 7 years ago. Initially, his blood pressure was 130/80 mmHg, pulse 84/minute, respiration 22/minute, and body temperature 37.2 °C. His oxygen saturation when breathing room air was 92%. Laboratory examination in the ED revealed creatine kinase myocardial band (CK-MB) 1.8 ng/mL (normal 3.6 ng/mL), troponin T high-sensitive (troponin T-hs) test <0.01 ng/mL (normal 0.1 ng/mL), and pro-B-type natriuretic peptide 132.0 pg/mL (normal 32.6–219.9 pg/mL). However, from an initial ECG, ST-segment elevation was seen in the leads II, III, aVF, V3, V4, V5, and V6 (Figure 1), and follow-up assessment of cardiac enzyme showed that CK-MB and troponin T-hs had increased to 5.5 ng/mL and 0.3 ng/mL, respectively. Chest radiography revealed left pneumothorax (Figure 2). Chest computed tomography

(CT) revealed an overall emphysematous change in both the lungs. Acute coronary syndrome was suspected, and pneumothorax was resolved by performing closed thoracostomy with 24 Fr chest tube. Although coronary angiography was planned, a coronary CT scan was performed due to the patient's strong refusal to an angiography. The CT scan did not reveal any specific stenosis, except for calcification of the left anterior descending artery. Transthoracic echocardiography showed hypokinesia of the mid- and apical- segments with sparing movements of the basal segment as typical characteristics of stress-induced cardiomyopathy (Figure 3). The patient was admitted to the general ward and received supportive treatment with chest underwater drainage system in the left pleural space. On follow-up laboratory examinations, cardiac enzymes showed a decreasing tendency with peak CK-MB and high-sensitivity troponin T levels at 7.0 ng/mL and 0.3 ng/mL, respectively, one day after admission to the general ward. The ECG showed changes in the ST-segment elevation to diffuse T-wave inversion. On the 9th day of admission, echocardiography showed that cardiac wall motion returned to be normal. On the 4th day of hospitalization, the chest tube was removed after confirmation of no air leak; however, minimal pneumothorax developed and was absorbed. Finally, the patient was discharged on the 17th day of hospitalization. Further treatments, including pleurodesis, were not considered as it may exacerbate the stress-induced cardiomyopathy.

DISCUSSION

Stress-induced cardiomyopathy was first introduced in Japan in 1990,^[12] and ever since many studies have been conducted.^[2] Although the precise pathophysiology

remains unclear, catecholamine cardiotoxicity is considered as the most important, with a prevalence of 1.7%–2.2% of patients with acute coronary syndrome.^[13] The Mayo Clinic diagnostic criteria were used to diagnose stress-induced cardiomyopathy. These criteria included: new ECG changes or elevated cardiac troponin; absence of coronary artery disease, recent significant head trauma, intracranial bleeding, pheochromocytoma, myocarditis, or hypertrophic cardiomyopathy; and transiently decreased ventricular systolic function extending beyond a single coronary artery.^[14] Stress-induced cardiomyopathy shows various patterns on an ECG, and ST-segment elevation is the most common change. T-wave changes, Q-wave, and prolongation of QT interval are also observed. In patients with pneumothorax, changes in the ST-segment in the ECG have been reported for a very long time, even without tension pneumothorax.^[3-11] Although the mechanism of the ECG changes in pneumothorax has not been clearly identified, there are several hypotheses to explain ECG alterations.^[3,4] Pneumothorax may cause a change in the heart position, which can cause ECG changes. More specifically, the air in the retrosternal space can change the distance between the heart and chest wall, which weakens electric conductivity. Alteration of cardiac contraction due to air between the heart and its surroundings also affects ECG baseline voltage, resulting in ST-segment elevation. Most mechanisms

described in previous studies elucidated it as the increase in intrathoracic pressure at the time of pneumothorax leading to a decrease in the venous return and increase in the pulmonary vascular resistance, which results in decreasing stroke volume that induces tachycardia with an increased oxygen demand in the myocardium; this makes it similar to ischemic heart disease.

The cause of ST-segment elevation in such patients remains unclear. Although the abnormalities of the ECG are resolved within a short time after resolution of the pneumothorax, as in previously reported cases,^[4-8] it may be different for cases in which abnormality of the ECG persisted for a long time, as in our case. Chan et al^[11] have reported that only 27% of right-sided pneumothorax recovered to normal ST-segment after closed thoracostomy in 25% of left-sided pneumothorax. Therefore, it may be difficult to identify the reason simply from changes in the ECG. With respect to myocardial enzymes in pneumothorax, normal category values have been reported for most cases.^[3-8] Although myocardial enzymes are elevated in some cases of pneumothorax,^[10,11] in our case, it could also be due to stress-induced cardiomyopathy. Considering the

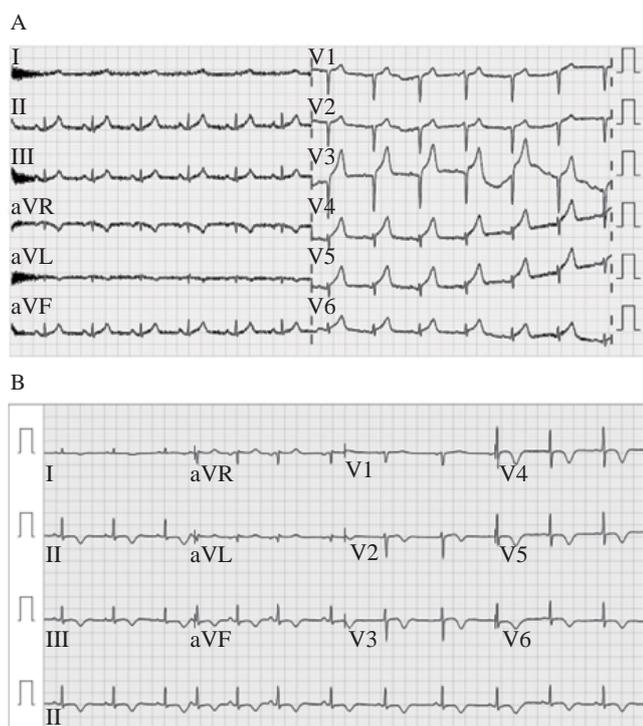


Figure 1. ECG findings at the initial (A) and last follow-up (B).



Figure 2. Chest radiography shows the left pneumothorax.



Figure 3. Echocardiography shows preserved movement of the basal segment of the left ventricle with decreased movement of apical- and mid-segments, a characteristic sign of stress-induced cardiomyopathy.

clinical situation of our case, myocardial enzyme levels were elevated for more than one week, indicating that ST-segment elevation was due to stress-induced cardiomyopathy rather than pneumothorax, as it would have resolved within a short time in the latter case.

Cases of combined pneumothorax and stress-induced cardiomyopathy are rarely reported. After searching in the English literature, only six cases were found to be reported.^[13,15-19] A brief analysis of these cases showed that, unlike in our case, all patients were women, with ages ranging from 58.0 to 83.0 years (average 69.5 years). Among these, three had tension pneumothorax, and four had pneumothorax on the right side. All these patients had a history of lung-related diseases. Three patients had COPD, one had asthma, and one had bronchogenic carcinoma. The other one was the oldest patient, aged 83 years old, and had a history of closed thoracostomy with pneumothorax, which occurred more than 50 years prior. All were expected to have limited lung function.

Although there is no scientifically proven mechanism between pneumothorax and stress-induced cardiomyopathy, the possibility of stress and hypoxemia occurring due to pneumothorax can affect the heart.^[17] Stress and hypoxemia may induce catecholamine release, which can in turn contribute to cardiac dysfunction. Particularly, stress-induced cardiomyopathy can occur in patients with limited pulmonary function owing to conditions such as COPD, even without tension pneumothorax.

CONCLUSIONS

In conclusion, serial echocardiographic evaluations and a thorough laboratory test for myocardial enzymes are required, and ST-segment elevation in patients with pneumothorax can be caused not only from pneumothorax but also from other heart problems, especially in cases of limited pulmonary function.

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