Internal Medicine
The Japanese Society of Internal Medicine
doi: 10.2169/internalmedicine.1422-18
Intern Med 58: 1131-1135, 2019
http://internmed.jp

【 CASE REPORT 】

Concomitant Spontaneous Tension Pneumothorax and Acute Myocardial Infarction

Kenshiro Arao[1], Takaaki Mase[1], Mori Nakai[2], Hiroshi Sekiguchi[2], Yasunori Abe[2], Naomi Kuroudu[3] and Ouji Oobayashi[3]

Abstract:
A 71-year-old man presented to our hospital for dyspnea lasting for the past 3 days. Chest X-ray and computed tomography demonstrated right tension pneumothorax, and an electrocardiogram suggested acute inferior myocardial infarction. Despite the relief of tension pneumothorax, the electrocardiographic findings were not completely resolved. Emergency coronary angiography demonstrated an occlusive lesion in the right coronary artery, and percutaneous coronary intervention was performed successfully. Thereafter, the chest tube was removed, and he was discharged. While rare, multiple life-threatening diseases that present with similar symptoms can coexist, so a re-evaluation after performing the initial treatment for one of these diseases is crucial.

Key words: acute myocardial infarction, dyspnea, electrocardiogram, tension pneumothorax

(Intern Med 58: 1131-1135, 2019)
(DOI: 10.2169/internalmedicine.1422-18)

Introduction

In daily practice, we sometimes experience patients with multiple concurrent diseases, each of which may demonstrate similar symptoms or laboratory findings. In such cases, a prompt and accurate diagnosis is crucial for providing appropriate treatment for each disease (1). We experienced a case with concomitant spontaneous tension pneumothorax and acute myocardial infarction, both of which were emergent and life-threatening, that was successfully treated.

Case Report

A 71-year-old man who had pulmonary emphysema, hypertension and a long-term history of smoking but no diabetes mellitus walked into the general outpatient department for dyspnea lasting for the past 3 days.

He had no chest pain or chest oppression on arrival. Since he showed severe hypoxemia with percutaneous oxygen saturation (SpO_2) of 68% on room air, oxygenation using a reservoir mask was immediately started. His blood pressure was 114/76 mmHg, and his pulse was regular at 115 beats per minute. His breath sound was diminished over the right chest, and no cardiac murmurs were audible. He showed no jugular vein distention and no edema in either lower limb. An electrocardiogram (ECG) showed ST-segment elevation in leads II, III and aVF and reciprocal ST-segment depression and negative T wave in leads I, aVL and V4-6 without significant axis deviation (Fig. 1), suggesting that he had acute inferior myocardial infarction. Chest X-ray demonstrated the collapsed right lung which was accompanied by leftward-shift of the mediastinum (Fig. 2a), indicating that he had right-sided tension pneumothorax. Immediately, a chest tube was inserted into the right thoracic cavity, and aspiration at the water-seal level was started (Fig. 2b). Thereafter, the level of SpO_2 increased from 90% to 98% under maximal oxygenation, and he remained in a hemodynamic steady-state condition. Subsequent computed tomography (CT) demonstrated residual right pneumothorax and diffuse multiple low-attenuation areas associated with pulmonary emphysema in both lungs as well as atelectasis in the right lower lobe (Fig. 3).

[1]Division of Cardiovascular Medicine, Nerima-Hikarigaoka Hospital, Japan, [2]Division of Internal Medicine, Nerima-Hikarigaoka Hospital, Japan and [3]Division of Respiratory Medicine, Nerima-Hikarigaoka Hospital, Japan
Received: April 21, 2018; Accepted: September 24, 2018; Advance Publication by J-STAGE: January 10, 2019
Correspondence to Dr. Kenshiro Arao, araon@omiya.jichi.ac.jp

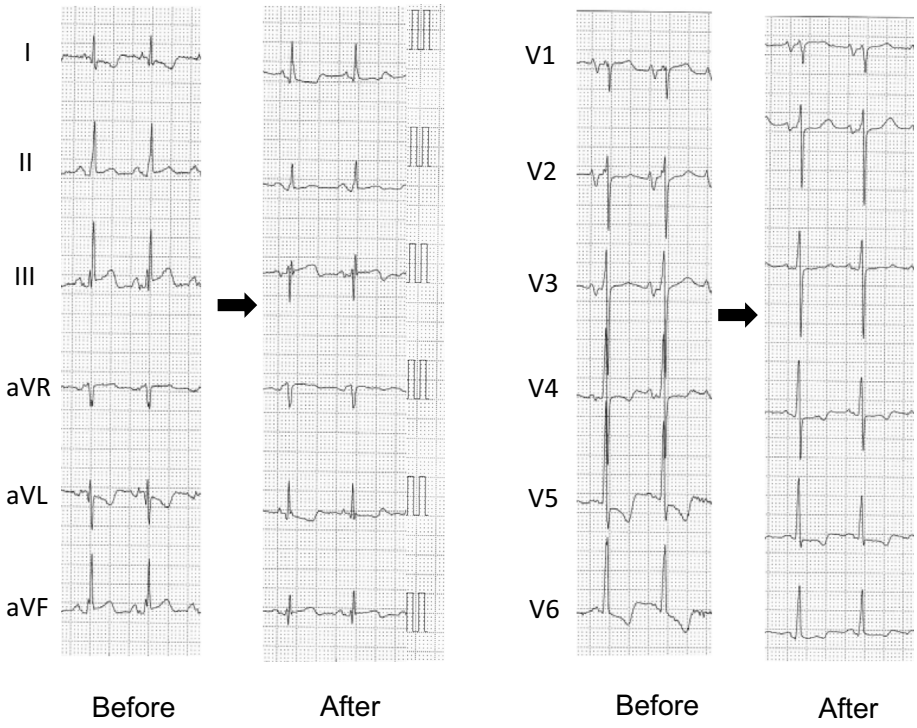

Figure 1. **Electrocardiographic changes before and after lung expansion. An electrocardiogram (ECG) before lung expansion demonstrated ST-segment elevation in leads II, III and aVF and reciprocal ST-segment depression and negative T wave in leads I, aVL and V4-6. An ECG after lung expansion revealed persistent slight ST-segment elevation in II, III and aVF and a reduced R-wave amplitude in V4-6 compared with before.**

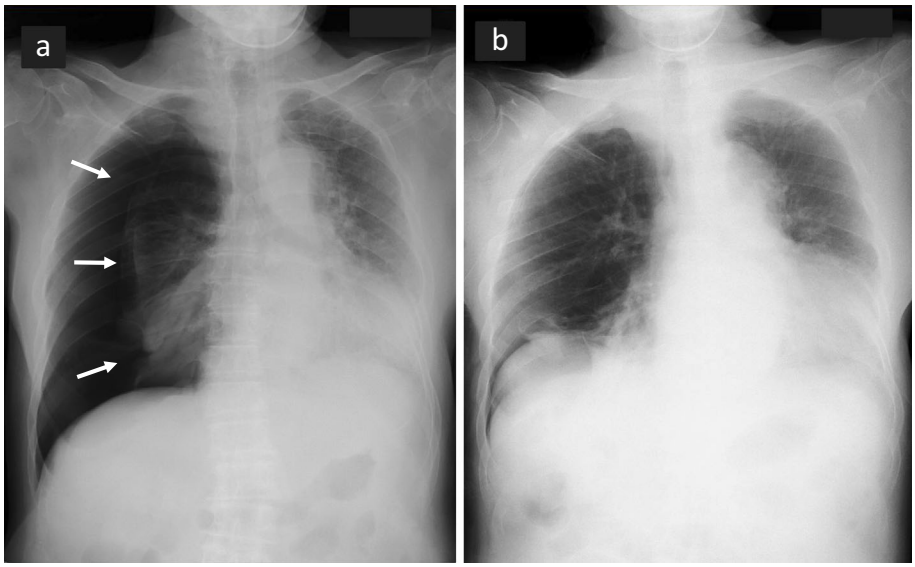

Figure 2. **Chest X-ray on arrival (a) and after expansion (b). Chest X-ray in (a) demonstrated right tension pneumothorax and leftward deviation of the mediastinum. The white arrows indicate the margin of the lung.**

However, despite the relief of tension pneumothorax, the ST-segment elevation on the ECG was not completely resolved (Fig. 1). Furthermore, blood laboratory tests on arrival revealed elevated levels of white blood cells (17,100/μL), creatine kinase (390 IU/L) and Troponin I (1.51 ng/mL), as shown in Table, and an echocardiogram showed a slight hypokinetic motion in the inferior wall of the left ventricle with a left ventricular ejection fraction of 50%, all of which indicated that he had acute inferior myocardial infarction. Emergency coronary arteriography demonstrated a thrombotic occlusive lesion in the proximal site of the right coronary artery, which was associated with a moderate degree of collateral supply from the left coronary artery.

Percutaneous coronary intervention (PCI) was therefore

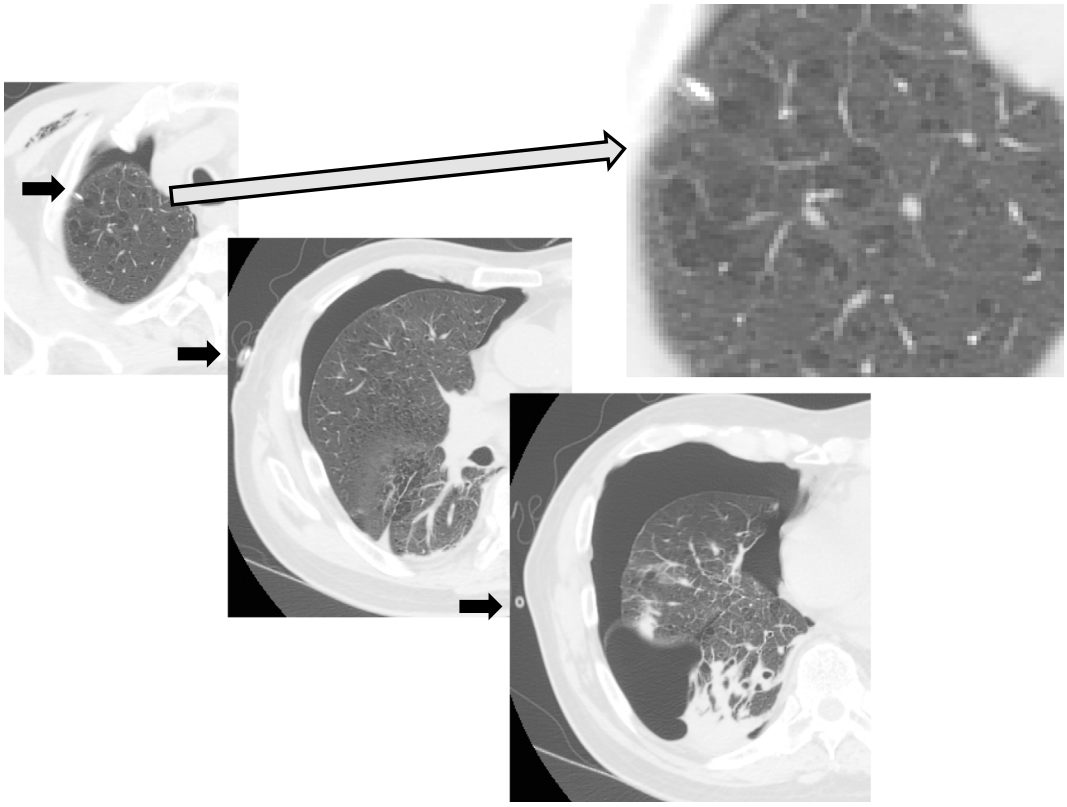

Figure 3. **Chest plain computed tomography (CT) findings soon after lung expansion. CT revealed residual right pneumothorax and low-attenuation areas associated with pulmonary emphysema in both lungs and atelectasis in the right lower lobe. The black arrows indicate the chest tube.**

Table. **Blood Laboratory Tests on Arrival.**

<Hematology>		Creatinine kinase	390 IU/mL
White blood cell	17,100 /μL	Troponin I	1.51 ng/mL
Red blood cell	508 $\times 10^4$/μL	BNP*	639 pg/mL
Hemoglobin	16.4 g/dL	Casual plasma glucose	221 mg/dL
Hematocrit	50.8 %	HbA1c	6.0 %
Platelet	19.9 $\times 10^4$/μL		
		<Arterial blood gas on room air>	
<Biochemistry>		pH	7.362
Total protein	7.6 g/dL	PCO_2	35.9 Torr
Albumin	4.2 g/dL	PO_2	37.6 Torr
Total bilirubin	1.0 mg/dL	HCO_3^-	19.9 mmol/L
AST	29 IU/L	Base excess	-4.6 mmol/L
ALT	28 IU/L	SaO_2	68.0 %
γ-GTP	21 IU/L		
Blood urea nitrogen	64.8 mg/dL		
Creatinine	2.18 mg/dL		
Na	143 mEq/dL		
K	3.7 mEq/dL		
Cl	103 mEq/dL		

ALT: alanine aminotransferase, AST: aspartate aminotransferase, BNP: brain natriuretic peptide, γGTP: γ-glutamyl transpeptidase, SaO_2: saturation of arterial oxygen

*A blood sample of BNP was taken on the same day after admission.

performed (Fig. 4). Initially, we intended to perform thrombectomy or balloon dilatation without stent implantation that would consequently necessitate dual anti-platelet therapy. Since the lung tissues on CT (Fig. 3) appeared very fragile due to emphysematous changes and a considerable amount of air was leaking from the chest tube, we assumed that surgical intervention to the pneumothorax would eventually be needed in order to halt the air leakage, in which case dual

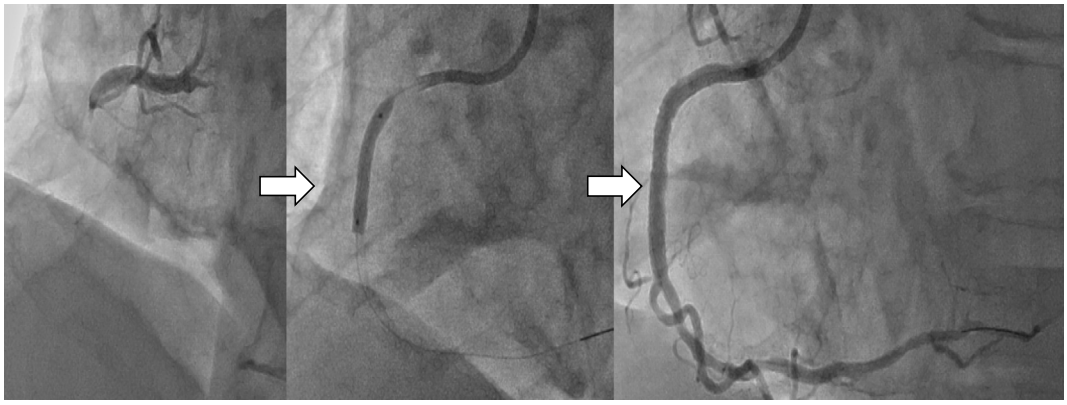

Figure 4. Percutaneous coronary intervention. Coronary arteriography showed the occluded lesion in the proximal right coronary artery, and optimal coronary intervention was performed.

anti-platelet therapy would complicate the prospective surgery. However, since thrombectomy alone was insufficient to restore the coronary blood flow across the thrombus and atherosclerotic stenotic lesion detected by intravascular ultrasound, we had no choice but to implant a stent, and optimal stent expansion and coronary blood flow without significant delay were obtained.

After PCI, the patient was managed in the intensive-care unit, and serum creatine kinase and its MB isozyme peaked at 1,086 and 121 IU/L within 24 hours, respectively. The right lung was kept expanded for three days, and the chest tube was withdrawn. However, since tension pneumothorax recurred immediately after a strong and intense cough on the fourth day, a chest tube was inserted again. Contrary to our initial assumption, his lung tissues were too fragile for surgical intervention. His right lung was thus kept expanded by chest tube drainage for two weeks, at which point the chest tube was finally removed without the recurrence of pneumothorax. After several days of observation, he was discharged. Since then, his condition has been uneventful for more than three years.

Discussion

We experienced a patient who simultaneously suffered from tension pneumothorax and acute myocardial infarction. Although both can cause similar symptoms, we were able to diagnose and treat both conditions promptly and successfully (1).

On arrival, the patient had no chest pain or oppression that would have suggested the presence of acute myocardial infarction (2). He did not have an apparent history of diabetes mellitus, and the level of HbA1c was 6.0%. The level of plasma glucose (226 mg/dL) on arrival was significantly elevated, which could be explained by a pathophysiological response sometimes seen in acute myocardial infarction (3). Therefore, it was unlikely that his chest symptoms that should have been associated with myocardial infarction were masked by diabetes neuropathy. Instead, we assumed that the absence of chest pain was due to the existance of dyspnea caused by tension pneumothorax or advanced age or that the pain had been slightly ameliorated by the presence of a coronary collateral supply (4-6).

Pneumothorax causes ECG changes in approximately 25% of cases, including QRS axis deviation, typically a right-axis deviation (in more than half of patients); clockwise rotation in the precordial leads; a reduction in the QRS amplitude; and T-wave inversion in the precordial leads (7, 8). In right-sided pneumothorax in particularly, the ECG demonstrates a reduction in the R-wave amplitude in V1-2; an increased R-wave amplitude in V5-6, which is attributed to the shortened distance between the heart and chest wall; and right bundle-branch block or negative T wave (7). In the present case, the R-wave amplitude in V5-6 before lung expansion was higher than that after the expansion (Fig. 1), and while a negative T wave was found, right bundle-branch block was not observed. Previous case reports on pneumothorax with ECG changes included relatively few cases with ST-segment changes (9-11). Anderson et al. described a case with right-sided pneumothorax and ST-segment elevation mimicking inferior myocardial infarction despite a normal coronary angiogram; the authors considered that the ST-segment change had been caused exclusively by pneumothorax (9). In tension pneumothorax, the following hemodynamic changes occur: transient hypoxemia, a reduction in the coronary arterial blood flow, an increase in the pulmonary vascular resistance and an increase in the intrapericardial pressure (8). In animal models of tension pneumothorax, the cardiac output decreases, the mean arterial pressure decreases at a collapsing rate of ≥56%, and complete cardiovascular collapse occurs at a collapsing rate of ≥94% (8). In the present case, serial changes in the ECG and in the levels of serum cardiac enzymes after admission indicated that his myocardial infarction was in the acute phase; however, dyspnea occurred three days before admission, suggesting that pneumothorax preceded the occurrence of acute myocardial infarction. Furthermore, we identified a coronary atherosclerotic lesion at the site of occlusion by angiographic and intravascular ultrasound imaging. Although it was not feasible to estimate the degree to which athero-

sclerosis and pneumothorax contributed to the onset of his myocardial infarction, pneumothorax can affect the onset of acute myocardial infarction by reducing the coronary blood flow and arterial blood oxygenation.

We experienced a case with concomitant tension pneumothorax and acute myocardial infarction, both of which are life-threatening diseases with similar and confounding symptoms and ECG changes. Delays in the diagnosis and treatment of such cases should be avoided (1). Rarely but occasionally multiple serious diseases presenting with similar symptoms can coexist. We should keep this in mind when we encounter patients in daily practice.

The authors state that they have no Conflict of Interest (COI).

References

1. McNamara RL, Wang Y, Herrin J, et al.; NRMI Investigators. Effect of door-to-balloon time on mortality in patients with ST-segment elevation myocardial infarction. J Am Coll Cardiol **47**: 2180-2186, 2006.
2. Cohn PF. Silent myocardial ischemia. Ann Intern Med **149**: 312-317, 1988.
3. Ceremuzynski L. Hormonal and metabolic reactions evoked by acute myocardial infarction. Circ Res; **48**: 767-776, 1981.
4. Niakan E, Harati Y, Rolak LA, Comstock JP, Rokey R. Silent myocardial infarction and diabetic cardiovascular autonomic neuropathy. Arch Intern Med **146**: 2229-2230, 1986.
5. Valensi P, Lorgis L, Cottin Y. Prevalence, incidence, predictive factors and prognosis of silent myocardial infarction: a review of the literature. Arch Cardiovasc Dis **104**: 178-188, 2011.
6. Sheifer SE, Gersh BJ, Yanez ND 3rd, Ades PA, Burke GL, Manolio TA. Prevalence, predisposing factors, and prognosis of clinically unrecognized myocardial infarction in the elderly. J Am Coll Cardiol **35**: 119-126, 2000.
7. Krenke R, Nasilowski J, Przybylowski T, Chazan R. Electrocardiographic changes in patients with spontaneous pneumothorax. Physiol Pharmacol **59** (Suppl 6): 361-373, 2008.
8. Janssens U, Koch KC, Graf J, Hanrath P. Severe transmyocardial ischemia in a patient with tension pneumothorax. Crit Care Med **28**: 1638-1641, 2000.
9. Anderson R, Costello B, Kilpatrick D. Transient ST-segment elevation resembling acute myocardial infarction in a patient with a right secondary spontaneous pneumothorax. Heart Lung Circ **22**: 149-152, 2013.
10. Alzghoul B, Innabi A, Shanbhag A, Chatterjee K, Amer F, Meena N. Iatrogenic right-sided pneumothorax presenting as ST-segment elevation: a rare case report and review of literature. Case Rep Crit Care **2017**: 3291751, 2017.
11. Slay RD, Slay LE, Luehrs JG. Transient ST elevation associated with tension pneumothorax. JACEP **8**: 6-8, 1979.