

Stress-induced Cardiomyopathy Associated with Non-Small Cell Lung Cancer Presenting as Hyponatremia

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Stress-induced cardiomyopathy, so-called Takotsubo cardiomyopathy, has recently been reported in Japan. Stress-induced cardiomyopathy is characterized by transient left ventricular apical dysfunction and ballooning, with normal coronary angiographic findings. We describe a rare case of stress-induced cardiomyopathy associated with lung adenocarcinoma presenting as hyponatremia. (**Ewha Med J 2015;38(2):90-93**)

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Key Words

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syndrome; Hyponatremia; Inappropriate
ADH syndrome; Lung neoplasms

Introduction

Stress-induced cardiomyopathy was first introduced in Japan under the name Takotsubo cardiomyopathy [1]. Since then, several cases with similar clinical features have been reported [2,3]. Stress-induced cardiomyopathy is accompanied by apical akinesia, along with apical ballooning, and is characterized by transient left ventricular dysfunction. In addition, it has clinical features that are similar to acute myocardial infarction but has normal coronary angiographic findings.

Clinical features of stress-induced cardiomyopathy include acute chest pain, ischemic echocardiographic change, and increased cardiac enzyme. Because these clinical features are similar to those of acute myocardial infarction, stress-induced cardiomyopathy can be mistaken for acute myocardial infarction in clinical settings. In most cases, coronary angiography is performed, thereby differential diagnosis can be made from acute myocardial infarction. Unlike acute myocardial infarction, most

patients with stress-induced cardiomyopathy show improvements in their symptoms within 1 week and a pattern of return to near-normal cardiac function within 1 month [4].

Authors encountered a patient with stress-induced cardiomyopathy that presented clinical features of hyponatremia, who was also diagnosed with lung adenocarcinoma, and hereby report this case along with a literature review.

Case

A 72-year-old woman visited the Kwangju Christian Hospital with a chief complaint of dizziness, nausea, and vomiting for the previous 3 days. She was being treated for 2 weeks before admission at a nearby hospital for cystitis with fever and dysuria, during which time she was transferred to our hospital with a chief complaint of dizziness, nausea, and vomiting.

According to her medical history, she was diagnosed with hypertension 15 years prior and had been treated with medica-

tion since then. She had no history of diabetes, tuberculosis, or liver disease. She had a right knee joint replacement surgery for degenerative arthritis 8 years earlier. Her family medical history indicated no particular findings. Upon admission, her blood pressure was 120/70 mmHg; pulse rate, 64 bpm, and body temperature, 36.2°C. She showed signs of acute conditions such as dizziness, nausea, and vomiting, but no signs of dyspnea or chest pain. Her pulse rate was regular, while no prominent rale was heard in the lung sound examination. She did not complain of tenderness or rebound tenderness during the abdominal examination.

Electrocardiography revealed normal sinus rhythm, and a T inversion of ≥ 9 mm was observed in the aVL, V1, V2, V3, V4, V5, and V6 leads (Fig. 1).

Chest radiography revealed cardiomegaly, along with enlarged right hilum. The result of the blood test performed at admission showed the following values: Na, 120 mEq/L and K, 3.8 mEq/L, indicating hyponatremia. BUN (13.0 mg/dL) was normal range, Cr (0.8 mg/dL) was normal range. Serum Osm (244 mOsm/kg) decreased, and urine Osm (530 mOsm/kg), urine sodium (150 mEq/L), creatine kinase (CK; 114 IU/L), creatine

kinase isoenzyme MB (CK-MB; 8.46 ng/mL), troponin I (0.96 ng/mL), troponin T (0.071 ng/mL), and D-dimer (11.1 $\mu\text{g/mL}$) increased. The urine analysis values were white blood cell count of 0–1/HPF and red blood cell count of 30–49/HPF. Thyroid function test was within normal limits and rapid adrenocorticotrophic (ACTH) stimulation test was unremarkable. On the transthoracic two-dimensional echocardiography performed on the first day of admission, the left ventricular ejection fraction was 56%, which indicated good left ventricular systolic function. However, left ventricular apical wall motion abnormality and ballooning were observed. Moreover, mild aortic valve regurgitation due to calcified aortic valve was also observed (Fig. 2).

Because the patient complained primarily of nausea and vomiting rather than chest pain, 3% hypertonic saline was administered for hyponatremia until serum sodium level 131 mEq/L, and the symptoms improved following conservative therapy. The patient was maintained on conservative management, including aspirin 100 mg, clopidogrel 75 mg orally. Three days after, cardiac enzyme were significant for CK 190 IU/L, CK-MB 11.25 ng/mL, troponin I 0.83 ng/mL, troponin T 0.065 ng/mL.

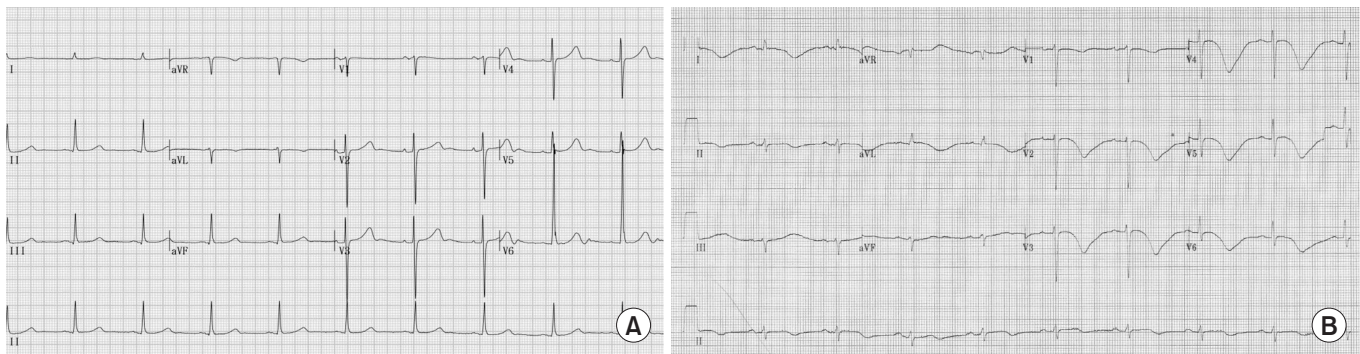


Fig. 1. Electrocardiogram (ECG). (A) ECG obtained two years ago shows sinus rhythm with left ventricular hypertrophy. (B) ECG obtained on arrival shows normal sinus rhythm with T inversion in all the leads.

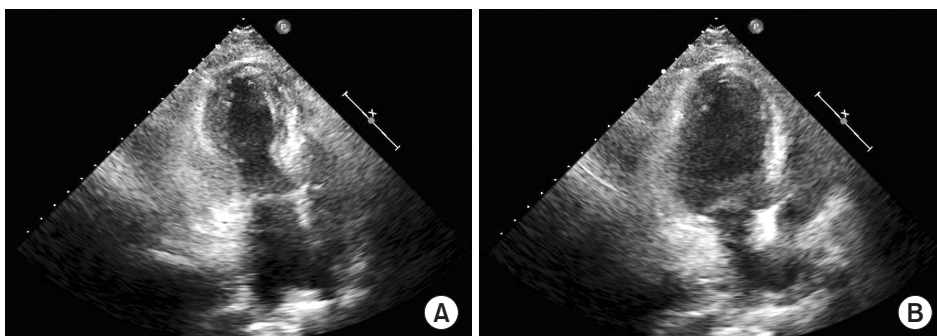


Fig. 2. Echocardiography during systole (A) and diastole (B), on the first day of admission. Initial echocardiography reveals left ventricular apical wall motion abnormality and ballooning.

Seven days after, patient remained stable and cardiac enzymes were decreased (CK 65 IU/L, CK-MB 2.09 ng/mL, troponin I 0.26 ng/mL, troponin T 0.037 ng/mL). On the coronary angiography performed on the seventh day after admission, no significant stenosis was found (Fig. 3). On the transthoracic two-dimensional echocardiography performed again on the seventh day after admission, improvements were observed in the left ventricular apical dyskinesia that was exhibited previously (Fig. 4). On the chest computed tomography, an indication of primary lung malignancy was found in the right upper lobe of lung (Fig. 5). Endobronchial ultrasonography-guided fine-

needle aspiration biopsy results indicated a poorly differentiated lung adenocarcinoma (Fig. 6). On positron emission tomographic computed tomography, indications of metastasis in both the pelvic bones and right femur were found, for which palliative systemic chemotherapy was to be administered. However, in consideration of the systemic condition, conservative therapy was administered instead.

Discussion

In this case study, we report a case of stress-induced car-



Fig. 3. Coronary angiogram. Coronary angiogram shows normal left (A) and right (B) coronary arteries.

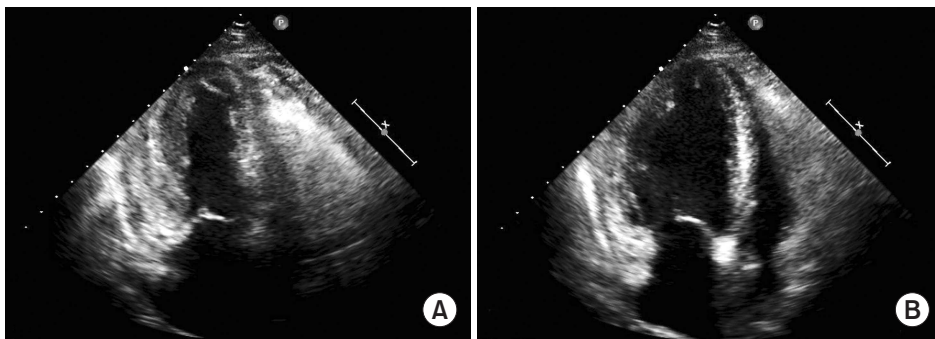


Fig. 4. Echocardiogram obtained during systole (A) and diastole (B) on the seventh day after admission. Echocardiogram shows the recovery of the previous wall motion abnormalities.



Fig. 5. Chest computed tomography image obtained on admission. Chest computed tomography shows an approximately 4.4-cm long, enhancing mass-like consolidation in the right upper lobe of the lung at serial axial image (A) and (B).

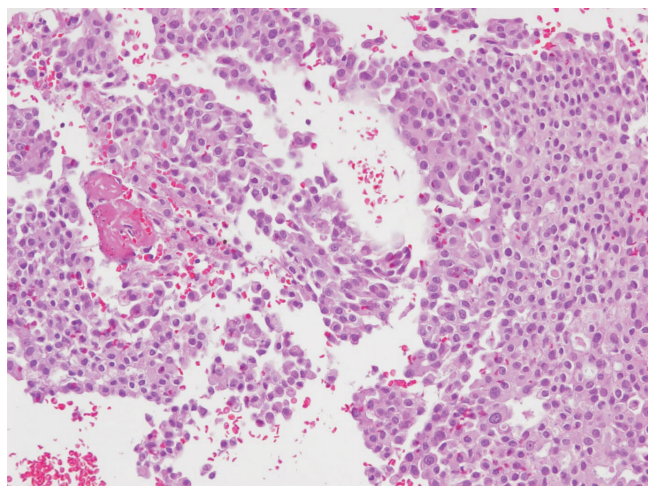


Fig. 6. Biopsy in the right upper lobe with endobronchial ultrasonography guidance. The adenocarcinoma is poorly differentiated. Histologic examination of the lung shows atypical cell proliferation with indistinct glandular structures (H&E, $\times 200$).

diomyopathy accompanied with lung adenocarcinoma that presented with clinical symptoms of hyponatremia. Stress-induced cardiomyopathy occurs in association with various conditions. Its causes include emotional stress, surgical disposition, and diseases such as septicemia and malignant tumors [5]. Most clinical cases of stress-induced cardiomyopathy show good prognosis. However, some cases are accompanied by various complications. Known complications include cardiogenic shock, thrombosis, heart failure, and cerebral infarction [6]. In lung cancer patients, hyponatremia caused by the syndrome of inappropriate antidiuretic hormone (SIADH) secretion is often known to develop into paraneoplastic syndrome. In particular, hyponatremia associated with the SIADH secretion occurs commonly in patients with small cell lung cancer [7,8]. Hyponatremia associated with the SIADH secretion is known to be rare in patients with non-small cell lung cancer, accounting only for <1% of patients, according to previous studies [9,10]. In this case, the patient was admitted owing to atypical symptoms such as nausea and vomiting, and the laboratory test results showed indications of T inversion and increased cardiac enzyme, besides hyponatremia. Based on these results, coronary angiography was performed to eliminate the possibility of acute myocardial infarction. The coronary angiography did not reveal significant indications of stenosis, and an indication of primary lung malignancy was discovered during the evaluation for hyponatremia. We considered

SIADH induced by lung cancer as a cause of hyponatremia. There was no other reason to cause of hyponatremia in this case. Although hyponatremia associated with the SIADH secretion is known to rarely occur in non-small cell lung cancer, an indication of lung adenocarcinoma was found on the endobronchial ultrasonography-guided fine-needle aspiration biopsy.

Stress-induced cardiomyopathy is a heart disease with various causes. Its prognosis may vary depending on the cause. The authors experienced a case of stress-induced cardiomyopathy with similar features as acute myocardial infarction in a patient with non-small cell lung cancer that presented with hyponatremia and hereby report the case.

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