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Cardiac Tamponade and Pericarditis in Type B Intramural Hematoma: a Case Report

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We report the case of a patient with cardiac tamponade and pericarditis in type B intramural hematoma. A 75-year-old woman was admitted to the emergency department presenting with general weakness and dizziness for several hours and hemodynamic collapse. Thoracic echocardiography and computed tomography (CT) showed a large pericardial effusion and aortic intramural hematoma but no evidence of aortic dissection. Therefore, we concluded that the intramural hematoma did not involve the ascending aorta and thus immediately performed pericardiocentesis. Follow-up CT showed no pericardial effusion or specific changes in the range or depth of the intramural hematoma, and she was discharged continuing colchicines and ibuprofen therapy for acute pericarditis. Cardiac tamponade in type B intramural hematoma is extremely rare. Prompt diagnosis and initial treatment resulted in a substantial improvement in clinical status. (Korean J Med 2015;89:695-698)

Keywords: Cardiac tamponade; Hematoma; Pericarditis

INTRODUCTION

Intramural hematoma (IMH) is a life-threatening disease whose pathophysiology is thought to be related to a contained hemorrhage within the medial layer of the aorta due to either rupture of the vasa vasorum or an atherosclerotic plaque [1]. IMH is classified as involving (type A) or not involving (type B) the ascending aorta. Patients with type A IMH usually present with more serious complications than those with type B IMH, including cardiac tamponade [2]. The initial management of IMH focuses on hemodynamic stability and clinical symptoms. Cases of acute type B IMH are usually treated conservatively, although open aortic surgery is usually considered in patients with type A IMH because of the risk of progression

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to aortic dissection or rupture [3]. Pericardial and pleural effusion, as well as mediastinal hemorrhage, are more frequent findings in IMH, especially in type A IMH, than aortic dissection [4]. Here, we report the case of a patient with type B IMH who developed cardiac tamponade and pericarditis after pericardiocentesis.

CASE REPORT

A 75-year-old woman was admitted to the emergency department presenting with general weakness and dizziness for several hours. She did not have any noteworthy medical history. She was initially in hemodynamic collapse (systolic blood pressure, 40 mmHg). Her body temperature was 36.1°C, and her heart rate was 97 bpm. After fluid resuscitation, an elevated jugular venous pulse was noted. Electrocardiography showed sinus tachycardia. Chest radiography showed a widened mediastinum and cardiomegaly. Laboratory results were as follows: white blood cell count, 11,790 cells/μL; hemoglobin, 11.7 g/dL; aspartate aminotransferase, 68 IU/L; alanine aminotransferase, 31 IU/L; brain natriuretic peptide, 47 pg/mL. Serum C-reactive protein and erythrocyte sediment rate were within their respective normal ranges. The patient's serum myoglobin level was elevated to 201.1 ng/mL. However, her serum troponin level had not increased repeatedly, ruling out acute myocardial infarction.

Bedside transthoracic echocardiography showed cardiac tamponade, which demonstrated a large pericardial effusion with significant respiratory variation in mitral and tricuspid inflows, diastolic right ventricle collapse, and inferior vena cava plethora. Thoracic computed tomography (CT) showed a hemorrhagic pericardial effusion and aortic IMH from the aortic arch to the descending aorta, and a 3.5-cm infrarenal type abdominal aortic aneurysm. No evidence of aortic dissection was observed. Therefore, we concluded that the IMH did not involve the ascending aorta and thus immediately performed pericardiocentesis. After 300 mL bloody drainage, intra-pericardial pressure decreased from 28 mmHg to 15 mmHg and the patient's vital signs stabilized. Over 1,000 cells were counted in the bloody pericardial fluid,

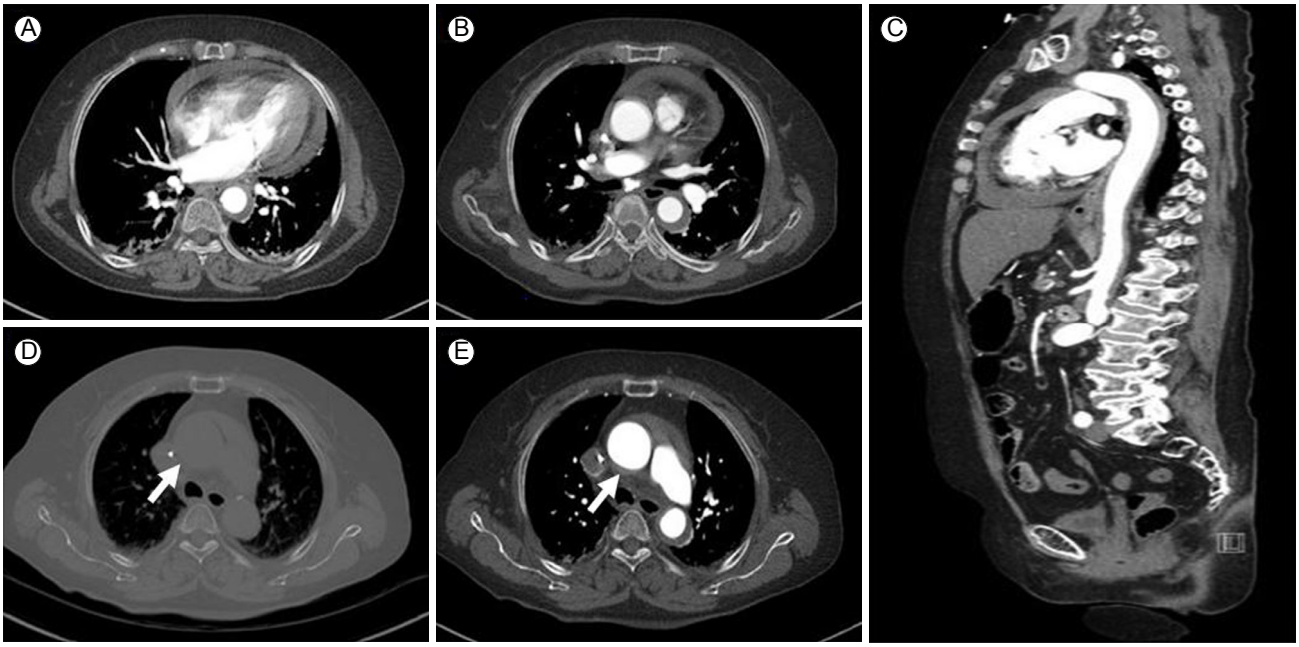

Figure 1. Thoracic contrast-enhanced CT (A) showing hemorrhagic pericardial effusion. Ventricular collapse indicates diastolic dysfunction. Axial CT scan at the left atrium level (B) shows a low-attenuation crescent-shaped lesion indicating intramural hematoma with no overt intimal disruption. Sagittal contrast image (C) through the descending thoracic aorta reveals crescenteric, high-attenuation material within the aortic wall, consistent with IMH. Non-contrast (D) and post-contrast (E) images demonstrate fluid collection in the superior aortic recess (arrow). High-attenuation in the superior aortic recess may appear as a hematoma due to descending aortic IMH. CT, computed tomography; IMH, intramural hematoma.

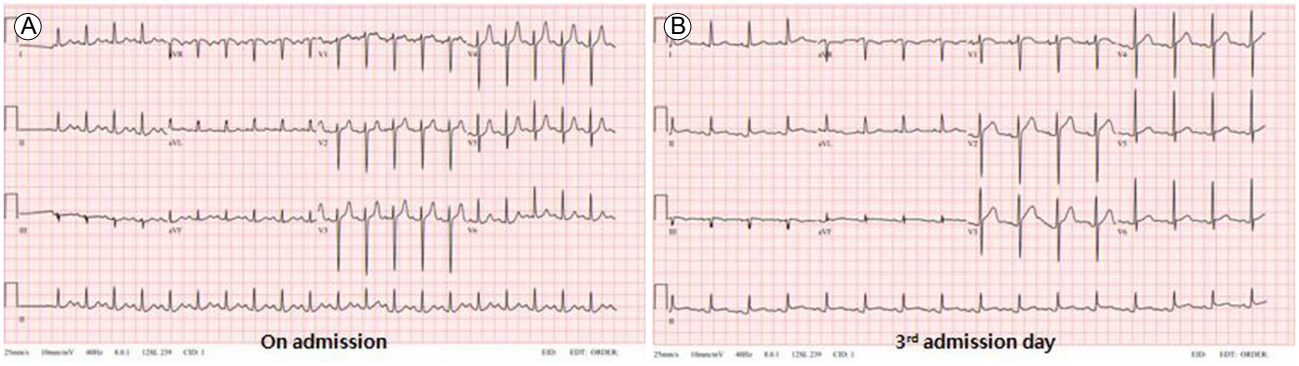

Figure 2. Electrocardiogram on admission showing sinus tachycardia. On day 3 of admission, electrocardiography showed ECG changes typical of acute pericarditis, with ST segment elevation on multiple precordial leads. The PR segment was depressed on lead I, and the PR segment was elevated on aVR lead. ECG, electrocardiography.

including old red blood cells and 33% neutrophils; meanwhile, the patient's fluid protein level was similar to serum protein level (7.4 vs. 6.0 g/dL, respectively). Coronary and left ventricular angiography were performed to rule out coronary artery disease and myocardial free wall rupture. No significant stenosis of the coronary artery or contrast leakage to the pericardium was noted. Pericardial fluid culture was negative for bacteria including *Mycobacterium tuberculosis*. The tuberculosis polymerase chain reaction result was also negative. CT scan and tumor markers were negative for malignancy. After carefully reviewing the CT scan, we concluded that the hemorrhagic pericardial effusion occurred from the aortic arch IMH via the superior aortic recess (Fig. 1).

The patient developed a mild fever 12 h after pericardiocentesis but did not complain of any specific symptom. Her serum erythrocyte sediment rate and C-reactive protein began to exceed normal ranges. Electrocardiography showed a normal sinus rhythm without any significant interval change. Blood culture and gram stain showed negative results. Empirical antibiotics (1st generation cephalosporin) were started. On day 3 after admission, routine electrocardiography showed ST segment elevation on multiple precordial leads, PR segment depression on lead I, and PR segment elevation on the aVR lead (Fig. 2). The clinical diagnosis was acute pericarditis. Colchicine and ibuprofen were administered for acute pericarditis. The patient's fever subsided the next day. Laboratory findings for inflammation and electrocardiography findings began to improve. A follow-up CT scan performed on day 3 after admission showed no pericardial effusion or specific changes in the range or depth of the IMH. She continued medical management for IMH and pericarditis, and was discharged in good condition while continuing colchicines and ibuprofen therapy.

DISCUSSION

IMH is being increasingly recognized in patients with acute aortic syndrome and is accepted as a variant form of classic aortic dissection. The pathophysiology of IMH is generally believed to be spontaneous rupture of the vasa vasorum or atherosclerotic plaque [1,4]. The prevalence of IMH in patients with acute aortic syndromes ranges from 5% to 23% [3]. Type B IMH is twice as prevalent as type A [1,5]. In previous reports, medical treatment was recommended for patients with uncomplicated type B IMH [3,6]. However, the appropriate treatment for patients with type A IMH has remained controversial until recently. Blood in IMH collects at a superficial location close to the adventitia. Bleeding from the intrapericardial portion of the ascending aorta will result in hemorrhagic pericardial effusion. Compared to patients with type A aortic dissection, patients with type A IMH exhibit a greater frequency of pericardial and pleural effusion, with a higher prevalence of cardiac tamponade [1,5]. IMH-related complications are more frequent in type A than type B IMH. Among patients with type A and B IMH, 29% and 2% were in shock on admission, respectively. Cardiac tamponade and aortic rupture occur in 24% and 10% of patients with type A IMH, respectively, whereas aortic rupture alone occurs in 2% of patients with type B IMH [2]. Cardiac tamponade in type B IMH is extremely rare.

Normal pericardial recesses occur owing to the contours of the heart and great vessels, and are closer to the visceral layer than the parietal layer. Moreover, portions of the left atrium are left uncovered by the pericardium. These factors result in normal fluid collection in pericardial recesses [7,8]. Our patient was diagnosed with a type B aortic IMH descending from the aortic arch combined with cardiac tamponade. We believe the pericardial hematoma was derived from the descending aorta rather than the ascending aorta via the superior aortic recess. The patient initially underwent pericardiocentesis because of her hemodynamic instability. Her vital signs subsequently stabilized, so we did not consider further surgical treatment. Early endovascular or surgical interventions are required for patients with type A IMH combined with cardiac tamponade because of the inherent risk of rupture, tamponade or compression of the coronary ostia.

Inflammation of the pericardium with or without pericardial effusion can occur as an isolated clinical problem or a manifestation of a systemic disease. Approximately 90% of isolated cases of acute pericarditis have idiopathic or viral causes [7,9]. Other causes of acute pericarditis include neoplasm, systemic autoimmune diseases, uremia, and pericardial inflammation after myocardial infarction, trauma or pericardial surgery [9]. In our patient, the acute pericarditis was considered an inflammatory reaction of the pericardium caused by hematoma and a pericardial procedure; this is because there were no findings indicating any other cause of acute pericarditis.

The present case presented with cardiac tamponade resulting from hemopericardium due to type B IMH. Even if hemodynamic stabilization is achieved after immediate pericardiocentesis, close observation for complications including acute pericarditis due to hematoma should be considered. Aortic IMH can lead to aortic dissection and rupture in 28-47% and 21-27% of patients, respectively [10]. Even if the patient remains asymptomatic, follow-up examinations should be performed if there is no evidence of progression.

중심 단어: 심낭압전; 혈종; 심막염

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