

[ CASE REPORT ]

## Acute Aortic Dissection Masquerading as Acute Pericarditis

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### Abstract:

We herein report 3 cases of acute aortic dissection (AAD) in which the initial 12-lead electrocardiogram showed typical ST elevation consistent with acute pericarditis. All patients exhibited small pericardial effusion but did not suffer from rupture into the pericardium or clinical tamponade. Slow leakage or exudate stemming from the dissecting hematoma appeared to have caused inflammation, resulting in pericarditis. Therefore, we highlight the fact that AAD may masquerade as acute pericarditis. Physicians should be aware of the possibility of type A AAD as an important underlying condition, since the early diagnosis and subsequent surgical treatment may save patients' lives.

**Key words:** acute aortic dissection, pericarditis, misdiagnosis, tamponade

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### Introduction

Acute aortic dissection (AAD) is a life-threatening condition that requires a prompt and accurate diagnosis. Indeed, potential delays in the treatment of this condition have been associated with an increased mortality rate (1). Due to the wide variety of clinical presentations, patients with AAD may have symptoms that are misdiagnosed or confused with other conditions, such as acute coronary syndrome, gastrointestinal diseases, and cerebrovascular accidents (1, 2).

Previous studies have demonstrated that acute electrocardiographic changes are commonly noted in patients with AAD (3, 4). ST elevation is observed in patients with coronary malperfusion, a condition that develops in 5-8% of patients with AAD (3, 4). Acute ST depression and T wave changes are common, especially when patients exhibit cardiac tamponade and shock, even without overt coronary malperfusion (3, 4). However, diffuse ST elevation suggestive of acute pericarditis has been seldom reported (4-9).

We herein report three distinct cases of AAD in which diffuse ST elevation was consistent with the development of pericarditis observed during the initial presentation.

### Case Reports

#### Case 1

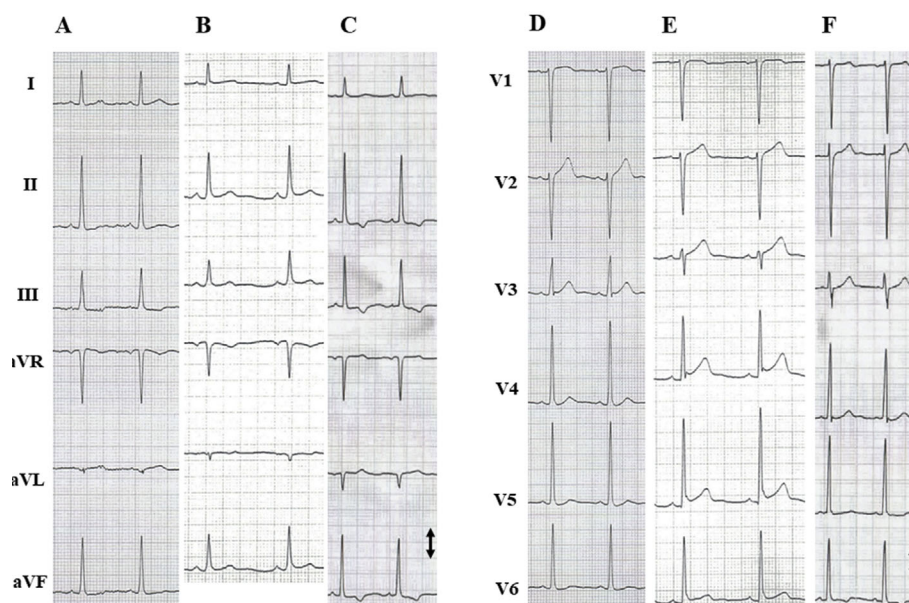
A man in his late 30s decided to visit the hospital due to abdominal pain and numbness in his right leg. While sitting on a chair in the waiting room, he suddenly developed blindness in the right eye and was consequently transferred to the emergency room (ER). His initial blood pressure (BP) was 190/100 mmHg, heart rate (HR) was 72 beats/min, respiratory rate (RR) was 24/min, and body temperature (BT) was 37.2°C. His blood oxygen saturation (Sat O<sub>2</sub>) was 98% while breathing ambient air. Cardiac auscultation did not reveal pericardial friction rub; however, the patient's right leg was cold, and the right femoral artery pulse was weak. The white blood cell count (WBC) was elevated at 15,600/μL. The C-reactive protein (CRP) level was 1.72 mg/dL (normal, <0.5 mg/dL). A 12-lead electrocardiogram (ECG) exhibited diffuse concave ST elevation of 1-3 mm and depression of the PQ segment, findings consistent with acute pericarditis (Fig. 1).

Chest X-ray revealed cardiomegaly and mediastinal widening. Contrast-enhanced computed tomography (CECT) showed Stanford type A classic AAD originating from the

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**Figure 1.** Serial 12-lead electrocardiograms (ECGs) in case 1. A and C: An ECG obtained 2 years before admission. B and D: An ECG obtained at initial presentation. C and F: An ECG obtained on day 7. High voltage in the precordial leads and diffuse ST elevation were noted at the initial presentation (B and D). The vertical arrow indicates 1 mV, and the paper speed is 25 mm/s (same for Figs 2 and 3). A, B, and C are limb leads. D, E, and F are precordial leads.

ascending aorta and extending to the iliac arteries, resulting in obstruction of the right innominate and right iliac arteries. A small amount of pericardial effusion was also noted. Emergency ascending aortic replacement and bypass grafting of the innominate artery were performed. There was no gross rupture of the ascending aorta, and the cause of pericardial effusion was assumed to be extravasation from the hematoma formed in the ascending aorta.

The postoperative course was complicated with pneumonia, and antibiotic treatment was performed. An ECG obtained on day 7 showed improved ST elevation (Fig. 1C, F). On day 63, the patient was transferred to another hospital for rehabilitation.

### Case 2

A man in his mid-30s already diagnosed with Marfan syndrome developed chest and dental pain 4 days before the presentation. The chest pain worsened with subsequent back pain and diaphoresis. Consequently, four days from the onset of these symptoms, the patient visited the ER.

The initial BP was 140/76 mmHg, HR was 104 beats/min, RR was 18/min, BT was 37.9°C, and Sat O<sub>2</sub> was 98% on 3 L/min of nasal oxygen. Auscultation revealed to-and-fro murmur at the left sternal border. The WBC count was 14,600/μL, and the CRP level was 4.02 mg/dL (normal, <0.45 mg/dL).

Chest X-ray showed mediastinal widening, and ECG revealed diffuse ST elevation and PQ depression consistent with acute pericarditis (Fig. 2). CECT and a bedside echocardiogram revealed a dilated ascending aorta at 6 cm. An intimal flap, moderate aortic regurgitation, and small amount

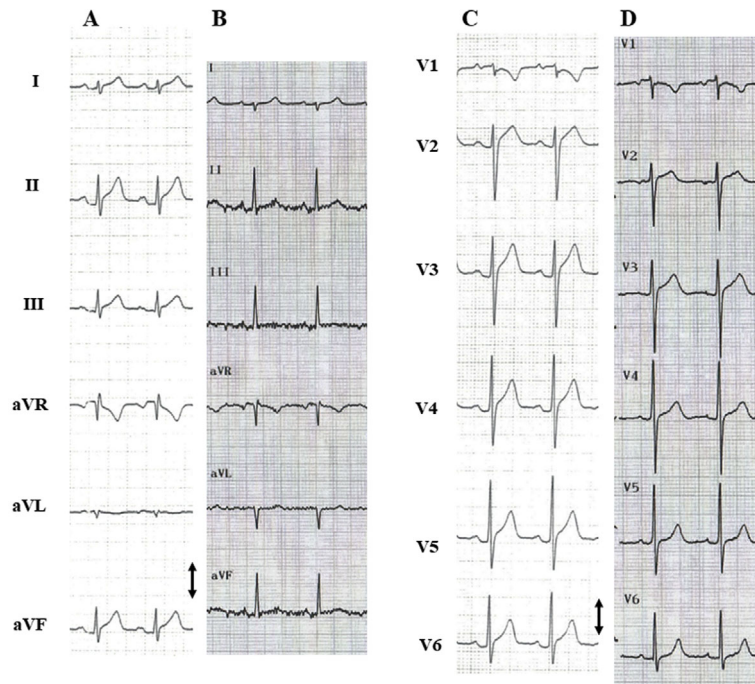
of pericardial effusion were also noted. The Bentall procedure was performed. A small amount of light reddish pericardial effusion was observed, which was considered exudate from the ascending aortic hematoma. There was no gross rupture of the ascending aorta. An ECG on day 3 showed improved ST elevation. The postoperative course was uneventful, and the patient was discharged home on day 24.

### Case 3

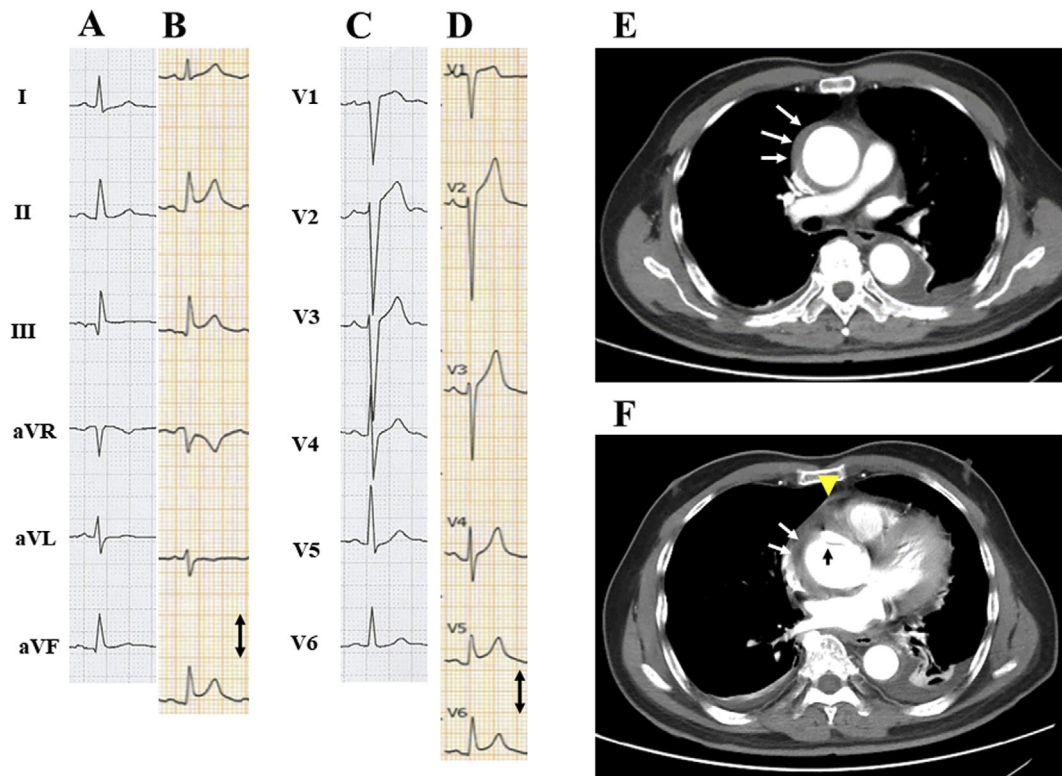
A man in his early 70s presented with symptoms of sudden chest pain while carrying heavy objects and was subsequently referred to our medical clinic. Cardiac enzyme levels were within normal range, and there were no new ECG findings except for chronic signs of left ventricular hypertrophy upon admission (Fig. 3A, C). An echocardiogram revealed a normal left ventricular systolic function and mild aortic regurgitation, but no pericardial effusion was noted. Furthermore, there was no visible intimal flap in the ascending aorta.

Three days after the initial presentation to the medical clinic, the patient was readmitted to the ER complaining of a persistent tingling sensation in the chest. His BP was 130/64 mmHg, HR was 104 beats/min, RR was 32/min, BT was 37.1°C, and Sat O<sub>2</sub> was 95% while breathing ambient air. No signs of murmur, friction rub, or gallop were observed. The WBC count was 12,600/μL, and the CRP level was 16.65 mg/dL (normal, <0.39 mg/dL).

An ECG revealed diffuse ST elevation and PR depression consistent with acute pericarditis (Fig. 3B, D). Echocardiography revealed a small amount of pericardial effusion



**Figure 2.** Serial 12-lead ECGs in case 2. A and C: The ECG obtained at the initial presentation shows diffuse ST elevation and PQ depression. B and D: The ECG obtained on day 3 shows improved ST elevation. A and B are limb leads. C and D are precordial leads.



**Figure 3.** A: Serial twelve-lead ECGs and contrast enhanced CT scan in case 3. A and C were obtained 3 days before admission. The QRS interval was slightly wide (110 ms), and deep S wave and mild ST elevation in V1-3 suggests left ventricular hypertrophy. These are chronic changes. B and D: The ECG obtained at admission shows diffuse ST elevation and PQ depression consistent with acute pericarditis. E and F: CECT exhibiting localized intimal flap (a black arrow) and intramural hematoma (white arrows) in the ascending aorta. A small amount of pericardial effusion was noted (yellow arrowhead). Modified from reference 8, with permission from JAMA.

that had not been identified during the initial admission three days before. This patient was therefore admitted following a diagnosis of acute pericarditis. Two days later, CECT was performed to evaluate the etiology of pericardial effusion, revealing a small amount of pericardial effusion, localized intimal flap, and intramural hematoma in the ascending aorta, indicating acute type A AAD (Fig. 3E, F). Consequently, emergency ascending aortic replacement was successfully performed. During the surgery, a small amount of light reddish pericardial effusion was identified, which was assumed to be exudate from the hematoma of the ascending aorta. No gross rupture in the ascending aorta was identified.

The postoperative course was complicated with deep vein thrombosis without pulmonary embolism, but the patient recovered and was discharged home on day 30.

## Discussion

The incidence of acute pericarditis in AAD is still unclear. Hirst et al. reported that 22 (4%) of 505 autopsied cases exhibited friction rubs, whereas 11 (6%) of 173 had ECG findings that were consistent with the development of acute pericarditis (10). We previously evaluated the incidence of acute ECG changes in 149 type A AAD cases and identified 2 cases (1.3%; cases 1 and 2 in the present study) (4) where diffuse ST elevation was consistent with the development of pericarditis.

The clinical manifestation of acute pericarditis related to AAD is variable. Typical symptoms of acute pericarditis, including pleuritic chest pain and a fever, along with the ECG change of diffuse ST elevation have been reported in some cases (5, 6). However, none of the three cases included in this study exhibited these typical symptoms, although the ECG changes were consistent with acute pericarditis. The underlying mechanism of pericardial effusion is not AAD rupture, as none of our cases had gross rupture of the ascending aorta. AAD is known to be closely associated with systemic and local inflammation of the aortic wall, as manifested by increased CRP and inflammatory cytokines (11, 12). Pericardial effusion and subsequent pericarditis can thus be attributed to slow leakage or exudate from the hematoma that facilitates pericardial inflammation (10). As a result, the amount of pericardial effusion is small, and overt tamponade is not apparent at the initial presentation. However, the amount of pericardial effusion may increase during the course and lead to clinical tamponade (5-7, 9). Indeed, sudden rupture of the aorta into the pericardium can sometimes develop with immediate tamponade (5, 6). Therefore, acute pericarditis may be a warning sign of tamponade developing in the subsequent few hours or even days (5).

The diagnosis is usually established with multimodality imaging that involves echocardiography and CECT. The diagnosis can be a more straightforward process in patients with predisposing factors, such as in case 2 (Marfan syndrome), and manifestations suggestive of AAD, such as in

case 1. However, in some patients, a fever, pleuritic chest pain, and ECG changes, which are consistent with the development of pericarditis, are the only clinical manifestations of AAD. The subsequent diagnosis may therefore be incorrect or delayed in such cases (5, 6). An accurate diagnosis in a timely manner is of remarkable importance for patients prior to developing true rupture in the pericardium resulting in tamponade, and surgical approaches are bound to be life-saving, as demonstrated in the cases reported here (7, 8).

## Conclusion

AAD may masquerade as clinical acute pericarditis or may present with acute ECG changes that are consistent with pericarditis. Therefore, making the correct diagnosis during the therapeutic window from the onset to the development of tamponade is bound to facilitate life-saving surgical therapies. Physicians should be aware of the possibility of AAD development as the underlying etiology of acute “idiopathic” pericarditis.

**Prior publication:** Case 3 of this case series was published as a “clinical challenge” in *JAMA Cardiology* (Mukaigawara M, Hirata K, Wake M. Diffusely elevated ST segments on electrocardiography. *JAMA Cardiol* 2016; 1: 229-30). doi:10.1001/jamacardio.2015.0338. Permission to use the figure was obtained from *JAMA* via the Copyright Clearance Center.

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

## References

- Spittell PC, Spittell JA Jr, Joyce JW, et al. Clinical features and differential diagnosis of aortic dissection: experience with 236 cases (1980 through 1990). *Mayo Clin Proc* **68**: 642-651, 1993.
- Hirata K, Wake M, Takahashi T, et al. Clinical predictors for delayed or inappropriate initial diagnosis of type A acute aortic dissection in the emergency room. *PLoS One* **10**: e0141929, 2015.
- Kosuge M, Uchida K, Imoto K, et al. Frequency and implication of ST-T abnormalities on hospital admission electrocardiograms in patients with type A acute aortic dissection. *Am J Cardiol* **112**: 424-429, 2013.
- Hirata K, Wake M, Kyushima M, et al. Electrocardiographic changes in patients with type A acute aortic dissection. *J Cardiol* **56**: 147-153, 2010.
- Greenberg DI, Davia JE, Fenoglio J, McAllister HA, Cheitlin MD. Dissecting aortic aneurysm manifesting as acute pericarditis. *Arch Intern Med* **139**: 108-109, 1979.
- Saner HE, Gobel FL, Nicoloff DM, Edwards JE. Aortic dissection presenting as pericarditis. *Chest* **91**: 71-74, 1987.
- Bains SR, Kedia A, Roldan CA. Pericarditis as initial manifestation of proximal aortic dissection in young patients. *Am J Emerg Med* **26**: 379.e3-379.e5, 2008.
- Mukaigawara M, Hirata K, Wake M. Diffusely elevated ST segments on electrocardiography. *JAMA Cardiol* **1**: 229-230, 2016.
- Soyer H. Acute pericarditis: a presenting manifestation of aortic dissection. *BMJ Case Rep* **2016**: bcr2016215853, 2016.
- Hirst AE Jr, Johns VJ Jr, Kime SW Jr. Dissecting aneurysm of the aorta: a review of 505 cases. *Medicine (Baltimore)* **37**: 217-279, 1958.
- Wu D, Shen YH, Russell L, Coselli JS, LeMaire SA. Molecular

mechanisms of thoracic aortic dissection. *J Surg Res* **184**: 907-924, 2013.

12. Komukai K, Shibata T, Mochizuki S. C-reactive protein is related to impaired oxygenation in patients with acute aortic dissection. *Int Heart J* **46**: 795-799, 2005.

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