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【 CASE REPORT 】

# Subendocardial Ischemia Caused by Acute Severe Aortic Regurgitation Due to Aortic Root Dissection: A Case Report and Literature Review

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**Abstract:**
Electrocardiogram (ECG) findings showing ST-segment depression in a wide range of leads and ST-segment elevation in aVR are found in patients with acute coronary syndrome with multivessel coronary lesions and left main trunk lesions. A 64-year-old man with a history of eosinophilic granulomatosis presented with chest pain and dyspnea. Although an ECG showed the above findings, he was diagnosed with acute severe aortic regurgitation (AR) complicating aortic root dissection and successfully underwent urgent Bentall operation. These ECG findings indicated that acute severe AR caused subendocardial ischemia.

**Key words:** acute severe aortic regurgitation, aortic root dissection, subendocardial ischemia

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## Introduction

Acute aortic regurgitation (AR) is usually caused by type A aortic dissection, infective endocarditis, or blunt trauma that leads to advanced heart failure and early death. Among the complications of acute type A aortic dissection, AR is common and is seen in 41-76% of patients (1). It is well known that flaps and acute AR are found on echocardiography and contrast-enhanced computed tomography (CT), especially in aortic root dissection. In contrast, there are few reports describing the electrocardiogram (ECG) findings when dissection has not progressed to coronary ostium. Acute AR may reduce myocardial perfusion by rapidly increasing the left ventricular (LV) end-diastolic pressure and decreasing the coronary blood flow (2).

We herein report a case of subendocardial ischemia with ECG findings due to acute severe AR complicating aortic root dissection.

## Case Report

A 64-year-old man had a history of eosinophilic granulomatosis with polyangiitis and had been taking prednisolone. He presented to the emergency department with chest pain and dyspnea that had persisted for an hour. He also had a history of bronchial asthma but had never had such symptoms before. His blood pressure was 98/23 mmHg, heart rate was 84 bpm, and percutaneous oxygen saturation was 91% under 8 L oxygen inhalation. A blood gas analysis revealed an elevated lactate level of 3.8 mmol/L. As basic physical findings, marked moist rales and diastolic heart murmur were confirmed. Troponin T was 0.019 ng/mL and creatine kinase (CK)-MB was 8 U/L on admission. Chest X-ray showed pulmonary congestion (Fig. 1). An ECG revealed ST-segment depression in a wide range of leads and ST-segment elevation in aVR (Fig. 2). These findings are known to be found in patients with acute coronary syndrome (ACS) with multivessel coronary lesions and left main trunk lesions. However, transthoracic echocardiography (TTE) showed a normal LV systolic function (Supplemental Material) but also revealed severe AR with flaps in the LV outflow tract (Fig. 3A, B). The patient was thus diagnosed with acute aortic root dissection on contrast-enhanced CT (Fig. 4A, B). Furthermore, CT showed findings of circumferential subendocardial ischemia (Fig. 5).

The patient underwent successful Bentall operation with a 27-mm Inspiris® and a 30-mm Gelweave Valsalva graft®. In-

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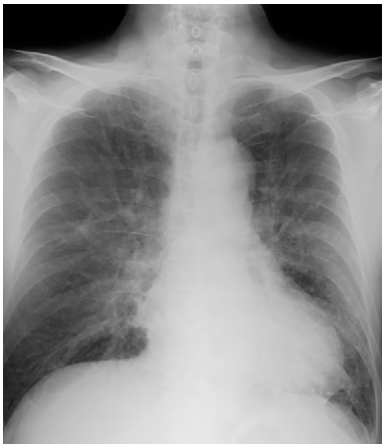

**Figure 1.** **Chest X-ray findings on admission. Chest X-ray showed pulmonary congestion.**

traoperative findings revealed that the left and right coronary ostium were intact. The CK-MB level on the day after surgery was 22 U/L. Coronary CT performed 10 days after surgery showed no significant stenotic lesions in the main trunk of the coronary arteries (Fig. 6). He was discharged 28 days later. The ECG at discharge showed only a high LV potential and a slight ST-segment depression (Fig. 7).

## Discussion

We reported a patient diagnosed with subendocardial ischemia due to acute severe AR complicating aortic root dissection. Subendocardial ischemia reportedly occurs in patients with chronic severe AR and normal coronary arteries (2). The causes are increased oxygen demand due to tachycardia and increased LV end-diastolic pressure as well as a decreased coronary blood flow and oxygen supply due

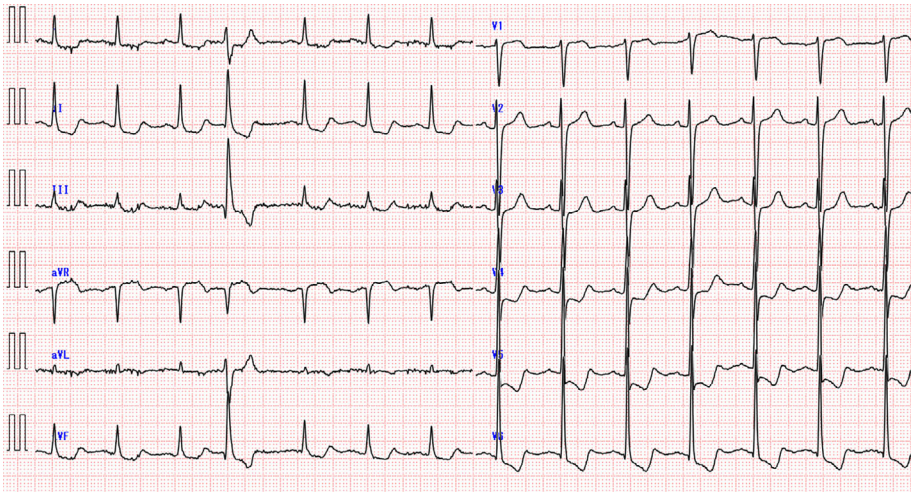


**Figure 2.** **ECG findings on admission. The ECG showed ST-segment depression in a wide range of leads and ST-segment elevation in aVR. Sweep speed was 25 mm/s, 10 mm/mV.**

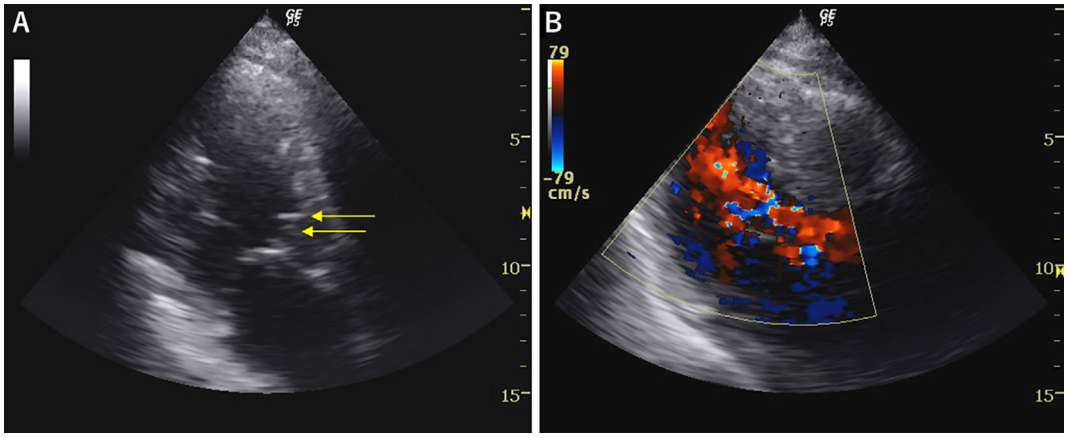


**Figure 3.** **Bedside echocardiography findings. (A) Flaps were found in the left ventricular (LV) outflow tract (yellow arrows). (B) Transthoracic echocardiography revealed severe aortic regurgitation.**

to low aortic diastolic pressure. The normal coronary flow pattern is predominantly diastolic but can change to systolic predominance in the setting of severe AR (3). Furthermore, low diastolic aortic root pressure and the Venturi effect of the regurgitant jet at the coronary ostium cause retrograde diastolic coronary blood flow. George et al. reported that transesophageal echocardiography (TEE) showed a retro-

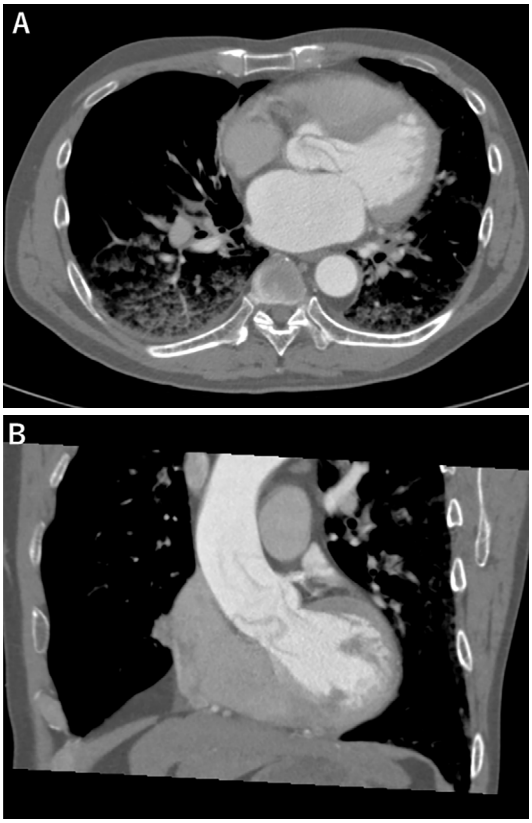


**Figure 4.** **Contrast-enhanced CT findings. (A) Axial CT scan. (B) Coronal CT scan. CT revealed type A aortic dissection with flaps in the LV outflow tract.**

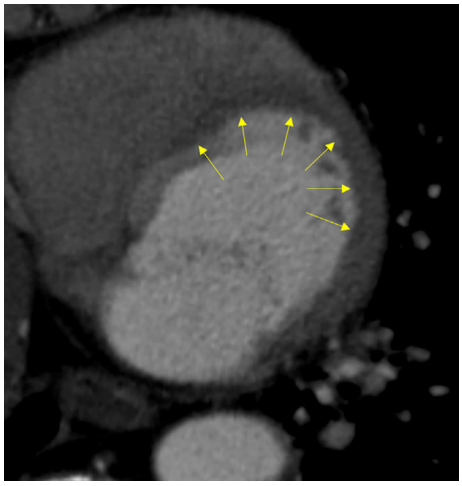

**Figure 5.** **Subendocardial ischemia. CT showed findings of circumferential subendocardial ischemia (yellow arrows).**

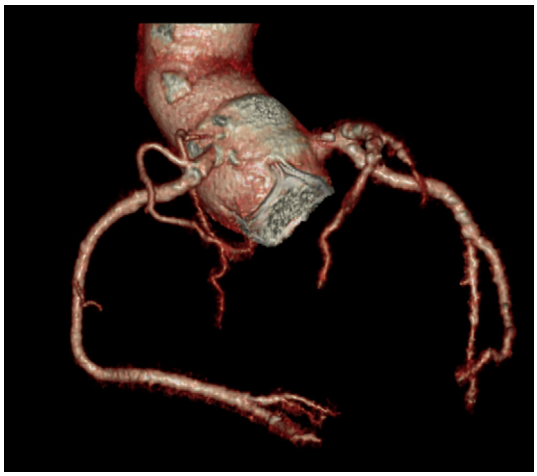

**Figure 6.** **Coronary CT performed 10 days after Bentall operation. CT showed no significant stenotic lesions in the main trunk of the coronary arteries.**

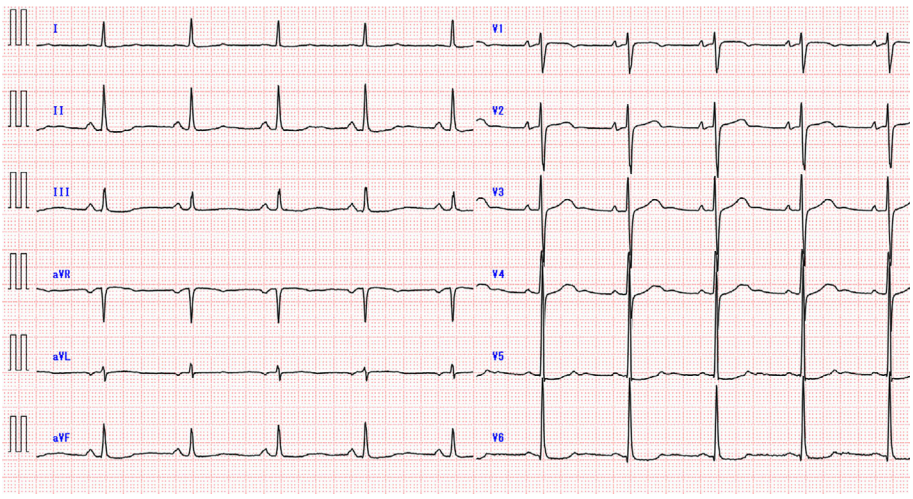


**Figure 7.** **ECG findings at discharge 28 days later. The ECG at discharge showed only a high LV potential and a slight ST-segment depression. Sweep speed was 25 mm/s, 10 mm/mV.**

**Table. List of Case Reports of Acute Aortic Regurgitation.**

| Reference | Age, sex | Clinical scenario and etiology | Symptoms and objective findings | ECG | TEE, others | Outcome |
|---|---|---|---|---|---|---|
| (5) | 73, M | Dilated aortic root and rupture of fibrous strand | Shock | Global ischemia | Intraoperative | AVR |
| (6) | 51, M | IE | Fever, back pain and diastolic murmur | NA | Preoperative | AVR |
| (7) | 30, M | IE | Fever, shortness of breath and unilateral pulmonary edema | NA | Preoperative | Bentall |
| (8) | 17, M | Traumatic aortic root rupture | Wide pulse pressure and diastolic murmur | New CRBBB | Intraoperative | Bentall |
| (9) | 46, F | Behçet disease, NCC prolapse and LCC shortening | Acute heart failure | NA | NA | Bentall with infliximab and prednisolone |
| (10) | 47, M | Intravenous drug abuse, IE | Lower back pain | Normal | NA | AVR |
| (11) | 71, M | Immediately after MV surgery, iatrogenic aortic incompetence | Wide pulse pressure and acute pulmonary edema | NA | Preoperative | TAVI |
| (12) | 53, F | Recurrent ovarian cancer and Trousseau's syndrome, NBTE | Acute heart failure | NA | NA | Death |
| (13) | 53, M | Dilation of Valsalva and ascending aorta, fibrous strand rupture | Acute dyspnea | NA | Preoperative | Bentall and ascending aortic replacement |
| (14) | 70, M | After AVR and CABG, rupture of prosthetic AV leaflet | Shock and heart failure | ST-T wave abnormalities | Preoperative | TAVI with VA-ECMO |
| (15) | 64, M | Bicuspid AV, rupture of anomalous cord | Dyspnea and diastolic murmur | NA | NA | AVR |
| (16) | 95, F | Complicating PTAV | Shock | NA | Intraoperative | TAVI |
| (17) | 80, M | Nontraumatic AV commissure avulsion | Dizziness and shortness of breath | Sinus tachycardia and CRBBB | NA | AVR |
| (18) | 65, M | IE | Severe sepsis | NA | Preoperative | AVR |
| (19) | 76, M | Fibrous strand rupture of LCC | Acute heart failure | left ventricular hypertrophy | Preoperative | AVR |
| (20) | 55, M | Intimo-intimal intussusception-circumferential aortic dissection | Retrosternal pain and dyspnea | Normal | NA | Died while preparing for surgery |
| (21) | 46, M | Type A dissection | Chest and back pain, Congestive heart failure | NA | Preoperative, MDCT | Replacement of ascending aorta |
| (22) | 34, M | Aortic dissection confined to Valsalva | Heart failure | NA | Preoperative | Surgery |
| (23) | 80, F | Type A dissection | Chest pain and diastolic murmur | Inferior and lateral wall myocardial infarction | NA | Death (refusal of surgery) |
| (24) | 51, F | AV rupture (LCC) caused by valvular myxomatous transformation | Progressive dyspnea and early diastolic murmur | NA | Preoperative | AVR |
| (4) | 57, M | NA | Acute pulmonary edema and syncope | NA | Preoperative | AVR |
| (25) | 69, M | Local avulsion of AV commissure | Precordial pain, dyspnea and shock | NA | Preoperative | AVR |
| (26) | 23, M | IE | Acute heart failure | NA | Preoperative | AVR, aortic root replacement Bentall |
| | 59, M | Type A dissection | Chest and back pain, syncope | NA | Intraoperative, CT | |
| (27) | 67, F | Immediately after MVR, AV injury | Hemodynamically unstable | NA | NA | Another operation |
| (28) | 63, M | Type A dissection | Chest pain and dyspnea | ST-segment depression and abnormal Q in leads I and II | NA, contrast enhanced ECG-gated MDCT | Ascending aorta replacement and AV repair |
| (29) | 68, M | Type A dissection | Back pain | NA | Preoperative | Bentall |
| (30) | 60, M | Spontaneous localized intimal tear of ascending aorta, flail NCC | Chest pain and subsequent shortness of breath | Normal | Preoperative | AVR |

ECG: electrocardiogram, TEE: transesophageal echocardiography, M: male, AVR: aortic valve replacement, IE: infective endocarditis, NA: not available, CRBBB: complete right bundle branch block, F: female, NCC: non-coronary cusp, LCC: left coronary cusp, MV: mitral valve, TAVI: transcatheter aortic valve implantation, NBTE: non-bacterial thrombotic endocarditis, CABG: coronary artery bypass grafting, AV: aortic valve, VA-ECMO: veno-arterial extracorporeal membrane oxygenation, PTAV: percutaneous transluminal aortic valvuloplasty, MDCT: multidetector computed tomography, MVR: mitral valve replacement

grade blood flow from the left main trunk to the aorta during LV diastole in a patient with acute severe AR (4). Our patient had a blood pressure of 98/23 mmHg on admission, and the diastolic blood pressure was extremely low. In addition, the patient had a high pulmonary capillary wedge pressure of 22 mmHg at the start of surgery, almost equal to the diastolic blood pressure. Intraoperative findings and postoperative coronary CT showed no organic stenosis or occlusion of the coronary arteries. Therefore, hemodynamic subendocardial ischemia due to acute severe AR may have resulted in such ECG findings. Fig. 5 suggests the occurrence of circumferential subendocardial ischemia. However, although the intraoperative findings showed that the coronary ostium was intact, it is possible that a decrease in the coronary blood flow occurred, as the dissection extended close to the coronary artery. Furthermore, Fig. 5 is unsharp because it is not ECG-gated, but myocardial ischemia of the ventricular septum appears to be biased towards the right ventricle (RV). This may mean that volume loading due to acute severe AR increases LV pressure followed by RV pressure, and the RV side, which is vulnerable to pressure loading, leads to significant myocardial ischemia. In this case, despite subendocardial ischemic condition, TTE showed a normal LV systolic function. When cardiac output decreases due to acute severe AR, compensatory sympathetic hyperactivity causes cardiac hypercontraction and tachycardia. This increases the cardiac work, but a low diastolic blood pressure leads to subendocardial ischemia without an adequate coronary blood flow. However, since it is not a transmural ischemia, the LV systolic function on TTE is maintained.

We searched the PubMed medical database on November 30, 2021, for all articles published on cases of acute AR. The following search terms were used: "acute aortic regurgitation". Among them, the case reports of acute AR were summarized (Table) (5-30). Type A aortic dissection, infective endocarditis, trauma, fibrous strand rupture of aortic valve, and iatrogenicity have been reported as causes of acute AR. The clinical course of acute AR varies from hours to weeks, depending on the etiology, but aortic dissection and fibrous strand rupture are particularly urgent and often present with shock and acute heart failure. Relatively few studies have described the ECG findings of patients with acute AR. Only George et al. reported the ECG findings of global ischemia, similar to our case (4). Although not only TTE but also TEE is extremely important in elucidating the etiology of acute AR, it is difficult to perform TEE when hemodynamics are unstable. In addition, TEE is relatively invasive and is not recommended preoperatively, especially for aortic dissection. For urgent conditions, ECG and TTE, which can be performed in a short time and at the bedside, are very useful diagnostic modalities. In our case, severe ACS was suspected based on symptoms and ECG findings, but aortic root dissection could be quickly and accurately diagnosed by TTE and CT and successfully treated. In the ECG showing subendocardial ischemia, it is recommended to evaluate hemodynamics associated with increased LV end-diastolic pressure as well as ACS.

Acute AR is an urgent condition, often with hemodynamic instability. If acute AR can be included in the differential diagnosis when subendocardial ischemia is detected on an ECG, it will lead to a prompt diagnosis and appropriate treatment. This case is a representative case with clinical implications. Furthermore, the findings suggest that not only coronary artery lesions but also proper control of blood pressure and the LV end-diastolic pressure are important for myocardial perfusion. This theory of myocardial oxygen supply-demand balance has academic significance in the management of severe heart failure.

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

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