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# Clinical manifestation as acute coronary syndrome without electrocardiographically ischemia: a clue for aortic dissection

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ABSTRACT

Aortic dissection is a critical condition requiring immediate assessment and management. Clinical presentation is commonly associated with severe chest pain and high blood pressure. However, misdiagnosis is frequent because of various features. We presented a case of 51-year-old woman who complained of dyspnea for 3 d after she experienced back pain for one week. She was presented with severe respiration distress with impending respiration failure on arrival to our hospital. Her chest X-ray showed cardiomegaly with acute pulmonary edema. The laboratory data revealed elevated cardiac enzyme and electrocardiography demonstrated sinus tachycardia. She was hospitalized under the initial diagnosis of acute coronary syndrome. The patient remained hemodynamically stable, and experienced one episode of chest discomfort. After electrocardiography, she was found with bigeminy ventricular premature beats without ST-T change. Follow-up cardiac enzyme demonstrated progressive declined. Cardiac catheterization was performed on the third day of admission, and coronary angiography revealed large intimal flap on aortic root with bilateral coronary artery involvement. Surgical management was arranged after immediate chest computed tomography study.

## 1. Introduction

Aortic dissection is a potential fatal condition and early diagnosis is critical to the prognosis. The management demands either surgical repair of the dissected aorta or medical reduce of arterial shear that force on the torn aortic site. Early diagnosis with minimal loss of time is the principle for prompt management[1]. However, misdiagnosis still remains an unresolved problem because of myriad and unpredictable clinical presentation. The initial symptoms usually present with severe and abrupt chest pain. However, symptom of chest pain can be easily confused with acute myocardial infarction in those who lacking clinical evidence of aortic dissection. Differential diagnosis between the two critical diseases can be more difficult if clinical manifestations present with myocardial injury. Clinically, both diseases present with acute, critical and rapid progress. In addition, managements in these two critical conditions are completely different. Early correct diagnosis is important for therapeutic strategy. We described a case of acute aortic dissection presenting with elevated cardiac enzyme mimicking acute coronary syndrome. The unusual presentations obscured the diagnosis. We emphasize the possibility of aortic dissection which should be kept in mind to avoid catastrophe.

## 2. Case report

A 51-year-old woman had a medical history of hypertension without regular medication. She experienced back pain for a week and did not pay much attention to it. Three days prior to arrival at the hospital, she developed dyspnea and her symptoms deteriorated progressively. On arrival to our emergency department, she showed severe respirational distress and drowsy consciousness level. Her blood pressure (BP) was 106/55 mmHg, and pulse rate was at 112 beats/min.

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Chest radiography (CXR) showed cardiomegaly, increasing interstitial markings, and blunting of bilateral costophrenic angles (Figure 1, left). And her electrocardiogram (ECG) revealed sinus tachycardia (Figure 2A).

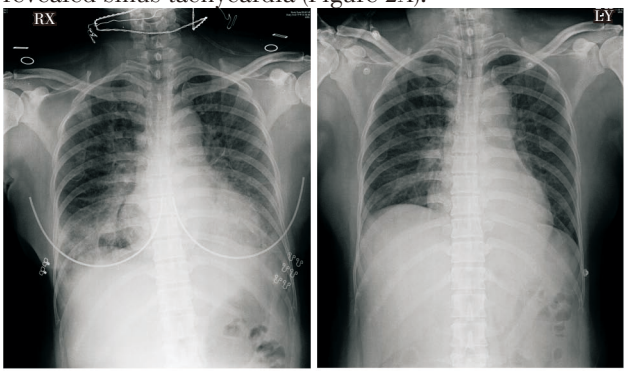


**Figure 1.** CXR on admission (left panel) shows cardiomegaly, increasing interstitial markings with blunted bilateral costophrenic angles, and on the following day (right panel) demonstrates pulmonary edema and pleural effusion improved after treatment.

Biochemical analysis demonstrated normal liver and renal function test. There were abnormal troponin I [3.06 ug/L (reference range: <0.01 ug/L)], total creatine kinase (CK) [628 U/L (reference range: 30-170 U/L)], and CK-myocardial band isoenzyme (CK-MB) [76 U/L (reference range: 0-16 U/L)]. She was recommended for admission to our intensive care unit under the impression of acute coronary syndrome with acute pulmonary edema. On admission to our intensive care unit, intubation with artificial ventilation was performed immediately due to persisted respiration distress. Six hours later, a follow-up cardiac enzyme demonstrated CK (985 U/L), CK-MB (88 U/L), and cardiac troponin I (3.28 ug/L). There was no further ECG change by continue monitoring (Figure 2B). On the following day,

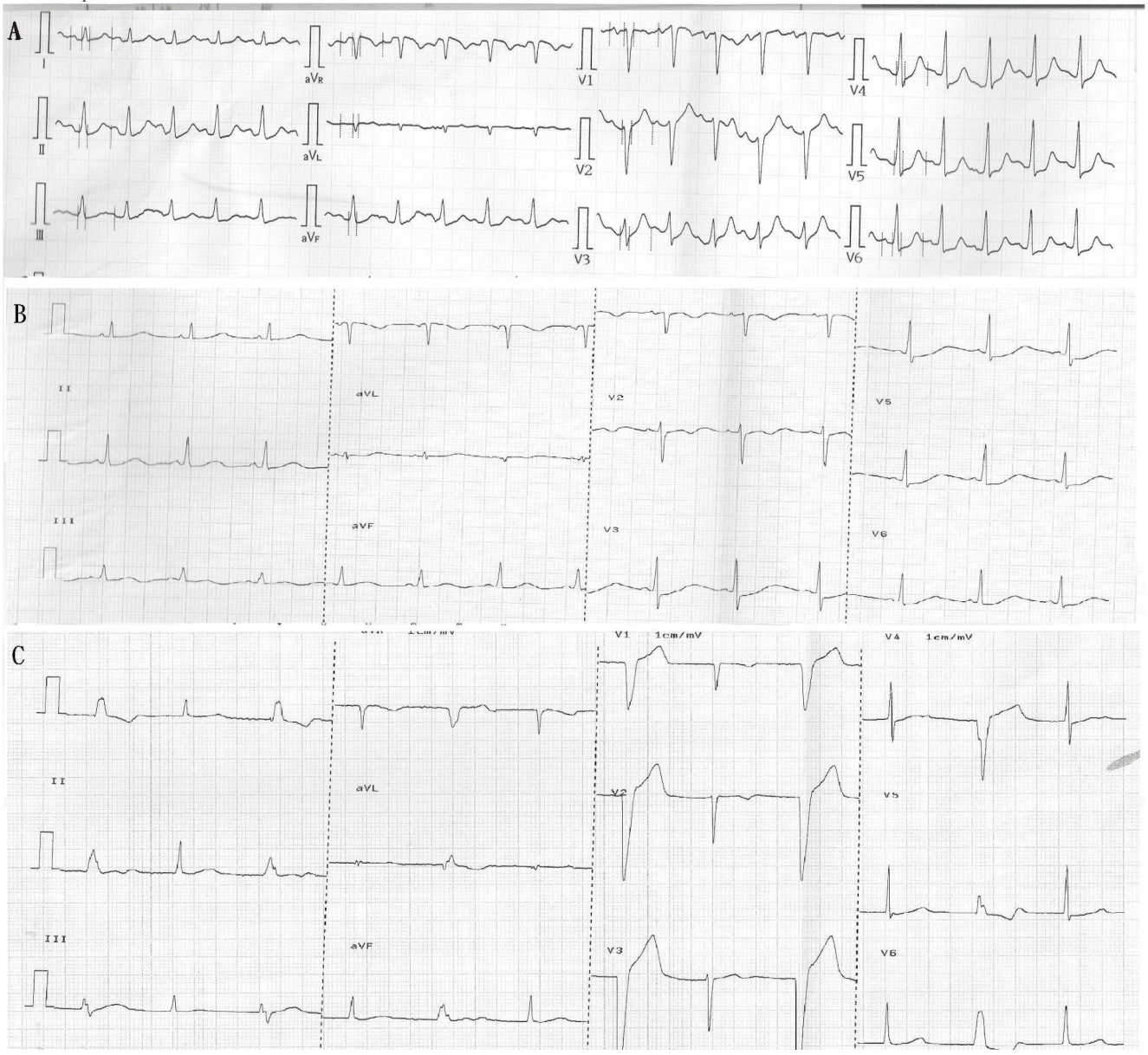


**Figure 2.** Electrocardiography at emergency room (A) demonstrates sinus tachycardia with slightly ST depression on V4-6, and six hours after admission (B). And the following day (C) shows bigerminy ventricular premature beats.

her conscious level was improved. She kept stable vital sign and remained symptom free during the intensive care unit, except with one episode of chest tightness complaint. Electrocardiography was checked and it revealed bigeminy ventricular premature beats without ST-T changes (Figure 2C). The arrhythmia was relieved by 150 mg infusion of amiodarone. Repeated CXR showed both pulmonary edema and bilateral pleural effusion which was resolved with decreased heart size after treatment (Figure 1, right). Echocardiography was done and the results demonstrated good left ventricular function without left ventricular dilatation. There was mild aortic regurgitation without pericardial effusion. There was no further abnormal finding as ST-T change by ECG follow-up. The cardiac enzyme also showed progressive declined by regular follow-up (CK: 743 U/L, 314 U/L and CK-MB 52 U/L, 20 U/L, and cardiac troponin 2.72 ug/L, 1.71 ug/L). Under the impression of acute coronary syndrome with acute pulmonary edema, coronary angiography was performed on the third day of admission. During the diagnostic procedure, it was difficult to engage the left coronary artery when the left Judkins catheter was used. The angiography showed a filling defect on aortic root from the origin of the left coronary artery extending to the right coronary artery. Aortography was performed and the image clearly revealed large intimal flap from ascending aortic root involving bilateral coronary artery orifice (Figure 3). The dissection extended to descending aorta. The patient was given chest computed tomography (CT) immediately. The chest CT findings, as images of aortography, demonstrated aortic dissection starting from 1.5 cm above the root level with bilateral coronary involvement and extended to the left iliac artery level (Figure 4). She was arranged for surgical management immediately.

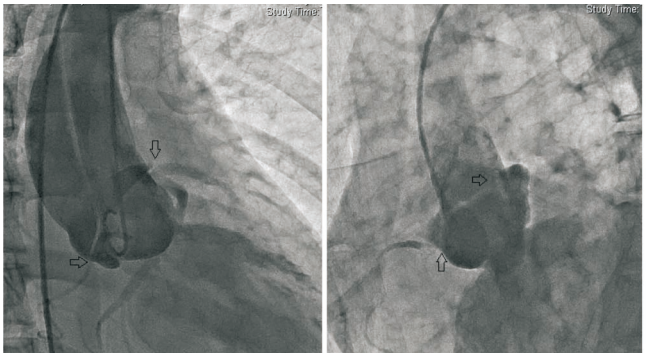

**Figure 3.** Large intimal flap from orifice of left coronary artery to right coronary artery (arrow) with incomplete obstruction of bilateral coronary artery, and the dissection extends to aortic arch (left panel: right anterior oblique view; left panel: left anterior oblique view) is showed by Aortography.

## 3. Discussion

Acute aortic dissection is a potential fatal condition requiring immediate assessment and therapy. Arterial hypertension is the single most important factor among the causes of aortic dissection and it is found in 80% of the patients[1]. The typical symptom in aortic dissection is a characteristic severe sharp pain. The pain may

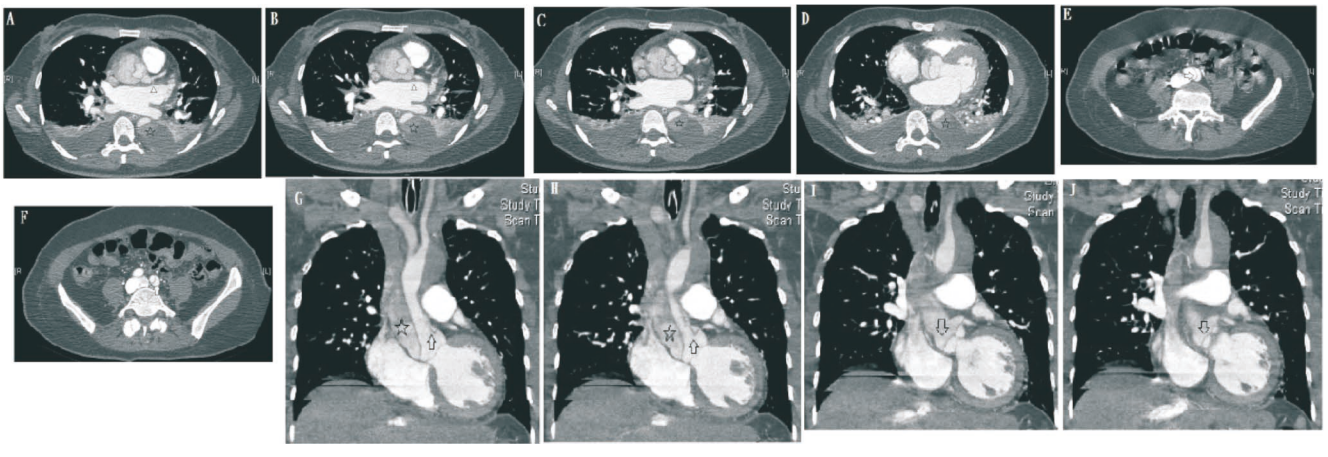

**Figure 4.** Chest CT (A-F: transverse view) demonstrates aortic dissection starting from 1.5 cm above aortic root, with false lumen compressing the origin of left main, left anterior artery and circumflex, and right coronary artery causing more than 70% narrowing of the lumen (arrowhead) (A, B). Aortic dissection extends through descending aorta site (asterisk) (A-D) to left iliac common artery level (arrow) (E, F). There are bilateral pleural effusion and minimal pericardial effusion. Chest CT (G-J: coronal view) demonstrates similar findings, large intimal flap from orifice of left coronary artery to right coronary artery (arrow) with huge false lumen formation (asterisk).

radiate to back or abdomen depending on the dissection site involved. Anterior chest pain indicates ascending aortic dissection, whereas patients with pain in the back, lower back and abdomen suggests descending aortic dissection. Widened mediastinum in a chest X-ray is a common finding. However, an estimated 20% of these chest X-ray results can have a negative finding[1]. Physical examination as BP discrepancy in two limbs can be an important sign for diagnosis. In a prior study, typical chest pain, abnormal chest X-ray finding, and BP difference permitted the identification of 96% of the aortic dissection[2]. However, misdiagnosis still remains an unresolved problem because of myriad and unpredictable clinical presentation.

Acute aortic dissection could be manifested as acute coronary syndrome. Because the prevalence rate of acute coronary syndrome is more common than aortic dissection, clinical manifestation with chest pain can be easily confused with those of acute coronary syndrome. Lacking clinical manifestation for aortic dissection makes it challenging for a correct diagnosis of an ascending aortic dissection involving the coronary artery. Acute pulmonary edema cases with elevated cardiac enzyme could arouse the suspicion of acute coronary syndrome. Myocarditis with acute pumping failure could be a possible diagnosis in the case. However, lacking of myocardial ischemia changes in ECG and presenting as a good left ventricular function by echocardiography made the true diagnosis uncertain. Acute aortic regurgitation could be a clue suggesting aortic dissection[3]. This may have hinted aortic dissection in our case, despite the lacking of pericardial effusion and visualization of the dissection information.

Coronary artery involved by aortic dissection was not rare. The true incidence is unknown because of limited cases in most studies. In a 14-year observational study, Chen *et al* found there were 14.1% incidence of coronary involvement in aortic dissection (20 patients in total 142 included cases)[4]. They also demonstrated that coronary malperfusion was rare, and myocardial ischemia was not always found in those of aortic dissection extending to coronary ostia. Other studies also demonstrated the majority of such cases were limited in surgical findings without clinical manifestation[5,6]. There were scatter case reported that acute ST-elevation myocardial infarction could develop when the coronary ostium is compressed. In our case, the serials changes of ECG and cardiac enzyme eluded to the fact that the myocardial ischemia could be a coronary malperfusion induced by coronary artery compressed by the intimal flap. This was evidenced by coronary angiography and CT image.

This case presented an unusual clinical complication of aortic dissection. We should keep aortic dissection as a differential diagnosis for patient with chest pain. Particularly for those clinical presentations suspected of acute coronary syndrome with atypical manifestation. We presented the case and emphasized it.

## Conflict of interest statement

The authors report no conflict of interest.

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