# Diagnosis and Management of Aortic Dissection Complicated by Coronary Malperfusion and Tamponade: A Case Report

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## *Abstract*

Aortic dissection (AD) is rare and often presents with atypical symptoms. Therefore, having a high index of suspicion is important as it can mimic other conditions and has a high mortality rate. We describe a patient with acute AD involving the coronary arteries, complicated by pericardial tamponade, and discuss findings using point-of-care ultrasound (POCUS), diagnostics, and treatment of this condition. Diagnosing acute AD is challenging and requires a high level of suspicion, as there is no single marker to make the diagnosis. Furthermore, the principles for definitive management vary widely when compared to other causes of chest pain such as acute coronary syndromes. Thus, POCUS is important to guide ourselves in the diagnosis in the Emergency Department.

**Keywords**: Aortic dissection, ischemia, coronary malperfusion, pericardial tamponade, case report

## Introduction

Acute aortic dissection (AD) is exceedingly rare, occurring in approximately 3 out of 100,000 patients per year[1]. Strikingly, it is estimated that 40% of patients with acute AD die before reaching the hospital[2] while the in-hospital mortality is approximately 20%[3]. Symptoms can be atypical and mimic other causes of chest pain such as acute coronary syndrome or pulmonary embolism, and classically includes sudden chest pain characterized as tearing or ripping that may radiate into the back or scapular region[4]. Physical exam findings can include blood pressure or pulse discrepancy of the right and left upper extremities. With the involvement of the coronary arteries, myocardial injury is a potential complication, and this may present with or without ST-segment elevation, which can confound the diagnosis.

## Case Report

A forty-year-old male presented to the Emergency Department (ED) by Emergency Medical Services (EMS) for chest pain that started one hour prior to arrival. The pain was pleuritic in nature and associated with shortness of breath. The patient's medical history included obesity, obstructive sleep apnea, hypertension, tobacco use, and right diaphragmatic palsy secondary to viral illness. He denied a history of deep venous thrombosis or pulmonary embolism.

On arrival, he was alert and oriented, but in respiratory distress. Vital signs were: heart rate 123 beats per minute, blood pressure 135/87 millimeters of mercury (mmHg) in the right arm and 123/66 mmHg in the left arm, respiratory rate 26 breaths per minute, temperature 36.6°C, oxygen saturation 89% on room air, which improved to 94% on a non-rebreather mask. Physical exam was notable for respiratory distress with diffuse expiratory wheezing. There were no cardiac murmurs, jugular venous distention, pulse deficits, or focal neurologic deficits. The remainder of the exam was unremarkable.

A 12-lead electrocardiogram (ECG) demonstrated sinus tachycardia with an incomplete right bundle branch block and poor R wave progression (figure 1). Bedside transthoracic echocardiography (TTE) was performed by the

emergency medicine resident and demonstrated a moderate pericardial effusion with more than 30% mitral inflow variability with inspiration and a plethoric IVC with minimal respiratory variation (figure 2). These findings were consistent with the findings of a TTE performed by the cardiology fellow. A TTE performed 5 months prior to the current presentation was notable for an ejection fraction of 65% and a dilated ascending aorta with a diameter of 5 cm.

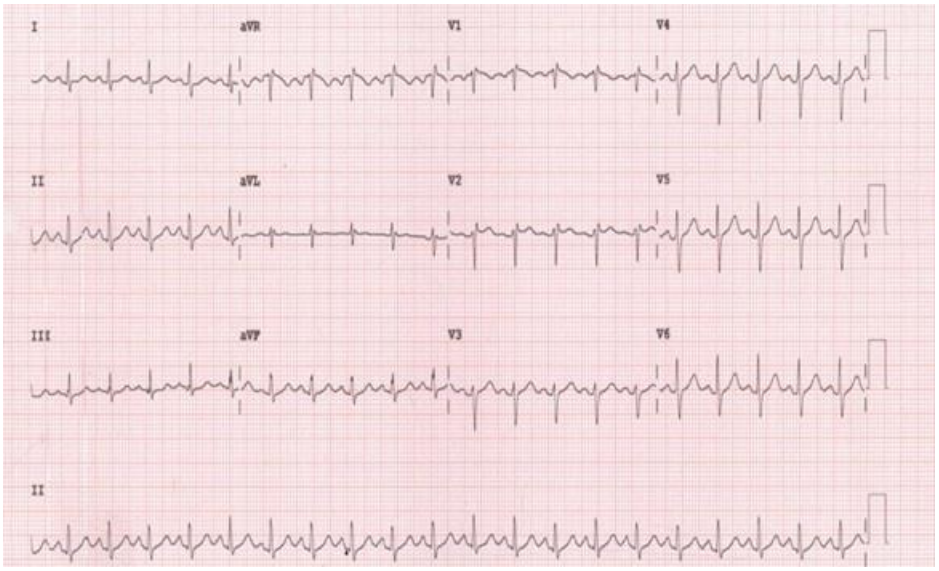


**Figure 1:** Twelve-lead electrocardiogram with sinus tachycardia and an incomplete right bundle branch block.

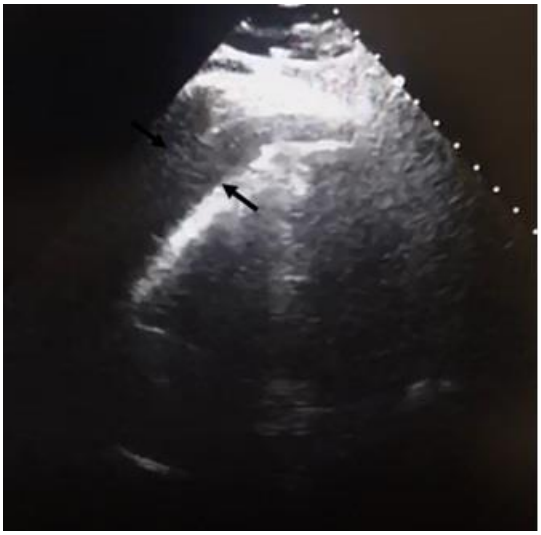

**Figure 2:** Transthoracic echocardiography apical four-chamber view of the pericardial effusion demarcated by the black arrow.

Laboratory studies demonstrated a white blood cell count of 12 x $10^9$ K/mcL (reference range: 4.5-11 K/mcL), hemoglobin 17.1 g/dL (reference range: 12.6-17.4 g/dL), platelets 190 K/mcL (reference range: 153-367 K/mcL), creatinine 1.3 mg/dL (reference range: 0.66-1.25 mg/dL), D-dimer 138 ng/mL (normal less than 255 ng/mL) and initial troponin I 0.017 ng/mL (normal less than 0.03 ng/mL), followed by a second troponin I 0.08 ng/mL five hours later. Emergent computed tomography (CT) angiography of the chest demonstrated moderate-sized pericardial effusion consistent with hemopericardium (mean density of 39 Hounsfield Units (HU)) with associated acute

intramural hematoma of the ascending aorta, aortic arch, descending thoracic aorta, right coronary artery, left coronary artery, and left vertebral artery (figure 3). The aneurysmal dilation of the ascending aorta measured 5 cm at the level of the right main pulmonary artery. There was no intimal flap in the aortic lumen nor any filling defect in the main and segmental pulmonary arteries.

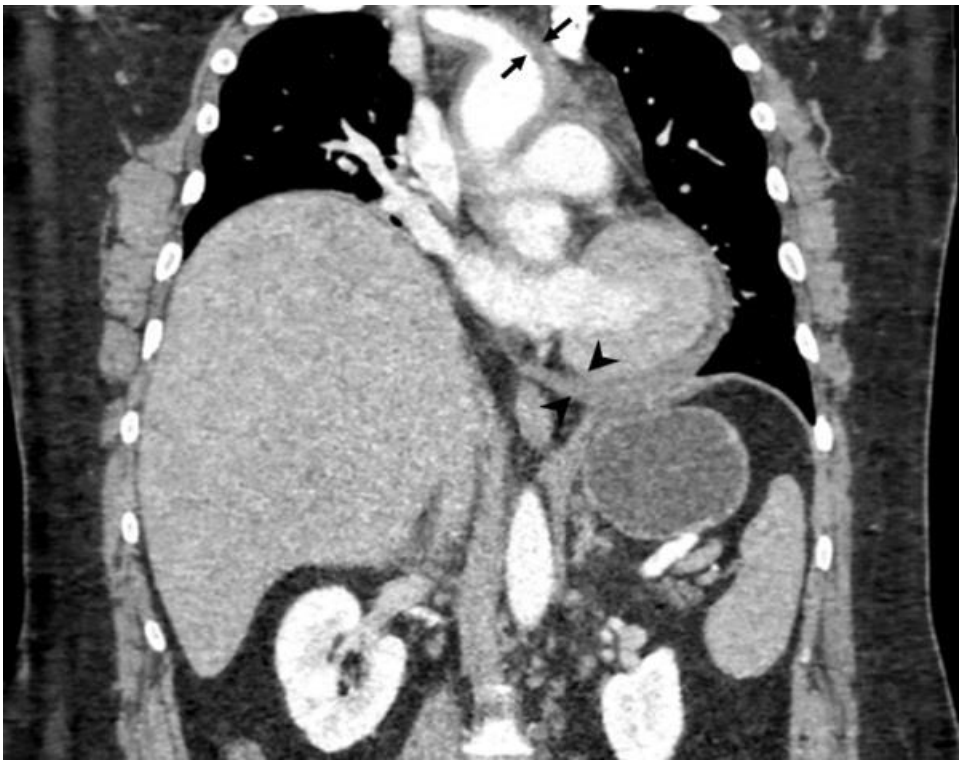

**Figure 3:** Coronal view of the Computed Tomography Angiography demonstrating involvement of the ascending aorta, aortic arch, descending thoracic aorta, and right and left coronary arteries.

The patient was started on an esmolol infusion at 50 mcg/kg/min and was taken to the operating room for an ascending aortic repair, in which an aortic tear above the right coronary sinus was seen with a thrombosed false lumen, as well as hemopericardium. His postoperative course was uncomplicated, and he was discharged home five days later in stable condition.

## Discussion

The muscular wall of the aorta is composed of 3 layers: the inner layer (tunica intima), the middle layer (tunica media), and the outer layer (tunica adventitia). A dissection occurs when a tear in the intima allows blood to leak into the media creating a false lumen. The Stanford Classification system is a commonly used classification system to describe the location of the dissection. Stanford Type A involves the ascending aorta proximal to the brachiocephalic artery and Stanford Type B only involves the descending aorta distal to the left subclavian artery[5].

Dissection into the pericardium with subsequent tamponade is one of the most common cause of death from AD[5]. Another cause of mortality is dissection leading to aortic free rupture into the pleural space or mediastinum[5]. Our patient was hemodynamically stable while in the ED despite the presence of a moderate-sized pericardial effusion. Therefore, a pericardiocentesis was not emergently performed and he proceeded to the operating room. Findings of tamponade using point-of-care ultrasound (POCUS) seen in our patient included a greater than 30% reduction of flow velocity with inspiration on pulse wave Doppler assessments of mitral flow. The presence of a distended inferior vena cava (IVC) is a non-specific finding in tamponade. The most specific finding is a diastolic collapse of the right ventricle.[6] The utility of bedside pericardiocentesis in unstable patients is debatable as it can aggravate the risk of leak

and complicate the surgical repair[6]. However, removal of even a small amount of blood can help maintain a systolic blood pressure of approximately 90 mmHg[7].

AD can also lead to coronary malperfusion which seen in up to one-third of cases. This refers to regional ischemia caused by the dissection flap extending into major branches of the aorta which can mimic several other conditions[8]. In fact, in cases of dissection affecting the aortic root, coronary artery malperfusion can present as a myocardial infarction with (STEMI) or without (NSTEMI) ST segment elevation. As a result, approximately one-quarter of all patients presenting with an AD will have a positive troponin, as is the case of our patient[9]. Additionally, up to 8% of patients with Type A dissection have acute ischemic changes[10]. In our patient, the dissection extended into the left and right coronary arteries, but the ECG lacked any ischemic findings.

Currently, there is no single biomarker or externally validated tool that is sensitive enough to aid in diagnosing acute AD. In terms of imaging, chest radiography has low sensitivity, but chest computed tomography (CT) angiography is fast, reliable, and highly specific for the diagnosis of aortic dissection. The role of POCUS is expanding, especially amongst emergency physicians. TTE is not specific for diagnosing AD but can be performed quickly at the bedside and is useful in determining left ventricular function as well as the presence of valve regurgitation and pericardial effusions. TTE can be combined with an abdominal aortic ultrasound (which may sometimes be limited due to bowel gas or body habitus) to quickly scan for a possible intimal flap. Transesophageal echocardiography (TEE) can also be performed at the bedside although it requires additional training and procedural sedation. Furthermore, it carries potential for airway management but provides immediate and more accurate information compared to a TTE.

Patients with AD are at risk of rapid deterioration and initial therapeutic considerations include strict blood pressure and heart rate control given the shearing forces in the aortic wall, as well as pain control. However, definitive treatment is largely based on the location of the dissection. Most guidelines describe therapeutic goals including a heart rate of lower than 60 beats per minute and systolic blood pressure less than 120 mmHg[11]. Beta-blockers are the preferred pharmacologic agent due to their concomitant heart rate and blood pressure lowering. Intravenous esmolol or labetalol are the preferred single agents. Emergent surgery is usually performed for type A dissection and frequently for type B when medical management has failed.

## Conclusion

AD is a life-threatening disease that accounts for a small fraction of the causes of chest pain that presents to the ED. Signs and symptoms can be atypical, but important features to consider are chest pain that is tearing or ripping, pain that radiates to the back, unequal pulses, limb ischemia, focal neurologic deficits, or limb discrepancies in blood pressure. The most commonly used imaging modality for diagnosis is CT angiography; however, the use of TTE and TEE may provide additional information or help make the diagnosis in a patient with hemodynamic instability that is unable to be transported to the CT scanner. Treatment is focused on strict blood pressure, heart rate, and pain management as well as prompt repair by a cardiothoracic surgeon.

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