

A unique case of acute aortic dissection mimicking myocardial infarction

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ABSTRACT

Introduction: Acute aortic syndrome (AAS) is a rapidly progressive and life-threatening disease of the aorta. Intramural hematoma (IM) is generally held to account for between 5–20% of patients admitted to hospital with the diagnosis of AAS. Acute aortic syndrome may have variable presentations and can mimic myocardial ischemia. **Case Report:** We present a case of a 69-year-old male with cardiac risk factors with sudden onset of severe, substernal chest pain radiating to the left shoulder associated with shortness of breath. Electrocardiography showed ST elevations as well as biphasic T waves in anterior lateral leads consistent with an anterior wall myocardial infarction. However, the cardiac catheterization showed normal coronaries. As the patient's chest pain persisted, an emergent computed tomography scan was performed that revealed an acute IM and underlying Stanford type B dissection. We focus on the difficulty in obtaining a diagnosis, the best diagnostic tools, and management options. Intramural hematoma is best seen on non-enhanced computed tomography scan. Acute dissections involving the ascending aorta are considered surgical

emergencies. Uncomplicated IM confined to the descending aorta are treated medically with intensive care unit monitoring, tight blood pressure and pain control. **Conclusion:** It is important to have a high index of suspicion for AAD in cases of chest pain. Aortic dissection is one of the differential diagnoses for acute chest pain syndrome, even in the presence of typical electrocardiography changes for acute myocardial infarction.

Keywords: Acute aortic dissection, Acute aortic syndrome, Intramural hematoma

How to cite this article

Siddiqui S, Kwan CNK, Concepcion J, Malik BA, Moskovits N, Hollander GM. A unique case of acute aortic dissection mimicking myocardial infarction. J Case Rep Images Med 2017;3:53–57.

Article ID: 100043Z09SS2017

doi: 10.5348/Z09-2017-43-CR-15

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Received: 14 September 2017

Accepted: 31 October 2017

Published: 16 November 2017

INTRODUCTION

Acute aortic syndrome (AAS) can be extremely difficult to diagnose and its characterization is of much controversy [1]. It is a rapidly progressive and life-threatening disease of the aorta and approximately 40% of patients with acute aortic dissection (AAD) die before reaching the hospital with in-hospital mortality of 1–2% per hour for those who survive to hospital-level of care [1]. Intramural hematoma (IM) is generally held to account for between 5–20% of patients admitted to hospital with the diagnosis of acute aortic syndrome

(AAS) or acute aortic dissection (AAD) [2]. Acute aortic dissection may have variable presentations in electrocardiography and symptoms and therefore, can mimic other conditions such as myocardial ischemia. When a patient presents with sudden onset of chest pain with electrocardiography showing ST segment elevation, it is often difficult to differentiate between myocardial ischemia and AAD. Here, we present a patient with chest pain with a strong initial clinical suspicion of a myocardial infarction which turned out to be a case of an AAD.

CASE REPORT

A 69-year-old male with a history of hypertension and type II diabetes mellitus presented with sudden onset of substernal chest pain and shortness of breath. He reported that his chest pain was severe, and radiated to the left shoulder. The patient was anxious and diaphoretic in the emergency room, and the vital signs showed a blood pressure reading of 130/90 mmHg and a heart rate of 68 beats per minute. The physical examination was otherwise unremarkable. In the emergency department, an electrocardiography performed showed ST elevations in the anterior leads and biphasic T waves in the anterior lateral leads (Figure 1). The patient was then taken to the catheterization laboratory emergently. The results of the angiogram revealed normal coronaries. After the procedure, the patient also complained of chest pain which radiated to the back. He was given pain medication and was emergently taken to have a CT scan performed. Computed tomography scan revealed crescentic high attenuation at the descending aortic arch which extends to the aortic hiatus compatible with acute IM and underlying Stanford type B dissection, and filling of the crescentic region via the intercostal arteries and suspected vasovagal vasorum (Figure 2).

Cardiothoracic surgery was consulted and the patient was transferred to the cardiac intensive care unit for medical management. The patient was treated with aggressive blood-pressure control with intravenous labetalol infusion and a nicardipine infusion was also added. Another CT scan was performed after 24 hours which showed the amount of intravenous contrast filling the intramural hematoma at the level of the left pulmonary artery had decreased since the prior study. The second focus of contrast material within the intramural hematoma just below the level of the carina was no longer present (Figure 3). The CT scan image findings where the intramural hematoma has reduced significantly from day-1 (Figure 4A–B) compared to day-2 (Figure 4C–D). The patient's intravenous blood pressure medications were eventually switched to oral antihypertensive medications with labetalol, amlodipine and losartan. The patient was free of chest pain, his blood pressure was well controlled and he was discharged home with close follow-up.

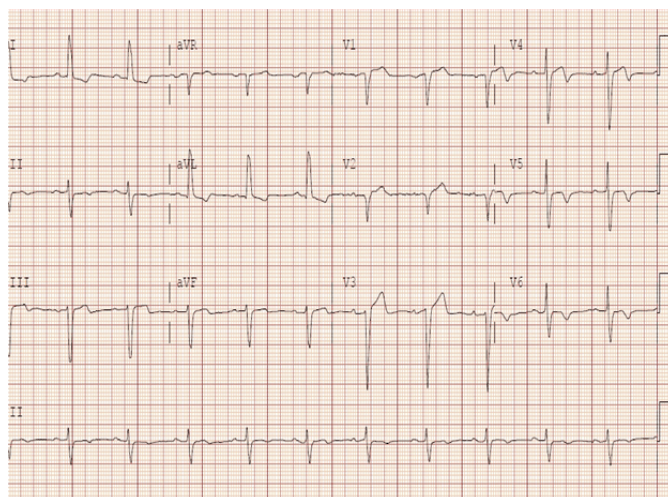


Figure 1: Electrocardiography showing ST elevations in the anterior leads and biphasic T waves in the anterior lateral leads.

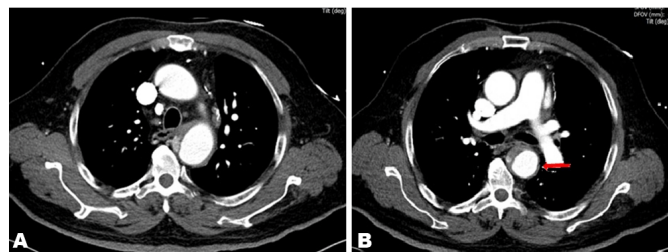


Figure 2 (A, B): Computed tomography scan revealing crescentic high attenuation at the descending aortic arch which extends to the aortic hiatus.

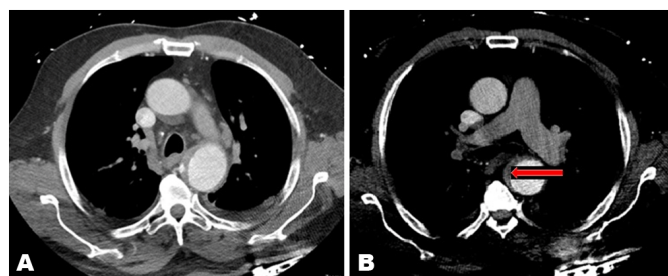


Figure 3 (A, B): Computed tomography scan images 24 hours after the second focus of contrast material within the intramural hematoma just below the level of the carina was no longer present.

DISCUSSION

Acute aortic syndrome, that encompasses, AAD, IM, and penetrating aortic ulcer, are difficult to diagnose [3]. It is defined as a separation within the medial layer of the aortic wall caused by an intimal tear [2]. The DeBakey and the Stanford systems have been used to classify aortic dissection. The Stanford system classifies dissections that involve the ascending aorta as type A, regardless of the site of the primary intimal tear; all other dissections are classified as type B [1]. Acute aortic dissection is an

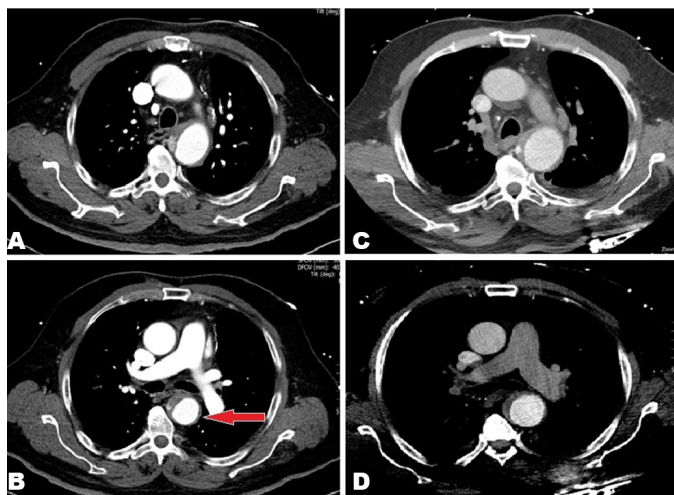


Figure 4: Computed tomography scan images 24 hours later the intramural hematoma has reduced significantly from day-1 (A, B) compared to day-2 (C, D).

uncommon disease in the United States, estimated to be at about 5–30 cases per one million people per year, and accounts for approximately 2000 new cases each year [4]. The risk factors associated with AAD include hypertension, atherosclerosis, known aneurysm and Marfan syndrome [1–3]. Dissection of the ascending aorta is two to three times more common than that of the descending aorta [4].

Intramural hematoma is a hematoma within the medial layer of the aortic wall without the presence of intimal injury [2]. According to literature, the mechanism by which an IM is created is not clearly elucidated [5, 6]. The demographic of IM is different than that of AAD. Patients with IM are older, it is more commonly present with aortic aneurysm, usually occurs in patients with severe atherosclerotic disease and rarely in those with Marfan syndrome. Fewer than 10% of events resolve spontaneously, whereas 16–47% progresses to dissection [6]. The average age ranges from 58–71 years old [6]. It is a serious and rare disease with a relative unpredictable course. Intramural hematoma is generally held to account for between 5–20% of patients admitted to hospital with the diagnosis of AAS or AAD [2]. Based on the international registry of acute aortic dissections (IRAD), of 2830 patients, 178 had [5] IM, of these 42% were classified as type A and 58% were type B [5]. Quick and accurate diagnosis of acute aortic syndromes are difficult due to the wide variety of clinical presentations such as acute coronary syndrome, gastrointestinal disease (such as cholecystitis or pancreatitis), musculoskeletal disease and respiratory diseases (such as pulmonary embolism) [7].

Computed tomography scan and MRI scan remain the gold standard to diagnose intramural hematoma [1, 2]. Computed tomography scan with intravenous contrast is vastly available and can be performed rapidly in the most emergency department, it has a sensitivity of 95% and

specificity between 85–100% [3, 5]. Magnetic resonance imaging scan has sensitivity and specificity of 100% [3, 5]. Its use is constrained by the lack of availability, length of time for image acquisition given unstable patients and contraindication in patients with metallic implants. Intramural hematoma is best seen on non-enhanced computed tomography scan and appears as an area of hyperattenuating crescentic thickening in the aortic wall which was seen on the CT scan findings of our patient. Once diagnosed, treatment options depend on disease severity. Acute dissections involving the ascending aorta are considered surgical emergencies.

In contrast, IM confined to the descending aorta are treated medically unless there is a high risk of end-organ ischemia or continued hemorrhage into the pleural or retroperitoneal space, which can be treated with an endovascular approach [6]. Acute uncomplicated type B dissection should be treated with medical therapy including intensive care unit monitoring, tight control of systolic blood pressure to 100–120 mmHg and pain control. This decreases aortic wall shear stress and reduces secondary adverse events such as aortic expansion, recurrent aortic dissection, and aortic rupture [8]. Beta-adrenergic antagonists (esmolol, metoprolol, or labetalol) represent the first-choice agents, and vasodilators (sodium nitroprusside) and calcium channel antagonists can be used as well [9]. Hydralazine and sodium nitroprusside are less favorable agents, vasodilator therapy should not be initiated prior to rate control so these agents are associated with reflex tachycardia that may increase aortic wall stress, leading to expansion of a thoracic aortic dissection thus, are relative contraindicated in patients with AAD [10].

In our case, the patient was treated appropriately with aggressive blood-pressure control with intravenous beta-blocker labetalol infusion and then a dihydropyridine calcium channel blocker nifedipine infusion was also added. Labetalol has alpha and beta adrenergic activity and is able to have an effect on the heart rate and blood pressure with a single agent. Esmolol is fast acting and has a short half-life and can be advantageous in patients with contraindications [11]. Acute aortic syndrome, including IM may demonstrate diverse symptoms of various types of severity, which may lead to misdiagnosis and delay in case of a life-threatening disease [12]. In the case of our patient, who had strong cardiac risk factors, his initial presentation strongly suggested an acute anterior wall MI fortunately; the diagnosis was made after CT scan was performed and the patient remained stable.

CONCLUSION

A high clinical suspicion and timely diagnosis are crucial to prevent the devastating sequelae of acute aortic syndrome (AAS). It is crucial to have a high index of suspicion for acute aortic dissection (AAD) in cases of chest pain. Clinicians should take into consideration

that aortic dissection is one of the differential diagnoses for acute chest pain syndrome, even in the presence of typical electrocardiography changes for acute myocardial infarction. If AAD is suspected in a patient with chest pain, confirming the diagnosis with the appropriate imaging studies such as computed tomography scan should be performed emergently to avoid administration of thrombolytic resulting in catastrophic results.

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Author Contributions

Sabah Siddiqui – Substantial contributions to conception and design, Acquisition of data, Drafting the article, Revising it critically for importance intellectual content, Final approval of the version to be published

Clara Kwan – Substantial contributions to conception and design, Acquisition of data, Drafting the article, Revising it critically for importance intellectual content, Final approval of the version to be published

Jose Concepcion – Substantial contributions to conception and design, Drafting the article, Revising it critically for importance intellectual content, Final approval of the version to be published

Bilal A. Malik – Substantial contributions to conception and design, Acquisition of data, Drafting the article, Revising it critically for importance intellectual content, Final approval of the version to be published

Norbert Moskovits – Substantial contributions to conception and design, Acquisition of data, Drafting the article, Revising it critically for importance intellectual content, Final approval of the version to be published

Gerald Hollander – Substantial contributions to conception and design, Acquisition of data, Drafting the article, Revising it critically for importance intellectual content, Final approval of the version to be published

Guarantor of Submission

The corresponding author is the guarantor of submission.

Source of Support

None

Conflict of Interest

Authors declare no conflict of interest.

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