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Case Report

Spinal cord infarction a rare complication of acute aortic intramural hematoma: a clinical case report and review of literature

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ABSTRACT

Aortic intramural hematoma is a rare and very serious condition, it can rapidly progress and lead to severe complications if there is a delay in the diagnosis and starting appropriate management. In this case report we are presenting a male 67 years old presented to the emergency department complaining of sudden severe worsening of his lower back pain, associated with left lower limb weakness for which he is unable to walk. A case report of spinal cord infarction secondary to acute aortic intramural hematoma (IMH) type A Standford classification. It represents an unusual clinical presentation of acute aortic IMH. Surgical management done resulting in a successful outcome.

Keywords: Acute aortic intramural hematoma complications, Spinal cord infarction

INTRODUCTION

Acute aortic syndrome is an umbrella of a variety of diseases including aortic dissection, aortic intramural hematoma (IMH), and penetrating aortic ulcer (PAU). Unlike aortic dissection which is classically characterized by an intimal flap and a false lumen, in aortic IMH there is a hematoma within the medial layer of the aortic wall but there is no intimal flap.[1] About 5 to 25 % of acute aortic syndromes are caused by IMH, with older age at presentation compared to Aortic dissection.[2,3] The pathogenesis of aortic (IMH) is not entirely understood, but it is thought to involve bleeding from the vasa vasorum rupture, the small vessels that supply blood to the aortic wall with subsequent mural hemorrhage.[1]

symptoms and risk factors associated with aortic IMH resemble those of aortic dissection, most commonly acute chest pain and or back pain. Additionally, the risk factors include hypertension, atherosclerosis, dyslipidemia, prior cardiac/aortic surgery, aortic aneurysm, and genetic disorders that involve the connective tissue, such as Marfan syndrome.[4,5]

An aortic IMH and its complications can be diagnosed using noninvasive diagnostic methods such as computed tomography angiography (CTA), magnetic resonance imaging (MRI), and transthoracic or transesophageal echocardiography.[2]

The classification of aortic IMH is based on the Stanford system (types A and B) and the De Bakey classification, which can provide the location and extent of the aortic area involved, and also guide the management and prognosis.[1,2] Most patients with IMH are classified as Stanford type B which involves descending aorta (60-70%), other areas involved including ascending aorta (30%), and the aortic arch (10%). ascending aortic IMH can result in aortic regurge, hemopericardium, or rupture, but malperfusion is less common.[1]

Early studies from Europe and the United States reported that individuals with type A IMH were at greater risk for

developing complications, including aortic dissection (25% to 50%), hemopericardium, and rupture, with mortality rate over 30% when treated with medical therapy alone. Given the high risk of fatal complications, surgical management is recommended for type A aortic intramural hematoma (IMH), whereas type B can be treated medically.[1,6]

The misdiagnosis rate of AAS can be as high as 58%. ASS is frequently misdiagnosed as acute coronary syndrome, more common in type A Stanford classification when ascending aorta is involved. this confusion with ACS can result in delayed diagnosis, and inappropriate management with antiplatelets and antithrombotic leading to clinically significant bleeding, for example, hemorrhagic pericardial effusion or periaortic hematoma, that can be fatal if untreated on an emergency basis.[2,7]

CASE REPORT

A 67-year-old married male, Active cigarette smoker 2 packs a day for 50 years, known to have hypertension not compliance to medications, chronic obstructive pulmonary disease (COPD) on Inhalers, and chronic history of cervical and low back pain with spondylopathy. His complaint when arrived at the emergency room was the following sudden severe worsening of lower back pain, and left lower limb weakness for which he could not stand up and walk. Associated with urinary retention. He denied any history of chest pain, palpitations, and shortness of breath.

He visited another hospital before the presentation to the hospital and CT brain done there, showed no evidence of acute brain insult. The patient was conscious, alert, and oriented but looked in pain.

Initially, when arrived at the emergency department his vital signs were stable: blood pressure was 175/94 mmHg, heart rate 69 beats/min, respiration rate 16/min, body temperature 36.8℃, and oxygen saturation 95% on room air.

An electrocardiogram (ECG) done immediately showed normal sinus rhythm with features of the right bundle branch block.

Physical examination revealed intact peripheral pulses bilaterally, Normal sounds, and rhythm with no murmurs in heart auscultation. And an unremarkable lung examination. There was a transient lower limb weakness.

Laboratory findings were as follows: creatine kinase (CK) 3035 U/l (20-200), troponin I high sensitive 0.096 ng/ml (0.00-0.026), D-dimer assay 3.7 mg/l (0.00-0.5), white blood cells 10.32×103/ul, hemoglobin 15.4 g/dl, platelets 187×103/ul, renal function test and electrolytes; serum creatinine 0.78 mg/dl, urea 35, sodium 135 mmol/l, potassium 4.32 mmol/l, prothrombin time 12.8 sec, international normalized ratio (INR) 0.94, and partial thromboplastin time 28.5 sec.

Laboratory investigations were all within normal limits except for an abnormal elevation of CK, D-dimer, and troponin level.

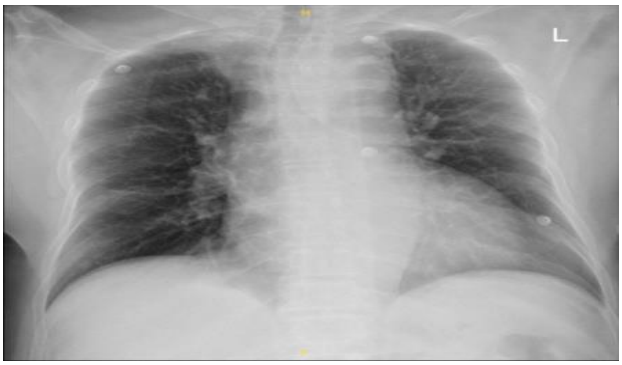

Figure 1: The chest X-ray PA view.

Chest X-ray PA view showing the widening mediastinum, increased cardiothoracic ratio, dilated unfolded aorta, and trachea shifted rightward.

Echocardiography revealed: normal systolic function with ejection fraction of 50%, grade I diastolic dysfunction, left ventricular hypertrophy, paradoxical septum, sclerotic aortic valve with mild aortic insufficiency, and mild mitral regurge, rest of cardiac valves were normal.

From the data above, the patient was admitted to the ward under the care of neurology as a case of back pain with lower limb weakness for evaluation, and the provisional diagnosis was dorsal compressive myelopathy.

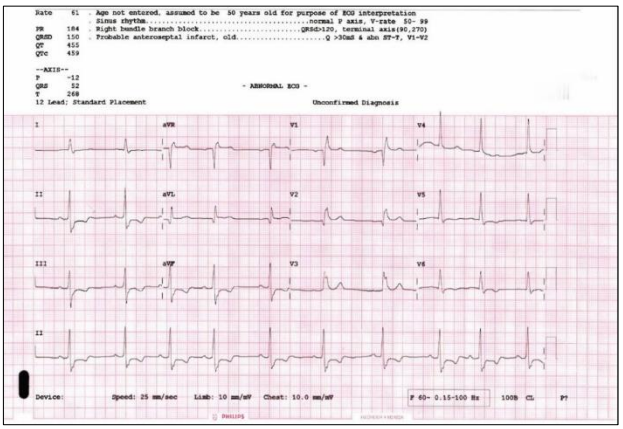

Figure 2: ECG showing sinus rhythm with features of right bundle branch block.

Therefore, the patient was seen by the orthopedic team, and additional imaging was done; contrast-enhanced multiplanar MRI of the dorsal spine with different pulse sequences. Surprisingly, the MRI spine result showed a focal area of abnormal intra-medullary signal opposite

the D5 vertebral body. The picture is highly suggestive of cord infarction, for clinical correlation. CT aortic angiography was recommended by the radiologist.

As the patient is diagnosed with type A aortic IMH, he is managed surgically. The patient was referred to the cardiothoracic surgery team and underwent an ascending aorta and aortic hemiarch replacement with a good outcome.

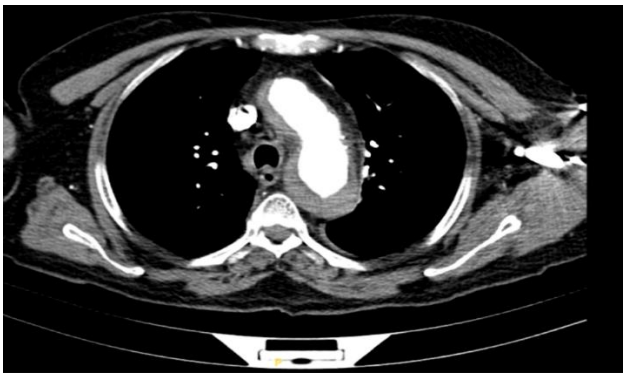

Figure 3: CT aortography, at the level of the aortic arch.

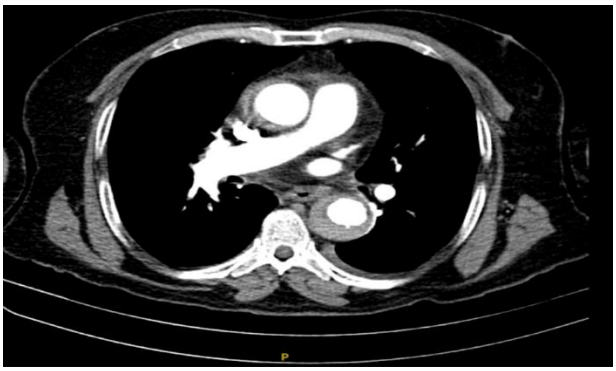

Figure 4: CT aortography, at the level of the ascending aorta.

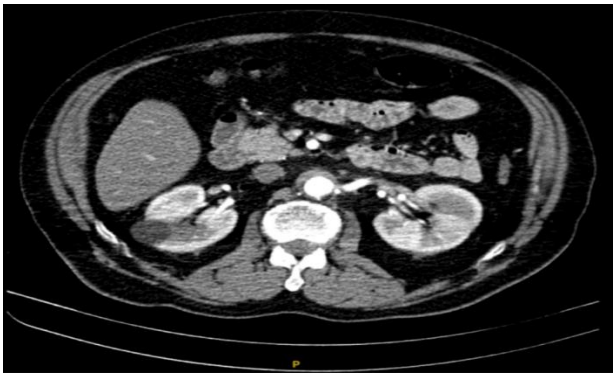

Figure 5: CT aortography, at the level of kidneys.

The above three levels of CT angiography with contrast of the aorta demonstrates a Stanford type A aortic intramural hematoma.

Pre- and post-contrast CT angiography of the aorta revealed intramural hematoma (atypical type of aortic IMH) of type A Stanford classification.

A significant mural hematoma extends along the entire course of the aorta. involving ascending part, arch, and throughout descending to the aortic bifurcation.

The mural hematoma is seen of high attenuation in the precontract phase with total filling of the lumen at the post-contrast phase. There are no signs of an intimal flap, and no contrast leak was detected.

DISCUSSION

Aortic IMH is a potentially fatal emergency that should be suspected and diagnosed early to have a better prognosis and avoid life-threatening complications. This case emphasizes that early recognition of acute aortic syndromes including aortic IMH is important, and to be taken into consideration within our differential diagnosis when a patient complains of sudden severe low back pain and other neurological symptoms such as urine incontinence and unilateral lower limb weakness as presented in our case.

Fortunately, in this case, aortic IMH didn't progress to aortic dissection or other catastrophic complications, even though there was some delay in reaching the diagnosis until it was guided by the radiologist.

Patients with AAS/IMH could present with various uncommon symptoms other than classically known chest or thoracic back pain. Some of the most common neurological presentations include seizures, and Horner syndrome. more severe and catastrophic presentations including stroke and coma.

The occurrence of spinal cord infarction (SCI) as a neurological presentation of AAS/IMH is very rare, with just a limited number of case reports published. Tsushima et al conducted a study in 2019 on the atypical complications of aortic IMH and found that only seven case reports have been published over the past decade.[8] The severity of SCI can range from mild weakness to paraplegia.[9,10] IMH presented with SCI associated with a poor prognosis due to delayed diagnosis.[9] Definitive management in AAS/IMH includes emergency surgery for type A IMH, and aggressive medical therapy with controlling blood pressure in uncomplicated type B IMH.[11] In the case of complicated IMH, surgical management, or less invasive procedure Thoracic endovascular aortic repair (TEVAR), can be considered.[11] According to the ESC guidelines, TEVAR is currently advised for complicated Type B aortic dissection characterized by persistent or recurrent pain, uncontrolled hypertension despite full medication, early aortic expansion, malperfusion, and signs of rupture such as hemothorax or increasing periaortic and mediastinal hematoma.[11,12] There are advocates for more aggressive intervention with

TEVAR plus medical therapy instead of medical therapy alone for uncomplicated type B IMH, due to high failure rates of optimal medical therapy (up to 72%). Emerging data suggest that TEVAR improves long-term survival and disease progression, leading to a shift in treatment approaches toward more aggressive interventional therapy for uncomplicated type B IMH.[13]

CONCLUSION

Acute aortic syndromes are a general term for life-threatening conditions. One of the subtypes is aortic intramural hematoma, which is presented in our case. Although neurological symptoms or complications are common neurological presentations and its prevalence is about 17-40% in AAS patients, spinal cord infarction remains very rare. Accordingly, when a patient has sudden severe back pain and new onset neurological deficit, AAS should be included in the differential diagnosis. For example, in our case, the patient had left lower limb weakness and urine incontinence.

As a physician, it is important to be aware of rare symptoms and atypical manifestations of diseases. It will provide better clinical practice with early diagnosis, fewer complications, and improved clinical outcomes.

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