Type A Aortic Dissection in a Young Patient Presenting with Pericarditis: A Case Report

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Acute or chronic aortic dissection is considered a rare emergency, with an estimated rate of 2.9 to 5 cases per 100,000 patients each year. This condition is most prevalent in males older than 65 years of age with a history of hypertension, atherosclerosis, and previous cardiac surgery [1,2]. To confirm the diagnosis, imaging is used, often by computed tomography angiography (CTA) of the chest. Although prompt treatment is required, patients often present with non-specific symptoms, such as abdominal pain or neurologic deficits, resulting in the early diagnosis of less than 20% and a high mortality rate [1].

We present the case of a 35-year-old man with a history of hypertension and diabetes mellitus. The patient presented to our emergency department (ED) with pleuritic chest pain and fever 3 days after he was diagnosed with gastroenteritis at his first visit. Based on his complaints, high inflammatory markers, and enlarged heart silhouette on chest radiography, pericarditis was our initial diagnosis. However, with the return of high troponin and D-dimer levels, the patient was urgently sent for a chest CTA, which revealed a Stanford type A aortic dissection involving all parts of the thoracic and abdominal aorta up to the bifurcation of the iliac arteries. The patient underwent emergency surgery.

PATIENT DESCRIPTION

A 35-year-old man was first admitted to the ED with lower back pain, which evolved into diffused abdominal pain near the time of his admission. Past medical history was positive for hypertension (treated with valsartan and intermittently amlodipine), which started at the age of 16 years with previous negative assessment for secondary causes and no relevant family history. Medical history also included diabetes mellitus type 2 treated by metformin, morbid obesity, and anxiety.

During his first visit, the patient denied any additional complaints other than non-specific abdominal pain. Vital signs were normal except for high blood pressure 183/95 mmHg. Physical examination revealed a moderate diffuse abdominal pain and a known umbilical hernia without signs of peritonitis or incarceration. Complete blood count, kidney functions, and serum electrolytes were normal; C-reactive protein (CRP) was 6.5 mg/L (normal < 5 mg/L). Urinalysis was negative for leukocytes, protein, or blood. The patient was treated with fluids, metoclopramide hydrochloride, and diclofenac. After improvement of his symptoms, he was discharged with the presumptive diagnosis of gastroenteritis.

Three days later, he was re-admitted to the ED with recurrent abdominal pain and a new chest pain. The patient described a stabbing left-sided chest pain, radiating to the upper back, relieved by leaning forward and aggravated by lying down or taking deep breaths. The pain was accompanied by shortness of breath and nausea without limitation of his regular activity. There were no complaints of vomiting, diarrhea, fever, or chills.

Vital signs included tachycardia of 113 beats per minute, fever of 38°C, blood pressure measured in right and left hands of 180/89 and 182/89 mmHg, respectively, and normal saturation in room air. Bilateral radial pulse was present and regular, heart and lung examination were without findings including non-distended jugular veins. Diffuse abdominal pain was noted without signs of peritonitis.

Initial lab results showed normal complete blood count, hypokalemia of 3.09 mmol/L (normal range 3.5–5.3 mmol/L) with normal kidney function and CRP of 216.7 mg/L (normal < 5 mg/L). Venous blood gases showed mild metabolic alkalosis with pH 7.437 (normal range 7.32–7.42). Sinus tachycardia of 114 bpm was present in electrocardiogram with S1Q3T3 pattern with no changes in ST segment or previous electrocardiogram for comparison. Chest X-ray revealed cardiomegaly and mild widening of the mediastinum [Figure 1A]. Considering the history and clinical manifestations, a leading diagnosis of pericarditis was determined.

During his stay in the ED, the patient presented with worsening chest pain and appeared to be sweating and in distress. His vital signs were without change. Troponin and D-dimer results, taken following the electrocardiogram findings, were 513 ng/L (normal < 50 ng/L) and 3500 ng/ml (normal < 490

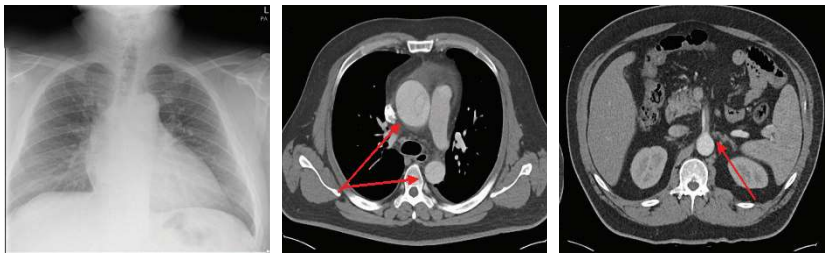
Figure 1. Chest X-ray and CTA showing signs of aortic dissection and pericarditis

CTA = computed tomography angiography

[A] Chest X-ray showing cardiomegaly and mild widening of the mediastinum

[B] Chest CTA with Stanford type A aortic dissection involving ascending and descending thoracic aorta

[C] CTA showing involvement of the superior mesenteric artery



ng/ml), respectively. After considering the diagnosis of pulmonary embolism in this patient, he was sent for an urgent chest CTA. During the test the radiologist noticed signs of aortic dissection and completed the test with CTA aortic protocol.

The chest CTA revealed a Stanford type A aortic dissection involving the ascending and descending aortic arch and abdominal aorta to the bifurcation of the iliac arteries. In addition, the dissection involved the superior mesenteric artery, left external iliac artery, and right internal iliac artery [Figures 1B and 1C]. The emergent cardiovascular surgery team was updated with these findings as per our institution protocol. The patient was immediately taken to the operating room, during which hemodynamic instability occurred following by cardiac arrest. Unfortunately, the patient died after all efforts of resuscitation failed.

COMMENT

Diagnosis of aortic dissection by emergency physicians is challenging [1,2]. An emergency physician seeing 3000 to 4000 patients each year may diagnose an aortic dissection approximately every 3–4 years [3]. Acute aortic dissection has a wide variety of clinical presentations. While chest pain is the most frequent presenting symptom, its low specificity can falsely lead to a different diagnosis [3]. Acute pericarditis may be an uncommon presenting feature of aortic dissection, probably caused by the slow leakage or exudate from the hematoma that facilitates pericardial inflammation, with unclear incidence [4]. In our case, the patient presented with features of acute pericarditis, previously described as a possible warning sign of sudden rupture and with high risk for tamponade development [4].

Only 5–7% of patients with type A aortic dissection are younger than 40 years old. The main risk factors among young patients are connective tissue disease, severe hypertension, and bicuspid aortic valve [5]. Our patient had a history of hypertension that was not well controlled with two first-line medications. The patient did not have any connective tissue disease related symptoms or history and no relevant family history. In a case series of more than 40 years, including young patients with aortic dissection, 25% had a history of severe hypertension, while only 21% had no known risk factors [5]. The mortality rate in this age group was 11%; however, compared with our case, most patients were diagnosed early and presented with intimal tear localized to the aortic root [5].

Considering the rarity of our case, several features in the patient's presentation and clinical course might have led to an early diagnosis. In his initial presentation, the patient had non-specific abdominal complaints. In a study describing consecutive cases of acute aortic dissection over 27 years, 9.7% had localization of abdominal pain at presentation [1]. While these complaints can be part of a viral disease leading to acute pericarditis, as was assumed, the presence of back pain should have raised suspicion of a different diagnosis [2]. The European Society of Cardiology guidelines recommend blood pressure measurement and comparison in both arms and pulses for all suspected patients [2]. Both tests were negative in our patient. However, negative results should not be used to rule out aortic dissection, especially pulse deficit, which was found in only 30% of the patients [2].

Laboratory testing and basic imaging can disclose other features relevant for aortic dissection. D-dimer levels are typically very high in the acute setting, compared with other disorders in which the D-dimer level increases gradually [2]. In addition, troponin levels can be used to assess for myocardial involvement and to suggest the need of further evaluation. Chest X-ray is usually used as the first imaging technique in patients with chest pain and can show the presence of widened mediastinum in some of the cases. While there are no specific electrocardiogram features in patients with aortic dissection, its use can help warrant a more thorough search for etiology, as was in our case.

Our case also highlights the possible role of bedside transthoracic echocardiography (TTE) in early diagnosis. TTE can be limited by abnormal chest wall or obesity, as was in our case. Although the visualization of the flap rupture requires a skilled operator, other more non-specific features such as aortic root widening or aortic regurgitation, can lead to an earlier consideration of the diagnosis, especially in a young patient.

CONCLUSIONS

While acute aortic dissection is a rare diagnosis, it should still be considered in the differential diagnosis of patients with chest pain, even in patients younger than 40 years of age. Younger patients (under 40 years old) with the relevant risk factors should be treated like older adults in the context of their initial diagnostic tests and examination in the emergency department. Basic tests such as chest X-ray, electrocardiogram, and troponin can lead to an earlier suspicion for the diagnosis of acute aortic dissection, which results in faster diagnosis and treatment.

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Capsule

New hope for treating atrial fibrillation

Atrial fibrillation (AFib), the most common type of heart arrhythmia, is a serious condition that can result in atrial blood clots and thromboembolic stroke. **Hulsmans** and co-authors performed single-cell RNA sequencing on atrial tissue from AFib patients and healthy controls to better understand how stromal and immune cells contribute to this disease. They found that recruited CCR2+ SPP1+ macrophages expanded in AFib patients. These cellular and transcriptomic changes were recapitulated in a mouse model of AFib that integrated hypertension, obesity, and mitral valve regurgitation (HOMER). Disrupting *Ccr2*, which coordinates inflammatory macrophage recruitment to atria, or *Spp1*, which helps to drive inflammatory fibroblast activation by macrophages, ameliorated disease burden in HOMER mice, suggesting two potential immunotherapy targets for Afib patients.

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Capsule

Gut microbial fatty acid isomerization modulates intraepithelial T cells

The human gut microbiome constantly converts natural products derived from the host and diet into numerous bioactive metabolites. Dietary fats are essential micronutrients that undergo lipolysis to release free fatty acids (FAs) for absorption in the small intestine. Gut commensal bacteria modify some unsaturated FAs, for example linoleic acid (LA), into various intestinal FA isomers that regulate host metabolism and have anticarcinogenic properties. However, little is known about how this diet–microorganism FA isomerization network affects the mucosal immune system of the host. **Song** et al. reported that both dietary factors and microbial factors influence the level of gut LA isomers (conjugated LAs (CLAs)) and that CLAs in turn modulate a distinct population of CD4+ intraepithelial lymphocytes (IELs) that express CD8αα in the small intestine. Genetic abolition of FA isomerization pathways in individual gut symbionts significantly decreases the number of CD4+CD8αα+ IELs in gnotobiotic mice. Restoration of CLAs increases CD4+CD8αα+ IEL levels in the presence of the transcription factor hepatocyte nuclear factor 4γ (HNF4γ). Mechanistically, HNF4γ facilitates CD4+CD8αα+ IEL development by modulating interleukin-18 signaling. In mice, specific deletion of HNF4γ in T cells leads to early mortality from infection by intestinal pathogens. The data revealed a new role for bacterial FA metabolic pathways in the control of host intraepithelial immunological homeostasis by modulating the relative number of CD4+ T cells that were CD4+CD8αα+.

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