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CASE REPORT

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A 43-Year Old Man with Complicated Stanford Type B Acute Aortic Dissection

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Abstract

Acute aortic dissection is a time-sensitive and rapidly fatal disease if left untreated, being early diagnosis and treatment decisive for survival. Although dissection of the ascending aorta requires immediate surgical attention, therapeutic strategies in case of Stanford type B acute descending aortic dissection may vary. Medical management is recommended for uncomplicated stable dissections while endovascular or surgical repair is indicated for the dissections complicated by rupture, malperfusion syndromes, refractory pain or rapid aortic expansion. We reported a rare case of a rapidly complicated Stanford type B aortic dissection associated with intramural aortic hematoma occurred in a 43-year old man from Apulia region of Southern Italy which was successfully submitted to endovascular stent graft placement and subsequently 12-months follow-up. We also indicated the need for early multidisciplinary assessment including acute classification having clinically relevant implications for subsequent management.

Keywords: *Type B acute aortic dissection, Aorta, Diagnosis, Management*

Introduction

Acute aortic dissection (AAD) represents the most frequent life-threatening disorder affecting the aorta [1,2] having an incidence in the industrialized countries likely to be about 3-6 per 100.000 inhabitants [2]. AAD occurs when the blood forces the tear in the intimal layer of the diseased aortic wall and dissects it along the media, forming a false lumen [1,3]. The dissection can propagate distally, proximally or both along the aorta wall. AAD is a time-sensitive and rapidly fatal disease if left untreated presenting high average mortality rates [1,4]. Despite this data, less than half of AAD are correctly diagnosed in their initial presentation [5] and over 50% of them are diagnosed 24 hours after hospitalization [6]. AAD is classified as complicated depending on the presence of rupture, malperfusion syndromes, refractory pain or rapid aortic expansion and is categorized anatomically according to the origin of the intimal tear [7]. In the Stanford classification system type A dissection involves the ascending and transverse aorta and type B dissection involves the descending aorta, although retro-A dissection was reported if type B dissection extends proximally to aortic arch. Stanford type A AAD generally occurs between 50-60 years age range, type B AAD in older subjects. Acute classification is important because it drives decisions regarding surgical versus medical management, requiring ascending type A aortic dissection emergency surgical repair, whereas descending type B dissection is generally managed medically [1,8].

We report a rare case of a rapidly complicated Stanford type B AAD associated with intramural aortic hematoma (IMH) occurred in a young subject from Apulia region of Southern Italy which was successfully submitted to endovascular stent graft placement and subsequently underwent 12-months clinical follow-up. IMH, considered the precursor of the classic dissection, is characterized by presence of bleed within the aortic wall without the evidence of an intimomedial flap.

Description

A 43-year old previously healthy Italian man was admitted to the Internal Medicine Unit of Bari Sud "Di Venere" Hospital, Azienda Sanitaria Locale Bari, Bari - Italy, in February 2017. Family history included arterial hypertension and diabetes mellitus, his personal history showed detection of refractory arterial hypertension from about two months. At admission the patient complained the sudden onset from about four days of shortness of breath associated with severe chest, back and abdominal pain rapidly radiating to the right lower limb. The patient reported sharp and tearing abdominal pain ascribing to the pain a migratory feature to inguinal region and right leg. He showed good functional, neurological and nutritional status and intact mental functioning. No comorbidities were reported.

Results

Patient physical general examination was remarkable for a blood pressure of 190/90 mmHg, heart rate of 100 bpm' and respiratory rate of 23 breath per minute. Thoracic examination was notable for reduced breath sounds in the medium-baseline seat of the left lung, abdominal examination for pain on palpation of umbilical and hypogastric region and right iliac fossa, cardiovascular examination was unremarkable. Laboratory tests were normal except for evidence of elevate levels of white blood cells (11.90 x10^3/uL), d-dimer (4135 ng/l), fibrinogen (646 mg/dl) and impaired fasting glucose (125 mg/dl). Electrocardiogram (EKG) detected sinus tachycardia and left-side ventricular hypertrophy. The ultrasound

scan (echography) was normal except for left basal pleural effusion that was confirmed by Chest X-ray that followed. The computed tomography angiography (CTA) performed revealed a Stanford type B thoracic-abdominal AAD with spiral course, intimomedial flap in the middle third of the thoracic aorta, enlargement of the descending aorta and finishing in the middle third of the right common iliac artery (Figure 1). CTA also showed the presence of 7.5 x 1.5 cm IMH located upstream of the origin of the left subclavian artery, occurring the entry point of the dissection at the distal portion of the IMH. The patient successfully underwent endovascular stent graft placement, the postoperative course was favourable and he was discharged in an improved condition on the 13th postoperative day. The patient subsequently underwent a 12-months medical follow-up presenting good general health and functional status and normal average blood pressure (120/70 mmHg) with the prescribed drug therapy (atenolol 100 mg/die, lercanedipine 10 mg/die). Patient was completely asymptomatic, performing a normal social and working activity.

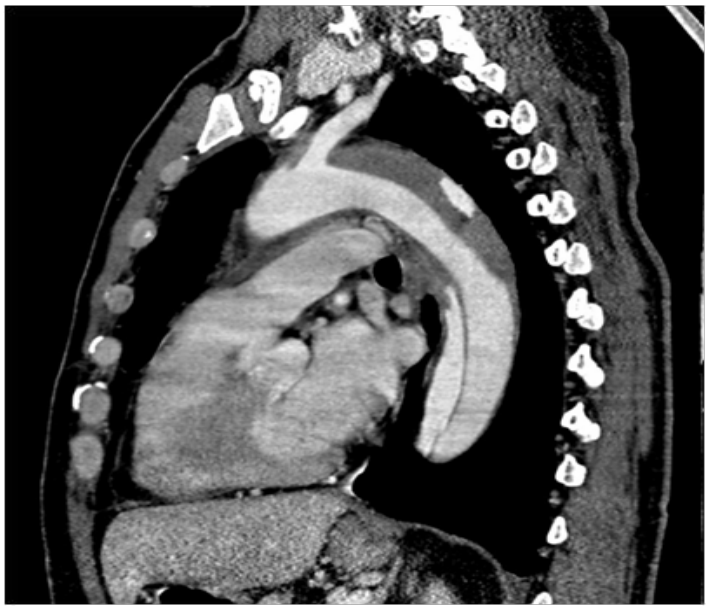

Figure 1: *Sagittal contrast computed tomography image demonstrating Stanford type B acute aortic dissection complicated by intramural aortic hematoma.*

Male sex
Age > 60 years
Hypertension
Atherosclerosis
Genetically triggered thoracic aortic disease (Marfan syndrome, Bicuspid aortic valve, vascular Ehlers-Danlos syndrome, Loeys-Dietz aneurysm syndrome, hereditary TAA/D)
Congenital diseases/syndromes (coarctation of the aorta, Turner syndrome, tetralogy of Fallot)
Inflammatory/infectious diseases (giant cell arteritis, Takayasu arteritis, Behçt disease, aortitis, syphilis)
Trauma, iatrogenic (Prior aortic valve surgery, catheter/stent, aortic manipulation)
Cocaine use
Pheocromocytoma
Pregnancy

Table 1: *Risk factors for acute aortic dissection according to the international registry of aortic dissection.*

Discussion

At present AAD remains a serious disease having multifactorial and unclear pathogenesis. We reported a rare case of a rapidly complicated Stanford type B AAD associated with IMH of the transverse aorta in a young hypertensive subject, although descending dissections occur more commonly in older individuals. This case underlines the utility of the Stanford classification system because the categories it describes are clearly defined on imaging and have clinically relevant implications for subsequent management and prognosis [1,9]. Stanford type B AAD is a rare but clinically relevant entity because of the potential for complications requiring emergency treatment and leading to poor outcome in up to 20% of the patients with estimated early mortality approximately 1-2% per hour [1,10]. A timely and accurate diagnosis and classification of AAD in the emergency remains a clinical challenge being crucial for decision making although more than half of the cases of AAD is yet diagnosed after 24 hours from hospitalization. This delayed diagnosis has become an important factor leading to increased risk of AAD-associated morbidity and mortality. Indeed in case of clinical suspicion of AAD is required to establish a set of highly sensitive and specific diagnostic examinations for the early diagnosis. Although typical clinical presentation of type B AAD includes sudden onset of severe chest, back or abdominal pain, the symptoms are variable and may mimic more common conditions, therefore especially when risk factors [11] are simultaneously present (Table 1), clinical suspicion must be placed and multidisciplinary diagnostic strategy including laboratory markers and diagnostic imaging techniques may be performed in order to early confirm the diagnosis of AAD [1,12]. Several biochemical markers of AAD has been reported in the last decade but up to now data are not conclusive for clinical use [13], representing plasma d-dimer levels a promising diagnostic marker with high sensitivity for exclusion of AAD [14]. Although the chest x-ray can detect abnormalities such us abnormal aortic contour, widening of the aortic silhouette, deviation of trachea or esophagus, pleural effusion, normal chest x-ray cannot exclude the presence of AAD [1,11]. The main auxiliary diagnostic approaches for AAD includes contrast computed tomography angiography (CTA), transesophageal echocardiography (TEE), aortography and magnetic resonance angiography (MRA) [1,11,15]. Although these methods have similar high sensitive and specificity, CTA is the most commonly used for its widely availability and rapidity [1,16]. In addition intravascular ultrasound (IVUS), a novel imaging modality recently proposed, demonstrated the highest accuracy, sensitivity and specificity during endovascular diagnosis and treatment particularly of complicated type B AAD [17]. Acute classification is crucial in decision-making, remaining Stanford type B AAD usually subject to medical treatment unless complicated by organ or limb malperfusion, progressive dissection, impending rupture, persistent intractable pain, uncontrolled hypertension despite adequate drug therapy, neurological deficits [1,11]. Initial medical management is recommended for uncomplicated dissection, representing up to now the anti-impulsive and anti-hypertensive therapy with beta-blockers the fulcrum of modern medical management of type B AAD [1,8,18,19]. Beta-blockers such us labetalol, metoprolol and atenolol are the first choice

drugs because of their mechanism lowering the left ventricular contraction force and stress on aorta in addition to controlling heart rate and blood pressure. Alternatively calcium channel blockers can be used to control blood pressure if beta-blockers cannot be administrated. In case of appearance of complications, endovascular interventions or surgical repair are recommended [9,10]. Today stent graft repair is developing as a strong alternative to open surgical repair for dissection with ischemic complications or impending rupture and it may evolve a method for definitive treatment for patients with appropriate indications [19,20].

Conclusion

We indicated the need for early multidisciplinary assessment of AAD including an accurate imaging identification of the site of the intimal tear. A timely and accurate diagnosis of AAD remains a clinical challenge particularly in the emergency departments. Although AAD is a time-sensitive and rapidly fatal disease if left untreated being early diagnosis and treatment decisive for survival, up to now less than half of AAD are correctly diagnosed in their initial presentation and many of them are diagnosed 24 hours after hospitalization. CTA represent the first choice for imaging in the work up of AAD due to its high sensitivity, specificity and widely availability also in the emergency departments. Given the recent results of medical and surgical therapy, today the use of anti-impulsive and anti-hypertensive therapy remains the first choice therapy in the management of uncomplicated stable type B AAD, while endovascular or surgical repair are indicated for the dissections complicated. Further intervention studies including a greater number of patients will undoubtedly be informative in this regard.

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