UNCOMMON ASYMPTOMATIC AORTIC DISSECTION WELL TOLERATED

UNE DISSECTION DE L'AORTE PAS COMME LES AUTRES : ASYMPTOMATIQUE ET BIEN TOLEREE

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Abstract

We report a case of 78-year-old man with a history of a workplace accident 54 years ago, hospitalized in our hospital with a type A aortic dissection involving brachiocephalic and sparing Valsalva sinus. The patient refused surgical treatment. After a follow up of 6 months, the patient has symptoms of dyspnea.
We discuss then the etiology, diagnosis and management of the type A aortic dissection.

Key words: Aortic dissection; Echocardiography; Computed tomography

Résumé

Nous rapportons dans cet article le cas d'un homme de 78 ans qui avait comme antécédent un accident de travail il y a 54 ans, et qui était hospitalisé dans notre service pour une dissection de l'aorte de type A s'étandant vers le tronc brachiocéphalique et épargnant le sinus de Valsalva. Le patient a refusé le traitement chirurgical. Actuellement après un recul de 6 mois, le patient est symptomatique de dyspnée.
Nous allons discuter les étiologies, le diagnostic et la prise en charge de la dissection de l'aorte de type A.

Mots clés : Dissection de l'aorte ; Echocardiographie ; Tomodensitométrie

ملخص

نقدم تقريرًا في هذاالمقال عن حالة رجل يبلغ من العمر 78 عامًا تعرض لحادث في العمل منذ 54 عامًا ، وتم نقله إلى المستشفى في قسمنا لإجراء تشريح للشريان الأبهر من نوع A ينتقل إلى الجذع العضدي الرأسي وتجنيب جيب Valsalva. و قد رفض المريض العلاج الجراحي. حاليا و بعد 6 أشهر المتابعة، يحمل المريض أعراضا من نوع ضيق التنفس.
سنناقش المسببات، والتشخيص، وإدارة تشريح الأبهر من النمط A.

الكلمات المفاتيح: تشريح الشريان الأبهري ; تخطيط صدى القلب ; التصوير بالمفراس الطبي.

Background

Aortic dissection (AD) is a severe life threatening disease which involves disruption of the madial layer with intramural hemorrhage [1]. Several conditions may lead to AD such as Marfan syndrome or other connective tissue diseases, bicuspid aortic disease, known thoracic aortic aneurysm and trauma [1]. We report a case of 78-year-old man with a chronic type A aortic dissection. We discuss herein the etiology, diagnosis and management of the chronic aortic dissection.

Case report

A 78-year-old smoker man presented a 5-month history of exertional dyspnea NYHA class III with paroxysmal orthopnea. He had a past history of poorly controlled arterial hypertension treated with Enalapril and paroxysmal atrial fibrillation treated with amiodaron. No oral anticoagulation was prescribed because of anemia.

Physical examination revealed a diastolic aortic murmur with pulmonary crepitations. Blood pressure was asymmetric in the arms (160/80 and 70/50 mmHg in the left and the right arms respectively). Electrocardiography showed a normal sinus rhythm with pathologic Q waves in the anteroseptal leads and incomplete left bundle branch block.

Chest radiography showed cardiomegaly with pulmonary congestion (Figure 1). Laboratory data pointed out a normocytic normochromic anemia (Hb = 8.7 g/dL). Serum creatinine was 246 µmol/L. Blood urea concentration was 21 mmol/L.

A transthoracic echocardiogram (TTE) revealed moderate concentric left ventricular hypertrophy with normal ejection fraction. Color-flow doppler mapping revealed mild mitral and aortic regurgitations. Aortic root was normal sized as Valsalva sinus was of 35 mm. Below sinotubular junction, ascending thoracic aorta was dilated (45 mm). No pericardial effusion or wall motion abnormalities were noted. We guessed the dissection flap as a slighltly moving membrane (Figure 2A) from the parasternal long axis view. The short axis window confirmed a tricuspid aortic valve and the dissection membrane was obsious from a modified view (Figure 2B). Suprasternal view showed the dissection flap (Figure 3A) with doubtful thrombus (Figure 3B).

Transoesophagal echocardiography (TEE) confirmed the presence of type A aortic dissection sparing the aortic root associated with mild aortic regurgitation. TEE revealed a partially thrombosed false lumen with small re-entry tears (Figure 4).

A careful questioning demonstrated that during a workplace accident 54 years ago, the patient took off a heavy load in order to rescue his coworker leading to a knife-like chest pain with dizziness and hemoptysis.

Regarding renal damage, isotonic saline hydratation was performed before CT angiography scan with an improvement of serum creatinine falling to 165 µmol/L. CT scans upheld Stanford type A aortic dissection involving ascending aorta and the brachiocephalic artery but sparing aortic valve and Valsalva sinus. There was no further extension to the descending aorta (**Figure 5**). The maximum diameter of the ascending aorta was 53 mm.

Blood pressure was controlled initially with intravenous infusion of nicardipin and then with acebutolol (200 mg daily) and Amlodipin (5 mg twice daily). Euroscore-II was 15% and open repair surgery was indicated but the patient refused. After 5 day hospital stay, he was discharged with antihypertensive therapy. After a follow up of 6 months, our patient complained about dyspnea but blood pressure was 120/60 mmHg.

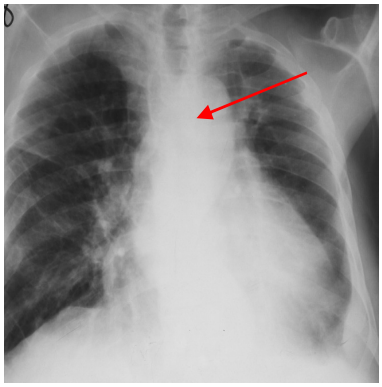

Figure 1: Chest X-ray revealed cardiomegaly with pulmonary congestion with enlarged ascending thoracic aorta (**Arrow**)

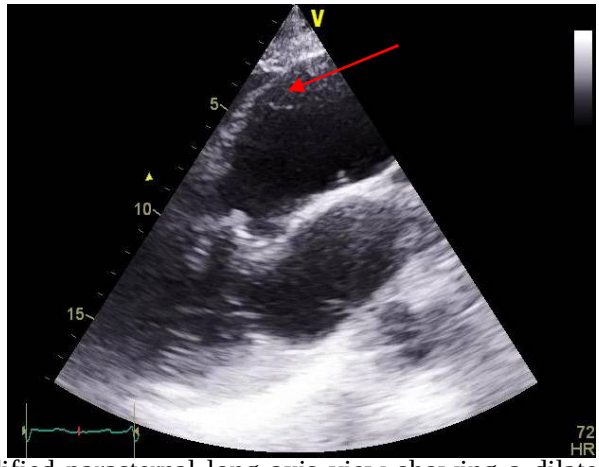

Figure 2A : Modified parasternal long axis view showing a dilated ascending aorta above Valsalva sinus with an intimal membrane (**Arrow**)

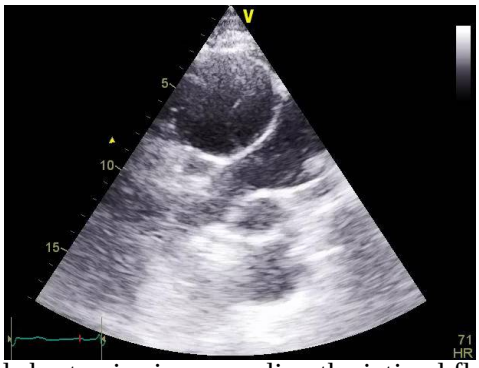

Figure 2B: Parasternal short axis view revealing the intimal flap within the aorta

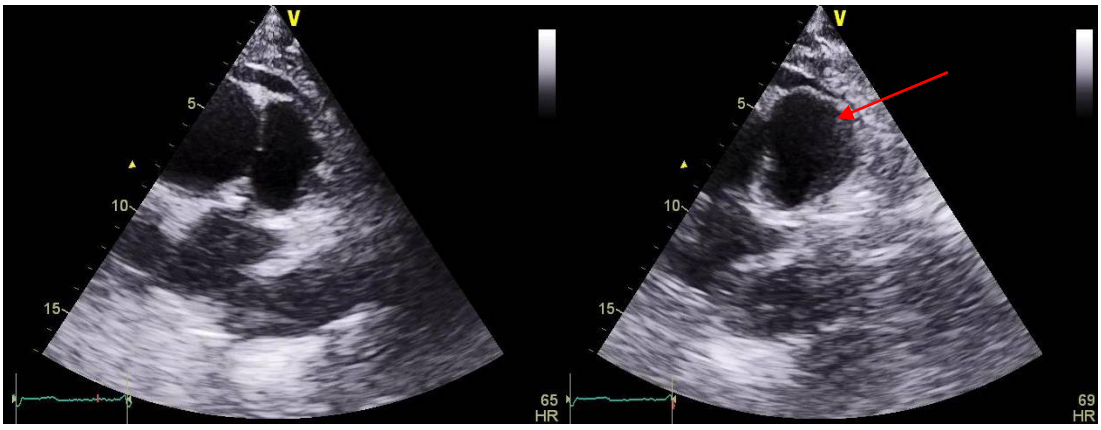

Figure 3: Suprasternal view showed the dissection flap with doubtful thrombus (**Arrow**)

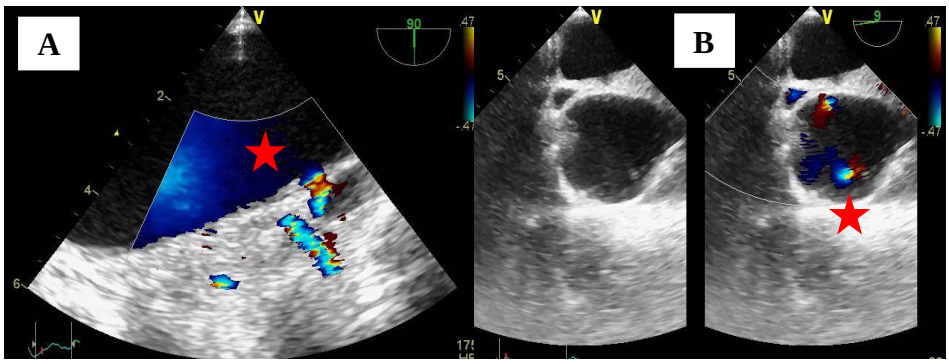

Figure 4 (A and B): Transœsophageal echocardiograms with color-flow Doppler imaging showing a dilated ascending aorta with entry tears (**Star**)

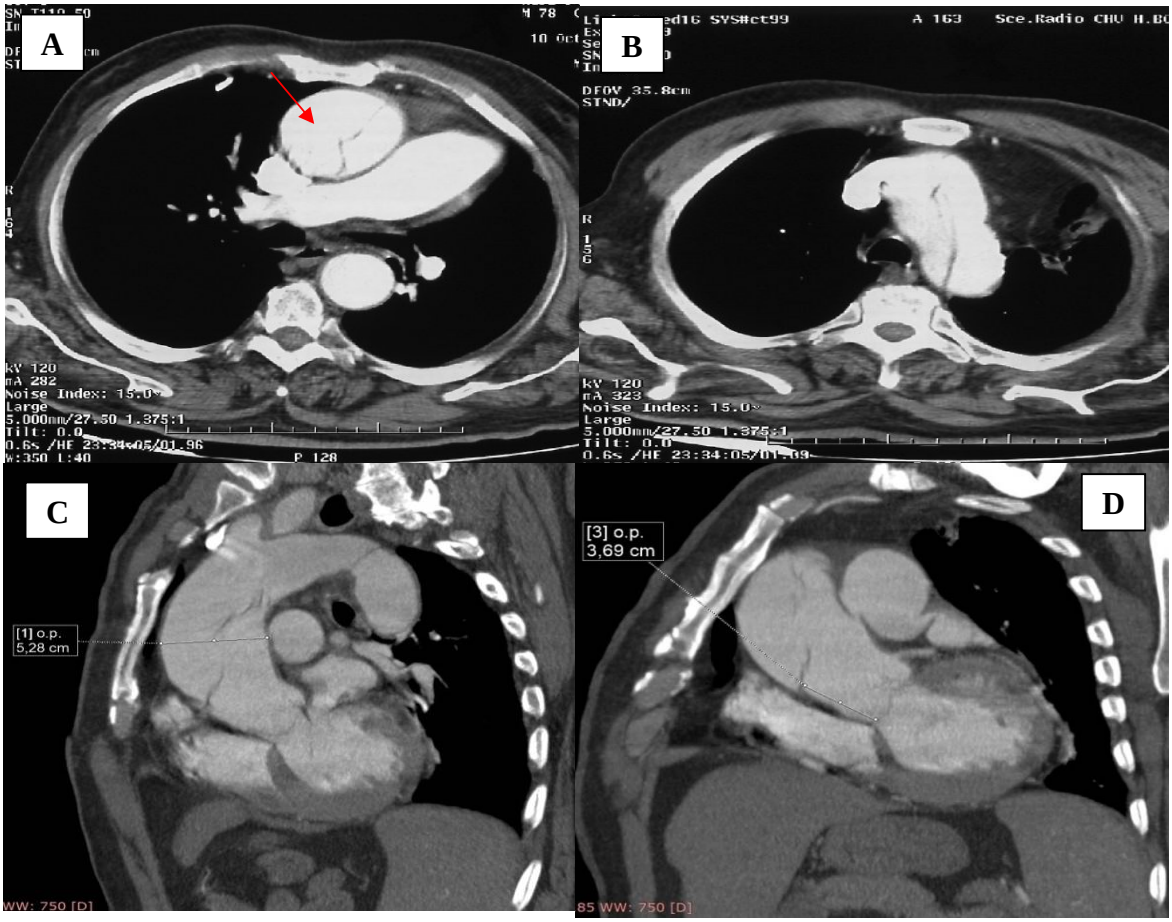

Figure 5 (A, B, C and D) : Contrast CT angiography with section axial images (**A and B**) demonstrating ascending aortic dissection with patent false lumen (**Arrow**). From oblique sagittal plane images, ascending aorta is dilated (**C**) and intimal flap appeared sparing the aortic valve and Valsalva sinus (**D**).

DISCUSSION

Aortic dissection is defined as disruption of the medial layer provoked by intramural bleeding, resulting in separation of the aortic wall layers and subsequent formation of true and an false lumens with or without communication [2]. In acute aortic syndrome, there are two classifications according localisation known as Stanford (A and B) and De Bakey (I, II and III) [2]. AD is commonly defined as acute when it occurs within 14 days after onset of symptoms [3,4]. Additionnaly, AD is defined as sub aucte (15-90 days) or chronic (> 90 days) [2]. Since the first reported case in 1760, AD is known as the most devastating complication of several aortic diseases [1]. The annual incidence of AD varies between registries. In the Oxford Vascular study, AD accounts 6 cases per 100 000 per year [5]. The annual incidence of AD was higher ranging from 9.1 and 16.3 cases per 100 000 in Swedich women and men respectively [1]. Acute AD may evolve towards chronic phase in nearly one third of patients [6].

Commonly, AD can result from intimal rupture with a cleavage formation and progression into the medial layer [4]. There is a medial degeneration in the aortic wall in most cases of AD [7]. We describe a case of type A AD which occured in a 78-year-old man survived from acute AD dating back 54 years. Our patient suffered solely from exertional dyspnea and orthopnea. Atypical manifestations may occur in elderly, such as palpitations or dyspnea [8]. In chronic AD, some patients suffered from prolonged fever, weight loss or anorexia mimicking infectious or neoplasic causes [8, 9, 10].

In the other hand, both acute and chronic AD may be completely asymptomatic [11,12]. Imaging techniques are of essential contribution in the diagnosis of AD. Although transthoracic echocardiography (TTE) is easier, cross-sectional imaging with transoesophageal echoardiography (TTE), CT scaning and magnetic resonance imaging (MRI) are highly accurate and reliable in

the diagnosis of AD [2,4,11]. In our case, AD was suspected from transthoracic echocardiogram and definitely confirmed by CT imaging. A patent false lumen and aneurysmal dilatation of the aorta are predictor of high mortality [13]. Our case is challenging because according to patient questioning, there was neither acute chest pain nor malperfusion syndrom and in the same way, the patient suffered from a violent chest trauma 54 years ago. In the other hand, some imaging characteristics reflecting chronicity are lacking. In fact, according to ESC guidelines dealing with aortic diseases, thickened and immobile intimal flap, thrombosed false lumen or aneurysms of the thoracic aorta indicated chronic AD [2]. In our patient, multislice CT advocated the diagnosis of acute AD with communicated true and false lumen and TEE revealed intimal flap was freely moving independently of aortal wall movement. In the other hand, there was no pericardial effusion in favor of acute AD. So far, more accurate imaging techniques may be helpful to determine the age of AD [14]. Typically, a type A AD mandates urgent open-heart surgery [15,16,17] despite of high mortality and morbidity [18]. Our patient refused surgical repair. Then, he was conservatively managed with intensive antihypertensive medications based on betablockade and calcium channel blocker. When dealing with elderly, aortic surgery may be harmful and thus endovascular repair is considered alternatively. This technique is still debating. In fact, some studies depicted no role for endovascular repair of Stanford type A AD [1] while other issues revealed successful management of type A AD with endograft placement [19].

In conclusion, AD is a potentially life-threatening condition associated with high mortality and morbidity. Survivors require careful watching and intensive blood pressure control. Surgical management remains the gold standard treatment of Stanford type A AD. Further studies are in need to clarify the place of endovasular repair in the management of ascending AD.

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