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Case Report

Successful medical management of a 16-month chronic type A aortic dissection ☆

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ABSTRACT

Stanford type A dissections usually require surgery because they are associated with high morbidity and mortality. However, there are situations where medical management becomes the definitive treatment. We report the successful medical management of a 16-month chronic type A aortic dissection in a 56-year-old male patient with a past surgical history of ascending aortic aneurysm repair. The dissection is unique because it is distal to the graft and does not extend into the main aortic branches. A review of a patient's surgical history and nonenhanced imaging studies is essential when a type A dissection is discovered. Ascending aortic grafts may preclude the most serious complications of type A dissections.

Introduction

Aortic dissections occur when blood enters the media of the aortic wall through an intimal defect. First, the most common etiology is cystic medial degeneration secondary to hypertension, atherosclerosis, and collagen disorders [1]. Second, cocaine may cause a sudden and severe elevation in blood pressure. This increased shear stress in the aorta leads to disruption of the intimal layer [2]. Third, aortic dissection may occur via iatrogenic causes that include catheter angiography, cardiac surgery, and valve replacements. This disease process commonly presents as a sudden tearing pain of the chest wall that radiates to the back.

Contrast enhanced CT demonstrates a true and a false lumina separated by an interposed intimal flap. The Stanford classification stratifies aortic dissections based on location. Type A dissections involve the ascending aorta, whereas Type B ones do not. Complications of acute type A dissections include pericardial tamponade, severe aortic regurgitation, and aortic rupture. Extension of the dissection into the aortic root may lead to the development of pericardial tamponade. Additionally, if aortic root dilation is present, retrograde dissection of the aortic cusps may result in acute aortic regurgitation.

☆ Conflict of interest: The authors have declared that no competing interests exist.

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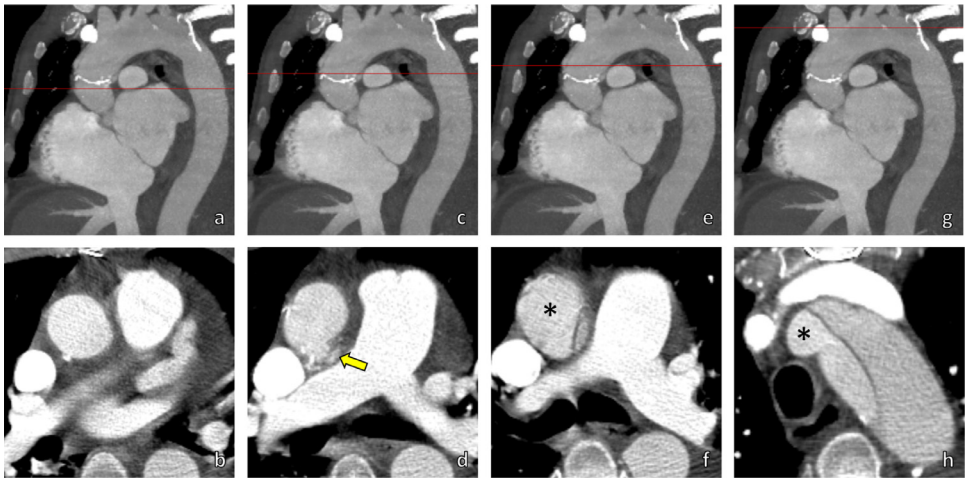

Fig. 1 – Sagittal MIP and correlated axial images of CTA chest sequenced caudally to cranially. (a, b) Inferior to the distal suture line of the supracoronary tube graft, (c) at the level of the distal suture line and (d) the origin of the CTAD (arrow), (e) superior to the distal suture line and (f) the true lumen of the dissection (*), (g, h) at the ostium of the right innominate artery.

Acute type A dissections can enter a chronic phase after 90 days and are labeled chronic type A dissections (CTAD) [3].

Traditionally, management of type A dissections is surgical intervention due to the high rate of complications and mortality [1]. However, there are reported cases of successful medical management of this condition. For example, it is not uncommon for patients with prior aortic valve replacements to present with type A dissections. The suture line prevents dissections from advancing into the right coronary artery, the prosthetic aortic valve protects against aortic insufficiency, and periaortic adhesions protect against aortic rupture [4]. In contrast, our case involves the development of a type A aortic dissection in the setting of an ascending aortic aneurysm repair for aortic insufficiency. This aortic dissection has been successfully managed with medical treatment, despite many risk factors, for 16 months.

Case Report

A 56-year-old African American male with a past medical history of hypertension, end-stage renal disease secondary to autosomal dominant polycystic kidney disease (ADPKD), prior myocardial infarction, cocaine use, liver cirrhosis, aortic insufficiency, and cardiomegaly presented with shortness of breath on exertion that limited his functional mobility. The patient had a past surgical history of ascending aortic aneurysm repair. He admitted to daily use of cocaine and tobacco. The review of symptoms was negative for chest pain, palpitations, and abdominal pain. The patient's renal disease was managed through hemodialysis. Initial laboratory results demonstrated renal failure with blood urea nitrogen 109 mg/dL and serum creatinine of 14.9 mg/dL.

Transesophageal echocardiogram on hospital day 5 revealed a type A aortic dissection. The patient was hemodynamically stable, his blood pressure was 104/68. Further evaluation with CT angiography (CTA) chest revealed Stanford type A dissection (Fig. 1). There was no extension of the CTAD into the right innominate artery, left common carotid artery, or left subclavian artery (Fig. 2). Findings related to ADPKD were seen in the upper abdomen. The enlarged kidneys contained innumerable cysts. Extrarenal manifestations included polycystic liver disease. The pleural and pericardial effusions may have been secondary to renal or cardiac pathologies (Fig. 3).

The dissection was first present on prior CTA chest 16 months earlier (Fig. 4a). In addition, nonenhanced CT chest at that time demonstrated a supracoronary tube graft (Fig. 4b). The graft was secondary to ascending aortic aneurysm repair performed within the past 4 years. Prior nonenhanced CT chest from approximately 4 years earlier demonstrated a 6.0 cm ascending aortic aneurysm (Fig. 5). The patient was discharged on hospital day 9 in stable condition on carvedilol 12.5 mg twice daily.

Discussion

Aortic insufficiency is caused by incompetent aortic valve leaflets or a dilated aortic root. Incomplete closure of the aortic cusps allows blood to flow retrograde into the left ventricle. When an ascending aortic aneurysm reaches 6 cm, the cumulative risks of aortic rupture, aortic dissection, and death are 14.1% [5]. Elective prophylactic surgery is appropriate when the diameter of the aneurysm reaches 5-5.5 cm or enlarges $\geq$ 1 cm/year [6]. One major risk factor for aortic insufficiency is ADPKD. Vascular manifestations of ADPKD include dolichoectasias, berry and aortic aneurysms, and dissections. Gene mutations that cause the loss of function of certain proteins expressed in vascular walls may be the underlying etiology for this disease process [7].

The development of aortic insufficiency in our patient is secondary to a dilated aortic root. He is at high risk of

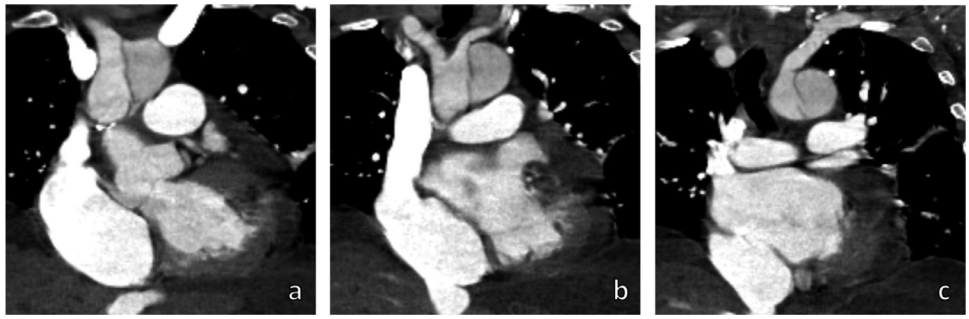

Fig. 2 – Coronal images of CTA chest sequenced anteriorly to posteriorly. No extension of the CTAD into (a) right innominate artery, (b) left common carotid artery, and (c) left subclavian artery.

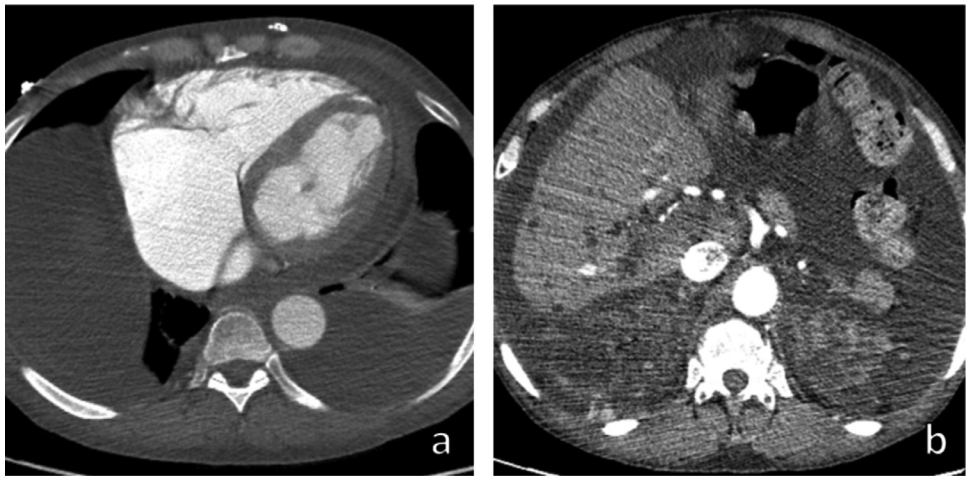

Fig. 3 – Axial images of CTA chest sequenced superiorly to inferiorly demonstrating (a) cardiomegaly, pericardial effusion, pleural effusion, (b) ascites, innumerable renal and liver cysts.

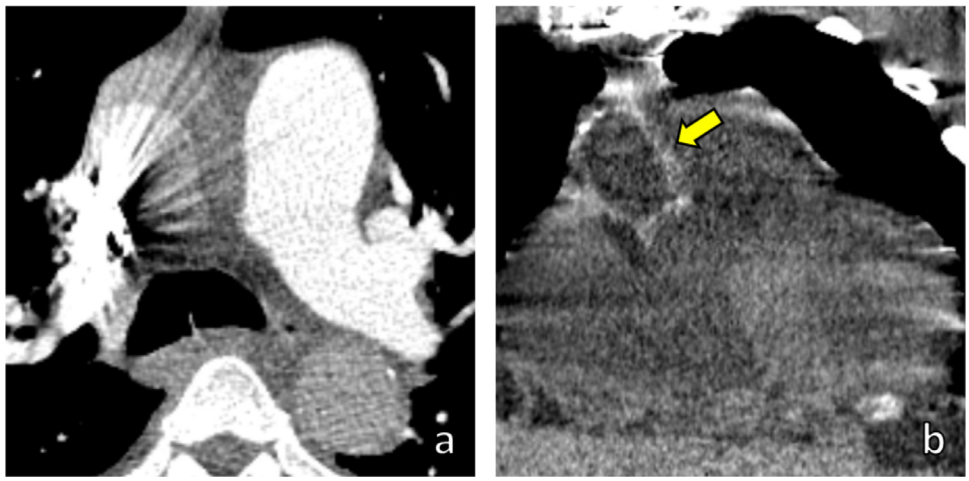

Fig. 4 – Priors from 16 months earlier. (a) Axial image of CTA chest demonstrating a type A dissection. (b) Coronal image of NECT chest demonstrating a supracoronary tube graft (arrow).

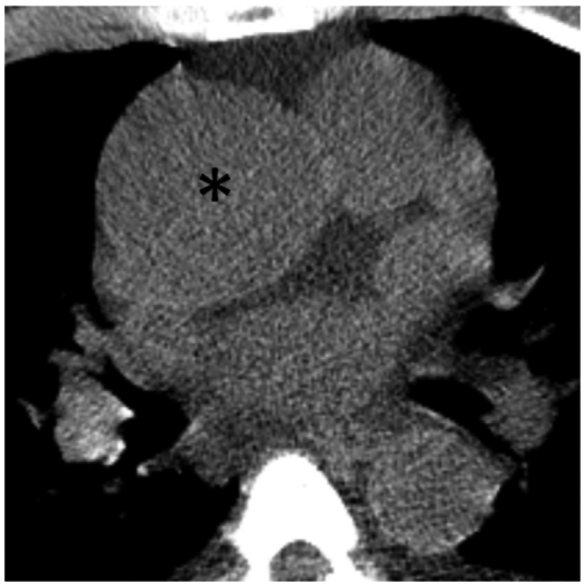

Fig. 5 – Prior from 4 years earlier. Axial image of NECT chest at the level of the ascending aorta demonstrating an ascending aortic aneurysm measuring 6.0 cm (*).

ascending aortic aneurysm because of ADPKD. Dilation of the ascending aorta that does not involve the sinus of Valsalva is termed supravalvular aortic aneurysm. Involvement of the entire aortic root is termed aortic root aneurysm. A supravalvular aortic aneurysm may be treated with the excision of the diseased native aorta and the placement of a supracoronary tube graft. The synthetic graft is typically composed of polyethylene (Dacron) [8]. The operation is technically less difficult and spares the native aortic valves when compared to replacing the entire aortic root [9]. However, the lack of compliance of the polyethylene graft may expose patients to worsening aortic insufficiency and root dilation in the chronic setting [9,10].

It is unclear if our patient's type A dissection originated from iatrogenic causes following the supracoronary tube graft placement for treatment of his aortic insufficiency. The patient is also at high risk because of ADPKD, hypertension, and cocaine use. Nevertheless, our case emphasizes the importance of carefully reviewing a patient's surgical history and nonenhanced imaging studies when a type A dissection is discovered [8].

Our case involves a CTAD distal to the supracoronary tube graft without involvement of the aortic root. It is not possible for the dissection to cross through the distal suture line and the synthetic polyethylene graft to involve the aortic root. Severe complications of cardiac tamponade, severe aortic regurgitation, and aortic rupture are unlikely to occur. The aortic root has been stable in size throughout the 16-month timeframe. Also, there is no dissection extension or static obstruction of the main aortic arch branches. Medical management has been effective in limiting the progression of this disease. This parallels the preferred treatment of type B aortic dissections, where antihypertensive agents reduce hemodynamic stresses on the aortic wall [11].

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