

Aggressive Progression of Penetrating Atheromatous Ulcer of the Descending Thoracic Aorta

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ABSTRACT

The treatment of acute aortic pathologies continues to evolve with enhanced imaging capabilities. This case report highlights the rapid progression of penetrating atherosclerotic ulcer to pseudoaneurysm development and subsequent treatment with thoracic endovascular stent graft.

INTRODUCTION

The treatment of penetrating atherosclerotic ulcer (PAU) disease continues to evolve. PAUs arise when atherosclerotic lesions rupture through the internal elastic lamina of the aortic wall with subsequent hematoma formation between the media and adventitia. This disorder represents one point in the spectrum of aortic pathologies. The exact pathophysiology of penetrating aortic ulcers and the relationship of this disease entity and other causes of acute aortic syndromes have received marginal attention until the last two decades. With the advent of computed tomography (CT), the radiological diagnosis of PAUs has become more prevalent. Therefore, as radiological imaging has improved, so has the recognition of this disease entity. However, although the recognition of PAUs has increased, the clinical management remains controversial due to limited clinical experience. This case report with relevant images represents the potential aggressive progression of this pathology.

CASE REPORT

A 55-year-old man presented to the emergency department with history of acute onset central chest pain radiating to the back. His past medical history was significant for hypertension and chronic renal disease.

A chest x-ray revealed widening of the superior mediastinum and a computed tomogram of the chest revealed an extensive soft tissue density within the wall of the descending

aorta measuring 45 to 65 Hounsfield units consistent with intramural hematoma. A small focal out-pouching along the infero-medial aspect of the descending aorta, consistent with a penetrating ulcer, was identified (Figure 1). There was no obvious intimal flap. A magnetic resonance angiogram demonstrated a small crescent appearing region of abnormal signal intensity consistent with intramural hemorrhage extending from the origin of the left subclavian artery to the aortic bifurcation and into the right common iliac artery.

At this point in time, optimal control of the patient's blood pressure was achieved with anti-hypertensive medication and an imaging study scheduled at 6 weeks follow-up. Magnetic resonance angiographic imaging at follow-up showed the interval development of a large bi-lobed pseudo-aneurysm originating from the poster-inferior aspect of the proximal descending aorta at the site of the previously noted ulcer (Figure 2). The overall caliber of the aorta was 5.3 cm 5.0 cm, and the lobulations measured 3.5 cm 3.1 cm and 2.3 cm 1.9 cm. An extensive intramural hematoma was again noted in the proximal descending thoracic aorta.

The patient underwent an initial left carotid to subclavian artery bypass followed by endovascular repair deploying a Gore TAG® (Gore Medical, Flagstaff, AZ, USA) endovascular stent graft with complete exclusion of the pseudo-aneurysm. A magnetic resonance angiogram carried out at 18 months follow-up revealed complete resolution of the intramural hematoma and the pseudo-aneurysm. In addition, stable position of the stent graft without any evidence of endoleak and a patent left carotid subclavian bypass graft were noted (Figure 3).

DISCUSSION

Intramural hematoma accounts for between 5% and 20% of patients admitted to the hospital with the diagnosis of acute aortic syndrome or acute dissection. The most commonly cited explanation for its occurrence is a rupture of the vasa vasora with intramural hemorrhage. This entity most likely presents as an acute dissection when the intramural component ruptures into the lumen [Sundt 2007].

A retrospective analysis among patients with an initial diagnosis of acute aortic dissection revealed that 7.6% were found to have a penetrating atheromatous aortic ulcer at presentation [Coady 1998]. Penetrating ulcers in the setting of

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an aortic intramural hematoma assume greater prognostic significance when compared to Type A or Type B dissections. They tend to have significantly progressive disease course versus an intramural hematoma alone, which typically run a stable course, especially when limited to the descending thoracic aorta [Ganaha 2002].

An aortic intramural hematoma may undergo no change in appearance over time, or it may show a progressive decrease in thickness, accompanied by a decreasing aortic diameter.

A complete resolution of a hematoma may occur as early as 1 month after the initial diagnosis. However, in view of their variable natural history, it is difficult to predict which of the hematomas would stabilize, regress/resolve, or progress to frank dissection or pseudo-aneurysm formation. In addition, it is difficult to define time frames for these various evolutionary patterns. As a result, long-term surveillance of aortic intramural hematomas is likely prudent even if there is evidence of improvement or complete resolution. In particular, if new symptoms occur or findings at routine surveillance imaging arouse suspicion, more frequent follow-up imaging may be necessary [Chao 2009].

Our report reiterates the need for a stringent follow-up protocol in patients with this intriguing entity and highlights successful long-term outcomes of this aggressive pathology following endovascular intervention.

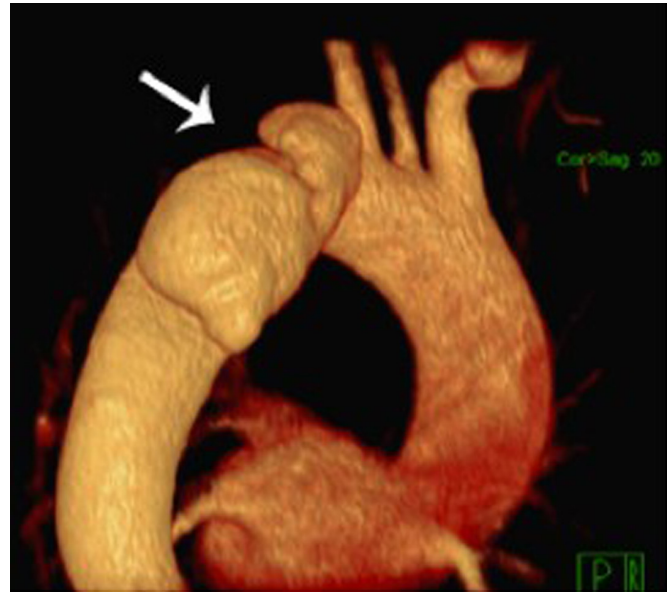


Figure 2. Magnetic resonance angiogram at 6-week follow-up showing development of a bi-lobed aneurysmal dilatation of the proximal descending thoracic aorta (arrow).

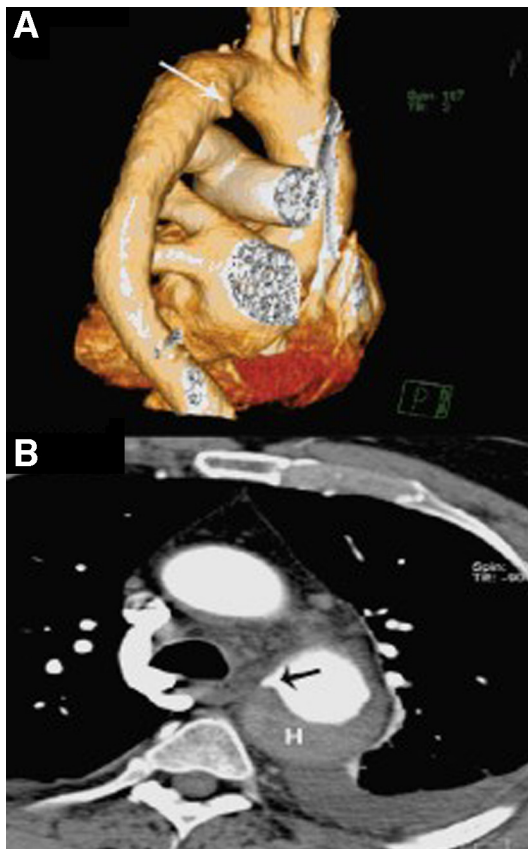


Figure 1. A, Reconstructed computed tomographic (CT) angiogram at admission showing out-pouching of the proximal descending thoracic aorta (arrow). B, Axial CT image demonstrating the out-pouching (solid arrow) and the intramural hematoma (H).

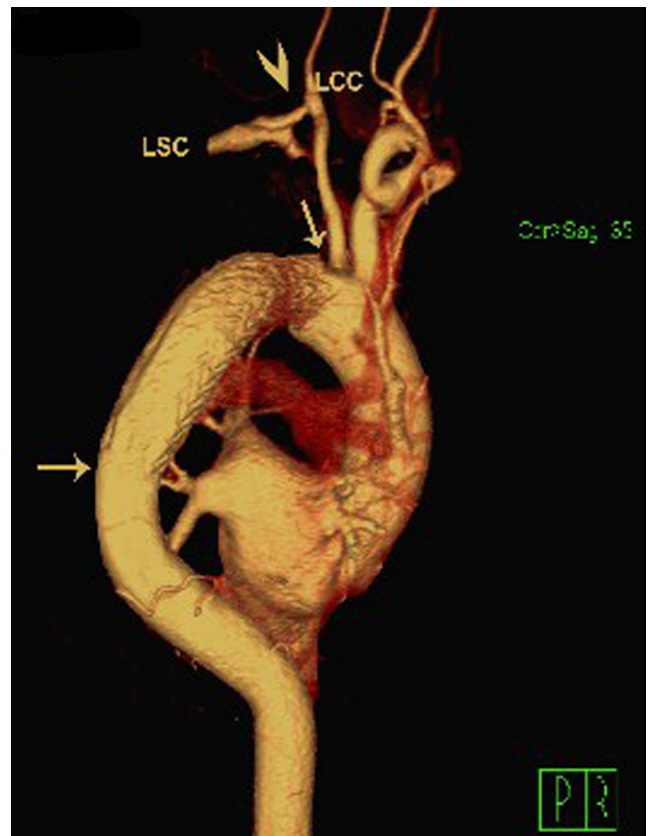


Figure 3. Magnetic resonance angiogram at 18-month follow-up showing stable stent graft (StG) and patent left carotid (LCC) to subclavian (LSC) bypass graft (arrowhead).

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