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Conservatively treated Type B intramural hematoma: Progression into acute aortic dissection followed by spontaneous resolution, assessed by CT

Guadalupe Buitrago, MD; Sabina Vasaturo, MD; and Lucia J. M. Kroft, MD, PhD

This case report shows the full spectrum evolution of type B intramural hematoma under conservative treatment, with initial progression into a true aortic dissection, followed by extremely rare near-complete healing of the dissection at followup.

Introduction

Aortic intramural hematoma (IMH), one of the acute aortic syndromes, implies hematoma formation into the aortic media. The natural evolution of acute IMH continues to be debated. Currently, cases of acute type B IMH (not involving the ascending aorta) are usually treated conservatively; however, progressive disease, including aortic dissection, has been described (1-4). Acute type B aortic dissections are also treated conservatively with antihypertensive medication, and a favorable course with false lumen-size reduction or thrombosis is frequent; however, a complete resolution of the aortic dissection has rarely been reported (5, 6).

Case report

A 78-year-old woman was admitted to the emergency department presenting with acute intense interscapular pain. Given the symptoms and onset, an acute aortic syndrome was suspected. Computed tomography (CT) showed a Stanford type B aortic IMH involving the descending aorta from the left subclavian artery to the diaphragmatic level (Figure, day 1). A small, penetrating, atherosclerotic ulcer (PAU) was evident in the distal descending aorta. No evidence of true aortic dissection was seen. Medical therapy (Labetalol) was initially given with resolution of pain.

CT was repeated for followup. At day 8, the aortic IMH had progressed into a Stanford type B aortic dissection at the level of the distal descending aorta (Figure, day 8). Medical treatment was continued, and the patient remained asymptomatic. After 15 days, the patient was discharged without significant changes on CT (Figure, day 15), and oral antihypertensive medication was continued.

Routine followup CT after 6 months showed complete resolution of the IMH and a remarkable reduction with complete thrombosis of the residual false lumen (Figure, 6 months). A small ulcer-like projection from the true lumen into the thrombosed false lumen was seen at the same level of the initial PAU.

Discussion

Acute aortic syndrome is a life-threatening condition that may include aortic dissection, IMH, or symptomatic PAU. Accurate and prompt diagnosis is required to determine the appropriate management. IMH represents 5 to 20 percent of the acute aortic syndromes (3, 7).

Classic aortic dissection (with dissection in the media) involves a tear in the aortic intima, with a rapid development of an intimal flap separating the true and false lumen. In contrast, IMH implies a hematoma formation in the media of the aortic wall and has no detectable intimal tear (7, 8). Dissection/IMH can be secondary to spontaneous rupture of aortic vasa vasorum, or to rupture induced

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Dr. Buitrago is in the Department of Radiology at the Hospital General Universitario Gregorio Maranon, Madrid, Spain. Drs. Vasaturo and Kroft are in the Department of Radiology at Leiden University Medical Center, Leiden, The Netherlands. Contact Dr. Kroft at L.J.M.Kroft@lumc.nl.

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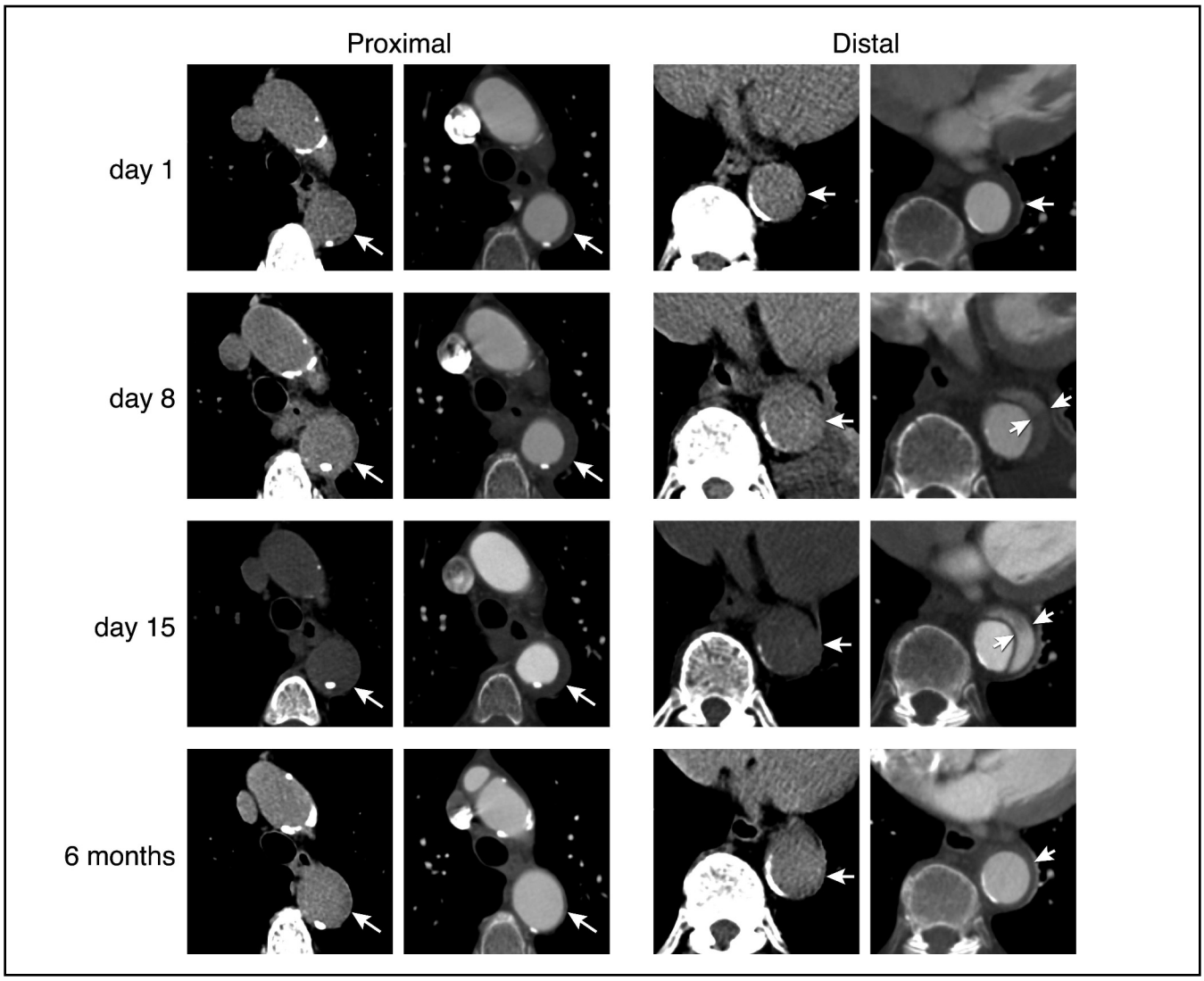

Figure: A 78-year-old woman with acute aortic syndrome presenting with intramural hematoma (IMH), or nonvisible dissection, progressing into true aortic dissection, and finally near-complete healing. Axial CT. Proximal column: descending aorta, proximal level. Distal column: descending aorta, distal level. Noncontrast CT (left) and contrast-enhanced CT angiography (right) images for each column. **Day 1:** Scan performed at admission showing a Stanford type B IMH extending along the descending aorta (arrows). **Day 8:** Progression of the IMH thickness at proximal level and evolution of the IMH into a true aortic dissection at distal level (arrows). **Day 15:** No change of IMH thickness at proximal level, but slight increase of false lumen size at distal level (arrows). **After 6 months:** Complete resolution of the IMH at proximal level, and a remarkable reduction with complete false-lumen thrombosis at distal level (arrows).

by a PAU, as in our case. The differentiation can be made only by imaging techniques and is relevant, as PAU leading to IMH may be associated with a higher rate of progressive disease (aneurysms, aortic rupture, hematoma expansion, or dissection) (9), as was observed in our case.

The clinical manifestation of IMH is similar to acute aortic dissection, with abrupt onset of severe chest pain (IMH affecting the ascending aorta) or back or interscapular pain (IMH affecting the descending aorta, as in our case) (7, 8). Aortic insufficiency, ischemic pain secondary to organ perfusion defects, and pulse deficits are far less common than in aortic dissection (3). Most IMHs are located in the descending aorta (60%), whereas classic aortic dissections affect the ascending aorta more frequently (65%) (3). IMH is usually associated with longstanding hypertension.

The diagnosis of aortic IMH relies on noninvasive imaging studies to demonstrate the aortic abnormality, to distinguish IMH from aortic dissection by exclusion of an intimal flap, and to identify the presence/absence of associated ulcer-like projections. Since chest radiography has a limited sensitivity in the diagnosis of acute aortic syndrome, additional imaging studies are necessary. The mo-

dality of first choice is CT; it is fast, has high sensitivity and specificity, and is widely available in the emergency department setting. CT also depicts involvement of visceral arteries and organs. On plain CT, IMHs are characterized by a high-attenuation, crescent-shaped area within the aortic wall that does not enhance after contrast administration (9). Other possible imaging diagnostic tools are transthoracic and transesophageal echocardiography and magnetic resonance imaging (MRI). Transthoracic echocardiography may be useful to identify proximal ascending aorta involvement, but it is mainly used to assess cardiac complications such as aortic insufficiency, pericardial tamponade, and systolic function. Transesophageal echocardiography allows the assessment of the entire thoracic aorta, but this technique requires esophageal intubation that usually implies sedation that may have adverse hemodynamic effects in unstable patients. MRI is highly accurate for distinguishing different types of acute aortic syndrome but has limited availability in the emergency setting, has limited applicability due to contraindications (for example, patients with pacemakers, aneurysm clips or metallic implants, and claustrophobia), and is hampered by limited access during prolonged scanning. Therefore, MRI is usually reserved as a secondary study when the diagnosis remains uncertain (7, 8).

The natural history of acute IMH is still debated. The progression of IMH to frank aortic dissection has been observed in nearly 16% of patients (3). Others studies suggest that IMH may progress to aortic dissection in as many as 47% (8). The mortality for IMH is similar to that of aortic dissection in both ascending and descending involvement (3, 7). As in aortic dissection, surgical aortic repair is recommended in patients with type A IMH (including ascending aorta), whereas aggressive medical therapy appears justified in descending aorta involvement (7, 10). Endovascular or surgical interventions are reserved for complications of acute type B aortic dissections, such as persistent or recurrent pain, aortic expansion, dissection progression, and end-organ malperfusion syndromes (7, 10). Since our patient remained stable, even after diagnosis of progression into a type B aortic dissection, medical treatment was continued.

The natural evolution of conservatively treated descending aorta dissection may have a favourable course with possible false-lumen thrombosis, although it may also be complicated by aneurismal formation or progression of the dissection (7, 10). However, a complete spontaneous resolution of the aortic dissection has rarely been reported (5, 6). Moreover, to our knowledge, the full spectrum of IMH progression into dissection followed by resolution has not been demonstrated previously.

The exact mechanism of a spontaneous resolution of aortic dissection is unknown, although it has been postulated that thrombus formation in the false lumen leads to obliteration of the false lumen and fibrosis between the two dissected layers (6).

In acute aortic syndrome, serial imaging of the aorta is recommended before discharge and at 3, 6, 9, and 12 months after discharge, even if the patient remains asymptomatic, so as to detect dissection progression, redissection, or aneurysm formation. After that, followup examinations should be performed every 12-24 months if there is no evidence of progression (7).

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