Complete regression of an intramural hematoma of the aorta after distal reperfusion

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A 58-year-old man with hypertension, severe abdominal pain, and pulseless extremities was diagnosed with an isolated abdominal intramural hematoma (IMH). The IMH extended from the distal descending thoracic aorta to just proximal to the renal arteries. β-Blockade treatment resolved the abdominal pain but induced progressive oliguria; decreasing β-blockade treatment increased urine output but caused return of abdominal pain. An axillobifemoral bypass allowed distal perfusion and retrograde visceral artery perfusion while maintaining normal blood pressure. The abdominal pain resolved, urine output increased, and the patient was discharged on day 7. Six months later the patient required an emergent thrombectomy of the axillobifemoral graft and normal antegrade aortic flow was found. A computed tomography scan showed resolution of the IMH. (J Vasc Surg 2005;42:149-52.)

Intramural hematoma (IMH) of the aorta is a dynamic pathological process with the potential to regress or to progress to aneurysmal formation, overt dissection, or aortic rupture. 1-12 The recommended initial therapeutic approach for patients with IMH is similar to that for aortic dissection. Disease involving the ascending aorta (type A) should be considered for urgent surgical intervention, whereas disease involving the descending aorta (type B) can be treated initially with medical management 5, 11, 12, 14 including aggressive antihypertensive therapy and (if appropriate) negative inotropic drugs, typically in an intensive care unit. Surgery is considered for type B IMH when there is evidence of distal malperfusion (with ischemic complications, involving the digestive tract, kidneys, spinal cord, or lower extremities) or progressive aortic enlargement with concern for impending rupture. 5, 6, 8, 10 Refractory hypertension and pain can also be considerations for surgical intervention.

We report a case of a type B IMH in a patient who presented with severe abdominal and back pain and pulseless extremities. Because medical treatment with a β-adrenergic antagonist caused oliguria, the patient was treated with an innovative surgical approach to maintain distal perfusion and retrograde visceral vessel perfusion yet maintain blood pressure at normal levels.

CASE REPORT

On November 21, 2003, a 58-year-old man was transferred from an outside facility with a diagnosis of an abdominal aortic aneurysm. On arrival, the patient was hypertensive (systolic blood pressure of 175/60 mm Hg, peaking at 201/65 mm Hg) and complaining of back and abdominal pain. His laboratory study results were notable for a creatinine level of 1.8 mg/dL (with no previous history of renal insufficiency) and hemoglobin of 12.5 g/dL. He related a 2-week history of lower back pain radiating to the left flank. He had a 40 pack-year history of tobacco use, and his cardiac status was unknown. The patient did not have palpable pulses in either lower extremity.

An emergency contrast computed tomography scan was performed, which showed the findings of an IMH extending from the level of the inferior pulmonary vein to just above the renal arteries. The involved aortic lumen was slit-like with minimal distal perfusion (Fig 1). Because of his severe hypertension, he was started on anti-impulse therapy with a β-adrenergic antagonist, which lowered the blood pressure to 136/65 mm Hg. Within 8 hours of starting the β-adrenergic antagonist, progressive oliguria developed, although the abdominal and back pain had resolved. The patient’s blood pressure was allowed to increase back toward admission levels, however, the resumption of urine production was concomitant with return of the abdominal and flank pain. His creatinine level also increased to 2.7 mg/dL.

Surgical options were discussed, which included replacement of the abdominal aorta with reimplantation of the visceral vessels. We sought to temporarily palliate the situation by restoring distal blood flow with a right axillobifemoral bypass. This decision was
made because we had no knowledge of the patient’s cardiac status or other medical problems as had been transferred from an outside hospital without medical records. Our original intention was to subsequently perform surgical replacement of the aorta at a later date after clinical stabilization and appropriate evaluation of cardiac function and other medical comorbidities. The axillo-bifemoral bypass was performed without complication, and by postoperative day 1 his pain had resolved, he was nonoliguric, his creatinine level had decreased to 1.5 mg/dL, and he had palpable bilateral peripheral pulses in each leg. On the fifth postoperative day, the patient’s serum creatinine level was 0.7 mg/dL, and he was discharged home 2 days later. A magnetic resonance imaging scan just before discharge showed perfusion of all visceral vessels. The patient continued to do well, with a normal serum creatinine level and bilateral palpable leg pulses at 1 and 3 months follow-up. A repeat magnetic resonance imaging scan at 3 months follow-up showed no change in the aortic IMH.

On May 17, 2004, the patient returned to the hospital complaining of acute ischemia of the right arm and leg. The patient was successfully treated with thrombectomy of the axillo-bifemoral bypass graft; however, native aortic blood flow was found. At the time of emergent thrombectomy, the thrombus was found to involve the limb of the right axillary to right femoral artery graft with thrombus extending into both the right axillary artery and the right common femoral artery. A postoperative computed tomography scan confirmed that the abdominal aorta was completely healed and that the graft thrombosis was presumably secondary to competitive flow now present in the native aorto-iliac system (Fig 2). Since discharge, the patient has been followed up at 3-month intervals for a 2-year period. The patient has been free from abdominal or back pain; has normal bilateral femoral, popliteal, dorsalis pedis, and posterior tibial pulses; and has normal renal function. Because of the long-term excellent postoperative result, we and the patient have not considered removal of the axillo-bifemoral bypass graft.

DISCUSSION

Intramural hematoma of the aorta represents a dynamic process, and as such investigators have attempted to classify this disease into subgroups that might aid in prognosis and treatment. One classification system uses the Stanford dissection classification: type A for disease involving the ascending aorta, type B for involvement of the descending
Aorta. Using this classification system is helpful to the clinician regardless of whether IMH is a variant of dissection or is a separate pathologic entity. With this classification scheme, comparison with treatment options for aortic dissection show a similar profile, although the mortality rates for IMH are lower.9,10 In a comparison of type B IMH with type B dissection, Kaji et al showed actuarial survival rates for IMH (n = 53) to be higher than those survival rates for dissection patients (n = 57) followed up for 5 years (100%, 97%, and 97% vs 83%, 79%, and 79% at 1, 2, and 5 years respectively, P = .009).9

Several investigators have attempted to define the pathogenesis of IMH of the aorta; however, this remains a somewhat controversial subject. Cambria, in a short review article in 2002, suggested that IMH is typically seen in the setting of extensive atherosclerotic degeneration of the thoracic aorta with subsequent intimomedial disruption and subsequent dissection of the hematoma either prograde or antegrade (for varying distances). This produces the typical radiographic feature of a crescent-like (or circumferential) ring in the aortic wall, which may or may not be accompanied by an ulcer-like projection (or localized dissection) on the imaging study.13 This hypothesis is in contrast to those formulated many years ago that theorized than an aortic IMH may either occur as a primary event in hypertensive patients in whom there is spontaneous bleeding from vasa vasorum into the media, or be caused by a penetrating atherosclerotic ulcer.13

Spontaneous regression of an IMH of the descending aorta has been documented with varying time courses. Ohmi et al describe a patient who experienced two separate episodes of IMH 10 months apart.1 In the first episode, the hematoma involved the descending aorta and regressed within 1 month. In the second episode, the hematoma involved the ascending and transverse arch and resolved within 24 hours. Both episodes were treated medically, although in the latter case the patient was being prepared for operative intervention because of the anatomic location of the hematoma. In a second study, the same investigators described the long-term follow-up of 94 patients with the diagnosis of IMH from their institution. Type B IMH (n = 53) was considered for medical treatment because only one patient was initially treated surgically.2 Evangelista et al noted that aortic IMH (thoracic or thoracoabdominal) regressed without dilatation in 34% of their patients.5 More recently, Sueyoshi et al have provided a more definitive analysis of the predictive factors for patients with type B IMH. Sueyoshi et al followed up 35 patients with type B IMH over a 17-year period. They described two factors that were associated with progression of the IMH, a maximum aortic diameter greater than 40 mm and an aortic diameter greater than 40 mm and an aortic diameter greater than 40 mm and an aortic diameter greater than 40 mm and an aortic diameter greater than 40 mm and an aortic diameter greater than 40 mm and an aortic
thickness greater than 1 cm. Patients with an aortic diameter of 40 mm or more had a 30-fold higher incidence of progression. Patients with an aortic diameter greater than 1 cm had a nine-fold increase in progression. Our patient had an aortic diameter of 31 mm and 7 mm of aortic wall thickening. Interestingly, of the 20 patients without evidence of progression of the IMH, 60% had regression of the IMH and aortic diameter and thickness.

Treatment of patients with IMH limited to the descending aorta remains unsettled. In a review of 24 patients from the Cleveland Clinic, 3 patients (13%) had symptoms of acute ischemia, for which two patients required surgical intervention. The third patient was not treated surgically because the patient had presented with paraplegia. Of the other 21 patients without acute complications, only one patient had confirmed long-term resolution of the IMH. Tittle et al also showed that intramural hematomas have a propensity to rupture, either at the initial presentation or late.

In a larger series of 50 patients followed up prospectively, 15 patients had an observed regression of the IMH. These were patients who either were judged not to be surgical candidates or did not have evidence of distal poor perfusion. Nine of the patients in this series had an IMH limited to the abdominal aorta; unfortunately, this article does not mention the natural history of this subset of patients. It is quite possible that if left alone, our patient may have experienced regression of the IMH without surgical intervention. However, his poor distal perfusion, as evidenced by abdominal pain, increasing serum creatinine, and progressive oliguria, probably would have led to significant morbidity and possible mortality.

REFERENCES


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