Long-term behavior of aortic intramural hematomas and penetrating ulcers

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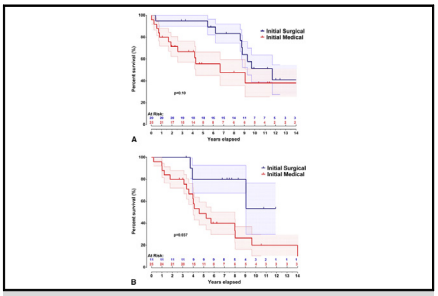
ABSTRACT

Objective: For intramural hematoma and penetrating atherosclerotic ulcer, long-term behavior and treatment are controversial. This study evaluates the long-term behavior of intramural hematoma and penetrating atherosclerotic ulcer, including radiologic follow-up and survival analysis.

Methods: Between 1995 and 2014, 108 patients (mean age, 70.8 ± 10 years; 56% female) presented with intramural hematoma or penetrating atherosclerotic ulcer to Yale-New Haven Hospital (New Haven, Conn). We reviewed the medical records, radiology, and online mortality databases.

Results: Ten of 55 patients (18%) with intramural hematoma and 17 of 53 patients (32%) with penetrating atherosclerotic ulcer had rupture state symptoms on admission, both greater than type A (8%) or type B dissection (4%) ($P < .001$). No branch vascular occlusion occurred. For patients with intramural hematoma with follow-up imaging, 8 of 14 (57%) worsened (mean follow-up, 9.4 months) and 6 (43%) underwent late surgery. For patients with penetrating atherosclerotic ulcer with follow-up imaging, 6 of 20 (30%) worsened and underwent late surgery, and 11 (55%) showed no change (mean follow-up, 34.3 months). Overall survivals were 77%, 70%, 58%, and 33% at 1, 3, 5, and 10 years, respectively. No operative deaths occurred for patients with nonrupture state. Patients with penetrating atherosclerotic ulcer with initial surgical treatment had better long-term survival than patients treated medically ($P = .037$). In the intramural hematoma group, no such difference was observed ($P = .10$).

Conclusions: At presentation, the incidence of early rupture of intramural hematoma and penetrating atherosclerotic ulcer was higher than for typical dissection. For branch vessels, intramural hematoma never occludes branch arteries. On imaging follow-up, patients with intramural hematoma and penetrating atherosclerotic ulcer rarely improved, with late surgery commonly needed. Better survival was observed for the initial surgical management of patients with penetrating atherosclerotic ulcer compared with initial medical management. (J Thorac Cardiovasc Surg 2016;151:361-73)



Kaplan–Meier survivals comparing treatment of patients with nonrupture state. A, IMH. B, PAU.

Central Message

IMH and PAU lesions rarely resolve, and surgery effectively restores survival. A more aggressive approach may be warranted.

Perspective

The long-term behavior and treatment of IMH and PAU are controversial. On the basis of substantial rates of worsening and late surgery, low operative mortality, and improvements in actuarial survival, we advocate a more aggressive surgical approach to these lesions. For experienced clinicians, the threshold toward surgical intervention should be lowered as evidence suggests that surgery is safe and effective.

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Read at the 95th Annual Meeting of The American Association for Thoracic Surgery, Seattle, Washington, April 25-29, 2015.

Received for publication April 27, 2015; revisions received Aug 22, 2015; accepted for publication Sept 3, 2015; available ahead of print Oct 20, 2015.

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0022-5223/$36.00

http://dx.doi.org/10.1016/j.jtcvs.2015.09.012

Supplemental material is available online.

Intramural hematoma (IMH) and penetrating atherosclerotic ulcer (PAU) are pathologic variants of classic aortic dissection that are distinct in their presentation, natural history, and prognosis.[1] Unlike classic dissection, IMH and PAU do not present with an intimal flap or with flow communication; instead, they are often described as lesions

Abbreviations and Acronyms

CT = computed tomography
ILVA = isolated left vertebral artery
IMH = intramural hematoma
MRI = magnetic resonance imaging
PAU = penetrating atherosclerotic ulcer
TEVAR = thoracic endovascular aortic repair

contained within the aortic wall. Penetrating ulcers are characterized by interruption of the intima of the aortic wall, with penetration into the media, whereas IMHs are characterized by hematoma contained within the aortic wall.[2,3]

IMH and PAU are typically diagnosed via computed tomography (CT) or magnetic resonance imaging (MRI).[4] IMH is thought to represent a rupture of the vasa vasorum into the media that results in a crescentic thickening of the aortic wall; it is differentiated from classic dissection by an intact intima that precludes flow communication.[2,3] Penetrating ulcers, as described by Cho and colleagues,[5] are characterized as ulcerations (commonly in an atheromatous plaque) involving intimal and partial medial disruption and possible formation of a hematoma surrounding the projection.

Both IMH and PAU are known to have complex, varied courses. It is well known that these lesions can resolve spontaneously, remain stable, or progress to aneurysm, classic dissection, or rupture.[6] Despite an increasing amount of literature reports, debate persists regarding the course and optimal treatment of IMH and PAU, summarized in Table 1.[3,4,16,17] Supporters of initial nonsurgical treatment cite data indicating that IMHs resolve readily and do not require aggressive surgical treatment, even when located in the ascending aorta.[7,8,10,12-14,18] However, conflicting results, including a prior study from this center, point to infrequent healing and a subsequent need for surgical repair.[6,11,19] Likewise, some retrospective studies on PAU demonstrate low rates of worsening and adverse events over time, permitting nonsurgical treatment, whereas other studies find high rates of radiographic progression, encouraging a lower threshold for early surgical repair.[5,6,15] These conflicting data are further complicated by a paucity of long-term follow-up of IMHs and PAUs.

In a previous report, we presented midterm follow-up results.[6] We extend this follow-up with an aim at elucidating the long-term natural history and progression of IMHs and penetrating ulcers. We focus on lesion progression and subsequent management, providing both presurgical and postsurgical follow-up with the intent to inform decision-making regarding these acute aortic syndromes.

MATERIALS AND METHODS

Patient Profile

From the Yale Aortic Institute database of patients (New Haven, Conn) with acute aortic syndromes, we identified a total of 108 patients with IMH (n = 55) and PAU (n = 53). All presented acutely (with chest pain and syncope) and were treated at Yale-New Haven Hospital (New Haven, Conn) between June 1995 and February 2014. Records for the study population were retrospectively reviewed and consisted of chart and electronic medical records, imaging records, telephone call follow-up, and Social Security Death Index mortality analysis.

We examined symptoms at initial presentation, demographic data, comorbidities, hospital course, treatment, interventions (if any), in-hospital mortality, late mortality, and other pertinent clinical follow-up. Radiologic analysis included review of angiogram, CT, and MRI and records for characterization of initial pathologic entity and subsequent radiographic course, both before and after intervention, to determine disease progression. Patient mortality was determined via hospital records and Social Security Death Index. Hospital discharge summaries and state death certificates were used to ascertain causes of death when possible. Follow-up was 100% complete. The study was approved by the Institutional Review Board of Yale University.

Long-term Radiology Follow-up Guidelines

Clinical and radiologic criteria were used to characterize lesions as follows:

- Patients were classified by their initial diagnosis (IMH or PAU) on earliest available imaging. If transition to another lesion type or to typical dissection occurs in later follow-up, this progression is recorded, but the original diagnosis is maintained for analysis.
- IMH is characterized by crescentic or circular shadowing and expansion within the aortic wall and is differentiated from dissection by the lack of an intimal flap. IMH is confirmed by contrast and noncontrast CT or MRI, when available, to demonstrate lack of flow communication. Patients with an ascending IMH that extended through the arch or descending aorta were considered as part of the ascending cohort.[20]
- Penetrating ulcers are identified as contract opacified projections into the medial wall, often with a mushroom-like appearance. PAUs surrounded by localized IMH were classified as PAU, reflecting our view that it is more likely that the PAU resulted in the located hematoma, rather than the alternative. In cases difficult to classify, the senior author directly reviewed the images to optimize classification.[20]
- Rupture state indicates rupture or impending rupture symptoms. Rupture was determined by the presence of extra-aortic blood confirmed by radiology, surgical examination, or postmortem examination; impending rupture was determined largely on the basis of radiologic findings (bloody or increasing pleural effusion, severe radiologic worsening of the aortic contour), clinical behavior (persistent pain, despite medical treatment), or intraoperative findings of impending rupture (periaortic hematoma without frank rupture, extreme wall thinning).

Progression of our observed pathologies was classified into 3 groups as follows (Figure 1):

- Decrease in size or disappearance of hematoma or ulcer was recorded as "resolution."
- "Worsening" refers to deterioration in aortic condition, including significant increases in the thickness or depth of the lesion, progression of PAU to IMH (or vice versa), progression to classic dissection, or rupture.
- Patients whose lesions did not improve or worsen significantly were noted to be "stable."

We also examined postsurgical imaging findings of repaired aortas. Notations and imaging evidence of aortic instability, including formation of aneurysm, pseudoaneurysm, dissection, endoleak, or contrast