Early and late outcomes of acute type A aortic dissection with intramural hematoma

Anthony L. Estrera, MD, FACS, Harleen K. Sandhu, MD, MPH, Samuel S. Leake, BS, Kristofer M. Charlton-Ouw, MD, FACS, Rana O. Afifi, MD, Charles C. Miller III, PhD, and Hazim J. Safi, MD, FACS

Introduction: Controversy remains regarding management of acute type A dissection with intramural hematoma (IMH). Our purpose was to analyze our experience and report outcomes after repair of acute type A aortic dissection with IMH.

Methods: We analyzed all patients from a single center who underwent open repair for acute type A aortic dissection with IMH.

Results: Between 2000 and 2013, we performed 418 repairs for acute type A aortic dissection. These were divided into 2 groups of patients: 64 patients (15%) with type A IMH and 354 patients (85%) with typical dissection. Those with IMH were older (62.4 ± 13.9 years vs 56.7 ± 14.7 years; $P < .0046$) and presented with reduced renal function (ie, glomerular filtration rate) ($P < .0341$), less frequently with distal malperfusion, and less frequently with rupture ($P < .0116$). With IMH, the time from presentation to repair was, by strategy, longer (median, 67 vs 6 hours; $P < .0001$), but no mortality occurred within 3 days of presentation. Mortality with IMH did not differ from typical dissection: 7 out of 64 patients (10.9%) versus 52 out of 354 patients (14.7%; $P = .4276$). A lower incidence of postoperative dialysis in the IMH group approached significance: 6 out of 63 patients (9.5%) versus 64 out of 347 patients (18.4%; $P = .0820$). When adjusted for age and renal function, late survival was improved with IMH ($P < .0343$).

Conclusions: Repair of acute type A aortic dissection with IMH is associated with significant early morbidity and mortality, differing minimally from typical aortic dissection. Although expectant repair within 3 days may be applied, the purposeful delay imparted little advantage. Improved late outcomes may be seen with IMH, but continued long-term surveillance is required for verification. (J Thorac Cardiovasc Surg 2015;149:137-42)

See related commentary on page 143.

Intramural hematoma (IMH), first described as ''aortic dissection without intimal flap'' by Krukenberg in 1920,[1] remains a therapeutic challenge. It is generally agreed that the management of descending thoracic, or Stanford type B, IMH is primarily medical, but the optimal treatment of ascending, or Stanford type A, IMH remains unclear.[2-7] Recent guidelines from the Society of Thoracic Surgeons recommend that type A IMH be ''reasonably treated with surgical intervention'' and that medical management only be applied to patients considered too high risk for open repair, under the conditions that they remain asymptomatic and radiographically stable.[8] However, some Asian series have demonstrated excellent early outcomes with medical management alone.[9-12]

We previously published our experience with acute type A IMH in 36 patients who underwent immediate surgical repair, delayed (expectant) repair, and medical management.[13] In this analysis, we also analyzed the hazard of conversion of the IMH to typical dissection in relation to time of presentation. We identified that 33% of cases converted to typical dissection by the time of open repair and that the risk of conversion was greatest at 8 days. Considering that the risk was low for early conversion, we adopted an approach to perform delayed open repair of acute type A IMH within 3 days of presentation, with the reasoning that delayed management might impart an advantage, as had been observed with traumatic aortic injury.[14] Thus, the purpose of our study was to analyze our experience with open repair for acute type A IMH and report early and late outcomes.

From the Department of Cardiothoracic and Vascular Surgery, Clinical Science Program, The University of Texas Medical School at Houston, and Memorial Hermann Hospital, Houston, Tex.

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Address for reprints: Anthony L. Estrera, MD, FACS, Department of Cardiothoracic and Vascular Surgery, The University of Texas Medical School at Houston, 6400 Fannin St, Ste 2850, Houston, TX 77030 (E-mail: Anthony.L.Estrera@uth.tmc.edu).

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Abbreviations and Acronyms
GFR = glomerular infiltration rate
IMH = intramural hematoma

METHODS

The Committee for Protection of Human Subjects for the University of Texas Medical School at Houston, the local institutional review board, approved this study and consent was waived.

Definitions

Type A IMH was defined as aortic dissection without identifiable intimal tear and lack of flow in the false lumen of the ascending aorta.[15] Identification of type A IMH was first obtained by computed tomography and confirmation that a dissection flap was not present was made by transesophageal echocardiography. Patients with identifiable flow in the false lumen of the descending and thoracoabdominal aorta were included for analysis. Acute dissection was defined as dissection occurring within 2 weeks of presentation. Conversion to typical dissection was determined at either operative examination or radiographic evaluation before surgery. The time of the initial onset of pain (in hours) determined the onset of IMH. Time of symptom onset is defined by the time identified by patient history as the first instance of symptoms. Time to operative repair was defined as time from onset of pain to operative repair. Typical dissection was dissection with any free flow into the false channel. Early mortality included in-hospital death and death occurring within 30 days of surgery. Stroke was defined as any gross focal neurologic brain injury, either temporary or permanent, identified on neurologic examination by a neurology consultant and confirmed with computed tomography or magnetic resonance imaging. The glomerular filtration rate (GFR) was calculated by the Cockcroft-Gault method.[16] Respiratory dysfunction was defined as prolonged intubation >24 hours, reintubation, or need for tracheostomy. Reoperation was defined as the need for any surgical procedure, including re-exploration for bleeding, an abdominal procedure, tracheostomy, or an aortovascular procedure. Hypotension was defined as systolic blood pressure <90 mm Hg, significant aortic insufficiency was defined as moderate to severe grade, and aortic rupture was defined as the presence of blood in the pericardial sac with tamponade. Malperfusion syndrome included any clinically evident organ malperfusion; for example, cerebral, spinal, visceral, renal, and limb.

Surgical Approach

All cases of typical dissection underwent urgent or emergent repair unless contraindicated for neurologic devastation, metastatic cancer, or patient refusal. Nonoperative cases were excluded. For patients with IMH, operative repair was performed emergently for patients in extremis and expectantly (eventual) in patients whose symptoms were controlled and whose vital signs were stable. Surgical management used cardiopulmonary bypass, deep hypothermic circulatory arrest, and retrograde cerebral perfusion, as described previously.[17] Cardiopulmonary bypass was routinely initiated via femoral or axillary artery cannulation and axillary artery cannulation was used in cases of femoral artery pulse deficit. Adequate arterial inflow perfusion was confirmed by monitoring cerebral oximetry and appropriate decreases in nasopharyngeal and bladder temperatures. Venous cannulation was via the superior and inferior vena cava. Both antegrade and retrograde cold blood cardioplegia provided myocardial protection. A left ventricular sump graft catheter was inserted into the right superior pulmonary vein. Once adequate cooling was achieved, cardiopulmonary bypass was discontinued and the circulation was arrested. Retrograde cerebral perfusion was started via the superior vena cava cannula. The patient was placed in slight Trendelenburg position. The ascending aorta was then opened and inspected. If a tear was identified, it was resected. The arch was replaced only if enlarged >5 cm; partial arch replacement (hemiarch) with an open distal anastomosis was performed otherwise. Any remaining thrombus within the wall was removed before performing the anastomosis. Once the anastomosis was completed, the graft was deaired and clamped, and antegrade perfusion was initiated via a sidearm branch of the aortic graft. After systemic warming was begun, the proximal reconstruction was performed. The remaining ascending aorta was resected to the sinotubular junction. If the aortic valve was intact and the aortic root was not enlarged >5 cm or compromised, the proximal anastomosis was performed after resuspension of the aortic valve.

Statistical Methods

Data were collected from chart reviews done by a trained nurse evaluator and were entered into a dedicated Microsoft Access database (Microsoft Corp, Redmond, Wash). Analysis was retrospective. Patient follow-up was obtained by direct patient contact, telephone interview, or the National Death Index and was complete for mortality and interventions. Data were managed under Health Insurance Portability and Accountability Act confidentiality guidelines in an Access database with encrypted patient identifiers.

Normally distributed continuous variables were compared by unpaired t test and are presented as mean ± standard deviation. Transfusion products, which arise from a zero-inflated skewed distribution, and other continuous variables not meeting normal assumptions are presented as median and interquartile range. Statistical tests for nonnormal continuous data were conducted by Wilcoxon rank-sum procedures. Dichotomous variables were analyzed by contingency tables methods, with χ^2 P values reported unless any expected cell value was less than 5, in which case Fisher exact P value is reported. Mortality hazard for the first 30 days (Figure 1) was computed using life table methods. Hazard was defined as the instantaneous probability of occurrence for each life table interval.[18] Long-term (10-year) (Figure 2) mortality predictors were identified using Cox proportional-hazards regression analysis. A formal test of the long-term proportional hazards assumption was performed. Figure 2 represents direct-adjusted survival functions from a Cox model, including terms for IMH, estimated GFR, and age. All computations were performed using SAS version 9.3 (SAS Institute Inc, Cary, NC). Long-term survival was compared between groups using Kaplan-Meier analysis.

RESULTS

Patients

Between 2000 and 2013, we performed 418 repairs for acute type A aortic dissection. Of this cohort, 15% (64 out of 418 patients) had associated IMH and 85% (354 out of 418 patients) had typical type A dissection. Patients with IMH were older than those with typical dissection (62.4 ± 13.9 vs 56.7 ± 14.7 years), with slightly more women (37.5% [24 out of 64 patients] vs 26.8% [95 out of 354 patients]; $P = .0819$). Patients with IMH presented less frequently with shortness of breath, malperfusion syndromes, pulse deficits, significant aortic insufficiency, hypotension, and rupture (Table 1). Cannulation strategy did not differ between IMH and typical dissection nor did aortic root replacement or complete transverse arch replacement (Table 1). In the IMH group, cardiopulmonary bypass time, aortic crossclamp time, and retrograde cerebral perfusion times were shorter when compared with the typical type A repair group (Table 2). Intraoperative blood use did not differ between groups (Table 2).