*Case Series*

# Open Repair of Acute Type A Intramural Hematoma in 3 Patients

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*Acute aortic syndrome encompasses classic aortic dissection and less common aortic phenomena, including intramural hematoma (IMH), a hemorrhage within the aortic media that occurs without a discrete intimal tear. We reviewed our experience with treating acute type A IMH to better understand this acute aortic syndrome. A review of our clinical database identified 1,902 proximal aortic repairs that were performed from January 2006 through December 2018; of these, 266 were for acute aortic syndrome, including 3 (1.1%) for acute type A IMH. Operative technique varied considerably. All IMH repairs involved hemiarch or total arch replacement. In all 3 patients, the IMH extended distally into the descending thoracic aorta. There were no operative deaths or major adverse events (stroke, paraplegia, paraparesis, or renal failure necessitating dialysis) that persisted to hospital discharge. Length of hospitalization ranged from 5 to 20 days. All 3 patients were alive at follow-up (range, 2-6 yr) and needed no aortic reintervention after their index or staged repairs. In our experience, repair of acute type A IMH was infrequent and could be either simple or complex. Despite our limited experience with this disease, we found that it can be repaired successfully in urgent and emergency cases. Following treatment guidelines for aortic dissection appears to be a reasonable strategy for treating IMH.* ***(Tex Heart Inst J 2020;47(4):290-7)***

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Acute aortic syndrome (AAS) encompasses a spectrum of life-threatening vascular conditions that necessitate emergency medical or surgical intervention.[1-3] Thus, AAS includes classic aortic dissection (AD), intramural hematoma (IMH), and penetrating aortic ulcer (PAU) (Fig. 1). Krukenberg[4] first described IMH in 1920 as "dissection without intimal tear" and attributed it to spontaneous hemorrhage of the vasa vasorum with subsequent crescentic or circumferential mural thickening. The Stanford system for AD classifies IMH as type A (ascending aorta involved) or type B (ascending aorta not involved).[5] Although some centers have reported that type A IMH constitutes as much as 22% to 44% of AAS cases,[6-9] others report lower proportions ranging from 4% to 15%.[10-13] This variation tends to fall along geographic lines; IMH reportedly represents a larger proportion of AAS in Asia than it does elsewhere.[14-16]

Overall, relatively little is known about how acute type A IMH should be treated; studies tend to range from dozens of cases to the low hundreds, so the available data are limited.[6,9,10,13,16-21] How to optimally manage this condition remains controversial.[9,10,16,18,19] Contemporary United States guidelines suggest that urgent surgical repair for acute type A IMH is reasonable; European guidelines also support this approach in most situations.[22-24] Nonetheless, some groups continue to favor a more conservative approach: initial medical management and diligent monitoring of stable patients, with surgery only for those who develop complications.[6-8,25] Thus, managing this condition

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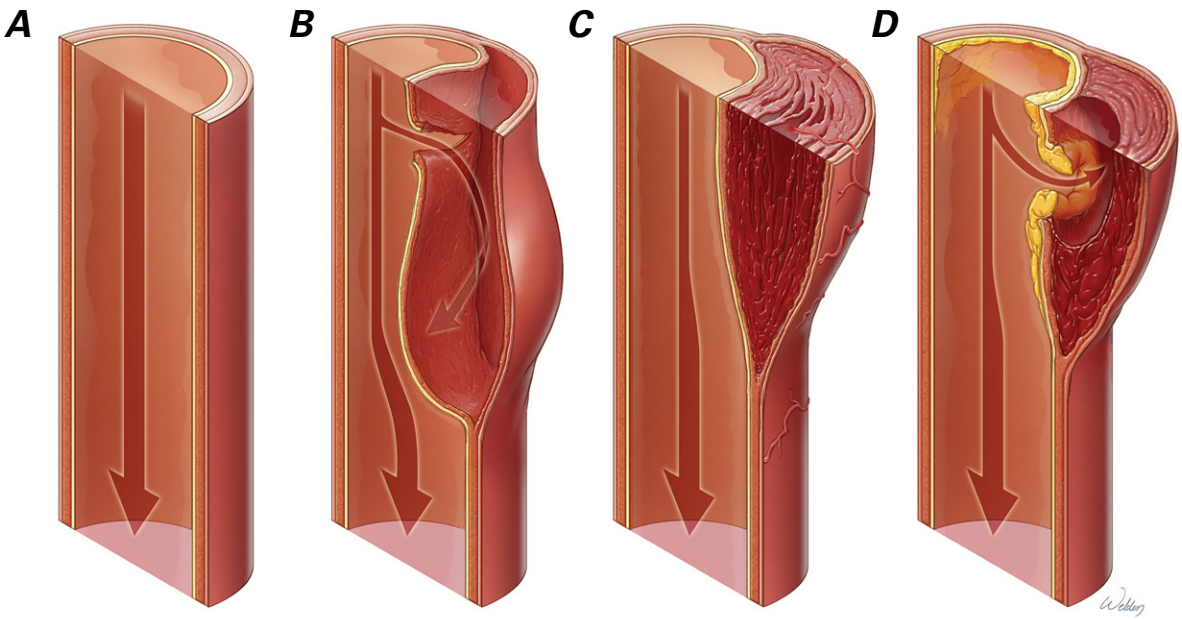


***Fig. 1*** *Illustration shows longitudinal sections of the aortic lumen and wall in acute aortic syndromes.* ***A****) In the normal aorta, blood flows freely downstream.* ***B****) In classic aortic dissection, blood passes into the media through an intimal tear to create a false lumen in the aortic wall.* ***C****) In intramural hematoma, the intima remains intact but blood collects within the media because of hemorrhage inside the wall.* ***D****) In penetrating aortic ulcer, deep atherosclerotic lesions burrow into the aortic wall and enable blood to pass into the media.*

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remains a moving target. We report our single-center experience treating type A IMH with early open surgical repair.

## Patients and Methods

The institutional review board of Baylor College of Medicine approved our clinical research protocol. Informed consent had been obtained whenever possible; waiver of consent was approved for patients who were too ill to provide consent and had no family members who could provide it on their behalf. Data were obtained from a clinical database of aortic operations performed at Baylor–St. Luke's Medical Center, a tertiary referral center in Houston, Texas.

Our study focused solely on patients who had IMH at the time of operation. From January 2006 through December 2018, 1,902 consecutive proximal aortic repairs (repair of the aortic root, ascending aorta, or aortic arch) were performed, including 266 (14%) for AAS. From these, we identified 26 patients whose outside medical records, imaging findings, or both suggested IMH. Initial diagnostic criteria typically consisted of crescentic or circumferential mural thickening within the ascending aorta in the absence of intimal tear or detectable blood flow within the false lumen; patients with classic AD or PAU as shown on computed tomographic (CT) images were excluded. Patient data, along with supplemental medical records, were reviewed by a senior cardiothoracic surgeon (SAL) to determine whether IMH was present at the time of repair. Confirmatory criteria were based on intraoperative findings detailed in the operative report. On review, 23 of the 26 patients proved not to have IMH, having instead classic AD (n=19) or PAU (n=2) identified on images or intraoperatively, or unclear pathology (n=2). Thus, only 3 (1.1%) patients met repair-based criteria for type A IMH.

### Analysis, Study Definitions, and Follow-Up

We examined the patients' preoperative characteristics, operative details, and early and late outcomes. All repairs were nonelective. Emergency repair was performed within 24 hours of transfer to our hospital, whereas urgent repair was performed after 24 hours. Adverse event was defined as operative death (in-hospital or 30-day death) or persistent (still evident at the time of hospital discharge) stroke, paraplegia, paraparesis, or renal failure necessitating dialysis. The overall hospital length of stay (LOS) was defined as the time from repair until hospital discharge. All patients were followed up by telephone; as needed, medical records were reviewed to determine whether they included reports of late aortic events.

### Surgical Technique

All IMH repairs involved replacing at least a portion of the transverse aortic arch and were performed through median sternotomy under moderate hypothermic circulatory arrest (HCA) with antegrade cerebral perfusion (ACP). The site of arterial cannulation varied depending on patient-specific factors such as any disease directly

**TABLE I.** Preoperative Characteristics of Patients With Type A Intramural Hematoma

| Variable* | Patient 1 | Patient 2 | Patient 3 |
|---|---|---|---|
| Age (yr) | 71 | 64 | 74 |
| Sex | Male | Male | Female |
| Maximum proximal aortic diameter (cm) | 5.0 | 3.7 | 3.1 |
| Maximum distal aortic diameter (cm) | 6.6 | 3.1 | 2.9 |
| Aortic valve regurgitation ≥3+ | No | Yes | No |
| Comorbidities | Hypertension | Hypertension | Hypertension |
| | Hyperlipidemia | Hyperlipidemia | Hyperlipidemia |
| | Pulmonary disease | | Peripheral vascular disease |
| | Tobacco use | | |
| Prior open aortic repair | No | No | Yes |

*All patients presented with acute symptoms

affecting branching arteries. Conduit grafts were used to facilitate cannulation, which is common practice. For total transverse aortic arch repairs, the brachiocephalic arteries were rerouted with small-diameter grafts (for example, a Y-graft approach).[26] The type of graft used (for example, straight, curved, collared, or with neosinuses) varied by extent of repair and other patient-specific factors. Cerebral perfusion was monitored with use of near-infrared spectroscopy. Additional repair of the aortic valve or root was performed when indicated by intraoperative findings.

## Results

### Preoperative Patient Characteristics

Preoperative characteristics of the 3 patients (age range, 64–74 yr; 2 men) are provided in Table I. All had a history of hypertension and hyperlipidemia, but few comorbidities overall. None had Marfan syndrome. All patients presented with acute symptoms at other facilities and were transferred to our center. No patient presented with cardiac tamponade or aortic rupture. Imaging indicated that IMH in all 3 patients extended from the proximal aorta to the distal aorta (descending thoracic, thoracoabdominal, or abdominal aorta).

### Preoperative Details

Patient 1 presented at another hospital with back pain and acute bilateral lower extremity weakness. The patient had a descending thoracic aortic aneurysm and was being monitored under an imaging surveillance protocol. A CT image showed diffuse aneurysmal dilation of the ascending and descending thoracic aorta with crescentic intramural density surrounding the entire ascending and descending thoracic aorta (Fig. 2).

Patient 2 presented at another hospital with chest pain 1 day after taking an over-the-counter medication for erectile dysfunction. An echocardiogram indicated moderate-to-severe aortic valve regurgitation and suggested AD with thrombosis or IMH. Repeated CT scans (Fig. 3) suggested rapidly progressing aortitis or AD. On day 3 of symptoms, the patient was transferred to our center, CT images were reevaluated, and IMH was identified and confirmed during operation.

Patient 3 presented at another hospital with chest pain radiating to her lower back, as well as epigastric and lower-neck pain. Computed tomographic images suggested an acute aortic event with IMH (Fig. 4). The patient was transferred to our center, where further evaluation confirmed IMH. At our center, more than 4 years earlier, the patient had undergone a Crawford extent IV thoracoabdominal aortic aneurysm repair with a 4-branched graft.

### Operative Details

All 3 patients underwent nonelective repair (2 emergency, 1 urgent) (Table II) after transfer to our center. Repairs varied substantially in complexity; aortic cross-clamp time ranged from 27 to 313 minutes.

Patient 1's ascending aorta and total transverse aortic arch were replaced during moderate HCA (22.6 °C), and bilateral ACP was provided through the brachiocephalic and left common carotid arteries. A 26-mm collared ET graft was used with a single Y-graft approach to reroute the brachiocephalic and left common carotid arteries using 14- and 8-mm grafts, respectively; the left subclavian artery was not debranched and remained in the distal arch. At the anatomic location of the left common carotid artery, the distal anastomosis was made, with the collar used to accommodate the size difference between the graft and the aortic arch and with the ET extending distally. A large left pleural effusion was drained before closing.

Patient 2's ascending aorta was resected during the cooling process, and a tissue sample was sent for pathologic analysis to rule out aortitis. The aortic valve exhib-

ited several large fenestrations, as well as leaflet prolapse; it was replaced with a 23-mm stented bioprosthetic valve. During a brief period of moderate HCA (23.8 °C), the hemiarch was replaced while bilateral ACP was provided through the brachiocephalic and left common carotid arteries. The proximal portion of the 26-mm curved graft was sutured to the sinotubular junction. Coagulopathy was treated with blood products, but bleeding was noted within the aortic root itself and the left atrium. The bioprosthetic valve was removed. The defect in the left atrial dome was repaired with a pericardial patch. The sinuses of the aortic root were friable and had a small perforation. Therefore, the sinuses were removed, and the coronary arteries were mobilized on buttons of tissue. A composite valve graft (CVG) to replace the aortic root was prepared tableside, with use of another 23-mm stented bioprosthetic valve and a 28-mm graft with neosinuses. The mobilized left coronary artery was reattached as a button to the CVG. Then, the distal aspect of the CVG was anastomosed to the previously replaced ascending aortic graft. The right coronary artery was reattached as a button to the CVG to complete the repair.

Patient 3 underwent a brief period of moderate HCA (24.1 °C) with bilateral ACP through the right axillary and left common carotid arteries, and a 24-mm curved graft was used to replace the lesser curvature of the transverse aortic arch. Mild aortic regurgitation was observed; the valve was resuspended at all 3 commissures, and a small wedge of felt was placed within the noncoronary sinus of the aortic root. The proximal end of the graft was sutured to the sinotubular junction.

### Early Outcomes

No operative deaths and no major adverse events occurred (Table III). One patient returned to the operating room because of bleeding requiring reoperation. The intensive care unit LOS ranged from 2 to 9 days, and the overall hospital LOS ranged from 5 to 20 days.

Patient 1 readily recovered without complication after the operation. His preoperative lower extremity weakness resolved. He was discharged home on postoperative day (POD) 5; second-stage completion repair of the dilated distal aorta was planned for after his recovery.

Patient 2 had a complicated recovery, which included encephalopathy and gastrointestinal obstruction. On POD 12, a pericardial effusion was drained. He was discharged from the hospital on POD 20. Of note, surgical pathologic results ruled out aortitis and inflammation.

Patient 3 had a brief episode of encephalopathy but otherwise recovered without major complications. The patient was discharged home on POD 8.

### Mid-Term and Late Outcomes

No patient died or had repair failure. Patient 1 returned to our center 11 weeks after his first-stage ET repair to undergo the second-stage completion repair as an extent I repair of thoracoabdominal aortic aneurysm. His left subclavian artery was bypassed with an 8-mm graft. On POD 1, left vocal cord paralysis developed and was subsequently treated with a laryngeal injection before the patient's discharge from the hospital on POD 9. Six years later, the patient was well and had needed no further aortic intervention.

Two years postoperatively, Patient 2 was well and had needed no further aortic intervention.

Two years postoperatively, Patient 3 was well, the residual IMH had appeared stable during imaging monitoring, and no further aortic interventions had been needed.

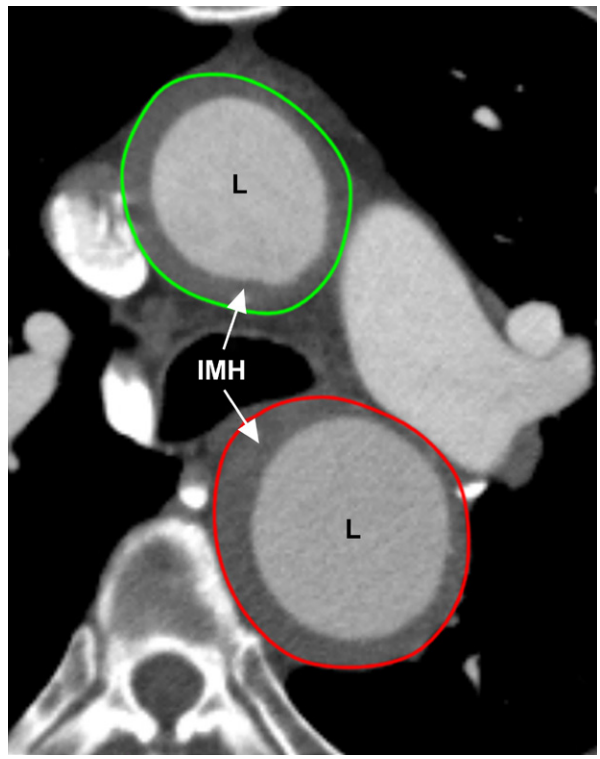


***Fig. 2*** *Patient 1. Computed tomographic image shows intramural hematoma (IMH) with diffuse aneurysmal dilation of the proximal (green outline) and distal aorta (red outline) in a 71-year-old man. The visible crescentic intraluminal density, without an intimal flap, is typical of IMH.*

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*L = lumen*

## Discussion

Although diagnosing IMH is challenging,[27] our experience suggests that the suspicion of IMH warrants transfer to a tertiary care center for urgent surgical repair. The crux of how to optimally manage type A IMH hinges on balancing the risks posed by surgery against the potential for disease progression if surgery is deferred. Proponents of early surgery point to the low

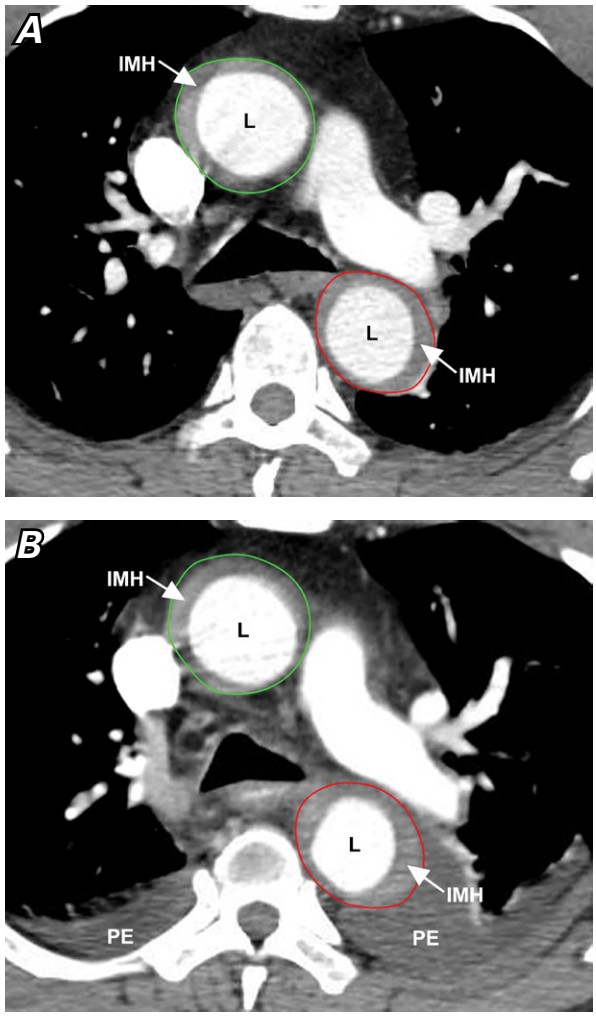


***Fig. 3*** *Patient 2. Computed tomographic (CT) images show* ***A****) an enlarged proximal aorta (green outline) and an intramural hematoma (IMH) extending into the distal aorta (red outline) in a 64-year-old man, and* ***B****) 2 days later rapid progression in the distal aorta, resulting in a pleural effusion (PE).*

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*L = lumen*

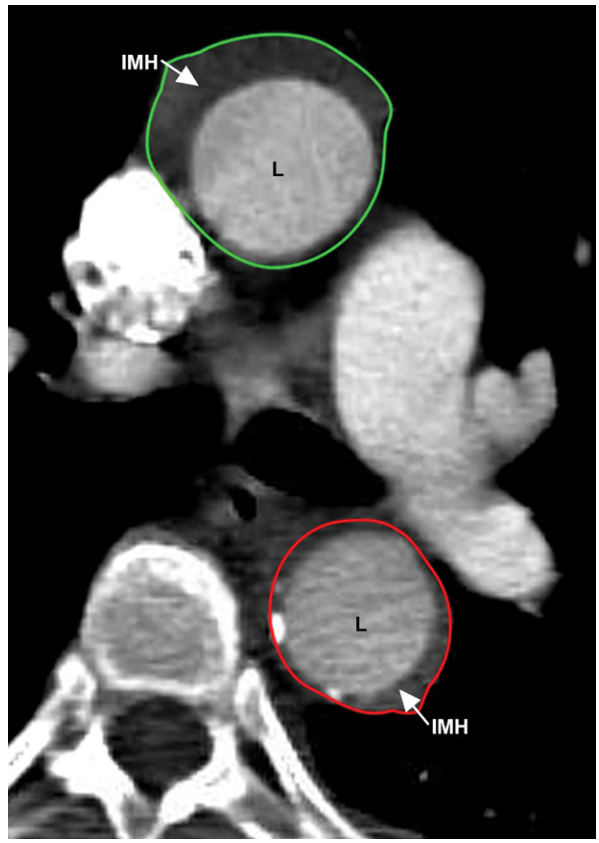


***Fig. 4*** *Patient 3. Computed tomographic image shows an intramural hematoma (IMH) extending from the proximal aorta (green outline) to the distal aorta (red outline) in a 74-year-old woman. The entire aorta is mildly dilated, and calcification is visible in the descending thoracic aorta.*

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*L = lumen*

operative mortality associated with surgical repair, along with the catastrophic risks associated with disease progression.[9,16,18,19] In contrast, proponents of conservative approaches suggest that the absolute risk of disease progression is relatively low, and that medical management may be reasonable initially, with surgical repair reserved for patients who develop complications later.[6-8,25] Our anecdotal experience suggests that the outcomes of early aortic repair in patients with type A IMH are acceptable, even for complex repair.

One of the predominant factors in deciding to opt for early surgery is mitigating the risk that the disease will progress to more grave aortic complications. In our Patient 2, the IMH appeared to be unstable and rapidly progressing (Fig. 3). Similarly, Xie and Bai[28] published a case report highlighting serial imaging in a patient whose extensive IMH affected both the proximal and distal aorta and progressed to AD within weeks. In a multicenter German study of 38 patients with acute type A IMH,[21] 28 patients (74%) had evidence of early progression (within 30 d of hospital admission) to frank dissection, contained rupture (disintegration of the outer layers of the aortic media without communication with the true lumen), or aneurysm. More recently, a study from the International Registry of Acute Dissection[10] corroborated this finding, showing that death or disease progression occurred in 6 of 10 medically managed patients with type A IMH. Likewise, in 2 larger studies of medically managed patients with acute type A IMH, a substantial proportion of patients progressed to surgical repair (25/85 [29%][6] and 15/50 [30%][8]). Of note, Hata and colleagues[9] observed a difference in early mortality between patients who were managed medically (17/66 [26%]) and those who were treated surgically (4/107 [4%]). In studies in which more than 100 patients with acute type A IMH underwent operative

repair, early mortality was acceptable and ranged from 1% to 12%.[9,16,19]

Taken together, these results highlight the aggressiveness of IMH involving the ascending aorta and the substantial risk associated with delaying or deferring surgery in these patients. However, optimal patient care may necessitate stabilizing patients with acute type A IMH and transferring them to a dedicated aortic center for surgery. The study by Estrera and colleagues[12] suggests that the risk of death in the first 3 days is lower in patients with acute type A IMH than in those with acute type A AD, which should enable sufficient transportation time.

Our study has limitations. The sample size was small, which precluded formal analysis. Although the incidence of type A IMH (3/266 AAS [1.1%]) in our experience is lower than that in previous studies,[6-13] our results must be interpreted with caution. Confirmatory diagnosis was repair based; therefore, it is plausible that the small sample size was due in part to cases of IMH that were missed because they progressed to frank AD before repair. We were therefore unable to accurately determine the incidence of acute type A IMH because our cohort comprised patients transferred to our center after initial diagnosis and progression of the AAS.

**TABLE II.** Operative Details

| Variable | Patient 1 | Patient 2 | Patient 3 |
|---|---|---|---|
| Year of repair | 2013 | 2017 | 2017 |
| Operative priority | Urgent | Emergency | Emergency |
| Procedures | Ascending Ao replacement | Ascending Ao replacement | Ascending Ao replacement |
| | Total Ao arch replacement + elephant trunk extension | Hemiarch replacement | Hemiarch replacement |
| | | Ao root replacement | Ao valve resuspension + annuloplasty |
| Cardiopulmonary bypass time (min) | 117 | 434 | 66 |
| Antegrade cerebral perfusion time (min) | 36 | 29 | 27 |
| Aortic cross-clamp time (min) | 27 | 313 | 35 |
| HCA time (min) | 36 | 29 | 27 |
| Lowest temperature during HCA (°C) | 22.6 | 23.8 | 24.1 |
| Cardiac ischemic time (min) | 63 | 342 | 62 |
| PRBC transfused (units) | 0 | 7 | 2 |

Ao = aortic; HCA = hypothermic circulatory arrest; PRBC = packed red blood cell

**TABLE III.** Early Outcomes

| Variable* | Patient 1 | Patient 2 | Patient 3 |
|---|---|---|---|
| Complications | None | Atrial arrhythmia | Encephalopathy |
| | | Encephalopathy | Pulmonary complications |
| | | Pulmonary complications | |
| | | Gastrointestinal obstruction | |
| | | Bleeding requiring reoperation | |
| Length of stay (d) | | | |
| ICU | 2 | 9 | 4 |
| Hospital | 5 | 20 | 8 |

ICU = intensive care unit

*No patients died or had a major adverse event (stroke, paraplegia, paraparesis, or renal failure necessitating dialysis) that persisted to discharge. All patients were discharged home.

## Conclusion

Acute type A IMH is relatively uncommon, and the optimal treatment of patients with this condition remains controversial. In our experience, repair of IMH typically requires urgency and techniques similar to those required to repair acute classic type A AD, the most prevalent form of AAS. Anecdotally, this study suggests that early, open repair is a safe and effective option for patients with acute type A IMH.

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