



# Clinical medical decision-making of acute aortic intramural hematoma: A non-randomized retrospective case study

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## ABSTRACT

**Objective:** This study explored the timing of interventional treatment for acute intramural aortic hematoma (IMH) and the corresponding high-risk factors for its development into local aortic dissection (AD).

**Method:** This retrospective case study method examined clinical follow-up data of 42 patients with acute IMH between April 2013 and October 2016 from the First Affiliated Hospital of Xi'an Jiaotong University. SPSS 17.0 and PPMS1.5 were used to analyze follow-up data spanning 3–12 months (mean,  $7.5 \pm 3.7$  months).

**Results:** Patients were divided into the conversion group and the hematoma group according to whether they developed AD. Among them, 16 patients (38.1%) developed AD and were treated with thoracic endovascular aortic repair (TEVAR). The remaining patients (61.89%) were treated conservatively. After 1 week, the mean aortic diameter of the conversion versus hematoma group was significantly widened. Hemodynamically unstable patients and those with hematoma to the abdominal aorta extension were more likely to develop AD. Patient outcomes after TEVAR were similar between groups.

**Conclusion:** Our findings suggest that aortic isthmus diameter  $\geq 3.0$  cm, hematoma extending to the abdominal aorta, and hemodynamic instability are associated with AD development in acute IMH patients. TEVAR should be considered if hematoma thickening, calcification ingression, ulcer progression, or contrast enhancement within the intramural hematoma is noted beyond 2 weeks after IMH onset.

Acute aortic syndromes (AAS) are vascular surgical emergencies with significant short- and long-term morbidity and mortality, including intramural hematoma (IMH), penetrating aortic ulcer (PAU), aortic dissection (AD), aortic rupture, and symptomatic aortic aneurysm.<sup>1</sup> Although the pathology and imaging findings of IMH differ from those of PAU and AD, IMH often occurs rapidly and can appear very similar to them. The three can coexist, and one can evolve into another. IMH is considered a direct consequence of vasa vasorum hemorrhage within the medial layer, and it is possible that microintimal tear(s) without an evident re-entry site create an “aortic dissection with a closed and thrombosed false lumen”.<sup>2</sup> In this regard, IMH imaging manifestations show a circular or crescent-shaped thickening hematoma  $>5$  mm of the aortic wall.<sup>3</sup> Clinical studies have shown that the mortality rates for 30 days and 3 years are 3.9% and 14.3%, respectively, if IMH patients were not treated actively. Although  $>50\%$  of hematomas in IMH patients are fully absorbed, some can progress; of them, about 33% progress to AD or aortic aneurysms, 25% progress to focal intimal disruption or PAU, and less than 5% rupture.<sup>4</sup> In this study, we

retrospectively analyzed the clinical follow-up data of 42 patients with acute IMH at the department of peripheral vascular disease of The First Affiliated Hospital of Xi'an Jiaotong University.

## 1. Subjects and method

### 1.1. Ethical approval

The study was approved by the ethics committee of the First Affiliated Hospital of Xi'an Jiaotong University. All clinical practices and observations were conducted in accordance with the Declaration of Helsinki. Informed consent was obtained from each patient before the study was conducted.

### 1.2. Subjects

The research subjects were 42 patients (34 men, 18 women; mean

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age,  $59.4 \pm 12.2$  years; range, 41–82 years) with acute DeBakey type III IMH treated between April 2013 and October 2016 in the department of peripheral vascular disease of The First Affiliated Hospital of Xi'an Jiaotong University. Among them, 16 developed AD. There were 42 cases of a history of hypertension, 28 of a history of diabetes, and 22 of a history of smoking. Of the 31 patients hospitalized with AAS, 18 had chest and back pain, 8 had pain below the xiphoid process, and 5 had both symptoms. Eleven patients were hospitalized for coronary heart disease, pancreatitis, or gallstones; of them, 8 had chest tightness and shortness of breath and 3 had abdominal pain. Inclusion criteria were as follows: within 2 weeks of IMH onset, the aortic wall below the descending aorta showed annular or crescent-shaped thickening with a hematoma thickness  $\geq 7$  mm as indicated by computed tomography angiography (CTA) imaging of the aorta with or without an ulcer-like bulge or focal enhanced lesions inside the hematoma and no vascular intima rupture. Classifications included (a) simple type, aortic wall annular or crescent-shaped thickening and a hematoma thickness  $\geq 7$  mm; (b) ulcer-like protrusion type, in which cystic enhancement of the hematoma is connected with the aortic lumen and there is no local atherosclerotic plaque; (c) focal enhancement type, in which focal enhancement occurs in the IMH and no vascular intima rupture. Exclusion criteria were as follows: (1) PAU, AD, and aortic deceleration injury; (2) Marfan syndrome, giant cell inflammation, multiple nodular arteritis, and systemic lupus erythematosus; and (3) drug addiction, treponema pallidum infection, or psychiatric disorders. According to the DeBakey classification method, there were 23 cases of type IIIA (hematoma not involving the abdominal aorta) and 19 cases of type IIIB (hematoma involving the abdominal aorta). Follow-up time was 3–12 (mean,  $7.5 \pm 3.7$ ) months.

### 1.3. Method

All patients were treated with medication, including antihypertensive therapy, heart rate control, analgesics, aggressive lipid lowering drugs, and sedatives for nervousness control. Sodium nitroprusside was used first for antihypertensive therapy, and a gradual transition was made to oral antihypertensive drugs, including calcium channel blockers, angiotensin converting enzyme inhibitors, and angiotensin II receptor blockers until the target blood pressure was reached (120/70 mmHg). Beta-blockers were used to control the heart rate to 60–80 beats/min. Pethidine was used for analgesia, aggressive lipid lowering was prescribed daily (40 mg atorvastatin or 20 mg rosuvastatin), and diazepam was used to control anxiety. The CTA was performed again 2 weeks after IMH onset and 16 patients were diagnosed with AD. Each was treated with TEVAR after family members provided consent. Conservative treatment remained unchanged for all other patients. TEVAR treatment was performed under local anesthesia, and the aortic rupture position was determined using the preoperative CTA results. In the course of TEVAR, ascending aorta angiography was performed through the radial artery puncture route to determine the anchoring zone. The femoral artery in cases of a suitable entry condition was selected for the puncture, after which two Proglide devices were installed. TEVAR treatment was performed through the selected femoral artery. The proximal end of the stent covered the aortic intima entry as much as possible. Coverage of the entire area of the aortic hematoma using a stent is unnecessary as long as one stent can cover the “entry,” while “two-stage” stent grafting was used if necessary and if the AD was located above the celiac trunk. The Valiant (Medtronic) was used in 8 cases versus the Ankura (LifeCare) in 5 and the Hercules (MicroPort).

### 1.4. Observe indicators

#### 1.4.1. Clinical manifestations

Comparison of blood pressure, pulse, pain level, pleural effusion, C-reactive protein level, and erythrocyte sedimentation rate in the intramural aortic hematoma and AD groups before and 2 weeks after IMH onset.

#### 1.4.2. Imaging data

Imaging data from CTA at the time of and 1 and 2 weeks after IMH onset were collected, with the focus on all the planes involved in intramural aortic hematoma. The analysis compared aortic isthmus diameter, hematoma thickness and length, ulcer size, interstitial hematoma contrast agent filling, and calcification ingression.

#### 1.5. Follow-up method

All data mentioned above were reviewed and updated at 1, 3, 6, and 12 months after discharge through telephone, WeChat, outpatient, and other methods of communication.

#### 1.6. Statistical method

SPSS17.0 and PPMS1.5<sup>5</sup> statistical software were used for the data analysis. The results are shown as mean  $\pm$  standard deviation. The chi-squared test was used to compare rates, differences in measured data were compared using a t-test, with values of  $P < 0.05$  being considered statistically significant.

## 2. Results

### 2.1. Comparison of clinical data

There were no statistically significant differences in the number of cases between the hematoma and conversion groups in sex, age, smoking (5 cigarettes per day for more than 1 year), hypertension, diabetes, hyperlipidemia, pleural effusion progress, or increased pain after 1 week. There were significant intergroup differences in the number of cases when the aortic isthmus diameter was  $\geq 3.0$  cm and in terms of parts affected by the hematoma (Tables 1 and 2).

### 2.2. Hemodynamics and pleural effusion in both groups

Unstable hemodynamic refers to a blood pressure  $\geq 140$  mmHg and/or heart rate  $\geq 80$  beats/min within 2 weeks of drug treatment. Pleural effusion progress refers to increased volume of fluid within 2 weeks versus the initial condition. There were significant intergroup differences in the hemodynamic indexes ( $P < 0.05$ ) (Table 3).

### 2.3. Comparison of the imaging data at and 1 and 2 weeks after IMH onset

Hematoma thickening: The maximum thickness of the intramural aortic hematoma  $\geq 10$  mm suggested an increased hematoma. Calcification ingression: The aortic atherosclerotic plaque calcification moved closer to the cavity, which also indicated an increasing hematoma. Ulcer aggravated: Ulcer-like protrusion increased on the original basis along the aortic or/and transverse diameter, suggesting ulcer progression. Enhancement lesion volume increased: the intramural aortic hematoma enhancement lesions increased, indicating increased bleeding volume. There were no statistically significant intergroup differences in the cumulative incidence of observation data at 1 week after IMH onset ( $P > 0.05$ ). However, there were statistically significant intergroup differences in the cumulative incidence of observation data at 2 weeks after IMH onset ( $P < 0.05$ ) (Table 4).

### 2.4. Post-discharge follow-up CTA imaging data

After discharge, follow-up CTA imaging data were received for 24 patients; of them, 11 were taken at more than 12 months after TEVAR. Measurement of the aortic isthmus hematoma thickness at and 1 and 2 weeks and at 1, 3, 6, and 12 months after TEVAR are shown in Table 5. There were no statistically significant intergroup differences at 3 months after TEVAR.

**Table 1**  
Comparison of general data between the two groups, cases (%).

Group	Cases, n	Male sex, n	Age (x ± s)	Smoking	Hypertension	Diabetes	Hyperlipidemia
Hematoma	26	21	70.1 ± 7.9	21 (80.7)	23 (88.4)	19 (70.3)	25 (96.1)
Conversion	16	13	71.08 ± 10.5	13 (81.2)	14 (87.5)	11 (68.5)	15 (93.7)

### 3. Discussion

IMH accounts for approximately 10–25% of AAS, and although the intima of the aorta is intact, intimal tears and flow communication between lumens is absent. However, the pathogenesis of IMH remains unclear.<sup>6</sup> It is commonly recognized that acute IMH is due to blood vessels rupturing in the middle of the aorta or upon entry to the intima causing blood to flow into the intima and form a hematoma. About two-thirds cases of acute IMH involve the descending aorta, which is more common in elderly, hypertensive, and atherosclerotic patients.<sup>2,7</sup> Almost all (90%) of the patients in this study were elderly, and in the AD conversion group, 68.7% of patients had atherosclerotic plaques in the aortic wall. Since the hematoma is near the adventitia, the patient has persistent chest and back pain, and the clinical manifestations and complications are very similar to those of PAU and AD. IMH can be completely absorbed, or it can evolve into AD.<sup>1,4</sup> DeBakey types I and II IMH have more comorbidities and higher mortality rates; thus, early surgical treatment is recommended.<sup>8,9</sup> DeBakey type III IMH can be treated conservatively since it is relatively stable, but if it evolves into DeBakey type III AD, interventional treatment should be considered. Some studies have found that the first 2 weeks after the initial IMH onset is the high-risk period during which the IMH may evolve into AD and/or aortic rupture.<sup>10</sup> To provide the basis for the timing of interventional treatment, this study focuses on risk factors that are connected with IMH evolving into AD within the first 2 weeks after IMH onset. A few high-risk factors of transformation into AD were identified.

Sueyoshi et al.<sup>11</sup> found that the diameter of the aorta involved in the hematoma was gradually expanding, possibly due to the aorta becoming more easily expandable as a result of separation between the endangium and the adventitia, which is again caused by the intramural hematoma. In this study, 68.7% of patients with IMH and an aortic isthmus diameter >3 cm developed AD. The reason for this may be that the Chinese aortic isthmus diameter is normally <3 cm.<sup>12</sup> Atherosclerosis causes the aorta to dilate and the intramural hematoma to gradually infiltrate, tears the arterial elastic fibers, and destroys the tunica media. As a result, the blood vessel wall becomes weaker and more susceptible to tearing. The longer the hematoma range, the wider the range of broken elastic fibers and the damaged tunica media becomes. Therefore, consider this one reason that DeBakey type IIIB IMH patients with involving abdominal aorta are more likely to convert to AD. Studies have shown that an IMH near the adventitia in the acute phase was more likely to lead to fluid leakage.<sup>12</sup> An estimated 73–75% of IMH cases in this study had pleural effusion, with a significantly higher volume in the conversion group than in the hematoma group. The IMH infiltrates and breaks the tunica media and elastic fibers but rarely affects the adventitia. This leads to separation of the intima and adventitia and a high chance of tearing due to inconsistency in the compliance of the intima and adventitia. At the same time, the nourishing artery is pulled to rupture, which thickens the IMH. Such

**Table 2**  
Intergroup comparison of patient signs and tests, cases (%).

Group	Cases, n	Sign			Laboratory test		Imaging	
		Chest pain	Back pain	Pleural effusion	CRP	ESR	Isthmus diameter ≥3.0 cm	Type IIIB hematoma
Hematoma	26	18 (69.2)	8 (30.7)	19 (73.0)	24 (92.3)	23 (88.4)	5 (19.2)	6 (20.3)
Conversion	16	10 (62.5)	6 (37.5)	12 (75.0)	15 (93.7)	14 (87.5)	11 (68.7)	13 (81.25)

CRP, C-reactive protein; ESR, erythrocyte sedimentation rate.

**Table 3**  
Intergroup comparison of hemodynamics and pleural effusion, cases (%).

Observation data	Hematoma group (n = 26)	Conversion group (n = 16)
Blood pressure ≥ 140 mmHg	5 (19.2)	14 (87.5)
Heart rate ≥ 80 beats/min	7 (26.9)	13 (81.2)
Pleural effusion progress	6 (23.1)	4 (25.0)

**Table 4**  
Intergroup comparison of cumulative incidence of CTA imaging observation data within 2 weeks after IMH onset, cases (%).

Observation data	1 week after onset		2 weeks after onset	
	Hematoma Group	Conversion Group	Hematoma Group	Conversion Group
Hematoma thickening	5 (19.2)	4 (25.0)	2 (7.60)	5 (31.20)
Calcification ingression	2 (7.60)	5 (31.2)	1 (3.84)	6 (37.5)
Ulcer aggravated	3 (11.5)	6 (37.5)	2 (7.60)	4 (25.0)
Enhancement lesions increased	3 (11.5)	5 (31.2)	2 (7.60)	7 (43.7)

**Table 5**  
Intergroup comparison of aortic isthmus intramural hematoma thicknesses by time point after IMH onset, mm.

Time	Hematoma Group	Conversion Group
Onset	9.5 ± 2.6	13.1 ± 2.4
1 week	9.3 ± 1.7	14.6 ± 2.8
2 weeks	8.5 ± 2.1	16.6 ± 2.6
1 month	7.4 ± 1.9	10.7 ± 2.5
3 months	6.2 ± 1.2	6.7 ± 1.6
6 months	4.1 ± 1.5	3.9 ± 1.3
12 months	2.7 ± 1.2	2.8 ± 1.4

IMH, intramural aortic hematoma.

cases are more pronounced under high-speed hemodynamics. This may also explain why some cases with poor blood pressure and heart rate control in this group had a significantly increased rate of intimal tearing.

IMH without a nourished artery rupture can also occur when blood flows into the arterial wall after the formation of aortic intima ulcer-like bumps. The rupture of these bumps in some patients were closed; the hematoma of this IMH type is located between the tunica media and the adventitia. As the endangium is intact, conservative treatment with oral prescriptions can achieve satisfactory results.<sup>4,13,14</sup> There are two possible causes for the formation of aortic ulcer-like bumps. First, aortic wall atherosclerotic plaque rupture is often surrounded by calcified

plaque. Second, it is caused by the aortic intimal inflammation and degeneration.<sup>4,15</sup> However, it remains unclear what mechanism causes the ulcer-like bumps to not close themselves easily. With advances of imaging technology and improvements in image resolution, about 70–80% of IMH cases are complicated by “microtears” or small intimal communications/ulcer-like bumps<sup>6,16,17</sup>; if the ulcer does not close, its progression often interferes with the effects of IMH medications. A gradually increasing ulcer may cause further intima tearing under the impact of high-velocity blood flow, which may progress to fatal AD. In this study, patients with thickened intramural hematomas, calcification ingression, or ulcer increases were relatively more likely to convert to AD.

In this study, irregular small nodular contrast-enhanced areas were seen in IMH in both groups. CTA findings appeared as a contrast lake in the intramural hematoma whose density was equal to that of the aortic lumen. Some studies considered<sup>6,18</sup> that the formation of nodular contrast-enhancing areas in the intramural hematoma resulted from small branch openings of the aorta such as the bronchial and/or intercostal artery that was torn by the hematoma. The aortic intima that had been lifted damaged these vessels and caused blood to flow into the hematoma. The data analysis demonstrated more patients with contrast area enhancement in the AD conversion group than in the hematoma group within the first 2 weeks after IMH onset.

Another important aspect of this study is that it evaluated the timing of interventional therapy in patients with IMH. In this study, patients with simple IMH were treated conservatively, while those with AD conversion were treated with TEVAR. The two groups of patients showed similar outcomes, with most IMH being absorbed within 12 months.

In conclusion, acute IMH patients with an aortic isthmus aortic diameter  $\geq 3.0$  cm and/or an IMH that extended to the abdominal aorta and/or unstable hemodynamics were at high risk of developing AD. After 2 weeks beyond IMH onset, if a hematoma is thickened, calcification ingression occurs, the ulcer progresses, or contrast areas in the IMH are enhanced, TEVAR therapy should be considered.

#### Patient consent

Written informed consent was obtained from patients for publication of these case reports and any accompanying images.

#### Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence

the work reported in this paper.

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