Long-Term Prognosis of Patients With Type A Aortic Intramural Hematoma

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Background—Recent studies have shown an favorable short-term prognosis of patients with type A acute aortic intramural hematoma (IMH). The difference of aortic pathology may have a different impact on clinical course compared with classic aortic dissection (AD). The purpose of this study was to elucidate clinical features and long-term prognosis of patients with type A IMH.

Methods and Results—Clinical data were compared retrospectively between 30 patients with acute type A IMH (IMH group) and 101 patients with acute type A AD (AD group) who were admitted to our institutions from 1988 to 1998. In AD group, 72 patients underwent surgical repair and 29 patients were treated medically. All patients in IMH group were treated initially with supportive medical therapy. Thirteen patients who demonstrated progression to AD or increase in size of hematoma underwent timed surgical repair except for 1 patient. The overall in-hospital mortality rate in IMH group was significantly lower than that in AD group (7% and 34%, \(P=0.004\)). Follow-up periods were 56±37 (IMH group) and 60±42 months (AD group), which revealed 1 and 6 late deaths, respectively. The actuarial survival rates in IMH group were all 90% at 1, 2, and 5 years, which were significantly higher than those in AD group (67%, 66%, and 62%, respectively; \(P=0.004\)).

Conclusions—Patients with type A IMH have better long-term prognosis than patients with AD. (Circulation. 2002; 106[suppl I]:I-248-I-252.)

Key Words: aorta ■ follow-up studies ■ mortality ■ prognosis ■ survival

Aortic intramural hematoma (IMH) has been recognized as a variant of aortic dissection (AD) and is detected with the use of noninvasive imaging techniques such as computed tomography (CT), magnetic resonance imaging (MRI), and transesophageal echocardiography (TEE).1–9 Isolated IMH is characterized by aortic wall hematoma without demonstrable intimal flap or penetrating ulcer,10,11 and it may cause potentially catastrophic clinical events, including the occurrence of overt AD and the rupture of aorta.

Studies to date have suggested that aortic IMH causes morbidity and mortality that are similar to those of classic AD.3,5–7,9 The site of IMH is an important parameter for determining prognosis. It has been reported that early surgery should be required for patients with IMH involving the ascending aorta (type A) because it tends to develop classic overt dissection or rupture.3,5–7,9,12 However, recent studies have shown favorable results of type A IMH with medical therapy.9,13–16 Song et al17 reported the midterm prognosis of patients with type A IMH and suggested that medical treatment with frequent imaging follow-up and timed elective surgery in cases with complications could be a rational option for those patients. The difference of aortic pathology may have a different impact on clinical course. However, the long-term clinical course of patients with type A IMH is not clearly known. The purpose of this study was to elucidate clinical pictures and long-term prognosis of patients with type A IMH.

Methods

Patient Characteristics

From 1988 to December 1998, 30 patients with acute type A IMH (IMH group) and 101 patients with acute type A AD (AD group) were admitted to our institutions within 2 days from the onset. All diagnoses were established with TEE and contrast-enhanced CT. Patients with chronic AD or IMH were excluded. Typical double-channel aorta with dissecting membrane or intimal tear was an imaging criterion for diagnosis of AD. IMH was defined as maximal crescent or circular thickening of the aortic wall >7 mm with TEE.4 Absence of dissection flap, intimal tear, or penetrating atherosclerotic ulcer was also a prerequisite for diagnosis of IMH.8

Treatment

For all patients in AD group, surgical intervention was strongly recommended. Seventy-two patients underwent emergent surgical repair. From 1988 to December 1998, 30 patients with acute type A IMH (IMH group) and 101 patients with acute type A AD (AD group) were admitted to our institutions within 2 days from the onset. All diagnoses were established with TEE and contrast-enhanced CT. Patients with chronic AD or IMH were excluded. Typical double-channel aorta with dissecting membrane or intimal tear was an imaging criterion for diagnosis of AD. IMH was defined as maximal crescent or circular thickening of the aortic wall >7 mm with TEE.4 Absence of dissection flap, intimal tear, or penetrating atherosclerotic ulcer was also a prerequisite for diagnosis of IMH.8

Treatment

For all patients in AD group, surgical intervention was strongly recommended. Seventy-two patients underwent emergent surgical repair.
repair and the remaining 29 patients were treated medically. Reasons for selecting medical treatment for the patients were refusal of surgery (n=3), high-risk surgical candidates (n=12), partial thrombosis of false lumen in ascending aorta (n=10), death just after admission (n=4).

However, medical therapy was selected for all patients in IMH group. Our therapeutic strategy for patients with type A IMH was summarized in Figure 1. We generally follow the blood pressure with an arterial line and monitor the electrocardiogram (ECG) in intensive care unit. Our initial therapeutic goal during the acute phase of IMH included the elimination of pain and the reduction of systolic blood pressure to 100 to 120 mm Hg. Close clinical follow-up using transthoracic echocardiography (TTE), TEE, and CT was performed to minimize the risk of fatal complications. TTE was performed daily during the initial 5 days to monitor pericardial effusion and aortic regurgitation. Follow-up TEE was performed within 3 days after the admission and generally once a week after that until the third week. CT examination was generally repeated at the first and third week after the admission. Patients who demonstrated regression or disappearance of hematoma were discharged with oral medication. If the size of the hematoma was unchanged, CT or TEE was repeated once a week after the third week. Patients who demonstrated the increase in the size of the hematoma or progression to overt dissection during the follow-up period were referred for surgical repair and underwent urgent operation. Pericardiocentesis was performed on admission in 5 patients with complications caused by cardiac tamponade. These patients were treated medically after the pericardiocentesis.

For all patients in both AD and IMH group, several antihypertensive drugs such as calcium-channel antagonists, angiotensin-converting enzyme inhibitors, or beta-blockers were administered orally during the course of hospitalization to achieve adequate blood pressure control.

Statistical Analysis
All values are expressed as mean±1 SD except for survival rates. Differences between categorical parameters were assessed by use of chi-square analysis or Fisher's exact test when appropriate. Continuous variables were compared by use of unpaired Student t test. Comparison of aortic diameter and wall thickness between on admission and follow-up was performed with Student paired t test. Survival analysis was performed with Kaplan-Meier analysis, and differences in survival between groups were examined with the log-rank test. Variability of survival rate was expressed as±1 SEM. P<0.05 was considered statistically significant.

Results
Table 1 summarizes the clinical features of all patients. Mean age of patients in IMH group was significantly higher than that in AD group. The prevalence of hypertension, diabetes mellitus, hyperlipidemia, history of smoking, cerebrovascular disease, ischemic heart disease, and hemodialysis did not show any difference between 2 groups. However, involvement of descending aorta was significantly more frequent in AD group than IMH group (P=0.0004). As for complications, there was no significant difference between 2 groups in the incidence of cardiac tamponade, whereas the incidence of moderate to severe aortic regurgitation and stroke were significantly higher in AD group than in IMH group.

In patients with type A IMH, 13 patients (43%) developed classic AD or showed increase in size of hematoma (Fig. 2). These patients underwent graft replacement of the ascending aorta (mean 37 days, ranged from 7 to 94 days, after the onset) except for 1 patient, who refused operation and died because of aortic rupture.

Operative mortality and morbidity are summarized in Table 2. There were 17 operative deaths: 1 (8%) in IMH group and 16 (22%) in AD group (P=0.444). The cause of the death in IMH group was uncontrollable bleeding. The cause of death in AD group were: cardiac failure in 4, stroke in 2, complications of uncontrollable bleeding in 6, and multiorgan failure in 4. There were 7 strokes: 3 in IMH group and 4 in AD group. Two of these 7 patients died. These 2 patients were all from AD group. Seven patients developed acute renal failure: 1 in IMH group and 6 in AD group. Four patients all from AD group required renal dialysis. Two patients developed sternal infection: 1 in IMH group and 1 in AD group.

There were 2 early deaths in IMH group and 34 deaths in AD group (P=0.004). The overall in-hospital mortality rate of the patients in IMH group was 7%, which was significantly lower than that of patients with type A AD (34%, P=0.004). Patients were followed from 2 to 120 months, with a mean of 55 months. Table 3 shows the follow-up periods for both groups and the cause of late deaths. There were 1 late death in IMH group and 6 late deaths in AD group (P=0.670). The actuarial survival rates in IMH were all 90±6% at 1, 2, and 5 years, which were significantly higher than those in AD group (67±5%, 66±5%, and 62±5%, respectively; P=0.004). Figure 3 shows the survival curve for IMH group and AD group.

In the IMH group, 17 patients (57%) were discharged without operation. Among them, follow-up imaging study could be obtained in all patients (mean 30 months after the onset). In these patients, the maximum aortic wall thickness (hematoma size) significantly decreased (from 9±3 mm to 1±3 mm, P<0.0001) (Fig. 4). In addition, maximum aortic diameter significantly decreased (from 48±5 mm to 45±6 mm, P=0.0006). Complete resolution of IMH in the ascending aorta occurred in 12 patients (40%) (Fig. 5). None of these patients died in follow-up periods.

Discussion
The main findings of this study were that patients with type A IMH were treated with supportive medical therapy with timed surgical repair and had better long-term prognosis than patients with type A AD. This finding suggests that supportive medical therapy with timed surgical repair in cases with progression can be a rational therapeutic strategy inpatients with type A IMH.

The previous studies reported that a formal distinction between type A and type B IMH might be justified with

Figure 1. Therapeutic strategy for patients with type A IMH.
TABLE 1. Patient Characteristics

<table>
<thead>
<tr>
<th></th>
<th>IMH (n=30)</th>
<th>AD (n=101)</th>
<th>P</th>
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</thead>
<tbody>
<tr>
<td><strong>Basic characteristics</strong></td>
<td></td>
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<tr>
<td>Age, yr</td>
<td>67±10</td>
<td>59±13</td>
<td>0.05</td>
</tr>
<tr>
<td>Sex, male/female</td>
<td>14/16</td>
<td>65/36</td>
<td>0.082</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>28 (93)</td>
<td>88 (87)</td>
<td>0.518</td>
</tr>
<tr>
<td>Diabetes mellitus, n (%)</td>
<td>4 (13)</td>
<td>8 (8)</td>
<td>0.469</td>
</tr>
<tr>
<td>Hyperlipidemia, n (%)</td>
<td>1 (3)</td>
<td>6 (6)</td>
<td>0.999</td>
</tr>
<tr>
<td>History of smoking, n (%)</td>
<td>13 (43)</td>
<td>55 (54)</td>
<td>0.284</td>
</tr>
<tr>
<td>Cerebrovascular disease, n (%)</td>
<td>2 (7)</td>
<td>2 (2)</td>
<td>0.225</td>
</tr>
<tr>
<td>Ischemic heart disease, n (%)</td>
<td>3 (10)</td>
<td>6 (6)</td>
<td>0.427</td>
</tr>
<tr>
<td>Hemodialysis, n (%)</td>
<td>0 (0)</td>
<td>2 (2)</td>
<td>0.999</td>
</tr>
<tr>
<td><strong>Location of aortic abnormality, n (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Involving the descending aorta</td>
<td>19 (63)</td>
<td>93 (92)</td>
<td>0.0004</td>
</tr>
<tr>
<td><strong>Complication, n (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic regurgitation (&gt;moderate)</td>
<td>2 (7)</td>
<td>28 (28)</td>
<td>0.016</td>
</tr>
<tr>
<td>Cardiac tamponade</td>
<td>7 (23)</td>
<td>21 (21)</td>
<td>0.766</td>
</tr>
<tr>
<td>Stroke</td>
<td>1 (3)</td>
<td>21 (21)</td>
<td>0.025</td>
</tr>
<tr>
<td><strong>Follow-up variables during the chronic phase, n (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean systolic BP&gt;140 mm Hg</td>
<td>9 (32)</td>
<td>27 (40)</td>
<td>0.455</td>
</tr>
<tr>
<td><strong>Antihypertensive therapy</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beta-blocker</td>
<td>16 (57)</td>
<td>19 (28)</td>
<td>0.008</td>
</tr>
<tr>
<td>Ca²⁺ antagonist</td>
<td>25 (89)</td>
<td>45 (67)</td>
<td>0.026</td>
</tr>
<tr>
<td>ACE inhibitor</td>
<td>8 (29)</td>
<td>25 (37)</td>
<td>0.415</td>
</tr>
<tr>
<td>Alpha-blocker</td>
<td>6 (21)</td>
<td>11 (16)</td>
<td>0.561</td>
</tr>
</tbody>
</table>

ACE=angiotensin-converting enzyme; AD=aortic dissection; BP=blood pressure; IMH=intramural hematoma.

TABLE 2. Operative Mortality and Morbidity

<table>
<thead>
<tr>
<th></th>
<th>IMH (n=12)</th>
<th>AD (n=72)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Operative procedure, n (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ascending aorta</td>
<td>7 (58)</td>
<td>51 (71)</td>
<td>0.501</td>
</tr>
<tr>
<td>Ascending aorta+hemiarch</td>
<td>3 (25)</td>
<td>7 (10)</td>
<td>0.150</td>
</tr>
<tr>
<td>Ascending aorta+total arch</td>
<td>2 (17)</td>
<td>14 (19)</td>
<td>0.999</td>
</tr>
<tr>
<td><strong>Operative mortality, n (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blending</td>
<td>1 (8)</td>
<td>3 (4)</td>
<td>0.467</td>
</tr>
<tr>
<td>Cerebral infarction</td>
<td>3 (25)</td>
<td>4 (6)</td>
<td>0.057</td>
</tr>
<tr>
<td>Renal failure</td>
<td>1 (8)</td>
<td>6 (8)</td>
<td>0.999</td>
</tr>
<tr>
<td>Sternal infection</td>
<td>1 (8)</td>
<td>1 (1)</td>
<td>0.267</td>
</tr>
</tbody>
</table>

AD=aortic dissection; IMH=intramural hematoma.

respects to both prognosis and treatment and that the site of IMH was a important parameter for determining prognosis. In the study by Robbins et al, all 3 patients with type A IMH ultimately underwent surgery. In the study by Mohr-Kahaly et al, 2 of 3 patients with type A IMH developed communicating dissection or outward rupture. The outcome of type A IMH appeared favorable with immediate surgical repair in these early investigations. However, recent reports showed excellent clinical course of type A IMH with medical treatment. In terms of surgical repair for ascending aorta, early mortality rate of patients for type A AD patients with operation ranges from 12% to 36%. In the current study, operative mortality rate for type A AD patients was similar to those in the previous reports. On the other hand, operative mortality rate of patients with type A IMH has been reported to be from 6% to 17%. In the present study, operative mortality rate for type A IMH patients was 8%. Considering relatively low mortality rate for type A IMH patients and the fact that 43% patients required operation in this study, it might seem to rationale to operate immediately on all patients with type A IMH. When diagnostic imaging

Figure 2. One example of the progression to overt AD in a patient with type A IMH. Initial CT scan showed characteristic crescent wall thickening in both the ascending and descending aorta (A). Two weeks after the onset, follow-up study revealed increase in size of hematoma (B) and contrast enhancement in the hematoma (arrow), which suggested flow communication (C).
modalities including TTE, TEE, CT, or MRI and urgent surgical intervention are not readily available, early surgery should be required. However, from an intention to treat analysis, only 2 (7%) early deaths occurred in 30 patients managed conservatively in this study. Given both the improvement of in-hospital mortality and long-term survival, it seems likely that supportive medical therapy may be a reasonable option as the initial treatment in patients with type A IMH when multiple imaging modalities and surgical backup are always available.

In the present study, 12 patients with type A IMH underwent surgical repair as a result of progression to overt dissection or increase in size of hematoma. The interval from the onset to the operation ranged from 7 to 94 days (mean 37 days). Therefore, we reasoned that close imaging follow-up by CT, MRI, or TEE is necessary to avoid overlooking the operative timing for these patients. We previously reported that maximum aortic diameter estimated by the initial CT images was the most significant risk factor for progression of type A IMH. Patients with an aortic diameter ≥50 mm had a tendency for progression to dissection or aortic enlargement. Thus, these patients could be monitored carefully with frequent imaging. Alternatively, early surgery might be required for these patients when various imaging modalities and urgent surgery are not always available.

The difference of survival rate between IMH and AD group may be related to the incidence of serious complications. Song et al reported that significant aortic regurgitation was observed more frequently in AD patients than IMH patients. This finding is similar to our results. In the current study, the incidence of stroke was also significantly higher in AD group than IMH group. It has been reported that severe aortic regurgitation and cerebral accident are risk factors for death for patients with medically or surgically treated ADs. Therefore, it seems likely that the higher survival rate in IMH group may be related to the lower incidence of aortic regurgitation and stroke than in AD group.

Spontaneous absorption is a frequent phenomenon encountered in IMH. Patients with resolved IMH have been reported
to have a good long-term prognosis.\textsuperscript{23} In this study, 12 of 17 patients who survive without surgery showed complete resolution of hematoma in the ascending aorta and none of these patients died in the follow-up periods. Therefore, it seems likely that the disappearance of IMH in the ascending aorta is a clinical sign of good prognosis.

In the setting of classic AD, cardiac tamponade is the most common cause of death. Isselbacher et al\textsuperscript{24} suggested that pericardiocentesis in treating cardiac tamponade might be harmful rather than beneficial. On the other hand, recent studies reported that patients with type A IMH complicated by cardiac tamponade can be stabilized successfully by pericardiocentesis alone.\textsuperscript{16,17,25} Given the considerable controversy surrounding the treatment of cardiac tamponade inpatients with IMH, further investigation is necessary to establish the risk and the role of pericardiocentesis. Although patients with cardiac tamponade could be treated with pericardiocentesis and after medical therapy in the present study, early surgery may be required for these patients when various imaging modalities and urgent surgery are not always available.

**Conclusions**

In conclusion, patients with type A IMH have better long-term prognosis than patients with type A AD. Considering mortality and morbidity for surgical repair of ascending aorta, supportive medical treatment with frequent follow-up imaging studies and timed surgical repair can be a rational therapeutic strategy in management of selected patients with type A IMH. With this strategy, it is essential that diagnostic imaging modalities including TTE, TEE, CT, or MRI and urgent surgical intervention are readily available.

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**References**

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