Aortic Intramural Hematoma: Assessment of Clinical and Radiological Features in Comparison to Acute Aortic Dissection

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Purpose: To compare the clinical and radiological features of aortic intramural hematoma (IMH) to those of acute aortic dissection (AD).

Materials and Methods: We analyzed the clinical and radiological features of 12 patients with aortic IMH and 43 patients with acute AD. In aortic IMH, the diagnoses were made by means of both CT and transesophageal echocardiography (TEE) and included two surgically proven cases. In acute AD, the diagnoses were made by means of CT and TEE and included 21 surgically proven cases. We compared patients' ages, etiologies, the extent of the disease, the presence or absence of aortic branch involvement, complications, and outcomes.

Results: Aortic IMH tended to develop in older patients (67.8 ± 7.9 vs. 50.4 ± 13.4, P < .0001) more often than acute AD. In aortic IMH, all patients had a history of hypertension; in acute AD, hypertension occurred in 37, Marfan's syndrome in three, and trauma in one. In aortic IMH, Stanford type A and B lesions were found in four patients (33%) and eight (67%), respectively. In acute AD, Stanford type A and B lesions were seen in 22 (51%) and 21 (49%), respectively (p > .05). In aortic IMH, there was no involvement of aortic branches, whereas in acute AD, 14 (33%) patients showed involvement of one or more aortic branches. Complications of aortic IMH included pericardial effusion (n = 2) and pleural effusion (n = 4); in acute AD, pericardial effusion (n = 7), pleural effusion (n = 4), aortic insufficiency (n = 8), cerebral infarction (n = 3), renal infarction (n = 4) and spinal infarction (n = 1) were seen. There was one (8%) death due to aortic IMH and ten (23%) deaths due to acute AD (p < .01).

Conclusion: Aortic IMH is characterized by its occurrence in older patients with hypertension, a less frequent incidence of complications, and a more favorable outcome than acute AD.

Index Words: Aorta, CT
Aorta, dissection

Aortic dissection is a life-threatening condition requiring immediate diagnosis and definitive treatment. An intimal flap and false lumen are the hallmark findings of dissection (1–3). Aortic intramural hematoma, described by Yamada et al (4), is a disease entity which has clinical symptoms similar to those of acute aortic dissection. It is important to distinguish between acute aortic dissection and aortic intramural hematoma, as the clinical course, prognosis and method of treatment of each condition is different. Yamada's report (4) emphasized that aortic intramural hematoma is a subgroup of aortic dissection without communication between the aortic lumen and the dissected cavity, and represented the early stage of dissection. Conversely, Robbins et al (5) postulated that aortic intramural hematoma was a distinct pathologic entity which should not be confused with aortic dissection. To our knowledge, there have been no reports directly comparing these entities. Our study attempts to compare the clinical and radiological features of aortic intramural hematoma to
those of acute aortic dissection in a patient undergoing CT and transesophageal echocardiography.

MATERIALS and METHODS

Between June 1993 and May 1995, a total of 55 patients aged 22–80 years (mean, 65) were examined while in the acute stage, i.e., less than 7 days after the onset of initial symptoms suggesting aortic dissection. For 12 of the 55 patients the diagnosis was aortic intramural hematoma, while for 43, acute aortic dissection was diagnosed. The 12 patients with aortic intramural hematoma were diagnosed by means of both CT and transesophageal echocardiography; diagnosis in all 43 patients with acute aortic dissection involved the use of CT, and for 22 patients transesophageal echocardiography was also used. Twenty of the 43 patients were examined with MRI and five with angiography, as supplementary tools for detecting the origin of the aortic branch vessels. Two patients with aortic intramural hematoma and 21 with acute aortic dissection underwent surgery. In nine patients with aortic intramural hematoma, follow-up CT or transesophageal echocardiographic studies were performed within 6 months of the initial imaging examination. Of the 43 patients with acute aortic dissection, 31 were followed up using CT or transesophageal echocardiography.

Three of the 12 patients with aortic intramural hematoma were scanned using conventional CT (CT/T 9800; GE, Milwaukee, USA), and for nine, spiral CT (Somatom Plus-S; Siemens, Erlangen, Germany) was used. Ten of the 43 patients with acute aortic dissection were examined using conventional CT; for 33, spiral CT was used. Both unenhanced and enhanced conventional CT scans were obtained; the enhanced scans from the aortic arch to the aortic bifurcation were performed at a continuous 10mm collimation and at 10 mm intervals after a 100cc bolus intravenous injection of contrast material (Ultravist-370; Schering, Berlin, Germany). Spiral CT scans were initially performed without contrast enhancement. Subsequent contrast-enhanced scans from the aortic arch to the aortic bifurcation (scan time 100 msec) were obtained at 10mm collimation at a table speed of 20mm/sec after an injection of 100ml of contrast material at a rate of 2ml/sec using an automatic power injector. In all patients, a scan delay time of 30 seconds was chosen after the start of injection.

Patients were examined with transesophageal echocardiography (Hewlett-Packard 500L, USA) using a 5-MHz transducer to obtain transesophageal two-dimensional and color-coded Doppler images. During transesophageal echocardiography examination, patients systolic blood pressure was carefully controlled. For mild sedation, 5–10mg diazepam or 10mg morphine was injected into a muscle; to suppress the gag reflex, local anesthesia of the pharynx was achieved with Lidocaine jelly or spray.

The diagnosis of aortic dissection was made when two lumens separated by an intimal flap were identified within the aorta on CT, transesophageal echocardiography or both (Fig. 1). The diagnosis of aortic intramural hematoma was made when the following conditions were present: (1) a continuous, crescentic high-attenuation area along the wall of the thoracic aorta on unenhanced CT (Fig. 2a); (2) a nonopacified crescentic area along the corresponding area of the aortic wall on enhanced CT (Fig. 2b); (3) a total absence of intimal ulceration or disruption; and (4) medial displacement of intimal calcification. On transesophageal echocardiography, the diagnosis of aortic intramural hematoma was made when there was an anechoic or echogenic space greater than 7mm with no evidence of double channel of flow signals along the aortic wall and no evidence of intimal disruption or distortion of the aortic lumen (Fig. 3a). CT and transesophageal echocardiographic findings were interpreted independently by two radiologists (K.H.Y, T.H.L) and two cardiologists (D.H.K, J.K.S), respectively. When the observers did not fully agree, the diagnosis was reached by consensus. Cases in which consensus was not reached were excluded from this analysis.

We considered the two entities along with various factors, including patients age at onset, etiology, extent of the disease according to the Stanford classification, the presence or absence of aortic branch involvement, complications, and outcome.

RESULTS

The clinical and radiological findings of the 12 patients with aortic intramural hematoma are summarized in Table 1. The age of these patients ranged from 50 to 80 years (mean ± SD, 67.8 ± 7.9 years) at the time of initial presentation of symptoms; the age of patients with acute aortic dissection ranged from 22 to 72 years.

![Fig. 1. A classical case of acute aortic dissection. Contrast enhanced CT scan demonstrates intimal flap (arrows) separating true and false lumen.](image-url)
(mean 50.4 ± 13.4). The differences in mean age of patients with aortic intramural hematoma and acute aortic dissection was statistically significant (p < 0.001). All patients with aortic intramural hematoma had a history of hypertension. Of the 43 patients with acute aortic dissection, 37 (86%) had a history of hypertension, three (7%) showed a classic stigmata of Marfan’s syndrome, and one (2%) had suffered acute trauma. The differences between the two entities according to the Stanford classification indicating the extent of the disease were not significant (p > 0.05). In aortic intramural hematoma, a type B (n = 8, 67%) lesion was more frequent than a type A (n = 4, 33%); in acute aortic dissection, the occurrence of type A (n = 22, 51%) and type B lesions (n = 21, 49%) was nearly equal. There was no involvement of major aortic branches in any patient with aortic intramural hematoma, whereas 14 (33%) of the 43 patients with acute aortic dissection demonstrated the involvement of one or more major aortic branches. There was involvement of the renal artery in 13 patients, the common carotid artery in five, the superior mesenteric artery in five, the inferior mesenteric artery in three, the celiac axis in three, and the subclavian artery in one patient. The complications between the two entities were markedly different. Of the 12 patients with aortic intramural hematoma, there was pericardial effusion in two (17%) (Fig. 4), and pleural effusion in four (33%). One (8%) of these patients was also in shock on admission. Of the 43 patients with acute aortic dissection, there was pericardial effusion in seven (16%).

Table 1. Data on 12 Patients with Aortic Intramural Hematoma

<table>
<thead>
<tr>
<th>Pt. No</th>
<th>Sex/Age</th>
<th>Extent*</th>
<th>History of hypertension</th>
<th>Sx at presentation</th>
<th>Duration of Sx before Dx</th>
<th>Diameter of IMH(cm)</th>
<th>Complications</th>
<th>Follow-up</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M/68</td>
<td>B</td>
<td>4 yrs</td>
<td>Chest pain</td>
<td>3 days</td>
<td>1.2</td>
<td>pleural effusion</td>
<td>Aortic dissection</td>
<td>Surgery</td>
</tr>
<tr>
<td>2</td>
<td>F/76</td>
<td>B</td>
<td>20 yrs</td>
<td>Abd. Pain</td>
<td>7 days</td>
<td>0.7</td>
<td>pleural effusion</td>
<td>Regression</td>
<td>CI</td>
</tr>
<tr>
<td>3</td>
<td>M/60</td>
<td>B</td>
<td>10 yrs</td>
<td>Chest pain</td>
<td>2 days</td>
<td>1.2</td>
<td>(-)</td>
<td>P &amp; R</td>
<td>CI</td>
</tr>
<tr>
<td>4</td>
<td>M/76</td>
<td>B</td>
<td>15 yrs</td>
<td>Back pain</td>
<td>4 hrs</td>
<td>0.8</td>
<td>(-)</td>
<td>Regression</td>
<td>CI</td>
</tr>
<tr>
<td>5</td>
<td>F/80</td>
<td>A</td>
<td>30 yrs</td>
<td>Back pain</td>
<td>8 hrs</td>
<td>1.2</td>
<td>pericardial effusion</td>
<td>Regression</td>
<td>CI</td>
</tr>
<tr>
<td>6</td>
<td>F/63</td>
<td>B</td>
<td>15 yrs</td>
<td>Back pain</td>
<td>2 hrs</td>
<td>1.0</td>
<td>pleural effusion</td>
<td>Regression</td>
<td>CI</td>
</tr>
<tr>
<td>7</td>
<td>F/72</td>
<td>A</td>
<td>50 yrs</td>
<td>Chest pain</td>
<td>7 days</td>
<td>1.0</td>
<td>(-)</td>
<td>P &amp; R</td>
<td>CI</td>
</tr>
<tr>
<td>8</td>
<td>F/71</td>
<td>A</td>
<td>5 yrs</td>
<td>Chest pain</td>
<td>6 hrs</td>
<td>1.2</td>
<td>pericardial effusion</td>
<td>Regression</td>
<td>Died</td>
</tr>
<tr>
<td>9</td>
<td>F/50</td>
<td>B</td>
<td>10 yrs</td>
<td>Back pain</td>
<td>4 hrs</td>
<td>1.8</td>
<td>(-)</td>
<td>Regression</td>
<td>CI</td>
</tr>
<tr>
<td>10</td>
<td>F/63</td>
<td>A</td>
<td>11 yrs</td>
<td>Chest pain</td>
<td>2 hrs</td>
<td>0.7</td>
<td>(-)</td>
<td>Surgery</td>
<td>CI</td>
</tr>
<tr>
<td>11</td>
<td>M/70</td>
<td>B</td>
<td>5 yrs</td>
<td>Back pain</td>
<td>6 hrs</td>
<td>1.2</td>
<td>pleural effusion</td>
<td>CI</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>F/65</td>
<td>B</td>
<td>10 yrs</td>
<td>Chest pain</td>
<td>2 days</td>
<td>1.5</td>
<td>(-)</td>
<td>Regression</td>
<td>CI</td>
</tr>
</tbody>
</table>

IMH: intramural hematoma, Sx: symptoms, Dx: diagnosis, CI: clinical improvement, yrs: years, hrs: hours, P & R: progression and then regression, (-): not present, Extent*: Stanford classification

Fig. 2. A 71-year-old female with aortic intramural hematoma.

a. Unenhanced CT scan shows crescentic high-attenuating area (arrows) along the aortic wall representing fresh hematoma.

b. Enhanced CT scan shows corresponding nonopacified crescentic area (arrows) along the aortic wall.
Fig. 3. A 76-year-old man with aortic intramural hematoma.
a. Initial transesophageal echocardiogram performed 1 day after the attack shows echogenic space (arrows) with extensive wall thickness measuring 10 mm. There is no evidence of intimal tear or intimal ulceration along the aorta.
b. Follow-up transesophageal echocardiogram performed 10 days later shows decreased wall thickness of the aorta (arrows) representing regression of the hematoma.

pleural effusion in 11 (26%), aortic insufficiency in eight (19%), renal infarction in four (9%), cerebral infarction in two (5%), and spinal infarction in one (2%). Seven (16%) of the patients with acute aortic dissection were in shock on admission. Of the 12 patients with aortic intramural hematoma, one (8%) who was in shock on admission died one day later. Of the 43 patients with acute aortic dissection, seven patients (16%) died despite emergency surgery in four and medical therapy in six within one week of admission. During the mean follow-up period of 12 months, none of the other patients with aortic intramural hematoma who underwent either surgery or medical treatment experienced a second attack. In contrast, three patients with aortic dissection who underwent medical treatment died within one year.

DISCUSSION

The pathologic mechanism of aortic dissection has been debated. Gore (6) suggested that spontaneous rupture of the aortic vasa vasorum is the inciting disturbance, which then leads to intimal tearing. Wilson et al. (7) suggested that the initial step in aortic dissection is actually two events, thereby supporting the theory that aortic dissection actually represents a spectrum of aortic pathology. The first event is rupture of the aortic vasa vasorum, which gives rise to intramural hemorrhage followed by dissection. The second event is intimal tearing, allowing blood to enter the media and establishing a plane of cleavage. Aortic intramural hematoma seems to represent a more confined or early stage of the dissecting process, with bleeding into the wall layers, and may progress to classical aortic dissection or to rupture of the aorta.

The prevalence of aortic intramural hematoma has previously been reported in the literature. Yamada et al. (4), Wilson and Hutchins (7), and Mohr-Kahaly (8) reported that aortic intramural hematoma existed in 41%, 13%, and 23% of all cases of aortic dissection, respectively. Twenty-one percent of our cases of acute aortic dissection were diagnosed as aortic intramural hematoma, and this correlates with those previous reports. In aortic intramural hematoma, the descending aorta may be more frequently involved than the ascending aorta. In Robbins’s report (5), Stanford type A and B lesions were three and ten, in Mohr-Kahaly’s report (8), three and ten, and in our cases, four and eight,
respectively. Mohr-Kahaly et al (8) stated that the longitudinal extent of the aortic intramural hemorrhage varied from 3 to 20 cm, and that maximum wall thickness at the site of the hemorrhage ranged from 0.7 to 3.0 cm. In our cases, the maximum thickness of the hematoma ranged from 0.7 to 2.0 cm (Table 1).

Many imaging modalities have been used to demonstrate the findings and characteristics of aortic intramural hematoma, although diagnostic problems still persist (4, 5, 8). Our criteria for the diagnosis of aortic intramural hematoma are based on these previous studies. Using spiral CT, we diagnose aortic intramural hematoma on the basis of the findings of aortic dissection on conventional CT (9) and helical or spiral CT (10, 11) as previously described. In diagnosing aortic intramural hematoma using imaging, a differential diagnosis of “aortic dissection with thrombosed and noncommunicating false lumen” is always problematic. We proposed that in the acute or hyperacute stage of the diseases, these two entities could be differentiated. In all cases examined within one week of the onset of symptoms, aortic intramural hematoma showed crescentic high-attenuating areas along the wall of the aortic lumen, which on unenhanced CT represented fresh hematoma. CT attenuation of an acute hemorrhage or clot appears typically hyperdense because the hematocrit of an acute retracted clot is around 90%. Occasionally, an acute hemorrhage may in certain cases appear isodense, with adjacent soft tissue; for example, extreme anemia and coagulation disorder. When the high-attenuating crescentic area could not be demonstrated on unenhanced CT, contrast-enhanced CT was useful for detection of the crescentic area at the acute stage. At the subacute and chronic stages, unenhanced CT scan did not demonstrate crescentic high-attenuating areas, but showed only low attenuating crescentic areas along the aortic wall. These could not be distinguished as either clotted hematoma or aortic dissection with thrombosed false lumen.

Mohr-Kahaly et al (8) reported that five (33%) of their 15 patients with aortic intramural hemorrhage developed classical aortic dissection and four (27%) developed to the point of rupture. Yamada et al (4) reported that one of their 15 patients demonstrated increased crescentic areas and pleural effusion and one other patient exhibited enlargement of the lumen of the descending aorta, indicating on follow-up images the formation of an aneurysm. Ide et al (12) reported serial changes on follow-up CT scans in 27 patients with intramural hematoma. They observed serial changes of intramural hematoma: four patients (14.8%) showed transition to classic dissection without resolution of intramural hematoma and one case (3.7%) developed an enlarging aneurysm without resolution of hematoma during follow-up. In two of our patients (16.7%), the thickness of the crescentic areas along the aortic wall increased on follow-up images. We thought that these findings represented the pathologic process of aortic dissection, with no communication between the aortic lumen and the dissected channel. Seven other patients of ours who were examined with follow-up CT or transthoracic echocardiography, showed decreased thickness of the aortic intramural hematoma (Fig. 3b). We propose that these findings suggest spontaneous regression of the hematoma.

In a review of the literature, almost all patients with aortic intramural hematoma had long-standing hypertension (4, 5, 8), whereas patients with classical aortic dissection may have had various underlying diseases, such as hypertension, Marfan’s syndrome, syphilis, cystic medial necrosis or trauma (1, 6, 7). Hypertension causes medial degeneration of the elastic tissue of the aorta, and eventually results in intramural vascular rhexis and dissecting aneurysm (6). In our patients, whose age ranged between 22 and 32, there were three cases of Marfan’s syndrome and one patient had a history of trauma. The mean age at which classical aortic dissection was present was, then, less than that of aortic intramural hematoma.

Complications of aortic intramural hematoma include aortic wall rupture and pericardial and pleural effusion. Intramural hemorrhage of the vasa vasorum into the outer layers of the media close to the adventitia may explain the rupture of the aortic wall. Pericardial and pleural effusion may result in increasing the permeability of the aortic wall and impending rupture (1). Fifty percent (2/4) of our patients in whom the ascending aorta was involved experienced pericardial effusion, and pleural effusion occurred in 33% (4/12) of all our patients. These findings correspond to those reported by Yamada et al (4) and Mohr-Kahaly et al (8). In contrast, classical aortic dissection may have more varied and severe complications, such as aortic rupture, pericardial or pleural effusion, aortic regurgitation, or cerebral, spinal, mesenteric, or renal infarction (1). These complications may result from the dissecting process as well as from obstructed major branch vessels arising from the false lumen of the dissected aorta. In patients with aortic intramural hematoma there was no involvement of major branches of the aorta, so there were no complications with the infarcted organ.

The mortality rate reported in the literature for aortic intramural hematoma was 21-30% (4, 5). The cause of death was intrathoracic hemorrhage, ventricular fibrillation or postoperative complications. Robbins et al (5) proposed that aortic intramural hematoma is a life-threatening condition for which early surgery should be considered for all patients with ascending aortic involvement and for any patient with persistent pain. In contrast, in our study only one (8%) of the 12 patients died of intrathoracic hemorrhage and shock. The other 11 patients, including two who had undergone surgery, were successfully treated with antihypertensive therapy and did not experience further symptoms during a
mean follow-up period of 12 months.
From this study, we conclude that aortic intramural hematoma is characterized by its occurrence in older patients with hypertension, a less frequent incidence of complications, and a more favorable outcome than in cases of acute aortic dissection. CT and transesophageal echocardiography provide accurate diagnosis of aortic intramural hematoma and acute aortic dissection and successfully demonstrate the complications which require surgical intervention.

REFERENCES