Detection of Intimal Defect by 64-Row Multidetector Computed Tomography in Patients With Acute Aortic Intramural Hematoma

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Background—Previous pathological and clinical studies demonstrated an intimal defect in patients with acute aortic intramural hematoma (IMH). The purpose of this study was to investigate the prevalence and clinical outcome of intimal defect detected by multidetector computed tomography (MDCT) in patients with IMH.

Methods and Results—We retrospectively analyzed 38 consecutive patients with IMH in whom 64-row MDCT was performed during the acute phase (median, 5 days from the onset). Intimal defect was defined as continuity disruption of the inner layer of thrombosed false lumen, which could be detected by 1-mm axial and longitudinal interactive multiplanar reformulation images. Clinical outcome of intimal defect was assessed in patients with type B IMH (n=32). A total of 48 lesions in 27 (71%) patients were recognized as intimal defects. The incidence of intimal defect was not affected by the timing of MDCT examination (1 to 3 days, 79%; 4 to 7 days, 58%; 8 to 14 days, 75%; P=0.56). In type B IMH, 16 (76%) of 21 patients with intimal defect showed progression (enlargement or progression to aortic aneurysm) in the chronic phase. In contrast, all 11 patients without intimal defect had complete resorption of hematoma. In lesion-based analysis, a depth of intimal defect of ≥5 mm predicted progression with sensitivity, specificity, and positive and negative predictive values of 84%, 95%, 94%, and 86%, respectively.

Conclusions—A considerable portion of patients with IMH showed intimal defect detected by MDCT even in the very early stage, and defects frequently enlarged. Patients with intimal defect should be carefully followed up with serial imaging. (Circulation. 2011;124[suppl 1]:S174–S178.)

Key Words: aorta ■ imaging ■ diagnosis ■ prognosis ■ follow-up studies

The recent introduction of noninvasive diagnostic imaging of the aorta has facilitated the diagnosis of aortic intramural hematoma (IMH), characterized by absence of intimal defect or direct communication between the true and false lumens.1,2 However, the appropriate management of IMH is not as well defined nor as widely understood as that of classic aortic dissection (AD). Much uncertainty is caused by the variable natural history of IMH, which is thought to be a precursor of AD, whereas a considerable number of cases show spontaneous regression.1,3–8 The degree to which IMH overlaps AD in cause, prognosis, and the best treatment requires further investigation.

Several reports have indicated that IMH associated with new development of ulcer-like projection (ULP) has a higher incidence of disease progression, whereas IMH without ULP may suggest a stable disease course.4,5,9 ULP may represent the site of the entry tear, but its existence as a distinct clinical and pathological entity is not fully understood. Moreover, complete identification of intimal defect in the entire aorta has been impossible with conventional imaging modalities. Recently, the 64-row multidetector computed tomography (MDCT) has become widely used and can provide detailed 3-dimensional information, which allows identification of small intimal defect in the entire aorta. A better understanding of the pathophysiology of IMH may lead to successful identification of high-risk patients and proper management of those affected patients before disease progression.10 Accordingly, the purpose of this study was to investigate the prevalence and clinical outcome of intimal defect detected by MDCT in patients with IMH and to determine the diagnostic accuracy of conventional axial images for the purpose of identifying intimal defect.

Methods

Patient Characteristics
A total of 60 patients who were diagnosed by conventional computed tomography (CT) within 48 hours from the onset as having IMH were admitted to our institution between January 2006 and March

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to the acute phase (2 weeks from the onset) and treated medically. We excluded patients who underwent surgical repair within 30 days from the onset. Approval for this study was obtained from the institutional review board.

Initial CT Evaluation
Initial CT scans were performed with a contrast-enhanced technique at the emergency room and were evaluated with conventional axial images. IMH was diagnosed by a crescentic or circular high-attenuation area along the aortic wall without contrast enhancement.\(^1,2\) We included the patients who showed a very small collection of contrast material (localized contrast pooling: LCP) within the aortic wall without apparent flow communication.\(^5,10\)

During the study period, there were 4 patients who showed thrombosed false lumen with apparent ULP in initial axial CT images. The depth of ULP ranged from 6.2 to 20.8 mm. Because the main purpose of this study was to detect intimal defects by MDCT in patients who were diagnosed as having IMH with conventional CT images, we excluded these patients from the analysis.

MDCT Evaluation
Sixty-four-row MDCT scans were performed with a Lightspeed VCT (GE Healthcare, Milwaukee, WI). In this study, intimal defect was defined as continuity disruption of the inner layer of thrombosed false lumen that could be detected by axial and longitudinal interactive multiplanar reformation (MPR) images with a 1-mm slice thickness (Figure 1). Localized contrast pooling in aortic wall, which was frequently connected to true lumen with very small orifice, was not regarded as intimal defect, because it has been considered a pseudoaneurysm and different from intimal defect associated with ULP.\(^10\)

As previously reported,\(^5,9\) we evaluated the number and location of intimal defects in the following: (1) ascending aorta, (2) aortic arch convexity, (3) aortic arch concavity, (4) proximal descending thoracic aorta, (5) mid-descending thoracic aorta, and (6) distal descending thoracic aorta. The aortic diameter was defined as the maximum diameter of the outer contour of the aorta. Width and length of intimal defect were defined as the maximum orifice diameter of the localized blood-filled pouch on axial and longitudinal MPR images, respectively. Depth of intimal defect was defined as the maximum depth of the blood-filled pouch.

Because therapeutic strategy and outcomes were different according to the Stanford classification, clinical outcomes of intimal defect were assessed in only 32 patients with type B IMH. Progression of intimal defect was defined as an increase in size (≥5 mm), progression to overt dissection (typical double-channel aorta with intimal flap) or aortic aneurysm (≥60 mm), and aortic rupture (Figure 2).

Diagnostic Accuracy of Conventional Axial Images in Detecting Intimal Defect
To evaluate diagnostic ability of conventional axial images, we reconstructed axial images with a 5-mm slice thickness from the same data set as MPR images. These images were analyzed by 2 independent readers blinded to the results, and diagnostic accuracy of axial images in detecting intimal defect was evaluated using diagnosis by MPR images as the gold standard. Consensus readout was appended in the case of disagreement.

Statistical Analysis
Categorical variables are described as number and percentage and were compared by the Fisher exact test. Continuous variables are described as mean±SD and were compared by unpaired \(t\) tests. Sensitivity, specificity, and positive and negative predictive values were calculated with a 95% confidence interval (CI). For lesion-based analysis, we classified intimal defects into quartiles on the basis of depth. For additional comparison of the prognostic value of intimal defect size regarding progression and determining cut-off
values, receiver operating characteristic curves were generated, and the areas under the curves (AUCs) were calculated. A probability value <0.05 was considered statistically significant. All analyses were performed with SPSS software (version 17.0, SPSS Inc, Chicago, IL).

### Results

#### Clinical Features of the Study Patients According to the Presence of Intimal Defect

Clinical features of the study patients according to the presence of intimal defect are summarized in Table 1. There were 17 men and 21 women, with a mean age of 68±10 years. Six patients were diagnosed as having Stanford type A IMH, and 32 patients were diagnosed as type B IMH. All patients, including those with type A IMH, were treated medically as previously reported.5,6

A total of 48 lesions in 27 (71%; 95% CI 54 to 85) patients were recognized as intimal defect by MPR images of MDCT. Of these, 20 lesions of 9 patients were identified only with MDCT, which might be considered the so-called microtear.

#### Diagnostic Accuracy of Conventional Axial Images in Detecting Intimal Defect

In 27 patients with intimal defect detected by MPR images, 5-mm axial images correctly identified 18 (67%) patients, but 9 patients were missed. Similarly, in 48 aortic segments with intimal defect, 5-mm axial images correctly identified 28 (58%) segments. The size of intimal defect in the 20 segments that 5-mm axial images could not identify ranged from 3.2 to 9.8 mm. The patient-based diagnostic accuracy of 5-mm axial images for detecting intimal defect was as follows: sensitivity 67% (95% CI 46 to 83), specificity 100% (95% CI 72 to 100), positive predictive value 100% (95% CI 81 to 100), and negative predictive value 55% (95% CI 32 to 77). The aortic-segment-based diagnostic accuracy was as follows: sensitivity 58% (95% CI 43 to 72), specificity 100% (95% CI 98 to 100), positive

### Timing of MDCT and Detection of Intimal Defect

MDCT examinations were performed at a median of 5 days (range, 1 to 12 days) from the onset. Fourteen patients underwent MDCT within 3 days from the onset, 12 underwent between 4 and 7 days, and the other 12 patients underwent between 8 and 12 days. The incidence of intimal defect was similar among the different timings of MDCT examination (1 to 3 days, 79%; 4 to 7 days, 58%; 8 to 14 days, 75%, P=0.56; Figure 3).

### Intimal Defect in Patients With Type A IMH

In 6 patients with type A IMH, all patients showed intimal defect with MDCT, including 1 in ascending aorta (Table 2). Follow-up CT was performed at a median of 11 months (range, 4 to 28 months) from the onset, and there were no patients who showed progression of ULP in the ascending

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### Table 1. Patient Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Intimal Defect (+) (n=27)</th>
<th>Intimal Defect (-) (n=11)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y, mean±SD</td>
<td>70±9</td>
<td>63±11</td>
<td>0.04</td>
</tr>
<tr>
<td>Gender, male/female</td>
<td>12/15</td>
<td>5/6</td>
<td>0.62</td>
</tr>
<tr>
<td>Stanford type, A/B</td>
<td>6/21</td>
<td>0/11</td>
<td>0.11</td>
</tr>
<tr>
<td>Risk factors, n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>23 (85)</td>
<td>9 (82)</td>
<td>0.57</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>5 (19)</td>
<td>2 (18)</td>
<td>0.68</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>10 (37)</td>
<td>3 (27)</td>
<td>0.43</td>
</tr>
<tr>
<td>Smoking</td>
<td>9 (33)</td>
<td>3 (27)</td>
<td>0.52</td>
</tr>
<tr>
<td>Chronic kidney disease, n (%)</td>
<td>12 (44)</td>
<td>2 (18)</td>
<td>0.12</td>
</tr>
<tr>
<td>Ischemic heart disease, n (%)</td>
<td>5 (19)</td>
<td>2 (18)</td>
<td>0.68</td>
</tr>
<tr>
<td>Prior history of stroke, n (%)</td>
<td>2 (7)</td>
<td>3 (27)</td>
<td>0.13</td>
</tr>
<tr>
<td>Maximal aortic diameter, mm</td>
<td>39±6</td>
<td>35±4</td>
<td>0.07</td>
</tr>
<tr>
<td>Maximal hematoma thickness, mm</td>
<td>11±3</td>
<td>9±2</td>
<td>0.07</td>
</tr>
</tbody>
</table>

**IMH** indicates intramural hematoma.

### Table 2. Numbers and Locations of Intimal Defect According to Involvement of Ascending Aorta

<table>
<thead>
<tr>
<th></th>
<th>Intimal Defect</th>
<th>Progression of Intimal Defect</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Type A IMH patients (n=6)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Locations of intimal defect, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ascending aorta</td>
<td>1 (10)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Aortic arch convexity</td>
<td>1 (10)</td>
<td>1 (14)</td>
</tr>
<tr>
<td>Proximal descending aorta</td>
<td>3 (30)</td>
<td>2 (29)</td>
</tr>
<tr>
<td>Mid descending aorta</td>
<td>2 (20)</td>
<td>2 (29)</td>
</tr>
<tr>
<td>Distal descending aorta</td>
<td>3 (30)</td>
<td>2 (29)</td>
</tr>
<tr>
<td>Total</td>
<td>10 (100)</td>
<td>7 (100)</td>
</tr>
<tr>
<td><strong>Type B IMH patients (n=21)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Locations of intimal defect, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic arch convexity</td>
<td>2 (5)</td>
<td>1 (5)</td>
</tr>
<tr>
<td>Aortic arch concavity</td>
<td>4 (11)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Proximal descending aorta</td>
<td>15 (39)</td>
<td>9 (47)</td>
</tr>
<tr>
<td>Mid descending aorta</td>
<td>11 (29)</td>
<td>5 (26)</td>
</tr>
<tr>
<td>Distal descending aorta</td>
<td>6 (16)</td>
<td>4 (21)</td>
</tr>
<tr>
<td>Total</td>
<td>38 (100)</td>
<td>19 (100)</td>
</tr>
</tbody>
</table>

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**Figure 3.** Relationship between multidetector computed tomography (MDCT) detection of intimal defect and the timing of MDCT examination.
Progression of Intimal Defect in Patients With Type B IMH

All patients with type B IMH were treated medically. In these patients, follow-up CT was performed at a median of 13 months (range, 2 to 36 months) from the onset. Among them, follow-up MDCT was performed in 22 (69%) patients. The proportion of intimal defects that showed progression associated with quartiles of their depth in 38 lesions of 21 type B intramural hematoma (IMH) patients is shown in Figure 4A, and the clinical outcomes of type B IMH patients (n=32) according to the presence of intimal defect are shown in Figure 4B. In 21 type B patients with intimal defect, 16 showed progression, including 3 with thoracic aortic aneurysm and 13 with an increase in size of intimal defect, whereas 5 showed regression of intimal defect. Of these, 1 with intimal defect in the arch convexity underwent surgical repair, and 1 with intimal defect in the proximal descending aorta underwent endovascular stenting. In contrast, all 11 patients without intimal defect in the proximal descending aorta underwent endovascular stenting. In the present study, intimal defect without flow communication were frequently observed, even at a very early stage. Thus, IMH may partially result from an entry tear similar to classic AD rather than bleeding of the vasa vasorum. This may imply another potential pathophysiology of IMH: intimal defect with closed and thrombosed false lumen. Thus, we think that IMH may not be a homogeneous condition but rather a heterogeneous array of pathologies with various potentialities. We previously proposed that the term “AD with closed and thrombosed false lumen” is better than “IMH” for these various pathophysiologies. Considering its heterogeneous pathology, the definition and diagnostic criteria of IMH might be reconsidered in the era of high-resolution imaging modalities.

Discussion

The present study reports the critical role of MDCT in the assessment of IMH and raises an issue about the pathogenesis of IMH. The main findings of this study were as follows. (1) Intimal defect was frequently detected by interactive MPR images of MDCT even at a very early stage. (2) Intimal defect that was ≥5 mm in depth tended to frequently show progression in the chronic phase. (3) Intimal defect was not completely identified by conventional 5-mm axial images in patients with IMH.

IMH has been thought to originate from the rupture of the vasa vasorum and hemorrhage in the medial layer of the aorta, with no blood flow within the media. Thus, the absence of intimal defect and flow communication between true and false lumens has been considered essential for the rigorous diagnosis of IMH. Cases of IMH observed without an intimal defect at autopsy or during surgery support this theory. However, in the present study, intimal defect without flow communication were frequently observed, even at a very early stage. Thus, IMH may partially result from an entry tear similar to classic AD rather than bleeding of the vasa vasorum. This may imply another potential pathophysiology of IMH: intimal defect with closed and thrombosed false lumen. Thus, we think that IMH may not be a homogeneous condition but rather a heterogeneous array of pathologies with various potentialities. We previously proposed that the term “AD with closed and thrombosed false lumen” is better than “IMH” for these various pathophysiologies. Considering its heterogeneous pathology, the definition and diagnostic criteria of IMH might be reconsidered in the era of high-resolution imaging modalities.

New development of ULP, identified during follow-up in patients with IMH, has been thought to be indicative of new intimal disruption and associated with higher incidence of disease progression. Although this process has been believed to be reentry tear formation secondary to hematoma expansion, it is also possible that these patients initially had intimal defect, but the false lumen was completely thrombosed without flow communication between true and false lumens. Likewise, previous study reported that plaque rupture may be the cause of IMH in a previously unrecognized subgroup of patients. If intimal defect is a secondary event followed by bleeding in the medial layer, the incidence of detection of intimal defects by MDCT should increase as the time interval between initial CT and MDCT increases. However, no such increase was observed in the present study. This may indicate that intimal defect exists at the onset of IMH. In the present study, intimal defect frequently enlarged in size, as typical ULP does. Therefore, we think that ULP
development may be closely associated with initial intimal defect.

Endovascular repair appears to be a promising option in patients who demonstrate high-risk features leading to potential rupture.\(^{15}\) Moreover, a recent study reported successful treatment with endovascular stent graft by targeting the intimal plaque rupture site in patients with IMH.\(^{14}\) We previously reported that newly developed ULP in patients with IMH frequently progressed and required surgical or percutaneous aortic repair during follow-up.\(^{5}\) Therefore, if intimal defect enlarges in size as ULP does, prophylactic surgical or endovascular intervention might be effective to prevent future aorta-related events.

**Study Limitations**

Our study has several limitations. First, because our study was based on the retrospective analysis, predetermined time schedule for follow-up imaging was absent. Besides, although MDCT examinations were performed during the acute phase, they were not performed exactly at the onset. However, MDCT examinations were carried out at a relatively early stage (median, 5 days from the onset), and the prevalence of intimal defect did not depend on the timing of CT. Therefore, we think that the intimal defect detected in the present study may reflect, to some extent, the pathogenesis of IMH. Second, in the present study, no patients underwent surgical exploration, and pathological specimens of intimal defect could not be obtained. Besides, the distribution of detected intimal defects in almost whole aorta is different from the primary entry site in classic AD. Therefore, it may be difficult to regard all of the intimal defects detected in IMH patients as the origin of disease process. Third, only 2 patients underwent surgical repair and endovascular stenting in the present study. Therefore, there was no significant difference in clinically “hard” event-free survival rates between patients with and without intimal defect. Besides, 5 patients showed complete resorption of intimal defect. This finding was consistent with a previous study\(^ {16}\) showing variable remodeling processes of focal contrast enhancement within a hematoma in patients with distal IMH. Additional studies with a larger number of patients and longer follow-up are needed.

**Clinical Implications**

On the basis of our data, 64-row MDCT is superior in detecting intimal defect, and a considerable portion of patients with IMH showed intimal defect detected by MDCT even in the early stage. In cases with the depth of intimal defect \(\geq 5\) mm, it may frequently enlarge in size or progress to aortic aneurysm or classic dissection, which are thought to be as risk factors for poor prognosis. Therefore, patients with intimal defect should be carefully followed up with serial imaging. Considering that intimal defects with a depth \(\geq 5\) mm can be missed by conventional axial images, our results suggest that accurate diagnosis of intimal defect in patients with IMH using MDCT must be important for risk stratification, which leads to better clinical management.

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**Disclosures**

None.

**References**

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