Intramural haematoma should be referred to as thrombosed-type aortic dissection

Keiji Uchida, Kiyotaka Imoto, Norihisa Karube, Tomoyuki Minami, Tomoki Cho, Motohiko Goda, Shin-ichi Suzuki and Munetaka Masuda

Cardiovascular Center, Yokohama City University Medical Center, Yokohama, Japan
Department of Surgery, Yokohama City University Hospital, Yokohama, Japan
* Corresponding author. 4-57 Urafune-cho, Minami-ku, PO Box 232-0024, Yokohama, Japan. Tel: +81-45-2615656; fax: +81-45-2619162; e-mail: k_uchida@yokohama-cu.ac.jp (K. Uchida).

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Abstract

OBJECTIVES: Intramural haematoma is defined pathologically as aortic dissection without an intimal tear. We therefore believe that this term is inappropriate as an acute clinical diagnosis, and instead, use the term ‘thrombosed-type acute aortic dissection’. We compared the features of thrombosed-type acute aortic dissection with those of classic dissection.

METHODS: Thrombosed type was defined as aortic dissection without flow in the false lumen of the aorta on contrast-enhanced computed tomography. Surgery was indicated for all cases of type A acute aortic dissection, and central repair operations were performed in 509 patients. We retrospectively studied these patients’ surgical records.

RESULTS: Three hundred and forty-four patients (68%) had classic dissection, and 165 (32%) had thrombosed type. Thrombosed type was associated with a significantly higher mean age (69 vs 60 years, \(P < 0.01\)), a higher incidence of cardiac tamponade (45 vs 28%, \(P < 0.01\)) and a lower incidence of malperfusion (6 vs 35%, \(P < 0.01\)) than classic dissection. Entry tears were located in the ascending aorta and the arch in 74 patients (45%) with thrombosed type. Since 2007, an intimal tear has been confirmed intraoperatively or on computed tomography in 39 (78%) of 50 patients with thrombosed-type aortic dissection. Mortality was significantly lower in patients with thrombosed-type dissection (6%) than in those with classic dissection (13%, \(P = 0.02\)).

CONCLUSIONS: Most cases of intramural haematoma are acute aortic dissections with an intimal tear without re-entry. Intramural haematoma should be referred to as thrombosed-type aortic dissection. Thrombosed type can be easily diagnosed on contrast-enhanced computed tomography and has features distinct from those of classic dissection. Our classification may be useful for the diagnosis of these types of aortic dissection.

Keywords: Acute aortic dissection • Thrombosed type • Intramural haematoma

INTRODUCTION

Aortic intramural haematoma (IMH) was reported to be a variant of aortic dissection [1], but its definitions and therapeutic strategies remain controversial. IMH was originally defined as aortic dissection without an intimal tear or typical moving intimal flap, and it was attributed to rupture of the vasa vasorum [2]. However, recent reports have described cases of IMH associated with an intimal tear [3], conflicting with the conventional definition. Moreover, the management of IMH involving the ascending aorta (type A) remains a matter of debate. Reports from Western countries often recommend early operation [4], whereas Asian groups have obtained good clinical results with medical treatment [5]. We have handled IMH as thrombosed-type aortic dissection and performed emergency surgery, similar to classic type A dissection. We analysed the clinical characteristics and surgical findings of these conditions to achieve a better pathophysiological understanding of IMH.

MATERIALS AND METHODS

We defined ‘thrombosed-type acute aortic dissection’ as aortic dissection without flow in the false lumen of the aorta on contrast-enhanced computed tomography, a thickened aortic wall, or crescentic or circular areas along the aortic wall. Localized ulcer-like projections of contrast medium into the false lumen, indicating an intimal tear, were included in this group (Fig. 1).

From August 1994 to April 2012, we performed emergency central aortic repair operations in 509 patients with type A acute aortic dissection. We categorized these patients into classic...
Acute aortic dissection usually refers to cases within 2 weeks after symptom onset. In our series, nearly all patients came to our hospital within 24 h after symptom onset and were thus in the ‘superacute’ phase.

Statistical analysis was done with the use of Student’s t-test and the χ² test. P-values of <0.05 were considered to indicate statistical significance.

RESULTS

The T group comprised 165 patients (32%), and the C group comprised 344 (68%). The preoperative characteristics of the T group are compared with those of the C group in Table 1. The patients in T group were significantly older, with a slight female predominance. The risk of overall visceral malperfusion was lower in the T group. Spinal-cord ischaemia occurred in a slightly, but not significantly, higher proportion of patients in the T group than in the C group.

In the T group, 113 patients (69%) had pericardial haemorrhage at operation, and 74 (45%) of them had cardiac tamponade. The incidences of pericardial haemorrhage and cardiac tamponade were significantly higher in the T group than in the C group (Table 2). In the T group, the entry was located in the ascending aorta in 51 patients and in the arch in 23. The frequency of no entry in the ascending aorta and the arch was significantly higher in the T group than in the C group (P < 0.01) (Table 3). The size of the intimal tear was significantly smaller in the T group (1.8 ± 1.0 cm) than in the C group (2.9 ± 1.2 cm, P < 0.01).

Operative procedures are presented in Table 4. Simple replacement of the ascending aorta was performed in a higher proportion of patients in the T group than in the C group. Operative mortality was significantly lower in the T group, with no operative death since 2007 (Table 5).

We further investigated intimal tears in the 50 patients in the T group who were treated since 2007, when we started to use multidetector computed tomography. An intimal tear in the ascending aorta or the arch was found at operation in 25 patients. Preoperative computed tomography detected tears in 13 (52%) of these patients. Among the 25 patients who had no entry site in the ascending aorta or the arch, intimal tears in the descending aorta were confirmed in 14 patients on preoperative or postoperative computed tomography. Overall, intimal tears were found in 39 (78%) of the 50 patients.

DISCUSSION

The patients with IMH had severe chest and back pain similar to that associated with classic dissection, but their computed tomographic findings clearly differed. Because the poor resolution of previously available imaging equipment could not detect intimal tears, the pathogenesis of IMH was attributed to rupture of the vasa vasorum [1]. Some pathological studies had supported this notion [6].

Because medical therapy for type A IMH resulted in a high risk of rupture or transition to classic dissection, early graft replacement was recommended in many reports from Western countries [7, 8]. Owing to recent progress in diagnostic imaging,
Table 3: Sites of entry tears at operation

<table>
<thead>
<tr>
<th></th>
<th>T Group</th>
<th>C Group</th>
<th>P-value</th>
</tr>
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<tbody>
<tr>
<td>Ascending aorta (%)</td>
<td>51 (32)</td>
<td>155 (45)</td>
<td></td>
</tr>
<tr>
<td>Aortic arch (%)</td>
<td>23 (14)</td>
<td>95 (28)</td>
<td></td>
</tr>
<tr>
<td>Proximal descending aorta (%)</td>
<td>3 (2)</td>
<td>23 (7)</td>
<td></td>
</tr>
<tr>
<td>Not found (%)</td>
<td>84 (52)</td>
<td>57 (17)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>No record (%)</td>
<td>4 (2)</td>
<td>14 (4)</td>
<td></td>
</tr>
</tbody>
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Table 4: Range of aortic replacement and simultaneous coronary artery bypass grafting (CABG)

<table>
<thead>
<tr>
<th></th>
<th>T Group</th>
<th>C Group</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic root + ascending (%)</td>
<td>6 (4)</td>
<td>27 (8)</td>
<td></td>
</tr>
<tr>
<td>Ascending (%)</td>
<td>148 (90)</td>
<td>234 (68)</td>
<td></td>
</tr>
<tr>
<td>Total arch (%)</td>
<td>11 (7)</td>
<td>79 (23)</td>
<td></td>
</tr>
<tr>
<td>Arch + descending</td>
<td>0</td>
<td>3 (1)</td>
<td></td>
</tr>
<tr>
<td>Aortic root + total arch</td>
<td>0</td>
<td>3 (1)</td>
<td></td>
</tr>
<tr>
<td>Simultaneous CABG (%)</td>
<td>4 (2)</td>
<td>24 (7)</td>
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Table 5: Operative mortality

<table>
<thead>
<tr>
<th></th>
<th>T Group</th>
<th>C Group</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality (total) (%)</td>
<td>10/165 (6)</td>
<td>45/344 (13)</td>
<td>0.02</td>
</tr>
<tr>
<td>1994–2000 (%)</td>
<td>2/51 (4)</td>
<td>17/90 (19)</td>
<td>0.01</td>
</tr>
<tr>
<td>2001–06 (%)</td>
<td>8/64 (13)</td>
<td>21/108 (19)</td>
<td>0.49</td>
</tr>
<tr>
<td>2007–12 (%)</td>
<td>0/50</td>
<td>7/118 (6)</td>
<td>0.09</td>
</tr>
</tbody>
</table>

many surgeons have become aware of the existence of intimal tears in some patients with IMH. Kouchoukos et al. reported a case of ‘intramural haematoma associated with an intimal tear’ [3]. It was a precious case report, but the title seemed to conflict with the definition of IMH. The use of the term ‘penetrating atherosclerotic ulcer’ as a pathological diagnosis created further confusion clinically [9, 10]. This term means a localized intimal defect, which is depicted as an ulcer-like projection on contrast-enhanced computed tomography in patients without dissection. Such intimal defects may become an entry tear in acute aortic dissection. It is considered inappropriate to diagnose acute aortic dissection as a penetrating atherosclerotic ulcer.

In our series, at least 78% of all cases of thrombosed-type aortic dissection had intimal tears. Because computed tomography cannot demonstrate tears in all patients, intimal tears are probably present in a higher proportion of patients. We have no data to refute rupture of the vasa vasorum, but the occurrence of this pathophysiological mechanism appears to be rare.

Our operative data showed that the size of the intimal tear was smaller in the T group than in the C group. We previously reported on the site of dissection in patients with IMH [11]. When compared with classic dissection, the dissection in IMH was located nearer the adventitial side of the media of the aortic wall. The mechanism of IMH may be as follows: when small intimal tears occur, and dissection develops on the side of the adventitia, re-entry to the true lumen is negligible, leading to a high probability of rupture. Finally, the false lumen is thrombosed.

Surgical results in the T group have been excellent in recent years. In addition to improved surgical techniques, good results were ascribed to the fact that patients in the T group had a very low incidence of visceral malperfusion, which is a serious risk factor in patients undergoing surgery for acute aortic dissection. The T group might have included patients with IMH who had no entry site. Lesions of the ascending aorta might be retrograde dissections resulting from tears in the descending aorta. In some patients, the tear may be so small that it undergoes complete thrombosis and heals after aggressive blood pressure control [12]. Operation would not be necessary for such patients, but during the superacute phase, it is difficult to distinguish this subgroup of patients from those who require emergency surgery. Given the excellent results of surgical treatment, the strategy of emergency operation for all patients with superacute type A thrombosed aortic dissection appears to be acceptable. Ideally, however, unnecessary operation should be avoided. If strict conservative treatment (medical treatment and surveillance) is a viable option in the future, such treatment should probably be indicated for patients with no motion artefacts due to heartbeats on high-resolution, multidetector computed tomography as well as no evidence of a tear in the ascending aorta or aortic arch or of pericardial effusion.

Most cases of IMH are acute aortic dissections with intimal tears without a re-entry site. IMH should therefore be referred to as ‘thrombosed-type acute aortic dissection’. Thrombosed type can be easily diagnosed on contrast-enhanced computed tomography and has features distinct from those of classic dissection. Our classification may be useful for the diagnosis of these types of aortic dissection. Surgical indications should be considered from this point of view.

Conflict of interest: none declared.

REFERENCES

APPENDIX. CONFERENCE DISCUSSION

Dr J. Bekkers (Rotterdam, Netherlands): You described the diagnosis of an acute dissection with a thrombosed false lumen as a CT scan diagnosis in which it is impossible to distinguish this phenomenon from an intramural haematoma without an intimal tear or without a visible intimal tear, at least that is what I understood from the abstract. I think the figures you presented here are a little bit different. In about half of the patients, you were not able to detect the intimal tear in the ascending aorta or in the aortic arch. Indeed, just a few days ago I operated on a patient with a similar CT scan image and he, as you showed, had only a very small (about 1 cm) intimal tear just at the junction of the ascending aorta and the aortic arch. So I confirm what you said from my own experience.

I do have a few questions for you. When you open up the aorta when operating on these patients and you are not able to identify any intimal tear, does that influence the further conduct of your operation? Does that influence the amount of aorta you resect and are going to replace?

Dr Uchida: When we don’t find an intimal tear in the operation, we just replace the ascending aorta for treating cardiac tamponade or coronary dissection.

Dr Bekkers: You are probably aware that there are some reports, and I think it was a Japanese series, in which they advocated conservative management of patients who had what they called an intramural haematoma. Do you see any place for conservative treatment in these patients, for instance, when there is no sign of pericardial tamponade?

Dr Uchida: How to treat these patients is a serious problem. We performed an emergency operation for all cases of type A intramural haematoma, but some papers from Japan and Korea strongly suggest not for all patients. I know that some patients with intramural haematoma do not need an operation, but in the superacute phase, we cannot distinguish the risky patients from other patients. So we performed an emergency operation for all cases. In the future, for example, with multidetector CT and ECG synchronized for a more precise ascending aorta examination, retrograde type A intramural haematoma might not need an operation.

Dr Bekkers: And my final question, in your series, patients with this thrombosed false lumen seemed to have better results and outcome. Do you have any explanation for that? I notice there was less malperfusion, for instance, in those patients. Could that be an explanation?

Dr Uchida: I previously measured the pressure of the false lumen. The pressure of the false lumen is low in every case. So malperfusion does not occur so often and simple replacement of the ascending aorta is enough for these patients, so the operative mortality is low.

Dr R. de Paula (Rome, Italy): Do you think that changing the definition of intramural haematoma to thrombosed-type acute dissection will change generally how people approach this disease, like doing more operations or waiting less? What do you think?

Dr Uchida: You mentioned about the surgical indication for intramural haematoma?

Dr De Paula: Yes.

Dr Uchida: In our series, the incidence of rupture was more frequent in intramural haematoma. In the superacute phase, we are safe to operate on every patient, but if the patient comes to our hospital two or three days later, we don’t operate on these patients.

Dr M. Grimm (Innsbruck, Austria): You pointed out the case of an intramural haematoma of the descending aorta with retrograde expansion into the ascending aorta. When you find a radiographic hint of a potential intimal tear in the distal arch, what is your therapeutic strategy in these patients now? If you have a type B intramural haematoma, expansion downstream to the bifurcation or whatever, and you have a retrograde expansion of the haematoma also in the ascending aorta, according to your new classification, this would require aggressive surgery, as in a type A dissection, also in line with the guidelines, and if you now find an intimal tear that is not causing dissection but haematoma in the distal arch, what is your therapeutic strategy in these patients?

Dr Uchida: Retrograde type A intramural haematoma sometimes causes cardiac tamponade. Our policy in the superacute phase is that we must operate on those patients.

Dr S. Takamoto (Tokyo, Japan): I always say that IMH is a pathological terminology. I think it should not be used clinically. We don’t know the exact cause of an intramural haematoma. Rupture of vasa vasorum? No one knows. But we could know just one simple thing: this is that the false lumen in aortic dissection is not imaged by the contrast medium. That is a diagnosis of the intramural haematoma. The important thing is the status of the false lumen. Sometimes thrombus in the false lumen is moving a little, sometimes it is completely fixed. In such a case, I think conservative therapy could be implemented, but if the thrombus is moving in the false lumen, surgery might be indicated. Therefore the terminology “intramural haematoma” might mislead the physicians.

Dr Laufer: I’m sorry, you have to come to the point because of time.

Dr Takamoto: I would say that the Japanese guideline of aortic dissection is that we have determined to abandon using IMH. I want to propose to all the audience and all the European surgeons not to use IMH. That misleads the physicians and patients.