Prognosis of Retrograde Dissection From the Descending to the Ascending Aorta

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Background—Natural history of aortic dissection (AD) with intimal tear in the descending or abdominal aorta and retrograde extension into the ascending aorta (retrograde AD) remains unknown. The purpose of this study was to elucidate medium-term prognosis of patients with retrograde AD.

Methods and Results—Study population consisted of 109 patients with acute type A AD. There were 27 patients (25%) with retrograde AD and 82 patients (75%) with intimal tear in the ascending aorta (antegrade AD). In antegrade AD patients, 60 patients underwent surgery and 22 patients were treated medically. In retrograde AD patients, 14 patients showed localized crescentic high attenuation area along the ascending aortic wall without enhancement in computed tomography. Transesophageal echocardiography revealed complete thrombosis of false lumen (FL) in the ascending aorta (retrograde thrombosed). The remaining 13 patients showed incomplete or no thrombosis (retrograde nonthrombosed). All retrograde nonthrombosed AD patients underwent surgery except for 1 patient with stroke, whereas all retrograde thrombosed AD patients were treated medically. In-hospital mortality rate of retrograde AD patients was significantly lower than that of antegrade AD patients (15% versus 38%, P=0.027). The survival rates in retrograde AD patients were all 85% at 1, 2, and 5 years, which were significantly higher than those of antegrade AD patients (63%, 62%, and 57%, respectively)(P=0.009).

Conclusions—Patients with type A retrograde AD have better medium-term prognosis than patients with antegrade AD. Retrograde AD patients with thrombosed FL in the ascending aorta could be treated medically with timely surgical repair. (Circulation. 2003;108[suppl II]:II-300-II-306.)

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

Dissections of the ascending aorta usually have a tear in the intima of the proximal aorta adjacent to the coronary arteries.1 These dissections extend in an antegrade direction to the aortic arch, then usually continue into the more distal aorta. On the other hand, dissections limited to the descending thoracic aorta usually have a tear in the intima of the aorta adjacent to the origin of the left subclavian artery. These dissections also extend in an antegrade direction, and they frequently continue into the abdominal aorta. However, it has been reported that aortic dissection (AD) might develop not only in an antegrade but also in a retrograde fashion.2−5 An increasing number of descending thoracic aortic dissection and retrograde extension of the dissection into the ascending aorta (retrograde AD) were identified and simultaneous repair of the ascending aorta and arch was recommended by several authors.6−9 The precise prevalence of this subtype is not clear. Lansman et al7 reported 7% of type A AD were of this type and Kazui et al10 reported 20% of type A AD were confirmed as of this type at the time of operation.

However, proper management of descending thoracic AD extending into the ascending aorta is controversial. Although the standard approach for a dissection involving the ascending aorta (Stanford type A) is nowadays surgical repair,2 a number of distal complications occurring in concurrent descending AD originating from an intimal tear in the descending aorta or abdominal aorta are difficult to manage through a median sternotomy.5,10,11 In addition, it has been suggested that primary medical therapy should be considered for patients with retrograde dissection with small ascending aorta, thrombosed false lumen in the ascending aorta, and neither pericardial effusion, aortic regurgitation, nor distal complication of the descending dissection.5 Controversy surrounding its prognosis and treatment exists and clinical features and medium-term prognosis remains unknown. The purpose of this study was to elucidate clinical pictures and medium-term prognosis of patients with retrograde AD.

Methods

Patient Characteristics

From 1988 to 2000, 109 patients were admitted to our institutions for acute type A AD. The extent of dissection was diagnosed with computed tomography (CT) in all patients. In addition, the entry site

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was identified by transesophageal echocardiography (TEE) in 57 patients and conventional cine angiography in 49 patients. In TEE, the entry site was defined by a disruption of the dissected membrane or by a typical communication between the two lumens on color Doppler echocardiography. In angiography, the entry site was identified by the shunting of contrast material between the true and false lumens. The diagnosis and the primary entry site were confirmed either by operation or by autopsy. Retrograde type A AD with a tear in the descending aorta and double-channel aorta with dissecting membrane in the descending aorta (A) and the abdominal aorta (B). One month after the onset, follow-up study revealed complete resolution of false lumen in the ascending aorta and increase in size of false lumen in the descending aorta (C). This patient underwent graft replacement of the descending aorta and follow-up study revealed no change in the ascending aorta 4 years after the onset (D).

Figure 1. An example of retrograde aortic thrombosed dissection. Initial computed tomography showed localized segmental, crescentic high attenuation area along the aortic wall without enhancement in the ascending aorta and double-channel aorta with dissecting membrane in the descending aorta (A) and the abdominal aorta (B). One month after the onset, follow-up study revealed complete resolution of false lumen in the ascending aorta and increase in size of false lumen in the descending aorta (C). This patient underwent graft replacement of the descending aorta and follow-up study revealed no change in the ascending aorta 4 years after the onset (D).

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Treatment

Type A Antegrade

For all patients with antegrade AD, surgical intervention was strongly recommended. Sixty (73%) patients underwent emergent (within 24 hours) surgical repair and the remaining 22 (27%) patients were treated medically (Figure 3). Reasons for selecting medical treatment for the patients were refusal of surgery (n=4, 5%), high-risk surgical candidates (n=12, 15%) including 9 patients complicated with stroke and 3 high age patients, and death just after admission (n=6, 7%). Forty-two patients had ascending aorta replacement only; 7 had a hemiarch replacement, 11 had ascending aorta plus total arch replacement.

Type A Retrograde Thrombosed

All patients with retrograde thrombosed AD were treated medically. The initial therapeutic goal during the acute phase included the elimination of pain and the reduction of systolic blood pressure to 100 to 120 mm Hg. Percardiocentesis was performed on admission in two patients complicated by cardiac tamponade. These patients were treated medically after the pericardiocentesis. 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 the increase in size of false lumen or aortic enlargement during the follow-up period were referred for surgical repair and underwent urgent operation. CT scans were then obtained twice or three times a year during the follow-up period.

Figure 2. Example images of 2 patients with retrograde nonthrombosed aortic dissection. Initial computed tomography showed false lumen with no thrombus formation (A, B) and incomplete thrombus formation in the ascending aorta (C, D).
Type A Retrograde Nonthrombosed

All patients with retrograde nonthrombosed AD underwent surgical repair except for 1 complicated with severe stroke. Seven patients had ascending aorta replacement only; 2 had a hemiarch replacement, 3 had ascending aorta plus total arch replacement. As a result, the primary intimal tear was resected in 2 patients.

Statistical Analysis

All values are expressed as mean ± 1 SD. 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’s t-test. Comparison of aortic diameter and diameter of false lumen between on admission and follow-up was done with Student’s 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. A probability value <0.05 was considered statistically significant.

Results

Table 1 summarizes the clinical features of all patients. Mean age of patients with retrograde AD was significantly lower than that of patients with antegrade AD.

In retrograde thrombosed AD patients, 1 showed increase in size of false lumen and aortic enlargement. This patient

TABLE 1. Patient Characteristics, In-hospital Mortality and Late Death

<table>
<thead>
<tr>
<th></th>
<th>Thrombosed (n=14)</th>
<th>Non-thrombosed (n=13)</th>
<th>Total (n=27)</th>
<th>Antegrade AD (n=82)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Basic Characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>54±11*</td>
<td>55±14*</td>
<td>54±12†</td>
<td>62±12</td>
</tr>
<tr>
<td>Gender, male/female</td>
<td>10/4</td>
<td>11/2</td>
<td>21/6</td>
<td>48/34</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>14 (100%)</td>
<td>10 (77%)</td>
<td>24 (89%)</td>
<td>71 (87%)</td>
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<tr>
<td>Diabetes Mellitus, n (%)</td>
<td>1 (7%)</td>
<td>0 (0%)</td>
<td>1 (4%)</td>
<td>7 (9%)</td>
</tr>
<tr>
<td>Hyperlipidemia, n (%)</td>
<td>2 (14%)</td>
<td>2 (15%)</td>
<td>4 (15%)</td>
<td>3 (4%)</td>
</tr>
<tr>
<td>History of smoking, n (%)</td>
<td>9 (64%)</td>
<td>9 (69%)</td>
<td>18 (67%)</td>
<td>39 (48%)</td>
</tr>
<tr>
<td>Cerebrovascular disease, n (%)</td>
<td>1 (7%)</td>
<td>0 (0%)</td>
<td>1 (4%)</td>
<td>1 (1%)</td>
</tr>
<tr>
<td>Ischemic heart disease, n (%)</td>
<td>2 (14%)</td>
<td>1 (8%)</td>
<td>3 (11%)</td>
<td>5 (6%)</td>
</tr>
<tr>
<td>Hemodialysis, n (%)</td>
<td>1 (7%)</td>
<td>0 (0%)</td>
<td>1 (4%)</td>
<td>2 (2%)</td>
</tr>
<tr>
<td>In-hospital mortality, n (%)</td>
<td>1 (7%)*</td>
<td>3 (23%)</td>
<td>4 (15%)*</td>
<td>31 (38%)</td>
</tr>
<tr>
<td>Follow-up variables, n (%)</td>
<td>n=13</td>
<td>n=10</td>
<td>n=23</td>
<td>n=51</td>
</tr>
<tr>
<td>Mean systolic BP&gt;140 mm Hg</td>
<td>4 (31%)</td>
<td>1 (10%)</td>
<td>5 (22%)</td>
<td>22 (43%)</td>
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<td><strong>Antihypertensive therapy</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>β-blocker</td>
<td>8 (62%)*</td>
<td>4 (40%)</td>
<td>12 (52%)*</td>
<td>21 (24%)</td>
</tr>
<tr>
<td>Ca²⁺ antagonist</td>
<td>10 (77%)</td>
<td>7 (70%)</td>
<td>17 (74%)</td>
<td>34 (67%)</td>
</tr>
<tr>
<td>ACE inhibitor</td>
<td>6 (46%)</td>
<td>3 (30%)</td>
<td>9 (39%)</td>
<td>18 (35%)</td>
</tr>
<tr>
<td>α-blocker</td>
<td>5 (38%)</td>
<td>1 (10%)</td>
<td>6 (26%)</td>
<td>7 (14%)</td>
</tr>
<tr>
<td><strong>Cause of late death, n (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rupture of chronic dissection</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>5 (10%)</td>
</tr>
<tr>
<td>Others</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>Total</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>6 (12%)</td>
</tr>
</tbody>
</table>

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

*P<0.05 versus Antegrade AD; † P<0.005 versus Antegrade AD.
underwent graft replacement of the ascending aorta 61 days after the onset. In the remaining 13 patients, size of false lumen decreased and complete resolution of false lumen in the ascending aorta occurred in 8 patients. Aortic measurements could be obtained in 11 (85%) patients (mean 40 months after the onset) in both initial and follow-up imaging study. In these patients, the maximum diameter of false lumen in the ascending aorta significantly decreased (from

11

mm to 2±3 mm, \( P=0.0008 \)) (Figure 4). In addition, maximum aortic diameter significantly decreased (from 47±6 mm to 42±4 mm, \( P=0.001 \)) (Figure 4). However, 4 patients showed descending aortic dilatation and these patients underwent graft replacement of the descending aorta (mean 46 days, ranged from 2 to 67 days, after the onset).

Operative mortality and morbidity are summarized in Table 2. The causes of death in retrograde AD patients were uncontrollable anastomotic bleeding and cardiac failure due to myocardial ischemia. The causes of death in antegrade AD patients were: cardiac failure because of myocardial ischemia in 3, stroke in 2, uncontrollable anastomotic bleeding in 4, and multigorgan failure in 5.

There was no significant difference in the length of hospital stay between retrograde and antegrade AD patients (52±35 versus 40±38 days, \( P=0.181 \)). There were 3 early (within 30 days) deaths in retrograde AD patients and 28 deaths in antegrade AD patients. The 30-day mortality rate of retrograde AD patients was 11%, which was significantly lower than that of antegrade AD patients (34%, \( P=0.021 \)). Similarly, the overall in-hospital mortality rate of retrograde AD patients was significantly lower than antegrade AD patients (Table 1, \( P=0.027 \)). Patients were followed from 2 to 166 months, with a mean of 61 months. There was no significant difference in follow-up periods between retrograde and antegrade AD patients (61±48 versus 61±43 months, \( P=0.979 \)). No patient with retrograde thrombosed AD underwent aortic reoperation. However, 4 antegrade AD patients and 2 retrograde nonthrombosed AD patients underwent aortic reoperation. Actuarial freedom estimates from aortic reoperation in retrograde nonthrombosed AD patients were 89±11%, 71±18%, and 71±18% at 1, 2, and 5 years; whereas those in antegrade AD patients were 98±2%, 95±3%, and 93±4%, respectively (\( P=0.074 \)). There were no late death in retrograde AD patients and 5 late deaths in antegrade AD patients (Table 1). The actuarial survival rates in retrograde nonthrombosed AD patients were all 77±12% at 1, 2, and 5 years, which were not significantly different from those in antegrade AD patients (63±5%, 62±5%, and 57±5%, respectively) (\( P=0.240 \), Figure 5). However, the actuarial survival rates in retrograde thrombosed AD patients were all 93±7% at 1, 2, and 5 years, which were significantly higher than those in antegrade AD patients (\( P=0.011 \)). As a result, the actuarial survival rates in retrograde AD patients were all 85±7% at 1, 2, and 5 years, which were significantly higher than those in antegrade AD patients (\( P=0.009 \)).

Table 3 shows the primary intimal tear sites, extension of the dissecting process and fatal complications. The incidence of moderate to severe aortic regurgitation was significantly lower in retrograde thrombosed AD than antegrade AD (\( P=0.016 \)).

**Discussion**

The main findings of this study were as follows: (1) Type A retrograde AD patients with thrombosed false lumen in the ascending aorta have different clinical features and better prognosis than patients with antegrade AD, whereas retrograde AD patients with nonthrombosed false lumen have similar clinical features and prognosis to patients with antegrade AD. As a result, patients with type A retrograde AD have different clinical features and better prognosis than patients with antegrade AD. (2) Type A retrograde thrombosed AD patients could be treated medically with timed surgical repair and had better medium-term prognosis than patients with antegrade AD.

Although retrograde AD occurs more commonly than is generally realized,\(^3\)\(^\text{-}\)\(^7\)\(^,\)\(^9\)\(^,\)\(^17\)\(^,\)\(^18\) there are few reports about natural history and specific therapeutic problems of patients with retrograde AD.\(^3\)\(^,\)\(^5\)\(^-\)\(^9\) The precise clinical, aortographic, and pathologic features of 6 patients who presented with acute retrograde dissection of the ascending aorta was reported by Cipriano et al.\(^3\) They recommended early surgery for these patients with retrograde AD. Roberts et al\(^9\) presented the largest autopsy series of aortic arch tear associated with ascending aortic dissection. In their study, clinical profile of patients with retrograde AD is quite similar to that of patients with antegrade AD.

**TABLE 2. Operative Mortality and Morbidity**

<table>
<thead>
<tr>
<th>Operative procedure, n (%)</th>
<th>Retrograde AD (n=13)</th>
<th>Antegrade AD (n=60)</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ascending aorta</td>
<td>8 (62%)</td>
<td>42 (70%)</td>
<td>0.532</td>
</tr>
<tr>
<td>Ascending aorta+hemiarch</td>
<td>2 (15%)</td>
<td>7 (12%)</td>
<td>0.657</td>
</tr>
<tr>
<td>Ascending aorta+total arch</td>
<td>3 (23%)</td>
<td>11 (18%)</td>
<td>0.705</td>
</tr>
<tr>
<td>Operative mortality, n (%)</td>
<td>2 (15%)</td>
<td>14 (23%)</td>
<td>0.720</td>
</tr>
<tr>
<td>Operative morbidity, n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bleeding</td>
<td>1 (8%)</td>
<td>6 (10%)</td>
<td>0.999</td>
</tr>
<tr>
<td>Cerebral Infarction</td>
<td>1 (8%)</td>
<td>7 (12%)</td>
<td>0.999</td>
</tr>
<tr>
<td>Renal failure</td>
<td>0 (0%)</td>
<td>6 (10%)</td>
<td>0.538</td>
</tr>
<tr>
<td>Sternal infection</td>
<td>0 (0%)</td>
<td>1 (2%)</td>
<td>0.999</td>
</tr>
</tbody>
</table>

\( AD = \text{aortic dissection.} \)
Erbel et al\textsuperscript{15} reported that there were 22 patients with retrograde dissection in the ascending aorta in a prospective follow-up study of 168 patients with aortic dissection including 52\% type A and 48\% type B AD. Surgical treatment was selected in 21 patients and medical treatment in one patient. Although the patient with medical treatment survived, surgical mortality was 19\% during the operation and 29\% after the operation in their study. They suggested that the poor results in patients with retrograde AD were because of the fact that present surgical techniques could not eliminate flow in the false lumen. Similarly, it has been reported that a patent false lumen correlates closely with variant late complications, including aneurysmal dilatation of the distal aorta, reoperation and aortic rupture.\textsuperscript{19,20} Recent advances in surgical techniques have led to successful one-stage repair of the entire dissected aorta in the acute phase.\textsuperscript{5,10,11,19} However, even when the latest surgical techniques are used, the operative mortality rate for simultaneous aortic arch replacement generally ranges from 13\% to 46\%, and the postoperative morbidity rate is extremely high.\textsuperscript{10,11} In addition, access to the

\begin{table}[h]
\centering
\begin{tabular}{|c|c|c|c|}
\hline
 & Retrograde Thrombosed (n=14) & Retrograde Nonthrombosed (n=13) & Antegrade (n=82) \\
\hline
\textbf{Location of intimal tear, n (\%)} & & & \\
Proximal descending aorta & 7 (50\%)\textsuperscript{\$} & 13 (100\%) & — \\
Distal descending aorta & 3 (21\%) & 0 (0\%) & — \\
Abdominal aorta & 4 (29\%) & 0 (0\%) & — \\
\textbf{Distal extension, n (\%)} & & & \\
Abdominal aorta & 3 (21\%) & 2 (15\%) & 12 (15\%) \\
Common iliac artery & 11 (79\%) & 11 (85\%)\textsuperscript{*} & 43 (52\%) \\
\textbf{Other vessels dissected} & & & \\
Innominate artery & 0 (0\%)\textsuperscript{†} & 4 (31\%) & 13 (16\%) \\
Left carotid artery & 0 (0\%) & 0 (0\%) & 9 (11\%) \\
Left subclavian artery & 1 (7\%) & 0 (0\%) & 8 (10\%) \\
\textbf{Complication, n (\%)} & & & \\
Aortic regurgitation (<=moderate) & 0 (0\%)\textsuperscript{\$} & 5 (38\%) & 25 (30\%) \\
Cardiac tamponade & 2 (14\%) & 2 (15\%) & 22 (27\%) \\
Stroke & 1 (7\%) & 5 (38\%) & 15 (18\%) \\
\hline
\end{tabular}
\caption{Comparison of Primary Intimal Tear Sites, Extension of the Dissecting Process and Fatal Complications Among Patients with Retrograde Thrombosed, Retrograde Nonthrombosed and Antegrade Aortic Dissection}
\end{table}

\footnotesize{*P < 0.05 versus Antegrade AD; †P < 0.05 versus Retrograde nonthrombosed AD; \$P < 0.01 versus Retrograde nonthrombosed AD.
descending thoracic aorta distal to the left subclavian artery by median sternotomy is a problem and the surgical approach selected for repair of the ascending aorta and aortic arch is different from that for repair of a thoracoabdominal lesion. Considering the technical difficulties of simultaneous repair of dissections of the ascending and the descending thoracic aorta, selected patients with retrograde AD might be managed surgically.

In this study, if we divided retrograde AD patients into two groups on the basis of thrombus formation in the false lumen in the ascending aorta, the incidence of moderate to severe aortic regurgitation was less frequent in retrograde thrombosed AD patients than antegrade AD patients. The difference of aortic pathology may have a different impact on clinical course. Therefore, central question remains controversial; should retrograde thrombosed AD patients be initially treated surgically as well as antegrade AD?

In this study, our indications for surgical intervention for retrograde AD were (1) nonthrombosed false lumen in the ascending aorta, (2) enlargement of the ascending aorta and arch, (3) fatal complications including aortic regurgitation and (4) rupture of the affected aorta. As a result, patients with thrombosed false lumen in the ascending aorta were initially treated medically, while 12 of 13 patients with nonthrombosed false lumen in the ascending aorta were treated surgically. This therapeutic approach is similar to that in the report by von Segesser et al. They recommended that descending thoracic aortic dissection extending into the ascending aorta should be managed in accordance with the site of the predominant lesion. They reported that five patients with predominantly distal aortic dissection, small ascending aorta, and absence of fatal complication were successfully treated with medical therapy and that there were no early mortality. In the current study, only 1 patient with retrograde thrombosed dissection underwent graft replacement of the ascending aorta due to aortic enlargement. Early and late results of medically treated patients with retrograde thrombosed dissection were favorable. 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 retrograde AD patients with thrombosed false lumen in the ascending aorta when multiple imaging modalities and surgical backup are always available.

Endovascular treatment with a stent-graft has been recently considered as a less invasive alternative to surgical graft replacement for patients with aortic dissection. Kato et al. reported successful stent-graft repair of type A AD with an entry in the descending thoracic aorta. Ishihara et al. reported successful one-stage repair of the dissected aorta by interpolating a synthetic graft with a self-expandable stent with only a median sternotomy. In this study, no patients were treated with endovascular stent-grafting. Considering the technical difficulties of one-stage repair of the dissected aorta, endovascular stent-grafting of the primary intimal tear may be an alternative to surgical graft replacement in selected patients.

In the setting of classic aortic dissection, cardiac tamponade is the most common cause of death. Isselbacher et al. suggested that pericardiocentesis in treating cardiac tamponade might be harmful rather than beneficial. In the current study, two retrograde thrombosed AD patients complicated with cardiac tamponade were treated with pericardiocentesis and following medical therapy. However, the numbers of patients who underwent pericardiocentesis in this study were small enough and early surgery should be required for these patients when various imaging modalities and urgent surgery are not always available.

There are several potential limitations in our study. First, a tear in the ascending aorta may be missed on imaging modalities due to many factors. In particular, the motion of aortic root may obscure a small intimal tear. Second, it is possible that the patients with a thrombosed false lumen actually had a primary intramural hematoma in the ascending aorta that led to a secondary tear in the descending aorta.

Conclusions

Patients with type A retrograde AD have better medium-term prognosis than patients with type A antegrade AD. In addition, retrograde AD patients with thrombosed false lumen in the ascending aorta could be initially treated medically with selective surgical management in case of complications.

References


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