Retrograde Type A Aortic Dissection After Endovascular Stent Graft Placement for Treatment of Type B Dissection

Zhi Hui Dong, MD; Wei Guo Fu, MD; Yu Qi Wang, MD; Da Qiao Guo, MD; Xin Xu, MD; Yuan Ji, MD; Bin Chen, MD; Jun Hao Jiang, MD; Jue Yang, MD; Zhen Yu Shi, MD; Ting Zhu, MD; Yun Shi, MD

Background—Retrograde type A aortic dissection has been deemed a rare complication after endovascular stent graft placement for type B dissection. However, this life-threatening event appears to be underrecognized and is worth being investigated further.

Methods and Results—Eleven of 443 patients developed retrograde type A aortic dissection during or after stent grafting for type B dissection from August 2000 to June 2007. Of these 11 patients, 3 had Marfan syndrome. The Kaplan–Meier estimate of the rate of freedom from this event at 36 months is 97.4% (95% confidence interval, 0.95 to 0.99). The new entry was located at the tip of the proximal bare spring of the stent graft in 9 patients, was within the anchoring area of the proximal bare spring in 1, and remained unknown in 1 patient. Eight patients were converted to open surgery, and 2 received medical treatment. One patient suddenly died 2 hours after the primary stent grafting, and 2 died within 1 week after the surgical conversion, so mortality reached 27.3%. During the follow-up from 3 to 50 months, type I endoleak was identified in 1 patient 3 months after the surgical exploration and disappeared at 6 months.

Conclusions—Retrograde type A aortic dissection after stent grafting for type B dissection appears not to be rare and results from mixed causes. Fragility of the aortic wall and disease progression may predispose to it, whereas stent grafting–related factors make important and provocative contributions. Avoiding aortic arch stent grafting in Marfan patients, preferably selecting the endograft without the proximal bare spring for patients with a kinked aortic arch or with Marfan syndrome (if endografting is used), improving the device design, and standardizing endovascular manipulation might lessen its occurrence. (Circulation. 2009;119:735-741.)

Key Words: aortic dissection ■ endovascular surgery ■ grafting ■ stents

Endovascular stent graft placement has been used increasingly in the treatment of Stanford type B aortic dissection since its safety and efficacy were reported at the end of the last century. Despite the advantage of low mortality and morbidity, its mid- to long-term durability has continuously been a source of concern. The midterm follow-up data at our center revealed 11 cases with retrograde type A dissection (RTAD) of 443 patients undergoing stent grafting for type B dissection from August 2000 to June 2007; its incidence reached 2.5% and was comparable to that of migration, which was regarded as one of the common complications with an incidence of 0% to 3%. Given that this catastrophic complication appeared not to be rare and lacked systematic investigation, the present study was performed by retrospective analysis on these 11 cases.

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Methods

Patient Demographics and Characteristics

From August 2000 to June 2007, 637 patients with type B dissection were treated in our center. 443 with stent graft placement, 158 medically, and 36 surgically. More than three quarters of the patients were referred from various hospitals throughout China for their complicated conditions, making up a large number of the patients managed at our institution. Among the 443 cases, 112 were in the acute phase, 196 were in the subacute phase, and 135 were in the chronic phase. The acute phase was defined as within 2 weeks after symptom onset; the subacute phase, as the following 2-month period; and the chronic phase, as anything thereafter. The indications for endografting are listed in Table 1. RTAD developed in 11 patients (8 men, 3 women; mean age, 43 ± 8 years; range, 32 to 53 years) intraoperatively or postoperatively, accounting for 2.5%. Of these patients, 10 underwent primary stent grafting during the subacute phase and 1 (patient 8) in the chronic phase after 10 years of medical treatment. Their clinical characteristics are shown in Table 2.

Stent Graft System

The Talent stent graft system (Medtronic, Minneapolis, Minn) was used in all 11 patients. It consists of a segmental nitinol stent skeleton covered with polyester (Dacron) and a straight nitinol connecting bar longitudinally linking the stent graft and preventing twisting or kinking. It has a proximal bare spring with strong radial force for strengthening its fixation and preventing migration. The delivery system contains a covering sheath and a placement catheter over which the self-expanding stent graft was compressed. Deploy
system back to the ideal location and then fully deployed it. If the proximal landing zone, the distance from the origin of the left subclavian artery to the primary entry site, measured <15 mm, 2 strategies were applied to create an extra anchoring area: intentional coverage of the left subclavian artery and right-to-left carotid and left-carotid-to-left-subclavian arterial bypass, both of which have been described in detail. Second, balloon inflation was avoided in all 11 patients.

### Treatment of RTAD

Both surgical and medical treatments were used in this series. The surgical conversion represented graft replacement of the ascending thoracic aorta with or without partial or total aortic arch replacement and was the first choice at the onset of RTAD. Medical management was chosen only if RTAD was limited and the patients remained clinically stable, with β-blockers, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, or calcium antagonists

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**Table 1. Indications for Stent Grafting in 443 Type B Dissection Patients**

<table>
<thead>
<tr>
<th>Indications</th>
<th>Patients, n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dissecting aneurysm</td>
<td></td>
</tr>
<tr>
<td>Maximal diameter &gt;55 mm</td>
<td>83</td>
</tr>
<tr>
<td>Increase of diameter &gt;5 mm/6 mo or &gt;10 mm/12 mo</td>
<td>39</td>
</tr>
<tr>
<td>Maximum aortic diameter ≥40 mm and a patent false lumen in the acute stage&lt;sup&gt;5&lt;/sup&gt;</td>
<td>86</td>
</tr>
<tr>
<td>End-organ ischemia</td>
<td></td>
</tr>
<tr>
<td>Lower extremity</td>
<td>42</td>
</tr>
<tr>
<td>Mesenteric</td>
<td>16</td>
</tr>
<tr>
<td>Renal</td>
<td>11</td>
</tr>
<tr>
<td>Contained rupture</td>
<td>28</td>
</tr>
<tr>
<td>Persistent or recurrent pain</td>
<td>48</td>
</tr>
<tr>
<td>Refractory hypertension</td>
<td>32</td>
</tr>
<tr>
<td>Prophylactic repair for patients with excessive worry about rupture</td>
<td>58</td>
</tr>
</tbody>
</table>

---

**Table 2. Characteristics of 11 Patients Complicated With RTAD After Endografting for Type B Dissection**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, y</th>
<th>Coexisting Conditions</th>
<th>Initial Indication</th>
<th>Stent Graft Oversizing, %</th>
<th>Onset Time</th>
<th>Location of the New Tear</th>
<th>Treatment</th>
<th>Duration of Follow-Up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>53</td>
<td>Hypertension</td>
<td>Recurrent pain</td>
<td>10</td>
<td>1 mo</td>
<td>TPBS</td>
<td>Surgery</td>
<td>18 mo</td>
</tr>
<tr>
<td>2</td>
<td>43</td>
<td>Hypertension</td>
<td>Refractory hypertension</td>
<td>15</td>
<td>6 mo</td>
<td>TPBS</td>
<td>Surgery</td>
<td>50 mo</td>
</tr>
<tr>
<td>3</td>
<td>41</td>
<td>Hypertension</td>
<td>Recurrent pain</td>
<td>10</td>
<td>29 mo</td>
<td>TPBS</td>
<td>Surgery</td>
<td>12 mo</td>
</tr>
<tr>
<td>4</td>
<td>34</td>
<td>Hypertension</td>
<td>Recurrent pain</td>
<td>15</td>
<td>12 mo</td>
<td>TPBS</td>
<td>Surgery</td>
<td>18 mo</td>
</tr>
<tr>
<td>5</td>
<td>52</td>
<td>Hypertension</td>
<td>Refractory hypertension</td>
<td>10</td>
<td>9 mo</td>
<td>TPBS</td>
<td>Surgery</td>
<td>30 mo</td>
</tr>
<tr>
<td>6</td>
<td>32</td>
<td>Marfan</td>
<td>Recurrent pain</td>
<td>10</td>
<td>1 mo</td>
<td>TPBS</td>
<td>Medical</td>
<td>21 d (lost)</td>
</tr>
<tr>
<td>7</td>
<td>39</td>
<td>Hypertension</td>
<td>Refractory hypertension</td>
<td>10</td>
<td>36 mo</td>
<td>TPBS</td>
<td>Surgery</td>
<td>3 mo</td>
</tr>
<tr>
<td>8</td>
<td>53</td>
<td>Hypertension</td>
<td>120-mm-diameter dissecting aneurysm</td>
<td>10</td>
<td>1 wk</td>
<td>TPBS</td>
<td>Surgery</td>
<td>1 d (died)</td>
</tr>
<tr>
<td>9</td>
<td>35</td>
<td>Hypertension</td>
<td>Refractory hypertension</td>
<td>10</td>
<td>Intraoperative</td>
<td>Proximal bare spring–covering area</td>
<td>Medical</td>
<td>16 d (lost)</td>
</tr>
<tr>
<td>10</td>
<td>44</td>
<td>Marfan</td>
<td>Recurrent pain</td>
<td>10</td>
<td>Intraoperative</td>
<td>TPBS</td>
<td>Surgery</td>
<td>5 d (died)</td>
</tr>
<tr>
<td>11</td>
<td>47</td>
<td>Marfan</td>
<td>Recurrent pain</td>
<td>10</td>
<td>2 h</td>
<td>Unknown</td>
<td>Medical</td>
<td>2 h (died)</td>
</tr>
</tbody>
</table>

TPBS indicates tip of the proximal bare spring.
administrated either alone or in combination to maintain the systolic blood pressure <140 mm Hg.

Follow-Up Protocol

Patients were followed up by CT at 1 month, 3 months, 6 months, and yearly thereafter both after the primary endografting and after the treatment of RTAD.

Statistical Analysis

The Kaplan–Meier method was used to estimate the rate of freedom from RTAD, and the Greenwood method was used for the corresponding 95% confidence intervals. The analysis was completed with STATA8.0 software (Stata Corp, College Station, Tex).

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Results

In all 11 RTAD cases, the stent graft was placed at the distal aortic arch. Of the remaining 432 non-RTAD patients, 399 also had arch placement, and 33 had the stent graft placed more distally. Two endografts were implanted with overlapping in patient 8 because of his large entry site of 45 mm and huge dissecting aneurysm. A >20-mm proximal landing zone was achieved in all RTAD patients, and an extra proximal fixing area was created in patients 10 and 11 by intentional coverage of the left subclavian artery and cervical reconstruction, respectively.

Balloon inflation had not been used in any of the RTAD patients, although slight proximal type I endoleak was revealed on the completion angiogram in 3 patients. All patients had good control of their hypertension after stent grafting.

RTAD developed intraoperatively in 2 patients, 2 hours after the procedure in 1 patient, at 1 week in 1 patient, at 1 month in 2 patients, at 6 months in 1 patient, at 9 months in 1 patient, at 12 months in 1 patient, at 29 months in 1 patient, and at 36 months in 1 patient. The Kaplan–Meier estimate of the rate of freedom from RTAD at 36 months was 97.4% (95% confidence interval, 0.95 to 0.99; Figure 1). The clinical manifestations included syncope, hypotension, dyspnea, hypoxemia, chest pain, and sudden death. Patient 11 died suddenly of pericardial tamponade 2 hours after endografting, and RTAD was highly suspected but could not be confirmed by autopsy because, disappointingly, the patient’s family refused consent. In the remaining 10 patients, the new entry site was identified at the tip of the proximal bare spring in 9 patients and within the area where the proximal bare spring anchored in 1 patient by intraoperative angiogram, CT, or surgical conversion.

Eight patients received graft replacement of the ascending thoracic aorta with or without partial or total arch replacement. Two patients were treated medically. Eight patients survived; 6 were followed up for 3 to 50 months (mean, 20 months), and 2 were lost. Type I endoleak was identified in patient 1 at 3 months after the surgical conversion but disappeared at 6 months (Figure 2). In patient 3, a new entry at the distal end of the stent graft that had been detected 1 month before the development of RTAD remained opacified 12 months after the open surgery (Figure 3), but the patient refused to have an additional stent graft implanted. Complete false lumen thrombosis of the thoracic aorta was demonstrated on CT 3 months after the surgical conversion in the remaining 4 patients. Three patients (patients 11, 10, and 8) died perioperatively; mortality reached 27.3%. Patient 11 was described above. Patient 10 was also a Marfan syndrome patient, and the stent graft was intended to be fixed immediately distal to the origin of the left common carotid artery with coverage of the left subclavian artery. During the deployment, the stent graft migrated slightly distal to the anticipated position after its first segment was partially released. We pushed the whole delivery system forward gently and then fully deployed it, achieving the anticipated alignment. However, the completion angiogram demonstrated limited extravasation of contrast at the origin of the

Figure 1. Kaplan–Meier estimate of the rate of freedom from RTAD at 36 months after endovascular stent grafting is 97.4% (95% confidence interval, 0.95 to 0.99).

Figure 2. A, Preoperative CT demonstrated type B dissection with the primary entry site at the aortic isthmus. B, One month after the stent grafting, RTAD developed with a new entry at the tip of the proximal bare spring. C, Three months after the graft replacement of the ascending thoracic aorta, type I endoleak was detected. D, Six months after the surgery, the endoleak disappeared.
innominate artery where the tip of the proximal bare spring reached (Figure 4). Given her hemodynamic stability and difficulty in endovascular management at this site, no immediate intervention was carried out. Two hours later, the patient presented with hypoxemia, and RTAD was detected by transthoracic echocardiography. The patient died of multiple organ failure 5 days after an emergency ascending thoracic aorta and total arch replacement. Patient 8 manifested dyspnea 1 week after the stent grafting, and CT revealed RTAD. Ultimately, he also died of multiple organ failure 1 day after emergency graft replacement of the ascending thoracic aorta and total arch.

Discussion
So far, most description of RTAD after stent grafting for type B dissection has been limited to isolated case reports7–13 or studies evaluating the general effects and complications of endovascular therapy.3,14–32 Its incidence varied from 1.4% to 20%.3,14–32 The Neuhauser et al33 study focused on RTAD but included only 4 dissection and 1 thoracic aneurysm patients. In all these published articles, the question of greatest concern remains unanswered: Is RTAD a true complication of the procedure or the natural progression of the disease itself? Results of this study suggest that RTAD would be attributed to mixed causes, including fragility of the aortic wall and disease progression as the pathological background and stent grafting–related factors as the important provocative aspect.

The fragility of the aortic wall and disease progression were deemed to predispose to RTAD because new type A dissection was not specific to stent graft placement. Rather, it developed after open surgery or even during drug management of type B dissection patients.34–37 Hata et al36 investigated the prognosis of 180 patients with acute type B dissection under medical management, and newly developed type A dissection was observed in 4 cases. Winnerkvist et al37 reported new type A dissection developing in 5 of 66 candidates in a prospective study of medical treatment of acute type B dissection. Furthermore, some of patients with post–stent grafting type A dissection had a spontaneous new entry irrelevant to the endoprosthesis. Scheinert et al29 presented 1 case with the new tear in the ascending thoracic aorta at 2 cm from the aortic valve 3 months after endovascular repair for ruptured pseudoaneurysm related to type B dissec-
tion. These data indicate that type B dissection is inherently vulnerable to progressively involving the ascending thoracic aorta.

Noticeably, 3 patients were diagnosed with Marfan syndrome according to the revised Gent criteria in this series,\(^3\) accounting for up to 27% (3 of 11), similar to that in the published articles (13.5%, 5 of 37).\(^3,16,18,22,26\) Interestingly, it was also observed in our center that post–stent grafting RTAD represented the most common complication among Marfan patients. In total, we have performed endografting in 6 type B dissections with Marfan syndrome, of which 4 had solitary type B dissection and 2 previously underwent the Bentall procedure for type A dissection. Among the former 4 cases, 3 experienced diagnosed RTAD presented in the present survey, and 1 had suspected RTAD; in this patient, satisfactory exclusion of the proximal entry had been achieved with 2 overlapped stent grafts (Talent proximally, Ankura distally), but the patient died suddenly 2 weeks after the procedure when she was discharged home. These findings suggest that Marfan syndrome predisposes to post–stent grafting RTAD, which appears consistent with Marfan’s well-recognized pathological and clinical characteristics, namely the aortic wall fragility and a tendency to progressively involve the residual aorta after either surgical or endovascular treatment for the primarily affected aortic segment.\(^39,40\) From this standpoint, the considerable proportion that Marfan made up among RTAD cases could, to a certain extent, support the inference that the aortic wall fragility and disease progression contribute to the occurrence of RTAD. Nevertheless, only a small number of cases are included in the present study, and the established association between Marfan and RTAD is pending further investigation with a larger sample.

As far as stent grafting–related factors are concerned, the injury from the proximal bare spring is taken into account first, which is designed to open widely without the constraint by polyester graft and to provide strong radial force to strengthen the proximal fixation. However, the stronger the radial force is, the higher the risk of injury on the aortic wall is. By reviewing the published articles,\(^3,7–33\) we found 37 patients who experienced RTAD after endografting for type B dissection; a stent graft with a proximal bare spring was used in at least 27 patients (73.0%),\(^7–10,14–17,19–25,30\) an endoprosthesis without a bare stent was implanted in 2 patients,\(^13,33\) and the type of device was unavailable in the remaining 8 patients. In our series, a prosthesis with a proximal bare spring was used in all 11 patients. Although this type of stent graft might be more readily available and widely used, it is still strongly suggested that the proximal bare spring plays an important provocative role in the development of RTAD, considering the fact that the new entry was located at the tip of the proximal bare spring in 81.8% (9 of 11) of the patients in our series and in 55.6% (15 of 27) of the patients in the published articles.\(^7,9–11,16,17,22,25,30,33\) On the other hand, however, it appears to be fairly rash to establish a specific association between the Talent device and RTAD in the absence of a randomized control study, although it was implanted in all 11 cases in the present survey.

Second, placement of the stent graft at the aortic arch probably correlates with the development of RTAD. In all patients in this series and most patients in the literature, the endograft was placed at the arch. When passively bent at the arch, the self-expanding endoprosthesis has the inherent tendency to spring back to its initial straight status, especially in the presence of the longitudinal connecting bar. Such spring-back strength could generate a stress on the greater curve, particularly at 2 ends of the graft, and create a new entry at one or both ends, such as the occurrence of new tear at both ends of the device in patient 3 (Figures 3 and 5). Generally, the pointed tip of the proximal bare spring could yield higher-intensity pressure than the fabric-covered portion of the graft and would more readily lead to injury, which would be more risky if the angulation is formed between the bare spring and the aortic wall in the setting of kinking of the arch. The overall stiffness of the endoprosthesis might also precipitate aortic wall injury by hampering its optimal arch alignment and intensifying its interaction with the aorta under the high-velocity pulsatile flow. Xu et al\(^46\) and Pitton et al\(^28\) considered the connecting bar to be partially responsible for RTAD because it sacrificed flexibility and increased the overall stiffness. The support spine of the Valiant device (Medtronic), currently used in our center, has been removed to offer better flexibility. Additionally, kinking of the arch could make unstable the deployment strategy of pulling the covering sheath caudal and cause forward and backward movements of the bare spring and resultant injury of the aortic wall. Pamler et al\(^33\) reported 2 cases of RTAD caused by arch kinking.
Third, the injury could result from endovascular manipulation. In patient 9, RTAD was ascribed to a cutting injury by the radiopaque gold marker of the angiographic catheter placed in the ascending thoracic aorta through the left subclavian artery across which the proximal bare spring was intentionally placed that thus pressed the catheter against the aortic wall. Consequently, when the catheter was pulled back, the gold marker protruding a little over the catheter surface generated a cutting force on the greater curve, and a new tear formed. In patient 10, the aortic wall was injured by pushing forward the partially released stent graft. RTAD in 2 patients presented by Won et al and Lee et al was attributed to guidewire-induced injury. Balloon inflation also could cause injury on the wall, particularly when the stent graft is attached proximally on the already involved aorta. During the early injury on the wall, particularly when the stent graft is attached forward the partially released stent graft. RTAD in 2 patients formed. In patient 10, the aortic wall was injured by pushing into the gold marker protruding a little over the catheter surface aortic wall. Consequently, when the catheter was pulled back, the gold marker protruding a little over the catheter surface generated a cutting force on the greater curve, and a new tear formed. In patient 10, the aortic wall was injured by pushing forward the partially released stent graft. RTAD in 2 patients presented by Won et al and Lee et al was attributed to guidewire-induced injury. Balloon inflation also could cause injury on the wall, particularly when the stent graft is attached proximally on the already involved aorta. During the early injury on the wall, particularly when the stent graft is attached forward the partially released stent graft. RTAD in 2 patients formed. In patient 10, the aortic wall was injured by pushing forward the partially released stent graft. RTAD in 2 patients presented by Won et al and Lee et al was attributed to guidewire-induced injury. Balloon inflation also could cause injury on the wall, particularly when the stent graft is attached proximally on the already involved aorta.7,41 During the early years in our center, balloon inflation was used on a regular basis to augment the fixation of the stent graft and to minimize endoleak until it resulted in terrible migration in several cases, which prompted us to investigate whether the instant mild type I endoleak was left without ballooning. Interestingly, spontaneous seal within 3 months was evidenced on follow-up CT in >90% of cases with slight type I endoleak, which had been detected in 17.6% of patients at the completion of the primary stent grafting such as patient 3 (Figure 3). Therefore, balloon inflation might be reasonably spared in most patients except for those with marked endoleak.

In consideration of these causes, some strategies could be conducive to the prevention of RTAD. First, close attention should be paid to patient and device selection. Stent grafting at the aortic arch should be avoided in Marfan patients unless the candidate has had a previous graft replacement of the ascending aorta, arch, or both. The device without the proximal bare spring is recommended when endografting is applied in patients with a kinked aortic arch or with Marfan syndrome (without previous graft replacement). Second, the design of the stent graft system should be improved; in particular, the development of a device specific for dissection is worth considering because currently available devices are designed mainly for atherosclerotic aneurysms. A marked difference exists between stent grafting for dissections and stent grafting for atherosclerotic aneurysms. In atherosclerotic aneurysms, the stable fixation of the stent graft relies on a relatively short but normal proximal and distal neck, which necessitates and tolerates the strong radial force of the stent graft. In contrast, in dissections, the entire length of the stent graft attaches to the aorta; hence, lower radial force is required. Furthermore, the aortic wall, especially the distal landing area, is usually involved by dissection and has worse resistance to the radial force of the device. Therefore, the endoprosthesis designed for dissections calls for lower radial force and higher flexibility. Additionally, given that the stent graft is frequently placed across the distal arch and curved in dissection patients, biomechanical properties are worth taking into account during device design to minimize the potential stress injury provoked by the passively bent endograft. Third, excessive oversizing of the stent graft should be avoided; 10% to 15% appears to be adequate. Fourth, standardized endovascular manipulation cannot be overemphasized. For example, the partially released stent graft should never be pushed forward. Finally, the stent graft ideally should be landed proximally on the uninvolved aortic wall. If it has to be landed proximally on the dissected wall, balloon inflation should be avoided.

Conclusions

RTAD is not rare after engrafting for treatment of type B dissection and could be life threatening. The fragility of the aortic wall and disease progression, combined with stent grafting–related causes, contribute to its occurrence. Careful patient and device selection, improvements in the design of the stent graft system, and standardized endovascular manipulation could be conducive to the prevention of RTAD, and both surgical conversion and medical management appear to be effective treatments.

Disclosures

None.

References

5. Marui A, Mochizuki T, Mitsui N, Koyama T, Kimura F, Horibe M. Toward the best treatment for uncomplicated patients with type B aortic dissection I: dissection–related causes, contribute to its occurrence. Careful patient and device selection, improvements in the design of the stent graft system, and standardized endovascular manipulation could be conducive to the prevention of RTAD, and both surgical conversion and medical management appear to be effective treatments.

Disclosures

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Disclosures

None.
CLINICAL PERSPECTIVE

Retrograde type A dissection after stent grafting for type B dissection is a life-threatening complication. However, it remains underrecognized, and the scattered reports have been based on isolated cases. Thus, we were prompted to initiate a systematic investigation into its cause and prevention through a retrospective analysis of 11 retrograde type A aortic dissections.

The outcomes revealed that its occurrence and mortality rates reached 2.5% and 27.3%, respectively, and that 2 main aspects collectively contributed to its development: fragility of the aortic wall and disease progression as the pathological background, particularly in patients with Marfan syndrome, and stent grafting–related injury provoked by the proximal bare stent graft of the descending thoracic aorta. This study suggests that the stent grafting technique should be carefully considered and that elective stent-graft treatment of type A dissections should be limited to carefully selected patients with Marfan syndrome or other connective tissue disorders. The final strategy is standardized and careful endovascular manipulation.
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**SUPPLEMENTAL MATERIAL**

**Supplemental Methods**

In this supplemental data, we are to present 3 recent cases complicated with retrograde type A aortic dissection (RTAD) following stent-grafting for type B dissection after the submission of our manuscript, given that their clinical features could further support 2 of the main points made in the paper. Their clinical characteristics are shown in Table 1.

Point 1 is that with the disease progression, a new entry tear may spontaneously occur irrelevant to the endograft. Patient 1 presented with acute chest pain 69 months after the stent-graft placement, and CT detected a new entry at the ascending thoracic aorta approximately 2 centimeters proximal to the stent-graft, which was also approved during the surgical conversion. It has also been observed that her new entry and false lumen were located along the lesser curve, different from RTAD caused by the proximal bare spring, in which the new entry and false lumen typically appear at the greater curve where the stress yielded by the passively bent endograft is probably concentrated (Figure 1).

Point 2 is that the kinked aortic arch could increase the risk of RTAD, and the device without the proximal bare spring might be preferable under such circumstances. Patient 3 had RTAD 1 month after the primary stent-grafting, manifesting dyspnea. CT revealed that the angulation was formed between the bare spring and the greater curve because of the obvious kinking of the arch. As a result, the bare stent failed to satisfactorily comply with the curve of the arch and created a new entry (Figure 2), which was also approved by the surgical exploration.

All 3 patients uneventfully recovered after graft replacement of the ascending aorta and partial/total arch and were still followed up.

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**Supplemental TABLE 1. Characteristics of 3 Recent RTAD Patients**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age</th>
<th>Gender</th>
<th>Coexisting Conditions</th>
<th>Initial Indication</th>
<th>Stage at the Endografting</th>
<th>Stent-graft &amp; Oversizing</th>
<th>Onset Time</th>
<th>Location of the New Tear</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>63</td>
<td>Female</td>
<td>Hypertension</td>
<td>Recurrent pain</td>
<td>Subacute</td>
<td>Talent, 15%</td>
<td>69 months</td>
<td>Ascending aorta</td>
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<tr>
<td>2</td>
<td>50</td>
<td>Male</td>
<td>Hypertension</td>
<td>Refractory hypertensive</td>
<td>Subacute</td>
<td>Valiant, 10%</td>
<td>1 week</td>
<td>TPBS</td>
</tr>
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<tr>
<td>3</td>
<td>59</td>
<td>Female</td>
<td>Hypertension</td>
<td>dissecting</td>
<td>Chronic</td>
<td>Valiant, 10%</td>
<td>1 month</td>
<td>TPBS</td>
</tr>
</tbody>
</table>

**TPBS** indicates the tip of the proximal bare spring.
Supplemental Figure 1: A, Patient 1 had RTAD 69 months after the primary stent-grafting, and CT detected the new entry at the ascending thoracic aorta and irrelevant to the stent-graft. B, During the surgical exploration in patient 1, it was approved that the new tear (arrow) was located approximately 2 cm proximal to the device. C, Patient 2 had RTAD 1 week after endografting, and CT clearly showed the proximal bare spring protruded into the false lumen. Compared with patient 1 whose entry and false lumen were at the lesser curve (A), his were located at the greater curve where the stress yielded by the passively bent endograft was probably concentrated. D, During the surgical conversion in patient 2, it was visualized that the bare spring (short arrow) made a huge tear (long arrow) at the greater curve, from which the spring protruded into the false lumen, consistent with the finding on CT.
Supplemental Figure 2: A, The preoperative CT demonstrated the significant kinking of the aortic arch in patient 3 with chronic type B dissection for 10 years. B&C, One month after the endovascular repair, the patient presented with dyspnea, and CT revealed the angulation formed between the proximal bare spring and the greater curve, at which site the spring caused a new entry and resultant RTAD involving the left common carotid artery. D, Three weeks after the graft replacement of the ascending thoracic aorta and total arch, CT showed complete thrombosis in the thoracic false lumen.