From the Society for Vascular Surgery

Stent graft-induced new entry after endovascular repair for Stanford type B aortic dissection

Zhihui Dong, MD,[a] **Weiguo Fu, MD,**[a] **Yuqi Wang, MD,**[a] **Chunsheng Wang, MD,**[b] **Zhiping Yan, MD,**[c] **Daqiao Guo, MD,**[a] **Xin Xu, MD,**[a] and **Bin Chen, MD,**[a] *Shanghai, China*

Background: Stent graft-induced new entry (SINE), defined as the new tear caused by the stent graft and excluding those arising from natural disease progression or iatrogenic injury from the endovascular manipulation, has been increasingly observed after thoracic endovascular aortic repair (TEVAR) for Stanford type B dissection in our center. SINE appears to be remarkably life threatening. We investigated the incidence, mortality, causes, and preventions of SINE after TEVAR for Stanford type B dissection.

Methods: Data for 22 patients with SINE were retrospectively collected and analyzed from 650 patients undergoing TEVAR for type B dissection from August 2000 to June 2008. An additional patient was referred to our center 14 months after TEVAR was performed in another hospital. The potential associations of SINE with Marfan syndrome, location of SINE and endograft placement, and the oversizing rate were analyzed by Fisher exact probability test or *t* test.

Results: We found 24 SINE tears in 23 patients, including SINE at the proximal end of the endograft in 15, at the distal end in 7, and at both ends in 1. Six patients died. SINE incidence and mortality reached 3.4% and 26.1%, respectively. Two SINE patients were diagnosed with Marfan syndrome, whereas there were only 6 Marfan patients among the 651 patients. The 16 proximal SINEs were evidenced at the greater curve of the arch and caused retrograde type A dissection. The eight distal SINEs occurred at the dissected flap, and five caused enlarging aneurysm whereas three remained stable. The endograft was placed across the distal aortic arch during the primary TEVAR in all 23 patients. The incidence of SINE was 33.33% among Marfan patients vs 3.26% among non-Marfan patients (*P* = .016). There was no significant difference in mortality between proximal and distal SINE (25% vs 28.6%, *P* > .99), incidence of SINE between endograft placement across the arch and at the straight portion of descending thoracic aorta (23 of 613 vs 0 of 38, *P* = .39), and the oversizing rate between SINE and non-SINE patients (13% ± 4.5% vs 16% ± 6.5%, *P* = .98).

Conclusions: SINE appears not to be rare after TEVAR for type B dissection and is associated with substantial mortality. The stress yielded by the endograft seems to play a predominant role in its occurrence. It is important to take this stress-induced injury into account during both design and placement of the endograft. (J Vasc Surg 2010;52:1450-8.)

Thoracic endovascular aortic repair (TEVAR) has been increasingly used in the treatment of Stanford type B dissection after its safety and efficacy were reported at the end of the last century.[1,2] Currently, the annual volume of TEVAR in many hospitals outnumbers that of graft replacement, and TEVAR has gradually become the mainstream procedure for complicated type B dissection. On the other hand, the long-term durability of this minimally invasive procedure has continuously remained a source of concern, and whether the stiff stent graft itself would bring about any potential injury on the aorta represents a main concern.

From August 2000 to June 2008, 650 patients underwent TEVAR for type B dissection in our center. Among them, a stent graft-induced new entry (SINE) tear developed perioperatively or during the follow-up in 22 patients, with 6 deaths. SINE was defined as a new tear caused by the stent graft itself, excluding those created by natural disease progression or any iatrogenic injury from the endovascular manipulation. Proximal and distal SINE represented the SINE at the proximal and distal end of the endograft, respectively. Its incidence and mortality reached 3.4% and 26.1%, respectively.

We initiated this study because to date, systematic investigation of this infrequent but high-mortality complication has been lacking. The limited information can only be obtained from solitary case reports[3] or extracted from studies about the general outcomes of TEVAR[4-22] or post-TEVAR retrograde type A dissection (RTAD).[23-25]

METHODS

Data collection and analysis. All-cause new entries after TEVAR were retrospectively collected among 650 patients undergoing TEVAR for type B dissection in our center from August 2000 to June 2008. The data of patients complicated with SINE were extracted for analysis. The proportion of SINE among all-cause new entries and among all TEVAR- or dissection-related deaths was calculated. MEDLINE was searched with the term "aortic dis-
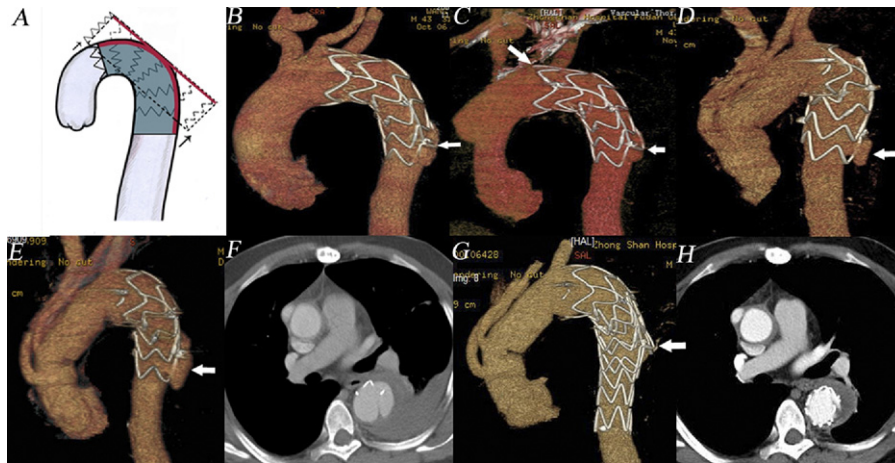
Fig 1. A, When passively bent at the arch, the self-expanding stent graft, like a spring, has the inherent tendency to spring back to its initial straight status, especially in the presence of the longitudinal connecting bar (*red*). Such spring-back strength could yield stress on the greater curve, particularly at two ends of the graft. **B,** In patient 3, computed tomography angiography (CTA) at 28 months detected an asymptomatic distal stent graft-induced new entry (SINE; *arrow*). **C,** CTA at 29 months showed a proximal SINE (*long arrow*) and resultant retrograde type A dissection, and the distal SINE (*short arrow*) remained stable. **D,** At 12 months after the graft replacement of the ascending thoracic aorta and partial arch, the distal SINE expanded (*arrow*). **E and F,** By 27 months after the open surgery, the distal SINE progressively enlarged, leading to contained rupture. **G and H,** At 3 months after an urgent secondary thoracic endovascular aortic repair, CTA evidenced complete seal of the distal SINE (*arrow*) and entire absorption of the pleural effusion.

section" to investigate all-cause new entries after TEVAR and the proportion of SINE among them.

Stent graft systems. Four stent graft systems were used in this series: Talent and Valiant (Medtronic, Minneapolis, Minn), Hercules (Microport, Shanghai, China), and Zenith TX2 (Cook, Bjaeverskov, Denmark). These self-expanding endografts have an inherent tendency to spring back to their initial straight status if passively bent, such as when placed across the aortic arch (Fig 1). Talent, Valiant, and Hercules have a proximal bare spring. Talent and Hercules have a longitudinal connecting bar that prevents twisting and kinking but sacrifices flexibility. Oversizing was calculated according to the diameter from the adventitia to adventitia of the proximal landing zone on computed tomography angiography (CTA).

Endovascular stent graft placement. The general procedure of TEVAR has been detailed elsewhere.[25,26] If the proximal landing zone, the distance from the origin of the left subclavian artery to the primary entry site, measured <15 mm, one of the two strategies would be applied to create an extra anchoring area:

1. Intentional coverage of the left subclavian artery, if it was proven beforehand that the right vertebral artery was patent and the left one was not dominant;
2. Right-to-left carotid plus left carotid-to-left subclavian arterial bypass and proximal ligation of the left carotid and subclavian arteries.

Both strategies have been described in detail.[27] A completion angiogram was performed at the end of the TEVAR to confirm the accurate endograft fixation at the anticipated location and satisfactory exclusion of the primary entry site.

Treatment of SINE. For proximal SINE that unexceptionally caused RTAD, graft replacement of the ascending thoracic aorta with or without partial or total aortic arch replacement was the first choice upon its onset. Medical management was chosen only if RTAD was limited and the patient remained clinically stable.

For distal SINE, medical treatment was preferred upon the diagnosis. The indication for reintervention included persistent enlargement of the false lumen, contained rupture, and becoming symptomatic. Secondary TEVAR was preferred for reintervention. Surgical graft replacement of the descending thoracic aorta was used if a large dissecting aneurysm was causing notable compression that would not be relieved simply by TEVAR. If the reintervention involved the distal descending thoracic aorta, the Adamkiewicz artery would be preoperatively located by CTA, and cerebrospinal fluid (CSF) pressure would be monitored intraoperatively and postoperatively, with CSF drainage if necessary.

Medical management included β-blockers, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, or calcium antagonists administrated alone or in combination to maintain the systolic blood pressure <140 mm Hg.

Follow-up protocol. Patients were followed-up by CTA at 1, 3, and 6 months, and yearly thereafter after the primary TEVAR and after the secondary procedure for SINE. For patients with SINE treated by medicine, CTA was performed at least every 3 to 6 months.

Statistical analysis. The Fisher exact probability test was used to evaluate significant differences in incidence of SINE between patients with Marfan syndrome and

Table. Characteristics of 23 patients complicated with stent graft-induced new entry *(SINE)* after thoracic endovascular aortic repair *(TEVAR)* for type B dissection

Patient	Age (year)	Coexisting conditions	Onset time	SINE symptoms	Device, oversizing	SINE location	Treatment	Follow-up duration
1	53	Hypertension	1 mo	Hypertension, syncope	Talent, 10%	Proximal	Surgery	43 mo
2	43	Hypertension	6 mo	Chest pain	Talent, 15%	Proximal	Surgery	80 mo
3	41	Hypertension	28 & 29 mo	Asymptomatic chest pain, fever	Talent, 10%	Distal proximal	TEVAR (56 mo) Surgery	65 mo
4	34	Hypertension	12 mo	Hypoxia	Talent, 15%	Proximal	Surgery	56 mo
5	52	Hypertension	9 mo	Chest pain	Talent, 10%	Proximal	Surgery	30 mo
6	32	Marfan	1 mo	Chest pain	Talent, 10%	Proximal	Medical	21 d (lost)
7	39	Hypertension	36 mo	Hypoxia	Talent, 10%	Proximal	Surgery	63 mo
8	53	Hypertension	1 wk	Dyspnea	Talent, 10%	Proximal	Surgery	8 d (died)
9	47	Marfan	2 h	Sudden death	Talent, 10%	Proximal	Sudden death	2 h (died)
10	63	Hypertension	3 mo	Chest pain	Valiant, 22%	Proximal	Medical	27 mo
11	50	Hypertension	60 mo	Asymptomatic	Talent, 15%	Proximal	Surgery	75 mo
12	59	None	1 mo	Dyspnea	Valiant, 13%	Proximal	Surgery	19 mo
13	50	Hypertension	1 wk	Chest pain, dyspnea	Valiant, 3%	Proximal	Surgery	17 mo
14	37	Hypertension	3 h	Dyspnea	Hercules, 12%	Proximal	Surgery	9 mo
15	57	Hypertension	3 h	Sudden death	Valiant, 10%	Proximal	Sudden death	3 h (died)
16	35	Hypertension	3 wk	Sudden death	Valiant, 15%	Proximal	Sudden death	3 wk (died)
17	59	Hypertension	3 mo	Asymptomatic	Zenith TX2, 10%	Distal	Medical	17 mo
18	45	Hypertension	3 mo	Asymptomatic	Zenith TX2, 20%	Distal	Medical	16 mo
19	71	Hypertension	15 mo	Asymptomatic	Talent, 18%	Distal and suspected proximal	TEVAR	16 mo (died)
20	64	Hypertension	12 mo	Asymptomatic	Valiant, 15%	Distal	Medical	40 mo
21	53	Hypertension	33 mo	Asymptomatic	Talent, 15%	Distal	TEVAR	36 mo
22	67	Hypertension	3 mo	Chest pain, fever	Valiant, 20%	Distal	Surgery	10 mo (died)
23	58	Hypertension	14 mo	Chest pain	Valiant, 20%	Distal	TEVAR	17 mo

non-Marfan patients, mortality between proximal and distal SINE, and the incidence of SINE between endograft placement across the arch and at the straight portion of descending thoracic aorta. The *t* test was used to analyze the difference in the oversizing rate between SINE and non-SINE patients. A value of *P* <.05 was considered significant. The analysis was completed with STATA 8.0 software (StataCorp, College Station, Tex).

RESULTS

Data for 27 dissection cases complicated with all-cause post-TEVAR new SINE tears have been collected in our center. Among them, 2 (7.4%) were caused by disease progression and diagnosed by follow-up CTA demonstrating the new entry at the proximal ascending thoracic aorta irrelevant to the endograft, 2 (7.4%) resulted from endovascular iatrogenic injury and were revealed by the intraoperative angiogram at the conclusion of the primary TEVAR (one was due to pushing a partially released delivery system forward at the arch, the other was ascribed to the cutting injury by the radiopaque gold markers of the angiographic catheter) and detailed previously,[25] and 23 (85.2%) were attributed to SINE and diagnosed according to the following criteria: (1) CTA or echocardiography showed the new entry located at either end of the endograft, (2) no new entry was detected by the completion angiogram at the end

of the primary TEVAR, and (3) during the surgical conversion for RTAD, the proximal bare spring of the stent graft was seen protruding into the false lumen through the new entry.

Among the 23 SINE cases (18 men, 5 women; mean age, 51 ± 11 years, range, 32 to 71), 22 (17 men, 5 women) underwent the primary TEVAR at our center, accounting for 3.4% among 650 patients, and an additional patient (patient 23) was referred to our center 14 months after TEVAR in another hospital. Of these 23 patients, 19 underwent primary TEVAR during the subacute phase for recurrent chest pain in 6, refractory hypotension in 4, >40-mm-diameter patent false lumen in 5,[28] malperfusion in 3, and contained rupture in 1. The remaining four patients (patients 8, 12, 22, 23) were in the chronic phase, with >10 years of history for >55-mm-diameter dissecting aneurysm. Their clinical characteristics are reported in the Table.

The oversizing rate was 13% ± 4.5% (range, 3%-20%) in SINE patients and 16% ± 6.5% (range, 3%-25%) in non-SINE patients, which was not significantly different (*P* = .98). Two of the 23 patients were diagnosed with Marfan syndrome according to the revised Gent criteria,[29] whereas there were only 6 Marfan cases among all 651 patients, of whom 4 had solitary type B dissection and 2 previously underwent the Bentall procedure for

type A dissection. The proportion of SINE among Marfan patients was 33.33% (2 of 6), which was significantly higher than the 3.26% among non-Marfan patients (21 of 645; $P = .016$).

Our center documented 12 procedure-related or dissection-related deaths after TEVAR for type B dissection. Among them, 6 (50%) were complicated with the new entry, 3 (25%) with stroke, 1(8.3%) with endotension, 1 (8.3%) with rupture, and 1 (8.3%) with stent graft failure to fully open, indicating that the post-TEVAR new entry made up the largest proportion.

A MEDLINE search collected 70 post-TEVAR new entry cases from 27 articles published between January 2002 and January 2009.[3-22,24,30-35] Among them, new entries were proximal to the endograft in 64 patients,[4-21,24,30-35] and were at the distal end of the stent graft in 6.[3,18,20,22] The primary TEVAR was for aortic dissection in 64 patients[3-22,24,30-34] and for descending thoracic aneurysm in 6.[13,17,21,24,35] SINE caused 28 cases (40%),[4-5,7-8,12-15,18,20,22,24,31-34] natural disease progression caused 7,[10,11,13,15,18] and endovascular manipulation-related injury caused 11 (15.7%), including 3 by guidewire[6,30,35] and 8 by ballooning.[9,17,18,24] The cause in the remaining 24 (34.3%) was not indicated. We thus inferred that SINE caused the largest proportion among all-cause post-TEVAR new entry tears, consistent with the data from our center.

The post-TEVAR onset of SINE was a mean of 11 ± 16 months (range, 2 hours-60 months). Proximal SINE developed in 15 patients, distal developed in 7, and 1 had both (patient 3). We hence collected 24 SINE tears in 23 patients, 16 proximal and 8 distal. The clinical manifestations included chest pain, hypoxia, dyspnea, syncope, fever, and sudden death. One proximal and six distal SINEs were asymptomatic when detected by follow-up CTA. All 16 proximal SINEs resulted in RTAD. Of these, 11 were treated with surgical conversion, which resulted in 1 postoperative death of multiple organ failure; 2 were treated medically, and 3 died suddenly of pericardial tamponade. Among 8 distal SINEs, 3 patients were given medical treatment because the false lumen remained stable, patients 3, 19, 21, 23 underwent a secondary TEVAR, and patient 22 received surgical conversion.

Distal SINE was incidentally detected in patient 19 by CTA at 15 months and was successfully excluded by placement of a Hercules endograft (36 × 34 × 160 mm). We were told during a follow-up telephone call 1 month later that he had died suddenly at home 2 days earlier. Despite the absence of an autopsy, we suspected that the proximal SINE and resultant RTAD could have developed subsequent to the secondary TEVAR, which might transfer the stress concentration from the distal to the proximal end of the endograft (Fig 2).

Patient 22 presented with chest pain and fever 3 months after the primary TEVAR for an 80-mm-diameter dissecting aneurysm, and CTA demonstrated the distal SINE. The patient refused immediate reintervention. Six months later, the aneurysm had expanded up to 104 mm and significantly compressed the left lung. Graft replacement of descending thoracic aorta was performed; however, the patient died of multiple organ failure on postoperative day 2. So, there were six deaths in the present series, and the overall mortality reached 26.1%. Mortality was 25% (4 of 16) in proximal and 28.6% (2 of 7) in distal SINE, which was not significantly different ($P > .99$).

Among the 17 survivors, 16 remained free from dissection-related events during a mean follow-up of 38 ± 23 months (range, 9-80 months), and 1 was lost to follow-up. The stent graft in the 23 SINE patients had been placed across the aortic arch. Among the 651 patients, 613 had across-arch endograft placement, whereas in 38, the endoprosthesis was fixed at the straight segment of the descending thoracic aorta. The incidence of SINE was not significantly different between these two groups (23 of 613 vs 0 of 38; $P = .39$).

In patient 3, asymptomatic distal SINE was revealed on follow-up CTA 28 months after the primary TEVAR. He presented with chest pain and fever 1 month later, and CTA demonstrated the proximal SINE and resultant RTAD. He underwent an emergency graft replacement of the ascending thoracic aorta and partial arch. At 41 months, the distal SINE expanded; however, the patient refused a secondary TEVAR. Acute chest pain occurred at 56 months, and CTA evidenced the contained rupture at the level of the distal SINE. A Hercules stent graft (40 × 38 × 120 mm) was urgently implanted, and the follow-up CTA at 3 months revealed satisfactory exclusion of the distal SINE, complete thrombosis of false lumen, and pleural effusion absorption (Fig 1).

In patient 23, the distal SINE was located at the level between T7 and T8, so the stent graft was estimated to be placed distally to the level of T10, raising the risk of spinal cord ischemia. The Adamkiewicz artery, which was located preoperatively by CTA at the level of T9, was unavoidably sacrificed during the secondary TEVAR. CSF pressure intraoperatively was 12 cm H_2O before deployment, increased to 19 cm H_2O directly after the stent graft was released, and then decreased to 14 cm H_2O after 8 mL of CSF was drained. Monitoring was continued for 48 hours after TEVAR and drainage was repeated twice, maintaining the pressure between 9 and 13 cm H_2O. The patient was free of neurologic events perioperatively and during 3 months of follow-up.

DISCUSSION

A variety of devices have been designed for TEVAR since the end of last century. The ability of the device to self-expand is a common basic feature that might create stress on the aortic wall in two manners. One is its radial force that, to a large extent, depends on how much the endograft had been oversized. The other is the "springback" force, by which we mean that the endograft, like a spring, has an inherent tendency to spring back to its initial straight status when passively bent at the aortic arch, thus generating stress on the greater curve, especially at its two ends (Fig 1). This situation exists in most
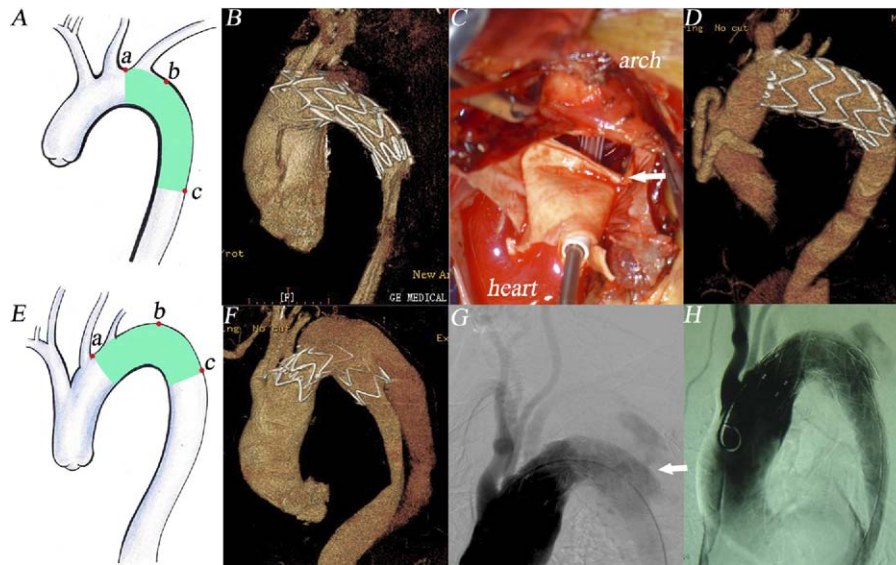
Fig 2. A, The stent graft is frequently placed across the aortic arch in type B dissection patients, and the proximal end (*dot a*) of the endograft (*green shade*) is usually much closer to the curving point (*dot b*) than the distal one (*dot c*); that is to say that *segment ab* is shorter than *segment bc*, probably making the stress more concentrated on its proximal tip and causing the proximal stent graft-induced new entry (SINE). **B,** As occurred in patient 13, the proximal SINE developed 1 week after thoracic endovascular aortic repair (TEVAR). **C,** During the surgical conversion, it was evidenced that the proximal bare spring (*arrow*) created a new entry and protruded into the false lumen. **D,** Twelve months after the David procedure and semiarch replacement, computed tomography angiography showed a stable arch and expanded distal true lumen. **E,** If *segment ab* were longer than *segment bc*, the new entry would probably develop at the distal end. **F,** As occurred in patient 19, the distal SINE occurred 15 months after TEVAR. **G,** The angiogram during the secondary TEVAR clearly revealed the distal SINE (*arrow*). **H,** An additional stent graft was implanted with distal overlapping with the original one, which, however, might change the distribution of the stress and transfer its concentration from the distal to the proximal end of the endograft, possibly causing the subsequent proximal SINE and resultant retrograde type A dissection, which was suspected to be responsible for his sudden death 1 month later.

type B dissections because the primary entry site frequently occurs adjacent to the aortic isthmus, necessitating the across-arch placement of the endograft, which was seen in 613 (including 23 SINEs) of the 651 patients in our center.

Even though the statistical analysis failed to show a significant difference in incidence of SINE between across-arch placement (3.8%, 23 of 613) and placement at a straight aortic segment (0 of 38), possibly because of the relatively small number of the patients in the latter group and the low incidence of SINE, the spring-back force was considered an important factor in the stent graft-induced injury, given that no SINE was observed in the group that did not undergo across-arch placement and the common feature of the "spring" to spontaneously turn back to straight if passively curved. It could also be estimated that the more the endograft is bent, the higher the stress might be, and that the stress would be stronger at the end closer to the curving point of the endograft than at the other end (Fig 2).

As for the formation of the proximal SINE, the stress from the spring-back force would contribute more than that from the radial force because:

1. All proximal SINE tears occurred at the greater curve, consistent with the route of the spring-back movement of the endograft.
2. Typically, the proximal end of the endograft is much closer to the curving point than the distal one, thus probably making the stress more concentrated on the proximal tip (Fig 2).
3. Even though the radial force was minimized through a very limited oversizing, such as only 3% in patient 13, proximal SINE still developed. Also, there was no significant difference in the oversizing rate between the SINE group and non-SINE group.
4. The injury by the strong radial force of the proximal bare spring has always been regarded as the predominant cause for proximal SINE.

Nevertheless, proximal SINE had also been observed in patients treated with the device without the bare spring.[18,23,24,36] In particular, a large-sample study reported in 2008 by Kpodonu et al[24] showed that RTAD developed in 7 of 287 cases of TEVAR using the Gore TAG thoracic endoprosthesis (W. L. Gore and Associates, Flagstaff, Ariz) without the bare spring, with
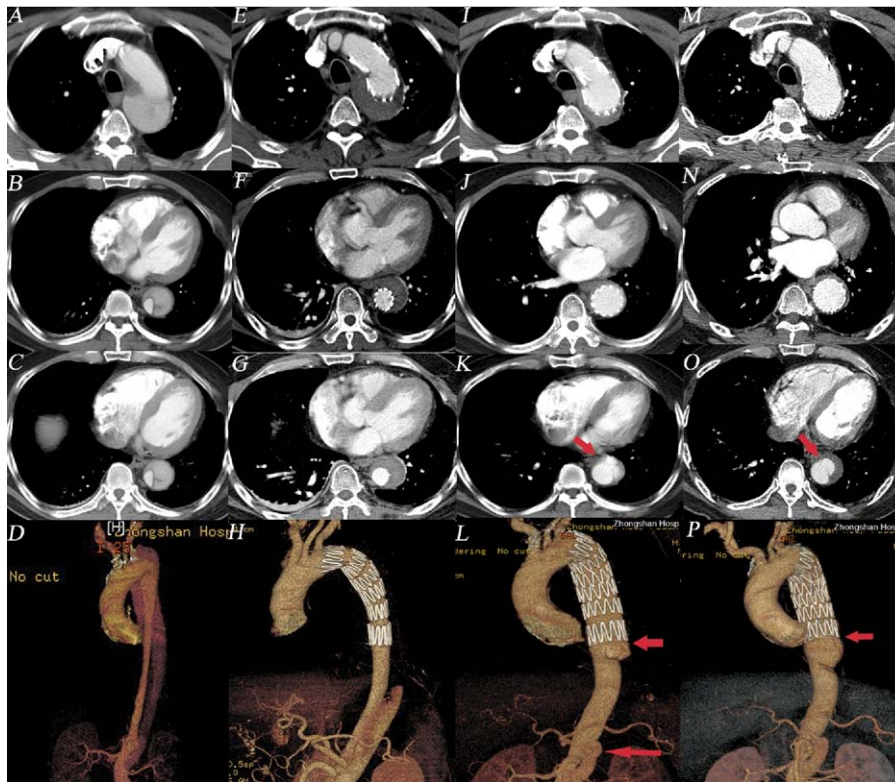
Fig 3. A to D, Preoperative computed tomography angiography (CTA) in patient 17 demonstrated the maximum aortic diameter of 50 mm and the evident gap of diameter between the proximal landing area (30 mm) and the distal true lumen (10 mm), at which level there was no entry thus far. **E to H,** The patient had chest pain 1 day after thoracic endovascular aortic repair with ZTEG-2P (34 × 15, Zenith TX2, Cook). CTA revealed a remarkable thrombosis of the false lumen, expansion of the distal true lumen up to 20 mm, and still no entry at that level where the stent graft distally fixed. **I to L,** CTA at 3 months showed that the maximum aortic diameter had shrunk to 45 mm, that the distal true lumen significantly expanded up to 36 mm, and that a distal stent graft-induced new entry (SINE) occurred (*short arrow*). The new false lumen was noted to be independent of the original false lumen (*long arrow*). **M to P,** CTA at 12 months evidenced that the maximum aortic diameter further decreased to 40 mm, and the distal SINE (*arrow*) had caused aortic enlargement from 36 to 40 mm.

an incidence of 2.4%, which was fairly similar to the 2.5% (11 of 443) we reported with the Talent device.[25]

In the seven cases of distal SINE, it is interesting that the new entry had been evidenced on the dissected flap at the either lesser or greater curve, unlike the proximal SINE tears, which were all at the greater curve. The stent graft was routinely oversized according to the proximal landing zone, which was typically much larger than the distal true lumen which thus had to endure a relatively higher radial force than the proximal segment (Fig 3). Moreover, the dissected flap was far more fragile than the uninvolved aortic wall and hence more subject to being injured. Therefore, the stress from the radial force was deemed predominantly responsible for the occurrence of the distal SINE. Xu et al[37] studied the difference in diameter between the aortic arch and the distal true lumen at the descending thoracic aorta in patients with type B dissection. The taper ratio, defined as [(arch diameter – distal true lumen diameter)/ arch diameter], was significantly larger in acute and chronic

dissection patients than in healthy people. The authors noted that oversizing according to the aortic arch meant excessive oversizing for the distal true lumen and potentially causing rupture of the already dissected membrane.[37]

Another aspect to be considered in assessment of the risk for SINE is the pathologic fragility of aortic wall. Marfan syndrome was well recognized for its effect on aortic wall weakness and a notable tendency to progressively involve the residual aorta after surgical or endovascular treatment for the primarily affected aortic segment.[38,39] The significantly higher incidence of SINE among Marfan patients in this series suggested a Marfan predisposition to this complication and implicated the fragility of the aortic wall would increase the risk for SINE. However, only a small number of Marfan patients were treated in the current survey, and a further investigation with a larger sample is needed to establish association between Marfan and SINE.

Given the substantial mortality of SINE, it would be meaningful to take measures to minimize its occurrence.
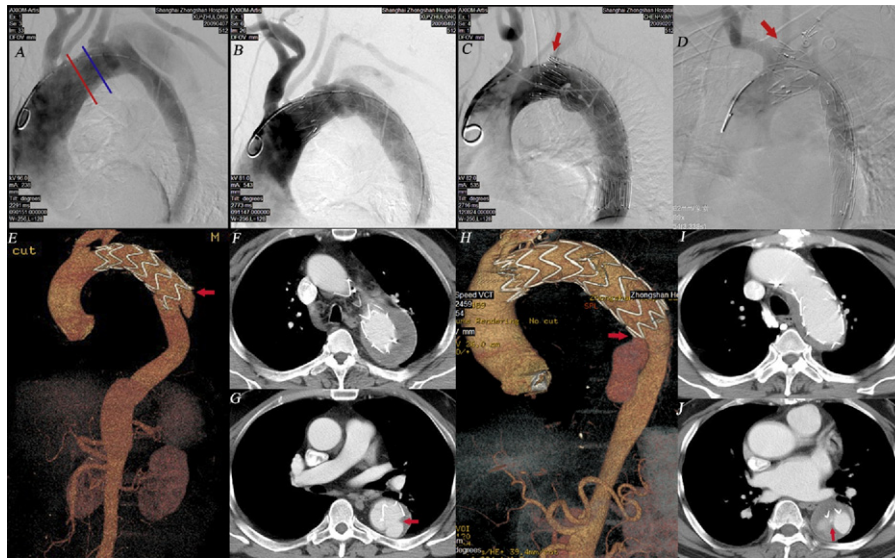
Fig 4. More attention needs to be given to an optimal compliance of the stent graft with the aorta in addition to its fixation distance. **A,** From the standpoint of the minimal requirement of the 15-mm proximal landing distance, it would have been enough to place the endograft right proximal to the left subclavian artery (*blue line*) in this case. However, it would achieve better compliance if the endoprosthesis could be deployed immediately distal to the left common artery (*red line*). **B,** So we ultimately fixed the endograft proximally at the red line, achieving an optimal compliance. **C,** Otherwise, as in this case, the proximal end of the stent graft would probably spring up and be angulated, with the greater curve (*arrow*) increasing the risk of wall injury. **D,** The proximal bare spring needs to be prevented from protruding into either origin of the three main branches. Such as in this case, the proximal bare spring went into the left common carotid artery, and its tip was almost perpendicularly pointed against the arterial wall (*arrow*), substantially increasing the risk of injury. **E to G,** In patient 21, computed tomography angiography (CTA) at 33 months demonstrated satisfactory proximal false lumen thrombosis but unsatisfactory compliance of the distal portion of the stent graft with the descending thoracic aorta and the distal stent graft-induced new entry (SINE; *arrow*). The distal SINE was located at the dissected flap and did not communicate with the original false lumen. **H to J,** In patient 20, CTA at 12 months revealed complete false lumen thrombosis at the proximal segment. However, the endograft distally stopped at the tortuous segment of the descending thoracic aorta and created a distal SINE (*arrow*), which was also at the dissected flap and independent of the primary false lumen. It would have been better in patients 21 and 20 to use a longer endograft that could extend more distally down to the relatively straight portion of the aorta and acquire better compliance.

First, try to achieve an optimal compliance. If necessary, the endograft could be placed more proximally to avoid angulation with the arch, even if the requisite minimal landing distance of 15 mm is already achieved. In addition, avoid placing the proximal bare stent into the origin of the three main branches of the aortic arch in case it might penetrate the arterial wall and create a new entry. For the distal landing, anchoring at a tortuous portion should be avoided by careful preoperative measuring and selecting an endograft with the appropriate length (Fig 4).

Second, the design of the device is pending improvement for dissection. A MEDLINE search found that much fewer post-TEVAR new entries were developing in aneurysm cases (n = 6)[13,17,21,24,35] than in dissection patients (n = 64).[3-22,24,30-34] Similarly, no SINE was observed after TEVAR in the 39 patients with thoracic aortic aneurysms in our center to date, implicating the probability that a dissection was more prone to development of SINE than an aneurysm. Most commercial devices currently available have been designed for thoracic atherosclerotic aneurysms with strong radial force, and the diameter of both ends is usually identical or just had 2-mm tapering. In contrast, a dissection-specific device calls for lower radial force, higher flexibility, and more tapering of the distal segment. Besides, the diameter of the distal true lumen would be added into the preoperative sizing, and a tapered device might be conducive if the distal true lumen is far smaller than the proximal landing zone.

Third, it would be best if the graft manufacturer could provide some mechanical indexes of the endograft, such as the radial force and the spring-back force when passively curved at a given angle. In this way, physicians can select a more appropriate device for each individual patient, particularly those with difficult anatomy or specific etiology.

Fourth, we currently try to avoid stent grafting in Marfan patients, but if one is to be used in patients with Marfan syndrome or a kinked aortic arch, we prefer the device without the proximal bare spring.

CONCLUSIONS

SINE appeared to be an infrequent but high-mortality complication after TEVAR for type B dissection. The stress-induced injury from the endograft seems to make predominant contributions to its development. If we could treat patients with TEVAR not only in a mechanical fashion but also from the perspective of biomechanics, SINE might be lessened and the minimally invasive TEVAR would be more minimally invasive.

REFERENCES

1. Dake MD, Kato N, Mitchell RS, Semba CP, Razavi MK, Shimono T, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. N Engl J Med 1999;340:1546-52.

2. Nienaber CA, Fattori R, Lund G, Dieckmann C, Dieckmann C, Wolf W, et al. Nonsurgical reconstruction of thoracic aortic dissection by stent-graft placement. N Engl J Med 1999;340:1539-45.

3. Riambau V, Guerrero F, Murillo I, Rivadeneira M, Montaña X, Matute P. Stent grafting-related acute type B redissection. Vascular 2008;16:101-5.

4. Xu SD, Huang FJ, Yang JF, Li ZZ, Wang XY, Zhang ZG, et al. Endovascular repair of acute type B aortic dissection: early and mid-term results. J Vasc Surg 2006;43:1090-5.

5. Fattori R, Napoli G, Lovato L, Grazia C, Piva T, Rocchi G, et al. Descending thoracic aortic diseases: stent-graft repair. Radiology 2003;229:176-83.

6. Lee KH, Won JY, Lee DY, Choi D, Shim WH, Chang BC. Elective stent-graft treatment of aortic dissections. J Endovasc Ther 2004;11:667-75.

7. Shim WH, Koo BK, Yoon YS, Choi D, Jang Y, Lee DY, et al. Treatment of thoracic aortic dissection with stent-grafts: midterm results. J Endovasc Ther 2002;9:817-21.

8. Pamler RS, Kotsis T, Görich J, Kapfer X, Orend KH, Sunder-Plassmann L. Complications after endovascular repair of type B aortic dissection. J Endovasc Ther 2002;9:822-8.

9. Li ZZ, Xu SD, Zheng SH, Li ZA, Du JH, Sun YQ. Interventional treatment of thoracic aortic dissection with stent-graft placement guided by transesophageal echocardiography. Zhonghua Nei Ke Za Zhi 2004;43:26-8.

10. Shimono T, Kato N, Yasuda F, Suzuki T, Yuasa U, Onoda K, et al. Transluminal stent-graft placements for the treatments of acute onset and chronic aortic dissections. Circulation 2002;106(suppl 1):I241-7.

11. Scheinert D, Krankenberg H, Schmidt A, Gummert JF, Nitzsche S, Scheinert S, et al. Endoluminal stent-graft placement for acute rupture of the descending thoracic aorta. Eur Heart J 2004;25:694-700.

12. Gawenda M, Brunkwall J. Device related problems of thoracic stent-grafts. Zentralbl Chir 2007;132:205-10.

13. Neuhauser B, Greiner A, Jaschke W, Chemelli A, Fraedrich G. Serious complications following endovascular thoracic aortic stent-graft repair for type B dissection. Eur J Cardiothorac Surg 2008;33:58-63.

14. Langer S, Mommertz G, Koeppel TA, Schurink GW, Autschbach R, Jacobs MJ. Surgical correction of failed thoracic endovascular aortic repair. J Vasc Surg 2008;47:1195-202.

15. Kaya A, Heijmen RH, Rousseau H, Nienaber CA, Ehrlich M, Amabile P, et al. Emergency treatment of the thoracic aorta: results in 113 consecutive acute patients (the Talent Thoracic Retrospective Registry). Eur J Cardiothorac Surg 2009;35:276-81.

16. Jing QM, Han YL, Wang XZ, Deng J, Luan B, Jin H, et al. Endovascular stent-grafts for acute and chronic type B aortic dissection: comparison of clinical outcomes. Chin Med J (Engl) 2008;121:2213-7.

17. Fairman RM, Criado F, Farber M, Kwolek C, Mehta ME, White R, et al; VALOR Investigators. Pivotal results of the Medtronic Vascular Talent Thoracic Stent Graft System: the VALOR trial. J Vasc Surg 2008;48:546-54.

18. Steingruber IE, Chemelli A, Glodny B, Hugl B, Bonatti J, Hiemetzberger J, et al. Endovascular repair of acute type B aortic dissection: midterm results. J Endovasc Ther 2008;15:150-60.

19. Inglese L, Mollichelli N, Medda M, Sirolla C, Tolva V, Grassi V, et al. Endovascular repair of thoracic aortic disease with the EndoFit stent-graft: short and midterm results from a single center. J Endovasc Ther 2008;15:54-61.

20. Pitton MB, Herber S, Schmiedt W, Neufang A, Dorweiler B, Duber C, et al. Long-term follow-up after endovascular treatment of acute aortic emergencies. Cardiovasc Intervent Radiol 2008;31:23-35.

21. Thompson M, Ivaz S, Cheshire N, Fattori R, Rousseau H, Heijmen R, et al. Early results of endovascular treatment of the thoracic aorta using the Valiant endograft. Cardiovasc Intervent Radiol 2007;30:1130-8.

22. Tespili M, Banfi C, Valsecchi O, Aiazzi L, Ricucci C, Guagliumi G, et al. Endovascular treatment of thoracic aortic disease: mid-term follow-up. Catheter Cardiovasc Interv 2007;70:595-601.

23. Neuhauser B, Czermak BV, Fish J, Perkmann R, Jaschke W, Chemelli A, et al. Type A dissection following endovascular thoracic aortic stent-graft repair. J Endovasc Ther 2005;12:74-81.

24. Kpodonu J, Preventza O, Ramaiah VG, Shennib H, Wheatley-GH 3rd, Rodriquez-Lopez J, et al. Retrograde type A dissection after endovascular stenting of the descending thoracic aorta. Is the risk real? Eur J Cardiothorac Surg 2008;33:1014-8.

25. Dong ZH, Fu WG, Wang YQ, Guo DQ, Xu X, Chen B, et al. Retrograde type A aortic dissection after endovascular stent graft placement for treatment of type B dissection. Circulation 2009;119:735-41.

26. Fu WG, Shi Y, Wang YQ, Guo DQ, Xu X, Chen B, et al. Endovascular therapy for Stanford type B aortic dissection in 102 cases. Asian J Surg 2005;28:271-6.

27. Fu WG, Dong ZH, Wang YQ, Guo DQ, Xu X, Chen B, et al. Strategies for managing the insufficiency of the proximal landing zone during endovascular thoracic aortic repair. Chin Med J 2005;13:1066-71.

28. Marui A, Mochizuki T, Mitsui N, Koyama T, Kimura F, Horibe M. Toward the best treatment for uncomplicated patients with type B acute aortic dissection: a consideration for sound surgical indication. Circulation 1999;100(suppl):II275-80.

29. De Paepe A, Devereux RB, Dietz HC, Hennekam RC, Pyeritz RE. Revised diagnostic criteria for the Marfan syndrome. Am J Med Genet 1996;62:417-26.

30. Won JY, Suh SH, Ko HK, Lee KH, Shim WH, Chang BC, et al. Problems encountered during and after stent-graft treatment of aortic dissection. J Vasc Interv Radiol 2006;17:271-81.

31. Rubin S, Bayle A, Poncet A, Baehrel B. Retrograde aortic dissection after a stent graft repair of a type B dissection: how to improve the endovascular technique. Interact Cardiovasc Thorac Surg 2006;5:746-8.

32. Panos A. Late retrograde aortic perforation by the uncovered part of an endograft: an increasing complication. Hellenic J Cardiol 2007;48:115-6.

33. Modine T, Lions C, Destrieux-Garnier L, Haulon S, Decoene C, Beregi JP, et al. Iatrogenic iliac artery rupture and type A dissection after endovascular repair of type B aortic dissection. Ann Thorac Surg 2004;77:317-9.

34. Misfeld M, Notzold A, Geist V, Richardt G, Sievers HH. Retrograde type A dissection after endovascular stent grafting of type B dissection. Z Kardiol 2002;91:274-7.

35. Zhang R, Kofidis T, Baus S, Klima U. Iatrogenic type A dissection after attempted stenting of a descending aortic aneurysm. Ann Thorac Surg 2006;82:1523-5.

36. Fanelli F, Salvatori FM, Marcelli G, Bezzi M, Totaro M, Vagnarelli S, et al. Type A aortic dissection developing during endovascular repair of an acute type B dissection. J Endovasc Ther 2003;10:254-9.

37. Xu SD, Huang FJ, Du JH, Li Y, Fan ZM, Yang JF, et al. A study of aortic dimension in type B aortic dissection. Interact Cardiovasc Thorac Surg 2008;7:244-8.

38. Gott VL, Greene PS, Alejo DE, Cameron DE, Naftel DC, Miller DC, et al. Replacement of the aortic root in patients with Marfan's syndrome. N Engl J Med 1999;340:1307-13.

39. Ince H, Rehders TC, Petzsch M, Kische S, Nienaber CA. Stent-grafts in patients with Marfan syndrome. J Endovasc Ther 2005;12:82-8.