Distal Stent Graft–Induced New Entry

The current paradigm and future management of dSINE.

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During the past 15 years, the treatment of type B aortic dissection has evolved after the introduction of thoracic endovascular aortic repair (TEVAR). With high success rates and acceptable clinical outcomes, stent grafting has been widely accepted as the treatment of choice for acute, complicated type B aortic dissection. The basic concept of this technique is to cover the proximal primary intimal tear of the aorta, to exclude the false lumen, and to initiate thrombosis of the false lumen and expansion of the true lumen. However, the distal landing zone of the stent graft is still often located in the diseased aorta, and potential intimal injury by the endograft is always a major concern. New distal intimomedial injury by the stent graft, so-called distal stent graft–induced new entry (dSINE), has been increasingly observed.1,2 dSINE may lead to a new patent false lumen, then aneurysmal degeneration, and eventually aortic rupture.

THE INCIDENCE, ONSET, AND MORTALITY OF dSINE

The incidence of dSINE ranged from 3.4% to 27%.1,3 A higher incidence was noted in patients treated for chronic aortic dissection. Our published data revealed an incidence of 18.9% in patients with acute type B aortic dissection and 35.7% in chronic cases.2 One recent study also reported that 89% of dSINE was noted in patients with chronic aortic dissection.4 In addition, dSINE usually develops late after endografting and patients may be asymptomatic for a long time. Our previous study showed that the average time from TEVAR to dSINE onset was 24.8 ± 5.9 months.2 Others reported onset times ranging from 11 ± 16 months1 to 31.5 ± 28.6 months.4 With a mortality rate as high as 28.6%,1 dSINE can be insidious in character and needs to be monitored by a long-term CT follow-up protocol.

THE MECHANISM OF dSINE DEVELOPMENT

The potential mechanisms for the development of dSINE are complex. Besides the natural progression of the aortic disease, aortic wall fragility and stent grafting–related factors may also play important roles. The pulsatile stimulus from the rigid end of the stent graft against the fragile intimal flap could eventually cause a tear and create a new entry. In chronic dissection, the intimal flap is more fibrotic and less mobile than in the acute phase. The rigid membrane would be less compliant to the expansion of the stent graft and thus carry a higher risk of a new intimal break.

Another important factor is distal stent graft oversizing (the ratio between the distal size of the selected stent graft and the true lumen size at the presumed level of the distal end of the stent graft before the procedure; Figure 1). Previous studies have shown that greater distal stent graft oversizing, in either area or diameter measurements, is related to the formation of dSINE.3,4 The possible mechanism is that the true lumen is usually narrower at the presumed distal landing zone than at the proximal landing zone, so stent grafts chosen according to the proximal landing zone will lead to substantial oversizing at the distal landing zone. This will create an excessive radial force and pose a risk.
of dSINE. Some studies demonstrated that a higher taper ratio (the ratio between the sizes of the true lumen at the proximal landing zone and that at the distal landing zone before the procedure) was seen in patients with dSINE. However, this phenomenon was not observed in our published studies. This means that distal oversizing may contribute more in the formation of dSINE than the discrepancy between the sizes of the proximal and distal landing zones.

Elastic recoil, the tendency of the stent graft to revert to its initial straight form, may also lead to intimal injury at the distal landing zone. In most cases of aortic dissection, the stent graft needs proximal landing in the aortic arch and is passively bent to conform to the curvature of the aorta. However, the endograft has the inherent tendency to spring back to its initial straight form, which generates stress along the outer curve and leads to an angulated aorta at the distal landing zone, posing a risk of dSINE formation.

**PREDICTION AND PREVENTION OF dSINE**

Because the incidence of dSINE is high, and it is associated with potentially life-threatening outcomes, prediction and prevention are crucial in its management. Our published study revealed that the oversizing ratio between the sizes of the selected stent graft and the true lumen at the presumed distal landing zone is a significant preoperative predictive factor of dSINE (Figure 1). This study also reported that the expansion mismatch ratio between the true lumen sizes at and adjacent to the distal landing zone is an important postoperative predictor of dSINE (Figure 2).

Several preventive procedures have been adopted at our institution to reduce the formation of dSINE. First, endografts with a tapered configuration, such as Zenith Dissection Endovascular Grafts with 4-, 8-, or 10-mm-diameter tapering (Cook Medical), can be used to avoid excessive distal oversizing. Alternatively, a bottom-up technique can also produce the effect of tapering in diameter or area. This technique is composed of deployment of a smaller stent graft distally, followed by deployment of a larger stent graft proximally. We have used this approach since 2010. Our unpublished analysis showed that the area oversizing ratio in the distal landing zone was reduced, and the incidence of dSINE decreased from 34.7% to 8.3% with the use of this endografting technique.

Second, restrictive bare stenting (RBS) or the modified PETTICOAT (provisional extension to induce complete attachment) technique may protect the intima at the distal edge of the stent graft from the excessive radial force of the stent graft. This technique involves the placement of a properly sized bare stent in the intended distal landing zone of the stent graft, prior to deployment of the stent graft. RBS was reported to be associated with a lower incidence of dSINE (0% vs 2.9%; P = .033) and fewer secondary interventions (3.9% vs 9.3%; P = .040). A recent study also demonstrated favorable results of the modified PETTICOAT technique.

Third, some studies suggested that avoidance of distal landing in a tortuous portion of the aorta or placement of several stent grafts until the distal end of the last stent graft is oriented parallel to the aortic wall would reduce the stress of the stent graft against the curvature of the aorta.

**MANAGEMENT OF dSINE**

Medical treatment with optimal blood pressure control is preferred upon confirming the diagnosis of dSINE. The indications for reintervention include persistent enlargement of the false lumen, contained rupture, pseudoaneurysm formation, malperfusion, or symptoms. In our institute, the procedure for the secondary intervention is performed in the same way as the standard TEVAR with proper device sizing. The artery of Adamkiewicz is located by CT preoperatively and is preserved as much as possible during the secondary procedure to reduce the risk of paraplegia, and cerebrospinal fluid drainage is set up immediately if there is any symptom of paraplegia. Our data showed that reendografting seems effective to treat complicated dSINE.
ROLE OF AORTIC BARE STENT IN dSINE

The Zenith Dissection Endovascular System (Cook Medical) is designed for treatment of aortic type B dissection utilizing the PETTICOAT technique. This device comprises stent grafts to cover the primary entry tear proximally and uncovered metal stents to promote true lumen expansion distally. Previous studies have demonstrated favorable clinical and anatomic results in the management of type B aortic dissection.

Interestingly, it may help reducing the risk of dSINE formation. In our initial experience with this device, in nearly 50 cases of complicated type B aortic dissection, only one dSINE formation was noted in a 2-year follow-up period. In addition, we started to use the modified PETTICOAT technique to treat recurrence of dSINE (Figure 3). No further redissection was noted in a 6-month follow-up period, but the effectiveness of this management needs further investigation.

CONCLUSION

dSINE is not rare and is possibly life-threatening. Stent grafts with a dissection-specified tapering design, a bottom-up technique, restrictive bare stenting, PETTICOAT, or modified PETTICOAT technique can be used to reduce the risk of dSINE. The preoperative distal oversizing ratio and postoperative expansion mismatch ratio appear to be predictive of the formation of dSINE. Endovascular reintervention seems an effective management for complicated dSINE. However, recurrence can occur, and long-term follow-up is mandatory.

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