Angioplasty and stenting for symptomatic stenosis of the left subclavian artery complicated with aortic dissection

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Abstract

Background: Aortic dissection is a rare but severe complication of percutaneous transluminal angioplasty and stenting (PTAS) for stenosis of the subclavian artery (SA). This retrospective study was designed to evaluate the risk factors and outcomes of patients with severe stenosis of the SA who underwent PTAS complicated by aortic dissection.

Methods: Between 1999 and 2018, 169 cases of severe symptomatic stenosis of the SA underwent PTAS at our institute. Of them, six cases complicated by aortic dissection were included in this study. We evaluated the demographic features, technical factors of PTAS, and clinical outcomes in these six patients.

Results: Aortic dissection occurred in 5.3% (6/113) of all left SA stenting cases but in none of the right SA stenting cases. All patients had hypertension and a high severity of SA stenosis (85.0 ± 13.0%, 60%–95%). Five of the six patients received balloon-expandable stents (83.3%). All patients had spontaneous resolution of the aortic dissection with conservative treatment. In a 63.33 ± 33.07 (7–118) month follow-up, five of the six patients (83.3%) had long-term symptom relief and stent patency.

Conclusion: Aortic dissection occurred in patients who underwent PTAS for severe stenosis of the left SA, mainly with balloon-expandable stents. We suggest using self-expandable stents and angioplasty with an undersized balloon during PTAS for severe stenosis of the left proximal SA to prevent aortic dissection.

Keywords: Angioplasty and stenting; Aortic dissection; Stenosis; Subclavian artery

1. INTRODUCTION

Severe stenosis of the subclavian artery (SA) can present with ischemic symptoms of the vertebrobasilar system, the upper limb, and even the coronary artery. The most common symptoms are due to subclavian steal syndrome, which means reverse vertebral artery (VA) flow resulting from a significant stenosis or occlusion of the ipsilateral proximal SA.[1] Most patients with SA stenosis are asymptomatic and do not require aggressive intervention.[2,3] In patients with severe stenosis of the SA associated with medically refractory arterial insufficiency, percutaneous transluminal angioplasty and stenting (PTAS) of the stenotic SA lesion is an effective alternative treatment.[3]

PTAS for stenosis of the SA is a procedure with high technical success and few major complications. The frequency of well-known major procedure-related complications is less than 3.4%. These complications include access site complications, cerebral ischemic insult, hyperperfusion syndrome, and arterial injury of the treated vessels.[4–7] Aortic dissection is a rare but severe complication of PTAS for stenosis of the SA, which has only been reported in few cases to date.[8,9] Acute symptoms of aortic dissection may be associated with sharp pain and dyspnea, which may increase the risk of periprocedural complications, such as stent migration. However, the factors and outcomes related to aortic dissection in PTAS for severe stenosis of the SA have not been clarified. This retrospective study was designed to evaluate the factors and outcome of PTAS for severe stenosis of the SA complicated by aortic dissection. We also compared the influence of different stent designs, including balloon-expandable stents and self-expandable stents, on the occurrence of this complication.

2. METHODS

This retrospective study was approved by the institutional review board at our institute. Preprocedural informed consent was obtained from each patient before performing the magnetic resonance image (MRI) examination and endovascular procedure.

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2.1. Patient selection

Between 1999 and 2018, 169 patients with symptomatic stenosis of the SA underwent PTAS at our institute. One of them had bilateral SA stenting, and therefore, there were 170 SA stenting procedures completed. Of them, six patients with cases complicated by aortic dissection during the procedure were included in this study. Aortic dissection was diagnosed by both clinical and imaging findings. The clinical findings indicating aortic dissection included sharp neck, chest or back pain, and dyspnea. The imaging diagnosis was performed by either angiography or postprocedural computed tomography (CT) of the chest. The imaging findings of aortic dissection included the presence of an intimal flap or false lumen. We evaluated the demographic characteristics and clinical data from the medical records and imaging system. A periprocedural MRI with magnetic resonance angiography (MRA) of the brain was also performed 3 days before and after PTAS to evaluate its neurological influence and intracranial embolic insult.

2.2. SA percutaneous transluminal angioplasty and stenting

The indication of PTAS for the stenosis of the SA included (1) the severity of the stenotic lesion >60% and (2) the presence of medically refractory ischemic symptoms.[4,6] These symptoms were cerebral ischemia (such as vertebrobasilar insufficiency or subclavian steal syndrome), limb ischemia (such as coldness or pain of the ipsilateral shoulder or upper limb), or coronary steal phenomenon.

All patients took dual antiplatelet premedication (300 mg of aspirin and 75 mg of clopidogrel) for at least 3 days before the procedure, continued dual antiplatelet therapy for 1–2 months postprocedurally, and were then switched to life-long aspirin monotherapy. The procedures were conducted under local anesthesia via a transfemoral arterial approach to obtain a complete angiogram of the supra-aortic arteries. After an intravenous bolus of 3000–5000 IU of heparin, a 7 or 8 Fr guiding sheath (Shuttle guiding sheath; Cook Co., Bloomington, IN) was placed in the target artery or in the aorta. Then, we navigated a 0.035 Fr extra-stiff wire (Amplatz wire; Cook Co.) into the ipsilateral axillary artery. Predilatation of the stenotic lesions was performed with a noncompliant balloon (Wanda; Boston Sci Co., MA). The diameter of the balloon was chosen to be approximately 80%–90% of the diameter of the adjacent normal segment. After predilatation, the vessels were stented with either balloon-expandable stents (Express LD; Boston Sci Co.) or self-expandable stents (Epic stent; Boston Sci Co., Fremont, CA) (Fig. 1). A control angiogram was performed to evaluate residual stenosis and any vascular injury. Any technical complications, including neurological symptoms, were recorded.

2.3. Clinical evaluation, imaging follow-up, and data collection

Postprocedural CT of the chest was performed to evaluate the severity and outcome of aortic dissection (Fig. 2). All patients were followed up with clinical and computed tomography angiography (CTA) or MRA in the first postprocedural month and every 3–6 months after the treatment. We evaluated the change in clinical symptoms before and after PTAS. Any recurrent ischemic symptoms or restenosis of the treated arteries were also recorded.

2.4. Statistical analysis

The data are expressed as absolute values and percentages.

3. RESULTS

3.1. Patient characteristics and periprocedural outcomes

The total 170 PTAS procedures for symptomatic stenosis of the SA included 113 left SA stenting procedures and 57 right SA stenting procedures. The Table shows the characteristics of the six cases (6/170, 3.5%) complicated by aortic dissection. All occurred at the left-sided SA. For the 113 left SA stenosis procedures, the complication rate of aortic dissection was 5.3% (6/113). The age of the six patients was 79.3 ± 5.4 (72–85) years old. The most common symptom of these six patients who accepted PTAS was dizziness. They had at least one atherosclerotic risk factor.

All six patients successfully underwent PTAS for stenosis of the left SA. No neurological complications other than aortic dissection were noted. Their stenotic lesions were located in the orifice or the proximal segment of the left SA. The severity of stenosis was 85.0 ± 13.0% (60–95%). The diameters of the stents used were 7–10 mm. The dissection was demonstrated by control angiogram and postprocedural CT of the chest. All patients with aortic dissection complained of various severities of back or shoulder pain. Five patients suffered from type B aortic arch dissection. One patient complained of chest pain and dyspnea because she had type A aortic dissection with hemopericardium (Fig. 3). Periprocedural MRI of the brain before and after PTAS was performed in four cases and showed 0–4 spots (average: 1.75 spots per patient) of restricted diffusion on diffusion-weighted image of the territory of the vertebrobasilar system (Fig. 2). The technical details are listed in Table.

3.2. Factors related to restenosis and outcomes

All six cases of aortic dissection resolved by conservative treatment without clinical sequelae (Figs. 2 and 3). In a 63.3 ± 33.1

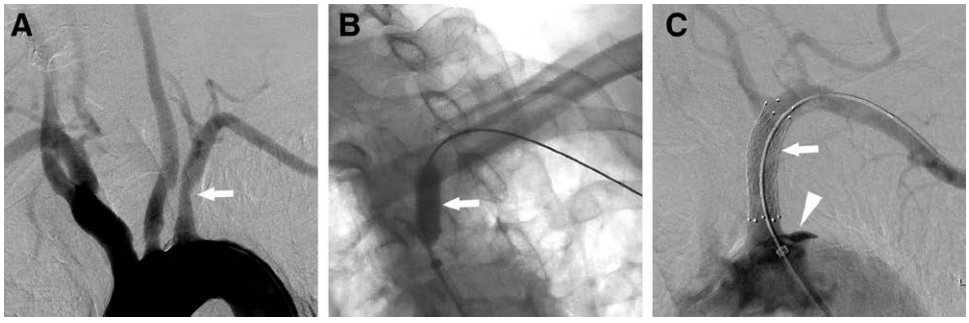

Fig. 1 A–C, PTAS for stenosis of the left SA complicated by type B aortic dissection. This 85-year-old gentleman had dizziness for 3 months. He underwent sonography, which showed biphasic flow of the left vertebral artery. The angiogram showed 60% stenosis of the left proximal SA (A, arrow). We first deployed a 9 × 30 self-expandable Epic stent in the left proximal SA and performed angioplasty with an 8 × 20 mm Wanda balloon (B, arrow). The control angiogram showed focal dissection in the descending aorta (C, arrowhead). PTAS = percutaneous transluminal angioplasty and stenting; SA = subclavian artery.

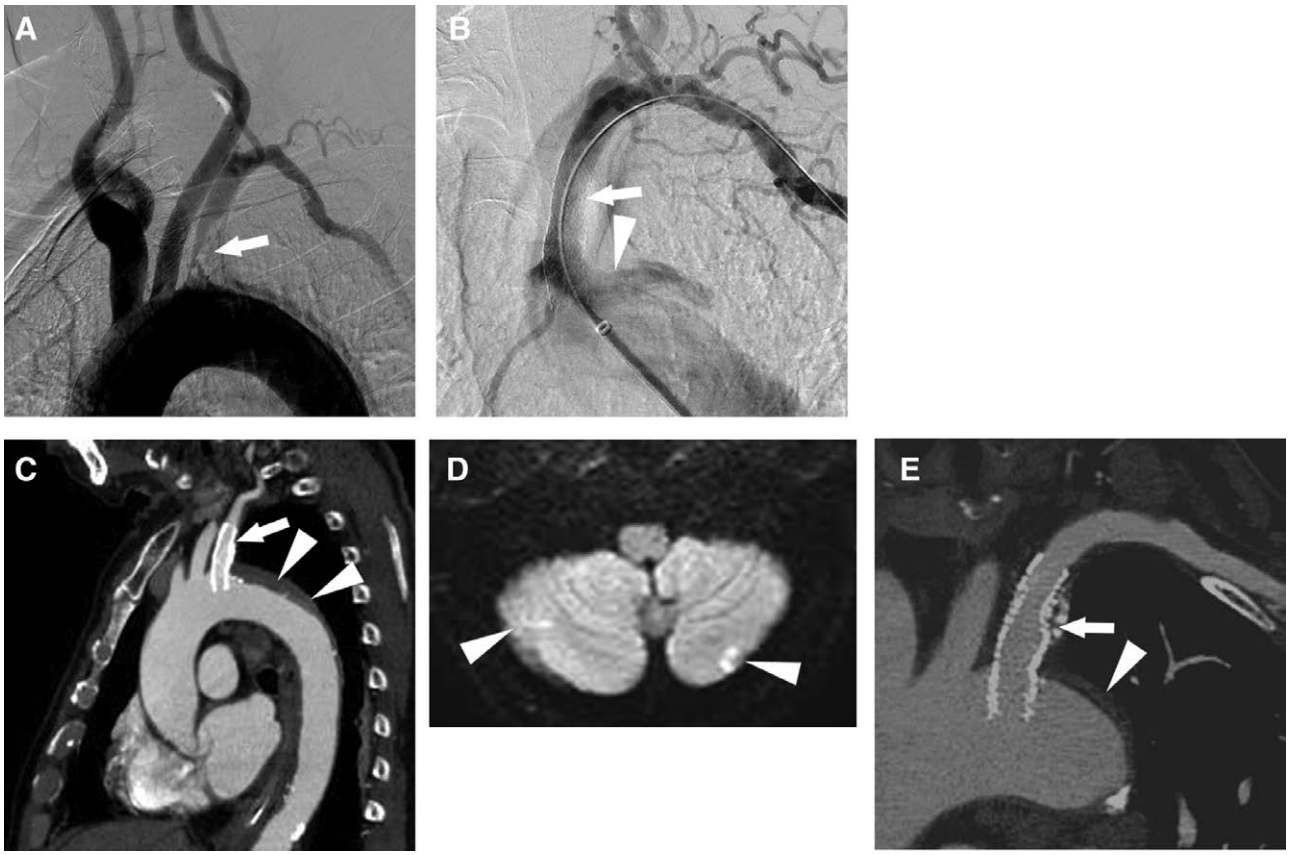

Fig. 2 A–E, PTAS for the stenosis of the left SA complicated by type B aortic dissection. This 76-year-old male patient had medically refractory syncope and dizziness. The angiogram showed 85% stenosis of the left proximal SA (A, arrow). After predilatation with a 7 mm balloon, a 9 × 37 mm balloon-expandable Express LD stent was deployed in the left proximal SA (B, arrow). The control angiogram revealed type B aortic dissection (B, arrowhead). Contrast-enhanced CT of the chest on the following day demonstrated aortic dissection (C, arrowheads). MRI of the brain on the next day also showed small recent embolic infarcts in the bilateral cerebellar hemispheres (D, arrowheads). Follow-up CTA of the neck 1 month later showed obliteration of the aortic dissection (E, arrowhead). Note that the stent migrated proximally after resolution of the intramural hematoma (E, arrowhead). CTA = computed tomography angiography; MRI = magnetic resonance image; PTAS = percutaneous transluminal angioplasty and stenting; SA = subclavian artery.

(7–118) month follow-up, five of the six patients (83.3%) had no recurrent symptoms and had stent patency. One patient had 70% in-stent restenosis and had recurrent dizziness at the 1-year follow-up. He preferred medical treatment for the recurrent symptoms. Further details are listed in Table.

4. DISCUSSION

The complication rate of aortic dissection during PTAS for the stenosis of the left SA was approximately 5.3% in this large-scale study, but aortic dissection did not occur at the right SA. This complication occurs just after balloon dilatation, mainly during the deployment of balloon-expandable stents. All patients presented with shoulder or back pain of variable severity. However, their prognosis with conservative treatment was quite good. Even when complicated by aortic dissection, this complication did not significantly affect technical or clinical success or long-term stent patency.

From a review of the literature, the prevalence of subclavian steal syndrome is approximately 0.6%–6.4%, and only 5.3% of these patients have neurological symptoms.[1] The lesion more commonly involves the left side, which accounts for approximately 82.3% of all cases.[10] The proposed explanation for this high lateralization is that the atherosclerotic process at the subclavian-aortic junction is faster because of increased flow turbulence resulting from the acute angle of origin of the left SA.[11] Among all cases of subclavian steal syndrome, the ratio between men and women is approximately 2:1.[1] The etiology is mainly due to atherosclerosis. In addition, there are some other rare causes, including aortic dissection, Takayasu's arteritis, external compression of the SA, and anatomical anomalies, such as an isolated innominate artery.[1,12] Symptoms related to subclavian steal syndrome are mainly vertebrobasilar insufficiency, which means decreased blood flow to the posterior circulation of the brain, upper limb ischemia, or both.[1] Aggressive intervention with surgical procedures or endovascular management is reserved for serious and medically refractory clinical symptoms.[10,13,14] PTAS for stenosis of the SA is a technique with a high success rate (93%–98%) and a low major complication rate (0%–3.4%). In addition, the long-term patency rate (59%–96%) was high, and the symptom recurrence rate (1.7%–3%) was low.[4,6,14]

Several complications due to PTAS for stenosis of the SA have been reported, including access site complications, cerebral ischemic insult, and arterial injury of the treated vessels.[4,5] The overall periprocedural complication rate has been reported to be as high as 15.1%.[15] Stroke is a less frequent complication with an approximately 2.2% occurrence rate, probably because of natural protection from reverse VA flow.[16] Some less common complications have been reported, such as nerve injury during access, stent infections, and SA rupture.[4] Aortic dissection is a rare but severe complication of PTAS for severe stenosis of the SA that has been reported in few cases to date.[8,9]

Table 1.

Demographic features and outcomes of the 6 patients of subclavian artery accepted angioplasty and stenting complicated with aortic dissection

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Demographic features						
Age	82	76	85	72	85	76
Gender	F	M	F	F	M	M
Symptoms	Dizziness Vertigo Syncope Nausea Weight loss	Dizziness Syncope	Dizziness Syncope	Dizziness Vertigo Upper limb weakness Headache	Dizziness	Dizziness
Risk factors	Hypertension Diabetes mellitus Hyperlipidemia	Hypertension Diabetes mellitus	Smoking Hypertension Hyperlipidemia	Hypertension Hyperlipidemia Coronary Artery Disease	Hypertension Diabetes mellitus Hyperlipidemia	Hypertension
Angioplasty and stenting						
Lesion side	Left	Left	Left	Left	Left	Left
Stenosis severity(%)	95	85	90	85	60	95
Stenosis location	Ostium	Proximal	Proximal	Proximal	Proximal	Proximal
Stent type and size	Express LD 9 × 25mm + 10 × 25mm	Express LD 9 × 37mm	Express LD 8 × 37mm	Express LD 9 × 25mm	Epic 9 × 30mm	Express LD 7mm
Complication	Aortic dissection Back pain	Aortic dissection Back pain	Aortic dissection Back pain	Aortic dissection Chest and chin pain	Aortic dissection Asymptomatic	Aortic dissection Asymptomatic
Number of acute embolic infarcts on post-stenting diffusion weighted image[a]	X	4	1	0	2	X
Outcome						
Symptom	Symptom-free 118 months	Recurrence 80 months	Symptom-free 55 months	Symptom-free 64 months	Symptom-free 56 months	Recurrence 7 months
Stent patency	Patent 32 months	Patent 67 months	Patent 54 months	Patent 57 months	Patent 38 months	Significant restenosis (70%) 8 months

[a]Only evaluate the diffusion-weighted image of the territory of vertebrobasilar system in the first postprocedural day.

Iatrogenic causes account for 5% of all aortic dissection cases, and the major risk factors are old age, hypertension, and diabetes, as compared with spontaneous aortic dissection.[17] Factors with increased risk for aortic dissection during vascular intervention also include stenosis eccentricity and heavy calcification as well as usage of oversized balloon. All our patients were elderly and had at least one atherosclerotic risk factor, which is consistent with the high-risk population for iatrogenic aortic dissection. This complication has been reported in several other procedures, including common carotid artery stenting, renal artery stenting, coronary artery bypass surgery, coronary angioplasty, aortic angioplasty and stenting, iliac artery stenting, and aortic stent-grafting procedures.[18,19] All target vessels are direct main branches of the aorta or aorta per se. In the present study, this complication only occurred in the left SA but not in the right side, which is anatomically reasonable because the left SA directly originates from the aortic arch. Although there are only a few case reports in the literature, the occurrence rate of our treated lesions of the left SA was 5.3%, which is not as low as previously considered. We suggest that this complication may also occur during interventions in the counterpart vessel, the innominate artery, although this complication has not yet been found in this artery in the literature.[19,20] Other documented complication rates of iatrogenic aortic dissection occur in approximately 0.06% of all coronary catheterization procedures, with a higher rate in the intervention group and 0.12% in the cardiac surgical procedure group.[21,22]

There are several proposed explanations and risk factors for iatrogenic aortic dissection during endovascular intervention for supra-aortic arteries in the literature. The three most important factors include (1) torque and shear forces on treated vessels during balloon angioplasty, (2) repeated dilatation or a short time interval between balloon dilatations, and (3) long-standing and poorly controlled hypertension.[23,24] We favored the first factor because all of our cases of aortic dissection occurred immediately after the full expansion of the balloon. Overdilatation of the arteries during balloon angioplasty can improve vascular long-term patency, and some suggest 20% overdilatation or using balloons with a diameter 1–2 mm greater than the angiographically estimated vessel diameter.[23,25] Nonetheless, during the balloon angioplasty process, there are radially directed forces from balloon inflation on the vessel wall, torque forces due to the straightening of the balloon that cause movement of vessels, and shear forces between balloon and vessel walls (Fig. 4). The combined results may lead to arterial dissection at the junction of the root of the left SA and aortic arch. Therefore, high-grade stenosis and vascular angulation are considered contributing factors for iatrogenic arterial dissection.[23,26] In addition, using oversized balloon is also considered a risk factor.[18] The risk associated with using oversized balloon can be avoided by scrutinizing the vessel diameter and selecting a balloon size that is not larger than 10% of the treated vessel. If full expansion of the vessel cannot be achieved after high-pressure balloon dilatation, repeated dilatation might not be necessary if the angiographic result is already satisfactory. If the decision regarding repeated dilatation is controversial, measurement of bilateral arm blood pressure and evaluation of stent apposition on intravascular ultrasound might be helpful.[18] Furthermore, during balloon dilatation, decreasing balloon migration, especially when in full expansion, might lower the risk of aortic dissection. We suggest deploying the balloon-expandable stent as slowly as possible to decrease balloon migration. In our patients, aortic

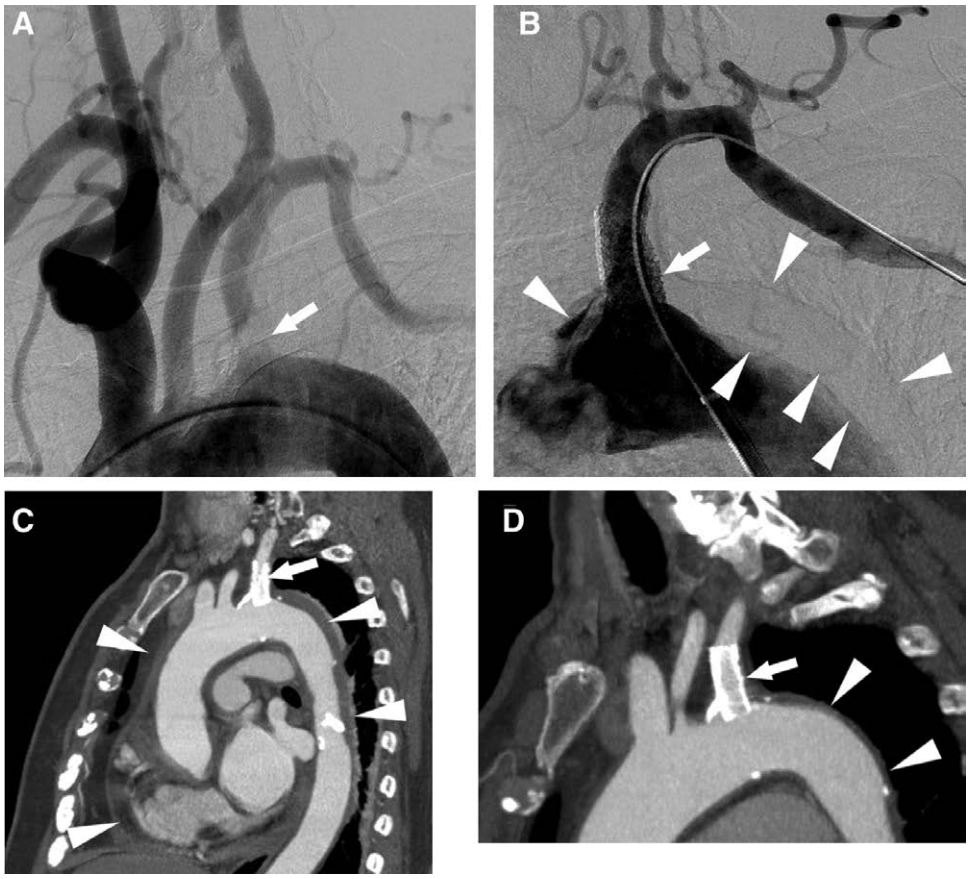

Fig. 3 A–D, PTAS for stenosis of the left SA complicated by type A aortic dissection and hemopericardium. A 72-year-old female was admitted for dizziness due to vertebrobasilar insufficiency from 85% stenosis of the left SA (A, arrow). Just as we deployed a 9×25 mm balloon-expandable Express LD stent in the orifice of the left SA (B, arrow), the patient complained of chest tightness and temporary atrial fibrillation. The control angiogram showed aortic dissection in the ascending and descending aorta (B, arrowheads). As the symptoms persisted, she underwent contrast-enhanced CT of the chest the next day, which revealed a type A aortic dissection and hemopericardium (C, arrowheads). Follow-up CTA of the neck 1 month later showed obliteration of the aortic dissection (D, arrowheads). Note good localization of the stent (C & D, arrows). CTA = computed tomography angiography; PTAS = percutaneous transluminal angioplasty and stenting; SA = subclavian artery.

dissection occurred after balloon dilatation in the presence of stent coverage, either using a balloon-expandable stent or while performing postdilatation for an existing stent, raising concern of a stent-related etiology. When the stent expands along with balloon inflation, the architecture of the stent will change and might cause more shear forces between the overall endovascular devices and the vascular intima than those between only a balloon and the vascular intima. As a result, the risk of aortic dissection might be elevated. The previously reported two cases are similar to ours; they occurred while using a balloon-expandable stent or during postdilatation after deployment of a self-expandable stent.[8,9] With appropriate predilatation, using self-expandable stents without postdilatation is helpful to prevent aortic dissection.

All our patients recovered fully from aortic dissection with conservative treatment. This suggests that additional intervention is not indicated for iatrogenic aortic dissection during PTAS for severe stenosis of the left SA. However, salvage intervention treatment may sometimes be necessary for persistent progression of an aortic dissection, which might compromise visceral arteries, peripheral arteries, and coronary arteries with resultant symptoms.[9,27] In addition, although all aortic dissections were identified during angiography in our study, sometimes they might occur hours after the procedure.[9] This suggests the importance of postprocedural follow-up. In addition to using aortograms during angiography to detect aortic dissection, on-table CT scans and calcium signs under fluoroscopy might be helpful.[8] In addition, CT angiography is useful to confirm and follow up the condition of aortic dissection (Figs. 2 and 3).

There are several limitations in this study, including a limited number of cases, the retrospective study design, and the different types of stents and balloons used. In the future, a prospective large-scale study design may be helpful to identify the complications of PTAS for stenosis of the SA.

In conclusion, aortic dissection in the left SA during the stenting procedure is not as rare as previously considered and can occur during or after this procedure. Most patients have a good prognosis with conservative treatment. We suggest using self-expandable stents after adequate predilatation for severe stenosis of the left proximal SA to prevent this complication. If postdilatation is necessary, undersized angioplasty is safer.

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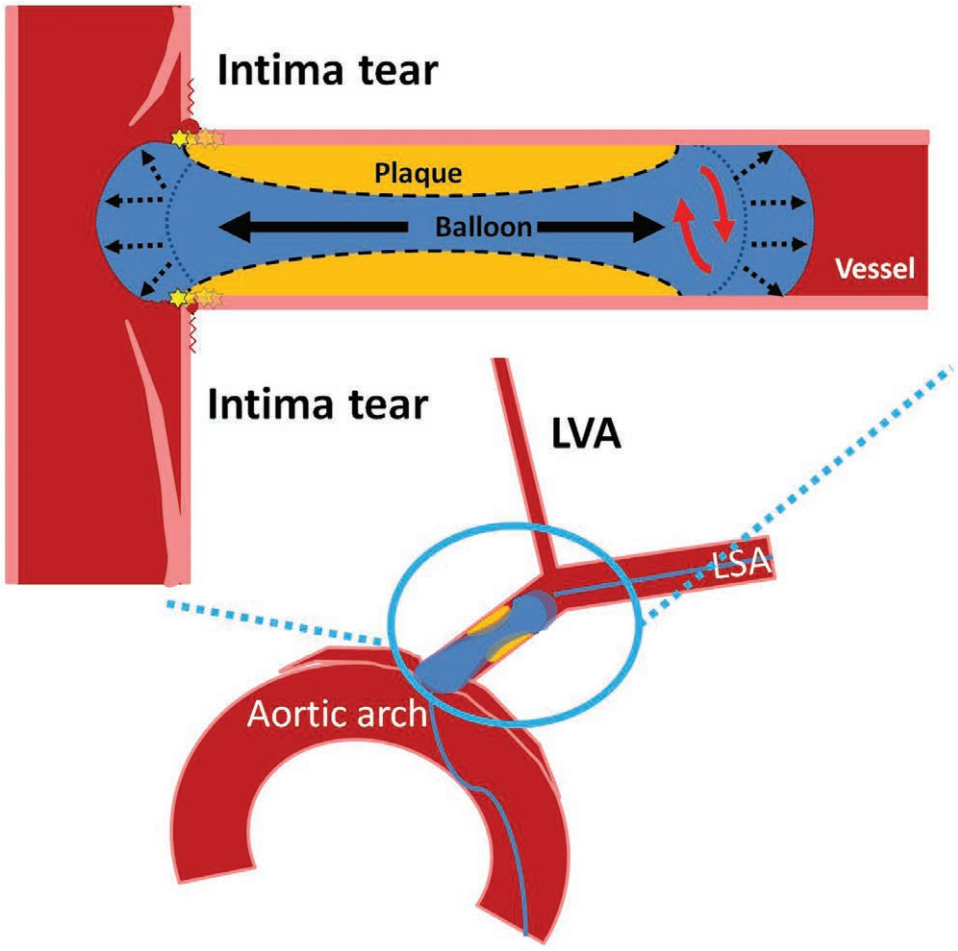

Fig. 4 Suggestive mechanism of the forces of the balloon during angioplasty. The torque force (black arrows) generated by straightening of the balloon causes movement of vessels or plaques. The radial force (dotted arrows) is generated directly from the expansion of the balloon on the vessel wall or plaques. The shear force (red arrows) is the rotating friction between the balloon with a stent and a vessel wall or plaque. The overall forces may cause intima tears at the junction of the left subclavian artery and the aorta, resulting in aortic dissection.

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