

Atherosclerotic Pseudoaneurysm of the Left Subclavian Artery: A Case Report

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A subclavian artery aneurysm is rare. We report the case of a 61-year-old man with a history of hypertension and angina pectoris who was diagnosed with an atherosclerotic aneurysm of the left subclavian artery. An incidental computed tomography scan revealed a 37-mm saccular aneurysm adjacent to both the proximal left subclavian artery and the distal aortic arch. He underwent an aneurysmectomy with total aortic arch replacement via a median sternotomy. Operative findings revealed that the aneurysm had originated from the left subclavian artery and was located 1 cm distal to the ostium. The final pathological diagnosis was a pseudoaneurysm. (Ann Thorac Cardiovasc Surg 2010; 16: 376–379)

Key words: subclavian artery aneurysm, pseudoaneurysm

Introduction

Isolated subclavian artery aneurysms (SAAs) are rare. Aneurysms resulting from atherosclerosis, trauma, thoracic outlet syndrome, infection, and congenital arterial anomalies, such as Marfan syndrome or von Recklinghausen's disease, have been previously described. Dent et al. reported that only 3.5% of atherosclerotic peripheral artery aneurysms were SAAs.¹⁾ Most of the reported atherosclerotic SAAs are true aneurysms. Here we present the case of an atherosclerotic pseudoaneurysm of the left subclavian artery.

Case Report

A 61-year-old man with a history of hypertension and smoking was admitted for unstable angina. An incidental chest computed tomography (CT) scan revealed a saccular aneurysm, which was 37 × 28 × 29 mm in size, adjacent

to both the proximal left subclavian artery and the distal aortic arch (Fig. 1A and B). Three-dimensional CT angiography revealed that the ostium of the left subclavian artery was dilated (Fig. 1C). He had no history of traumatic lesions, infective disease, or congenital arterial anomalies and no upper extremity embolic symptoms. Percutaneous catheter intervention in the coronary arteries was carried out. The patient was carefully monitored for 4 months, and there was no alteration in the aneurysm size during this period. Therefore the patient was readmitted.

The patient underwent surgical management. Because his preoperative CT scan did not reveal the exact origin of the aneurysm, and since dilatation of the ostium of the subclavian artery was observed, we diagnosed the aneurysm as a distal arch aneurysm.

The operation was performed via a median sternotomy with a 2-cm left supraclavicular extension. A cardiopulmonary bypass was established with ascending aorta and bicaval cannulation. After systemic deep hypothermia (25°C) and circulatory arrest followed by transverse aortotomy and antegrade selective cerebral perfusion through the innominate and left carotid arteries, the left subclavian artery was exposed and incised. The walls of the proximal left subclavian artery and distal aortic arch were severely atherosclerotic, and ulcerlike projections (ULPs) were observed. A saccular aneurysm measuring 3 × 3 cm derived from the left subclavian artery and located 1 cm distal to

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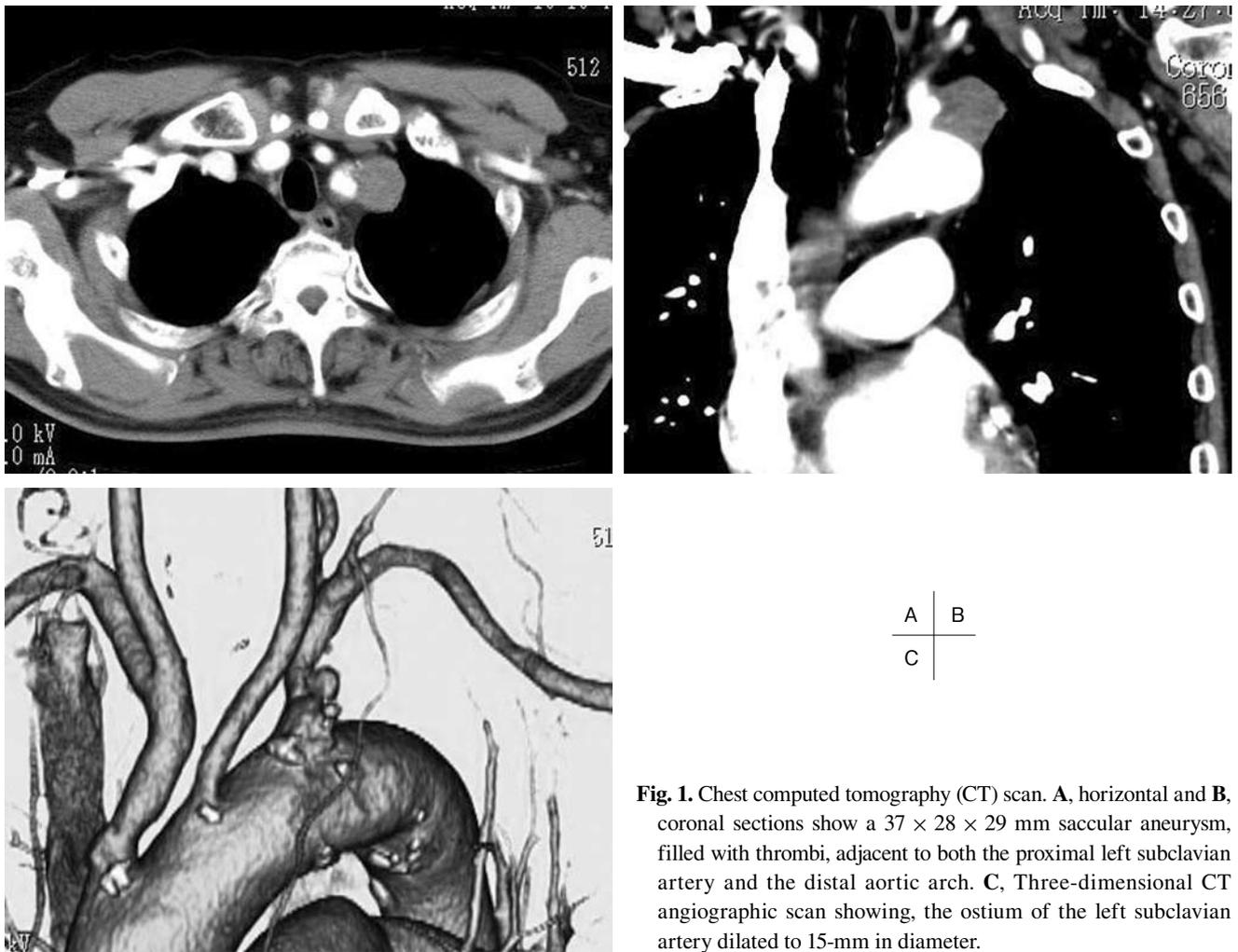


Fig. 1. Chest computed tomography (CT) scan. **A**, horizontal and **B**, coronal sections show a $37 \times 28 \times 29$ mm saccular aneurysm, filled with thrombi, adjacent to both the proximal left subclavian artery and the distal aortic arch. **C**, Three-dimensional CT angiographic scan showing, the ostium of the left subclavian artery dilated to 15-mm in diameter.

its origin was observed (Fig. 2A and B). A large ULP, approximately 6 mm in diameter, was identified on the left side of the arterial wall (Fig. 2B). The ostium of the left subclavian artery was dilated, but no direct continuation to the main aneurysm was observed. The aneurysm was replaced by an 8-mm Dacron tube graft, and a total aortic arch replacement was conducted with a 4-branch Dacron graft using an open distal anastomosis technique by standard procedure. Histological study revealed that the left SAA was an atherosclerotic pseudoaneurysm (Fig. 3). The postoperative course was uneventful, and the patient was discharged 22 days after the operation.

Discussion

Dougherty et al. identified 64 reported cases of patients with a total of 70 nontraumatic SAAs in the literature since 1926.²⁾ The most common etiology of SAAs was

atherosclerosis, constituting of approximately 60% of the reported causes, followed by infectious aneurysms, i.e., syphilis, tuberculosis, mycotic, or bacterial infections. Congenital arterial anomalies, such as Marfan syndrome and cystic medial necrosis, constituted 10% of the causes. Among recent reports, it was found that true SAAs in intrathoracic lesions were mainly due to atherosclerosis, whereas in the case of extrathoracic lesions, thoracic outlet syndrome was the main cause of SAAs.^{2,3)}

A nontraumatic pseudoaneurysm of the subclavian artery is extremely rare. Furthermore, there have been only a few case reports of pseudoaneurysms in other arch vessels caused by Beçhet disease, von Recklinghausen's disease, Marfan syndrome, or mycotic or idiopathic infection.⁴⁻⁷⁾ Our patient had no history of trauma, infections, arterial anomalies, or systemic vasculitis. Histological examination showed the existence of ULP that penetrated the elastic lamina and was associated with hematoma

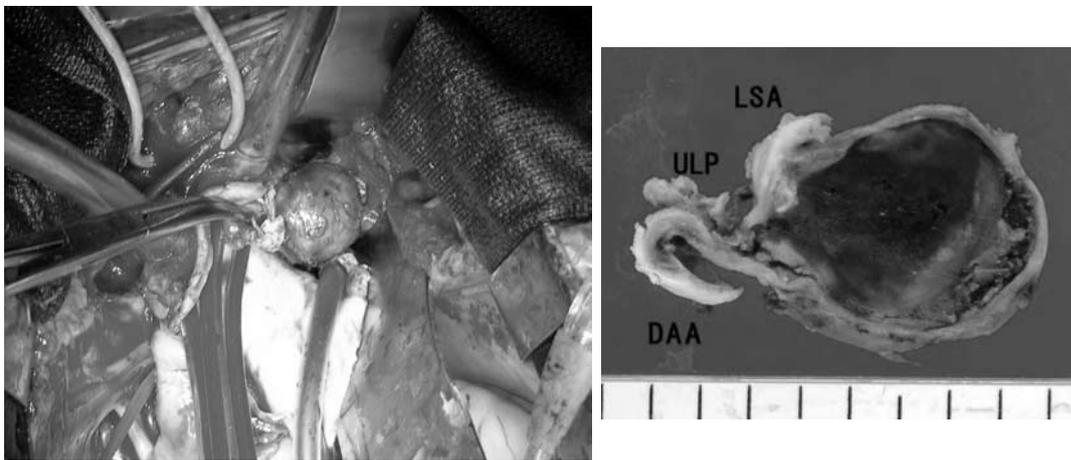


Fig. 2. Operative findings. **A,** Preoperative view of the saccular aneurysm. **B,** Image of the excised specimens. The sagittal section of the saccular aneurysm demonstrates that it is derived from the left subclavian artery and is located 1-cm distal to its origin. A large ULP with a diameter of 6 mm is observed at the left side of the subclavian artery wall.

LSA, left subclavian artery; DAA, distal aortic arch; ULP, ulcer-like projection.

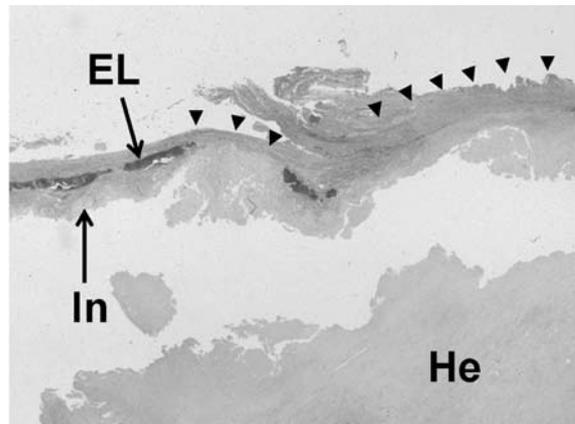


Fig. 3. Microscopic findings of the subclavian artery aneurysm demonstrating atheromatous degeneration with disruption of the intima, elastic lamina, and media (Elastic van Gieson stain, $\times 10$).

In: intima, EL: elastic lamina, He: hematoma. Triangles indicate disruption of the elastic lamina.

formation within the aortic wall. These findings indicated that the most likely cause of the pseudoaneurysm development was a penetrating atherosclerotic ulcer (PAU), even though it was not clear whether the ULP existed before aneurysm formation. We found only 1 case report of spontaneous rupture of the innominate artery caused by PAU.⁸⁾ But no reports of SAA were found. PAUs are uncommon and mainly observed in descending thoracic aorta.^{9,10)} Lesions are mainly symptomatic, but 26 of 105 PAUs in the descending thoracic aorta were asymptomatic.

⁹⁾ Our patient was asymptomatic, and the wall of proximal descending thoracic aorta was severely ulcerated compared to those of the arch and ascending aorta. Our case might be considered as a variant of chronic PAU in the descending thoracic aorta.

Surgery is the main treatment for SAAs,^{2,3)} Endoluminal stent-graft replacement is also performed in limited cases.¹¹⁾ The surgical approach depends on the location of the aneurysm. For extrathoracic SAAs, supraclavicular incision is adequate for aneurysmectomy. An intrathoracic

aneurysm on the right side is best managed by median sternotomy, whereas lateral thoracotomy is preferred for the left intrathoracic aneurysms.^{2,12,13} Resection of the aneurysm and direct graft replacement is recommended. However, in many cases of proximal left SAAs, the origin of the left subclavian artery is fragile and dilated, and reconstruction by anastomosis or direct closure of the origin with partial clamp is difficult.^{2,12} Salo performed aneurysmal resection by left thoracotomy for 6 proximal left SAA patients, and 2 of these experienced two major strokes, two recurrent nerve palsies, and one hemorrhage. Because the aneurysmal resection is associated with a high risk of major complications, some physicians performed hybrid treatments consisting of the extra-anatomical bypass and exclusion of the aneurysm, using endoluminal stent grafting of the aorta, and they achieved good outcomes.^{12,14} These methods are attractive not only because of their minimal invasiveness, but also because they prevent embolisms from the aneurysmal thrombus. The patency of a carotid-subclavian bypass graft will have a suitable patency, but the long-term outcomes have not been completely evaluated. On the other hand, despite the relatively high risk of complications, conventional aortic arch replacement with circulatory arrest and selective cerebral perfusion is the established method. In our case, it was easy to expose the aneurysm with minimal risk of embolism during circulatory arrest. For the treatment of proximal left SAAs with dilated origin, arch replacement via a median sternotomy is one of the suboptimal procedures.

In conclusion, we report a 61-year-old male patient with an atherosclerotic pseudoaneurysm of the proximal left subclavian artery.

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