Coil embolization of bilateral internal mammary artery aneurysms is durable in a patient with Marfan syndrome

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ABSTRACT

Internal mammary artery (IMA) aneurysms are very rare, have a high risk of rupture, and can cause hemothorax. Here, we report the case of a 33-year-old man with metachronal and bilateral IMA aneurysms. He had Marfan syndrome diagnosed by genetic testing. We carried out endovascular repair with coil embolization. He has survived without additional treatment for 7 years. Endovascular repair of metachronal and bilateral IMA aneurysms is feasible even in a patient with Marfan syndrome. (J Vasc Surg Cases and Innovative Techniques 2018;4:216-9.)

Keywords: Internal mammary artery aneurysms; Marfan syndrome; Endovascular repair

Internal mammary artery (IMA) aneurysms are very rare, with only 40 cases reported in the last 40 years.[1] Although IMA aneurysms are small, they often rupture and cause hemothorax, which can be life-threatening.[1,2] Although the cause of IMA aneurysms is iatrogenic or traumatic in most cases, it can be inflammatory vasculitis or connective tissue disease, including Loeys-Dietz syndrome, Marfan syndrome (MFS), Ehlers-Danlos syndrome, neurofibromatosis type 1, and fibromuscular dysplasia.[1] Here, we describe an extremely rare case of metachronal, bilateral IMA aneurysms due to MFS successfully treated by endovascular therapy. The patient consented to participation and publication of this case report.

CASE REPORT

We report the case of a 33-year-old man with a history of medical and surgical interventions for aortic anomalies. In August 2002, he presented with symptoms of acute type B aortic dissection and was treated by anti-impulse therapy using beta blockers, angiotensin II receptor blockers, and an angiotensin-converting enzyme inhibitor. In April 2006, he underwent an open repair of the proximal descending thoracic aorta for the treatment of an aneurysmal dilation of the false lumen of the descending aorta. In August 2006, he underwent a second-stage thoracoabdominal aortic repair with a reconstruction of intercostal arteries.

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In May 2010, a follow-up computed tomography (CT) scan revealed a new aneurysmal dilation in the left IMA (LIMA; Fig 1, *A-C*). Hence, coil embolization of the aneurysm was performed. Furthermore, puncture of the left brachial artery and selective catheterization of the LIMA were performed using a 4F catheter followed by a microcatheter. Angiography revealed the aneurysm at the midportion of the LIMA (Fig 2, *A*). The LIMA was occluded with 37 detachable coils (Tornado; Cook Medical, Bloomington, Ind) that were distally and proximally placed to the aneurysm, and the aneurysm was packed with 20 detachable coils. Completion arteriography demonstrated the absence of filling in the coiled aneurysmal sac.

In October 2012, a CT scan revealed two aneurysmal dilations in the right IMA (RIMA; Fig 1, *D*). In May 2012, coil embolization of the aneurysms was performed using a 0.016-inch guidewire and a 0.018-inch microcatheter. Retrograde cannulation of the RIMA was performed through the right brachial artery. Angiography revealed both aneurysms at the midportion of the RIMA (Fig 2, *B*). Coil packing of the aneurysms was performed with detachable coils. A total of 35 detachable coils were placed at the proximal and distal necks of the aneurysms. Completion arteriography demonstrated the absence of filling in the coiled aneurysmal sac. The follow-up images revealed successful embolization of the RIMA and subsequent complete thrombosis of both aneurysms (Fig 3). Five years after the final intervention, three-dimensional CT revealed no recurrence of the aneurysms (Fig 1, *E*).

Diffuse and rapidly progressing vascular disease was diagnosed in 2017 in our patient who met the criteria for MFS, namely, a height of 181 cm, family history, dural ectasia, clubfoot, and hypertelorism. Genetic testing also revealed a novel frameshift mutation of the *FBN1* gene (Fig 4).

DISCUSSION

Only a few studies have reported true IMA aneurysms in the medical literature.[1-6] The noniatrogenic or nontraumatic etiology, including vasculitis, connective tissue diseases, neurofibromatosis type 1, fibromuscular dysplasia, atherosclerosis, and idiopathic causes, is very rare.[3-7] MFS is an autosomal dominant inherited connective tissue

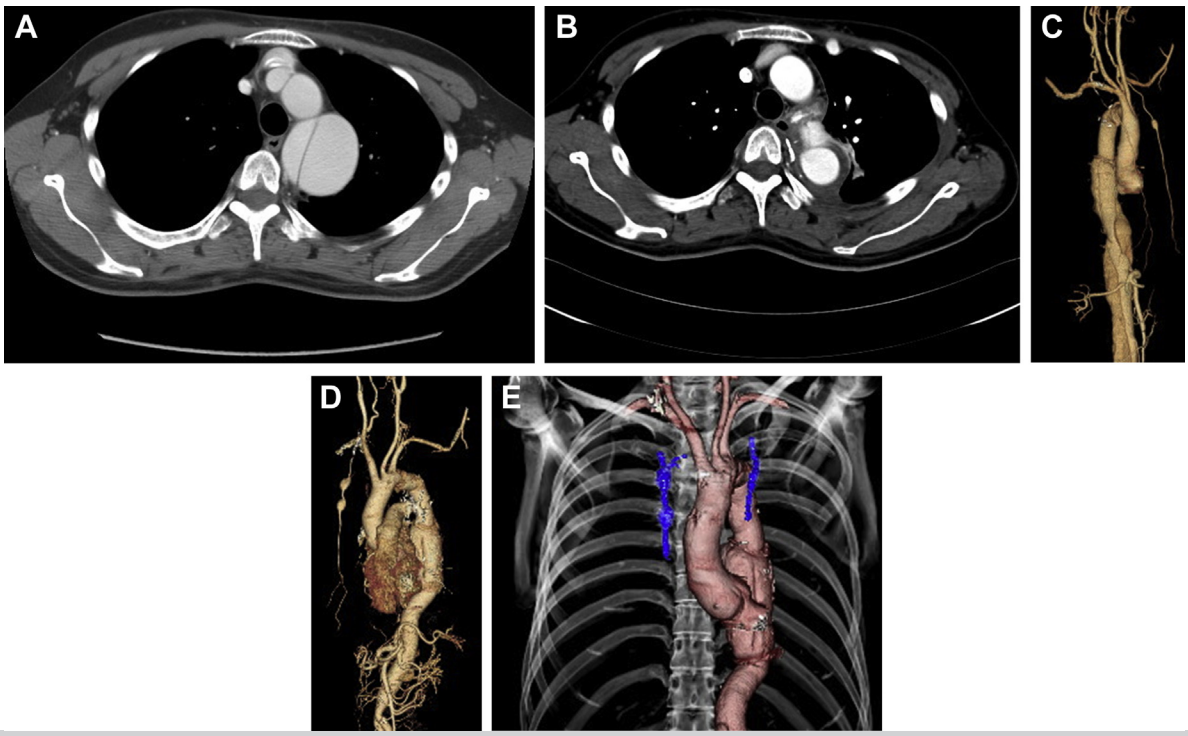

Fig 1. Computed tomography (CT) scan image. **A,** Before 6 months, an aneurysmal dilation in the left internal mammary artery (LIMA) was not detected. **B,** Axial CT showing a new aneurysmal dilation of the LIMA. **C,** Three-dimensional CT showing a new aneurysmal dilation of the LIMA. **D,** Three-dimensional CT showing a new aneurysmal dilation in the right internal mammary artery (RIMA). **E,** The most recent three-dimensional CT revealed no recurrence of the aneurysm.

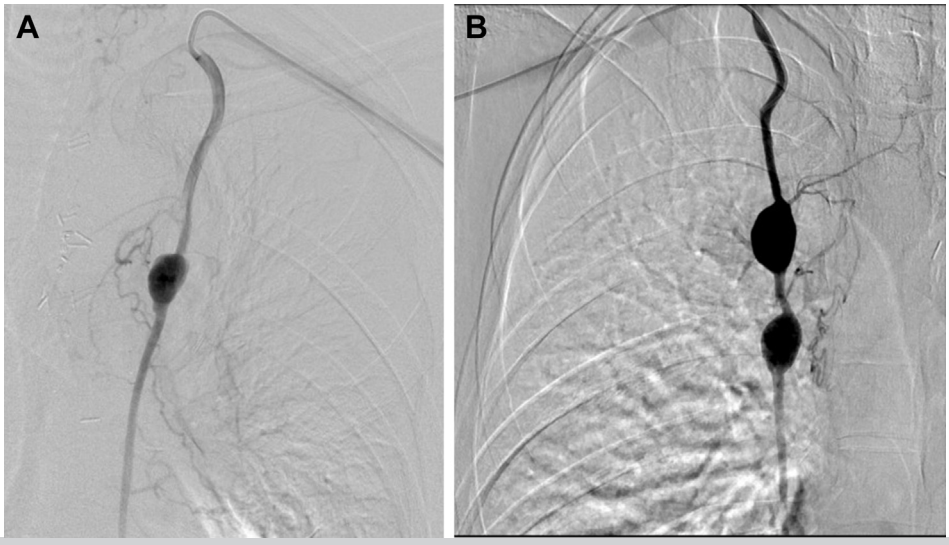

Fig 2. Angiography image. **A,** An aneurysmal dilation in the left internal mammary artery (LIMA). **B,** An aneurysmal dilation in the right internal mammary artery (RIMA).

disorder due to genetic mutation in the FBN1 receptor. The likely causes of mortality are aortic aneurysms, dissections, and ruptures. Moreover, MFS is associated with multiple aneurysms and arterial tortuosity throughout the body.[8] In this case report, we describe an MFS patient with metachronal and bilateral IMA aneurysms.

An appropriate selection of interventions is essential because arterial tortuosity may preclude an endovascular intervention. Proper diagnosis is necessary to avoid selecting unsuitable interventions in patients with connective tissue diseases that may phenotypically mimic more common connective tissue diseases. Before surgical