Texas Heart Institute Journal

Case Reports

Endovascular Treatment of Acquired Atheromatous Postsubclavian Aortic Coarctation

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Coral reef aorta is a rare calcifying obstructive disease that involves the thoracoabdominal aorta. Similar presentations in the postsubclavian aorta may result in acquired atheromatous aortic coarctation leading to systemic hypertension and heart failure. The associated calcification makes surgical anatomic or extraanatomic bypass and thromboendarterectomy challenging. Extensive circumferential calcification often precludes endovascular intervention. We present the case of a 25-year-old man with an acquired atheromatous coarctation of the postsubclavian aorta who underwent successful endovascular treatment with use of a balloon-expandable covered stent. ***(Tex Heart Inst J 2021;48(5):e207270)***

Coral reef aorta is a rare calcifying obstructive aortic disease that can cause lower limb and visceral ischemia, renovascular hypertension, and heart failure.[1-3] The pathogenesis of coral reef aorta and the extensive calcification and metaplastic bone formation associated with it is uncertain.[2] Surgical treatment is challenging and can result in highly morbid complications and death.[3] Extensive and circumferential calcification poses difficulties for endovascular therapy.[4,5] Calcification of the postsubclavian aorta can lead to acquired atheromatous coarctation.[6,7] We present the case of a young man with an acquired atheromatous coarctation of the postsubclavian aorta.

Case Report

A 25-year-old man presented with effort dyspnea of 2 months' duration, systemic hypertension, and weak femoral pulses. One year previously, he had been diagnosed with diabetes and hyperlipidemia. He reported no angina. His current medications included bisoprolol, amlodipine, telmisartan, and hydrochlorothiazide for hypertension; and insulin, vildagliptin, and metformin for diabetes. On examination, the patient's blood pressure was 190/70 mmHg in his right arm and 138/60 mmHg in the legs, along with radiofemoral delay. Laboratory tests revealed a glycosylated hemoglobin level of 8.6%, indicating suboptimal diabetes control; a low-density-lipoprotein cholesterol level of 169 mg/dL; and a triglyceride level of 215 mg/dL.

An electrocardiogram indicated left ventricular hypertrophy. An echocardiogram revealed a large calcific mass protruding into and obstructing the aorta distal to the left subclavian artery, as well as diffuse atheromatous luminal irregularities in the abdominal aorta. A contrast-enhanced computed tomographic aortogram confirmed near-circumferential calcification and a large mass protruding from the anterior wall of the aorta distal to the left subclavian artery (Fig. 1).

During cardiac catheterization, a fluoroscopic angiogram showed substantive calcification of the ascending aortic arch that extended into the coronary tree. Coronary angiograms revealed noncritical narrowing of the ostia to the left anterior descending coronary artery and dominant right coronary artery (RCA). An intravascular ultrasonogram of the RCA revealed an atheromatous plaque and intimal calcification at the ostium. An intravascular ultrasonogram of the left coronary artery could not be obtained because the calcifications in the aortic arch prevented the guide catheter from engaging the coronary ostium. An intravascular ultrasonogram of the aorta revealed severe luminal narrowing due to protruding calcified masses (Fig. 2). The systolic

Citation: Sonawane BS, Pavithran S, Sivakumar K. Endovascular treatment of acquired atheromatous postsubclavian aortic coarctation. Tex Heart Inst J 2021;48(5):e207270. doi: 10.14503/THIJ-20-7270

Key words: Endovascular procedures; plaque, atherosclerotic; stents; vascular calcification

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gradient across the aortic isthmus was 54 mmHg. An aortogram showed a filling defect due to a calcific mass protruding into the aortic lumen (Fig. 3A).

We decided to perform endovascular intervention. A 9F sheath was used to position a 12- × 41-mm, balloon-expandable covered stent (Advanta V12; Atrium Medical Corporation) at the left subclavian artery proximally, and then across the calcified occlusion within the coarcted aortic segment. The stent was then completely expanded, without recoil, by inflating a 14-mm balloon. Even though an irregular luminal calcification protruded from the distal end of the stent, unobstructed flow was restored (Fig. 3B), and the pressure gradient was eliminated. At one-year follow-up, the patient's symptoms had improved, he was receiving optimal diabetic and lipid-lowering medical therapy, and he had no arm-leg pressure gradient.

Fig. 1 *Contrast-enhanced computed tomographic aortogram shows diffuse dense calcification of the ascending aorta, aortic arch, arch vessels, and descending aorta that extends beyond the aortic bifurcation and into the iliac vessels. Note the near-circumferential calcification and a large mass protruding from the anterior wall of the aorta distal to the left subclavian artery (arrow).*

Discussion

Coral reef aorta, which was first observed in the suprarenal aorta, can occur anywhere in the thoracoabdominal aorta.[1,2] The condition affects both sexes equally and is associated with comorbid conditions such as syphilis, neurofibromatosis, rubella, and amyloidosis.[3] An acquired atheromatous coarctation can result when protruding calcified masses occlude the postsubclavian aorta.[6,7]

The heavily calcified obstructive plaques characteristic of coral reef aorta can lead to hypertension, renal hypoperfusion, claudication, and heart failure. Their pathogenesis has been attributed by some to calcification of a fibrin-platelet thrombus, but atherosclerotic risk factors do not sufficiently explain the extensive calcification and metaplastic bone formation.[4,5] Surgical treatment of coral reef aorta—with thromboendarterectomy, calcified thrombus resection, stent-graft placement, or extraanatomic bypass—is associated with high rates of operative mortality and postoperative complications.[3] Frequent postoperative complications include acute leg ischemia, pleural effusion, bleeding, brain infarction, myocardial infarction, spleen rupture, terminal renal insufficiency, necrotizing pancreatitis, and colon ischemia.[3]

In cases of atherosclerotic but noncalcific aortic narrowing, percutaneous angioplasty with or without stents may be technically and clinically advantageous.[8] However, endovascular treatment is not routinely attempted in a coral reef aorta because the risk of aortic rupture is high and because incomplete stent expansion can limit the effectiveness of stent-grafts.[4,5,9]

In our patient, we used a balloon-expandable covered stent to avoid rupture, despite the risk of dissection proximally into the subclavian artery. Such a complication would have necessitated emergency plugging of

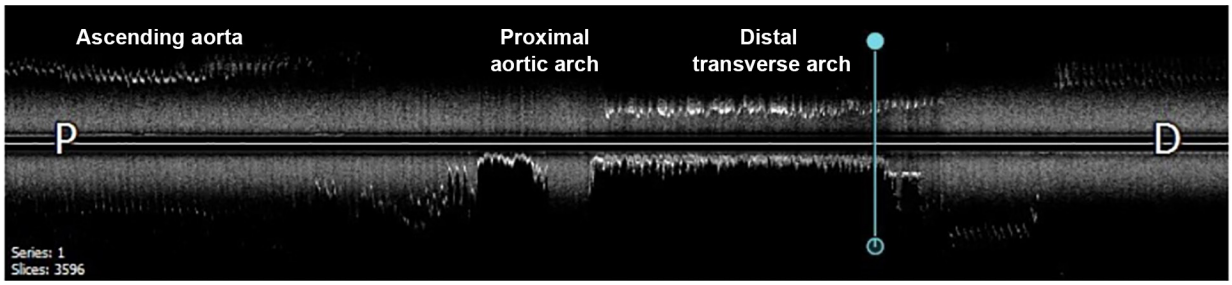

Fig. 2 *Intravascular ultrasonogram obtained during pullback from the ascending aorta to the descending thoracic aorta reveals a dense, linear calcified mass protruding into the aortic lumen. The minimal luminal diameter is 6 mm at the vertical blue marker line.*

D = distal; P = proximal

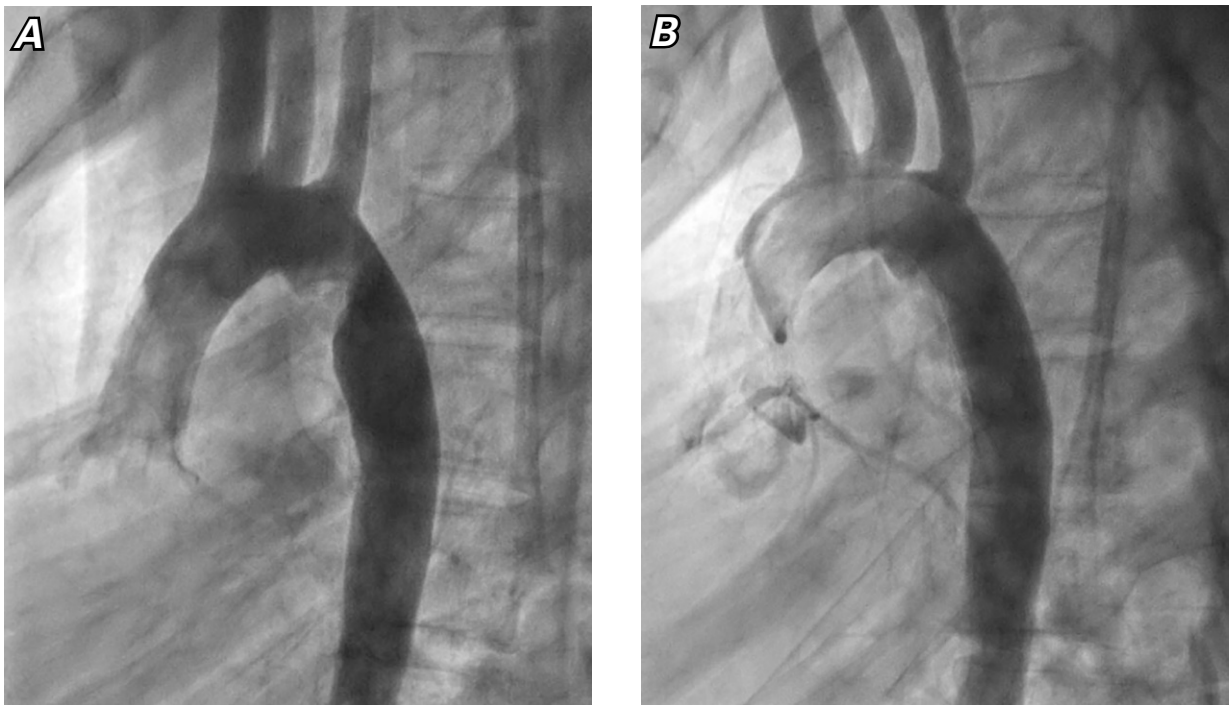

Fig. 3 *Aortograms show **A**) a filling defect caused by a calcified mass protruding into the aortic lumen and **B**) unobstructed flow after deployment of a 12- × 41-mm balloon-expandable covered stent.*

Supplemental motion image is available for Figure 3.

the left subclavian artery origin through the left radial artery and extending the covered stent to the distal transverse aortic arch. Deploying covered stents and excluding circumferential luminal calcifications are mandatory steps to avoid serious procedural complications during endovascular interventions. If the origin of any arch vessel (for example, the subclavian artery) is compromised during an intervention involving covered stents, physician-modified endografts may help maintain patency.[10]

Conclusion

Acquired atheromatous coarctation of the postsubclavian aorta by protruding calcified masses has a pathology similar to coral reef aorta. It can be treated surgically, but the high risks associated with thromboendarterectomy or extraanatomic bypass necessitate alternative options. Endovascular intervention with stents is a viable alternative in selected patients.

Published: 13 December 2021

Conflict of interest disclosures: None

Funding/support: None

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