

Endovascular treatment of acquired atheromatous aortic arch coarctation

Stephan Kische, MD, PhD, Giuseppe D'Ancona, MD, PhD, Jasmin Ortak, MD, PhD, Yannik Stoekicht, MD, and Hüseyin Ince, MD, PhD, *Berlin and Rostock, Germany*

Acquired aortic coarctation is a rare condition. Its treatment using a percutaneous approach can be challenging, especially when severe calcifications and concomitant aneurysmal disease are present. We report a patient with symptomatic thoracic aorta acquired coarctation and aneurysm that was successfully treated using endovascular technique. After left subclavian artery transposition, a self-expanding endograft was implanted percutaneously, with complete abolishment of the transaortic gradient. Follow-up evaluation at 12 months revealed perfect position of the endograft, persistent reduction of the coarctation, and exclusion of the concomitant aneurysm. A noninvasive pressure reading demonstrated significant systemic blood pressure reduction, with no change in antihypertensive medications. (*J Vasc Surg Cases* 2015;1:3-5.)

Acquired aortic coarctation is a rare condition,^{1,2} and the term refers to noncongenital obstructive lesions of the thoracic aorta producing brachiocephalic hypertension and relative hypotension in the lower half of the body. The etiology and anatomical variants of this condition are heterogeneous. One subgroup comprises patients with symptomatic focal obstructive calcified lesions of the aorta. These patients should be differentiated from those presenting with congenital aortic narrowing and superimposed acquired atheromatous calcified thrombi.

When severe aortic calcifications and aneurysm are present, percutaneous treatment can be challenging.³ We report a patient with acquired symptomatic coarctation accompanied by aneurysm formation that was successfully treated using an endovascular approach. The patient consented to personal data handling, including clinical case publication.

CASE REPORT

A 65-year-old woman was admitted to the emergency department for a hypertensive crisis resulting in dizziness, severe headaches, and left arm discomfort. Her past medical history included hypercholesterolemia, type 2 diabetes mellitus, moderate renal impairment, and severe chronic obstructive pulmonary disease (Global Initiative for Chronic Obstructive Lung Disease III). Her present medical history was notable for onset of arterial hypertension, within the last 6 months, that was poorly controlled by a

combination of two antihypertensive drugs. At physical examination, a 2/6 systolic heart murmur was present over the second left intercostal space. In addition, a difference of 50 mm Hg was noticed between the right and left arm blood pressures.

Results of a 12-lead electrocardiogram and transthoracic echocardiography were within normal limits, without any sign of myocardial ischemia, left ventricular dysfunction, or hypertrophy. Results of routine blood analysis were also unremarkable.

To elucidate the vascular anatomy, we performed magnetic resonance angiography of the thoracic aorta and neck vessels. A severe aortic coarctation was noticed between the left subclavian and the left common carotid artery. Immediately after the coarctation, the aorta had developed a convoluted aneurysm, proximal to the origin of the left subclavian artery (Fig 1, A). A computed tomography (CT) scan was performed to better clarify the anatomy of the lesion and its relationship with the neck vessels and thoracic organs (Fig 1, B-E). Particular attention was given to the degree and distribution of calcifications to guide any possible operative approach. CT imaging demonstrated a localized calcified plaque circumferentially protruding >5 mm within the aortic lumen. The overall extension of aortic calcifications measured 6 cm (Fig 1, B-E). The remaining aorta appeared within normal limits. In particular, no collateral circulation originating before the coarctation and revascularizing the lower part of the body was noticed. An invasive diagnostic workup, performed through right radial and femoral artery access, revealed normal coronary arteries and a systolic pressure gradient of 55 mm Hg across the coarctation.

In consideration of the significant risk of embolization from the atheromatous aortic arch, the patient was prescribed high-dose simvastatin, and a multidisciplinary discussion was held to investigate possible treatment strategies of the acquired aortic coarctation.

In light of the increased operative risk derived by her complex comorbid profile (Society of Thoracic Surgeons Score, 24%), the patient was referred for a minimally invasive percutaneous approach and consented to the procedure. In a first stage, a subclavian artery transposition was performed to avoid its unprotected coverage.

In a second phase, within the same hospitalization, a 24-mm Valiant thoracic endoprosthesis (Medtronic, Minneapolis, Minn) was implanted via transfemoral access. The procedure was performed in a hybrid suite with surgical backup to manage

From the Vivantes Klinikum im Friedrichshain und Am Urban, Berlin; and the University Medical Center, Rostock.

S.K. and G.D.A. contributed equally to this work.

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Reprint requests: Giuseppe D'Ancona, MD, PhD, Department of Cardiology, Vivantes Klinikum Friedrichshain, Landsberger Allee 49, 10249 Berlin, Germany (e-mail: rgea@hotmail.com).

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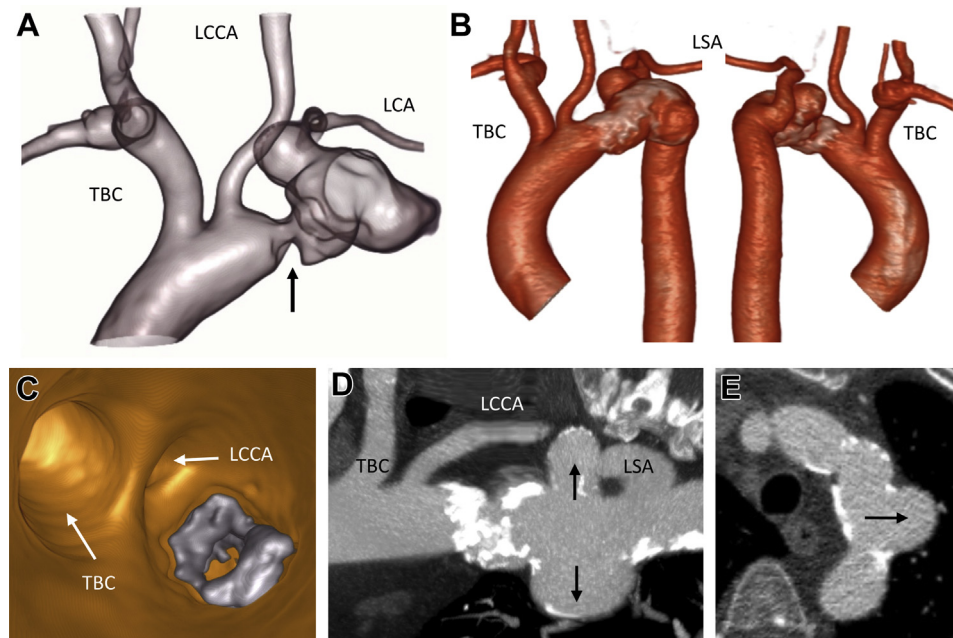


Fig 1. **A**, Three-dimensional magnetic resonance image shows severe stenosis of the arch (*black arrow*) distal to the left common carotid artery (*LCCA*) with a concomitant aneurysm in close vicinity to the aortic obstruction. **B**, Three-dimensional reconstruction from an electrocardiogram-gated computed tomography (CT) confirms extensive calcification within the middle-distal arch. **C**, The accompanying aneurysm presents with an irregular shape. Virtual angiography depicts the target lesion as viewed from the ascending aorta, thus demonstrating a calcified subocclusive plug proximal to the origin of the left subclavian artery (*LSA*). The *white arrows* indicate the unaffected ostia of the brachiocephalic trunk (*TBC*) and the *LCCA*. The spatial relations between the intraluminal calcification, the poststenotic aneurysm (*black arrows*), and the supraaortic branches are further clarified by (**D**) a virtually sketched view and by (**E**) the axial slices.

inadvertent aortic rupture. The endoprosthesis was cautiously advanced across the arch to prevent atherosclerotic debris embolism and was deployed under rapid ventricular pacing.

Immediately after release, a residual stenosis was noticed with a persistent transaortic gradient of 30 mm Hg. At this stage, a progressive stent-protected postdilatation of the endoprosthesis was performed, under rapid ventricular pacing and using noncompliant 8-mm and 10 mm Tyshak-X (NuMed, Hopkinton, NY) and 14-mm Maxi-LD (Cordis, Bridgewater, NJ) balloons. Although a residual stenosis was present, the transcoarctation gradient was finally reduced to 0 mm Hg, and the poststenotic convoluted aneurysm was completely excluded.

The patient's postoperative course was uneventful, and she was discharged home within a week. A 12-month follow-up CT scan confirmed a perfect position of the endoprosthesis, drastic and persistent reduction of the aortic coarctation degree, and exclusion of the concomitant aneurysm (Fig 2). A 24-hour noninvasive pressure reading demonstrated significant reduction of systemic blood pressure with dual-antihypertensive therapy.

DISCUSSION

Acquired aortic coarctation is rare, and its etiology is often related to atherosclerosis.⁴ The evolution of the lesion is normally slow, allowing for compensatory ventricular hypertrophy and development of collateral circulation to vascularize the distal body. When severe calcifications are present within the coarctation and in the bordering

segments of aorta, the macroscopic image resembles a coral reef.^{5,6} In this condition, treatment of the coarctation may be cumbersome as a result of the embolization and rupture risks.

The patient in the case reported here presents some peculiarities. First, the origin of the lesion was unclear. In fact, the sudden onset of symptoms and the complete absence of compensatory signs to overcome the coarctation may all suggest an almost subacute evolution. If this were the case, a different etiology could be hypothesized to explain the rapid evolution of the coarctation. Whether the lesion was of atherosclerotic or other origin (infective, inflammatory) cannot be answered from the collected information.

Second, the location of the lesion is also unusual. In fact, most acquired coarctations are postisthmic.^{5,7} In this particular case, the stenosis was located immediately before the origin of the left subclavian artery.

Finally, the treatment that we selected was not conventional for this type of pathology. Although percutaneous treatment of acquired aortic coarctation has been proposed,^{8,9} management of such a heavily calcified and complex lesion can be treacherous. Conventional surgery should be advocated for most patients, especially when the expertise in percutaneous treatment of such complex cases is not available.^{5,10} The risks of endovascular treatment include embolization of calcified debris, iatrogenic

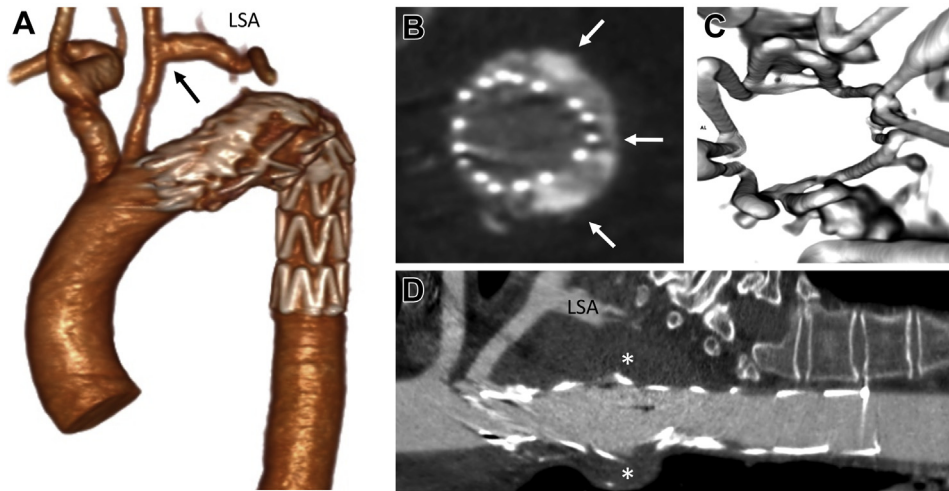


Fig 2. Follow-up computed tomography (CT) scan shows the thoracic aorta anatomy 12 months after endovascular repair of the acquired aortic coarctation. **A**, A three-dimensional reconstruction shows the endoprosthesis within the distal aortic arch. To avoid unprotected side branch coverage, a left subclavian artery (LSA) transposition was performed before the endoprosthesis placement (*black arrow*). **B**, Discrete residual compression of the proximal stent by compact calcified debris (*white arrows*) is displayed in a cross-sectional plane and (**C**) further clarified by a virtual angioscopy. **D**, Note that the obstructed lumen has been adequately reconstructed with exclusion of the convoluted poststenotic aneurysm (*).

aortic rupture, and recurrent stenosis.⁷ When a percutaneous strategy is planned, two important goals should be achieved. First, the immediate and direct trauma within the aorta should be minimized, reaching progressively the desired pressure gradient reduction. Second, complete exclusion of the concomitant poststenotic aortic dilatation should be simultaneously obtained.¹¹

In our experience, a self-expandable covered stent (endoprosthesis) presents the required characteristics to treat this condition. Its radial force will lead to partial dilatation of the coarctation and immediate aneurysm exclusion. A progressive and protected (from the endoprosthesis fabric) balloon dilatation can be performed whenever residual aortic stenosis is noticed. In particular, further dilatation should be aimed mainly at correcting the transaortic gradient and, for this reason, should be guided more by the hemodynamic measurements than by the angiographic imaging.

CONCLUSIONS

Acquired coarctation may result from severe calcification and atherosclerosis of the aortic wall. Although conventional surgery must be advised in most patients, tailored endovascular approaches could be proposed. In this context, when extensive aortic calcifications are present, endovascular treatment can be an option, if performed within an adequately experienced environment and using the appropriate material.¹²

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