Calcified obstructive disease of the aortic arch
Laura Palcau, Djelloul Gouicem, Lucie Cameliere and Ludovic Berger*

Department of Vascular Surgery, CHU Côte de Nacre, Caen, France

* Corresponding author. Department of Vascular Surgery, CHU de Caen, Faculty of Medicine EA4650, 14000 Caen, France.
Tel: +33-231064445; fax: +33-2-31065165; e-mail: berger-l@chu-caen.fr (L. Berger)

Received 19 August 2013; received in revised form 30 October 2013; accepted 5 November 2013

Abstract
Coral reef aorta is described as rock-hard calcifications usually localized in the visceral part of the aorta. Rare cases of acquired coarctation of the descending aorta due to coral reefs have been mentioned in the literature. A more uncommon entity is the coral reef of the aortic arch. We are presenting a rare case of a 55-year-old woman referred to our vascular department for bilateral lower limb claudication associated with resistant hypertension and anisotension. A thoracoabdominal computed tomography scan was subsequently performed and showed a preocclusive calcified lesion located at the termination of the aortic arch, involving the ostia of the left subclavian artery. An open surgical approach was decided upon and an aortic endarterectomy by transversal aortotomy was successfully performed. Although uncommon, acquired coarctation should be considered in all hypertensive patients presenting with bilateral lower limb claudication and blood pressure differences between the upper and lower extremities.

Keywords: Atherosclerotic occlusive disease • Coral reef aorta • Aortic arch

INTRODUCTION
Coral reef aorta (CRA) is described as rock-hard calcifications usually localized in the visceral part of the aorta. Acquired coarctation, in which a non-congenital obstructed thoracic and/or abdominal aorta causes a pressure gradient between the upper and lower half of the body with atherosclerosis, is rare. Heavily calcified plaques grow into the lumen and can cause significant stenosis, leading to malperfusion of the lower limbs, visceral ischaemia, severe congestive heart failure or hypertension due to renal ischaemia.

CASE REPORT
A 55-year-old woman was referred to our vascular department for bilateral lower limb claudication associated with resistant hypertension and anisotension. Cardiovascular risk factors included dyslipidaemia and smoking. An aortic and lower limb artery ultrasound was performed and showed no atherosclerotic lesion, but there was a collapsed ankle–brachial pressure index of 0.57 on the left side and 0.65 on the right.

A thoracoabdominal computed tomography (CT) scan was subsequently performed and showed a preocclusive calcified lesion located at the termination of the aortic arch, involving the ostia of the left subclavian artery (Fig. 1). To complete the examinations, a coronary angiography and cardiac evaluation were performed but showed no abnormalities.

Open surgical treatment was decided upon and performed by a left anterolateral thoracotomy centred on the fourth intercostal space. The end of the aortic arch and the left subclavian artery were exposed. Extracorporeal circulation by left femoro-femoral bypass under normothermia with a beating heart was used during aortic repair. Aortic clamps were applied proximally between the left common carotid artery and the left subclavian artery, and distally. Only the anterior aortic wall was opened transversally in order to perform a complete endarterectomy of the calcified lesion with a better visualization of the subclavian artery ostia. As classical, only the inner layer of the aortic wall was removed. The aortic wall was then closed without the need of prosthetic graft interposition with a reinforced pledgeted suture using Teflon material. The aortic clamping time was 37 min with 53 min of extracorporeal circulation.

The recovery of the patient was satisfactory, and the patient was discharged 5 days after surgery.

The 3-month CT scan showed a good result of the endarterectomy (Fig. 2). The patient was asymptomatic and her blood pressure was controlled on only one hypotensive drug. The postoperative ankle–brachial pressure index was 0.80 on the left side and 0.83 on the right.

DISCUSSION
Acquired coarctation due to coral reefs is a rare condition characterized by extensive calcification of the thoracic aorta. Occlusive lesions of the descending aorta or the aortic isthmus are described in the literature [1]. The aortic arch localization is uncommon. One similar case was described in the literature [1]. The particularity of our presentation was the localization of the lesion and the fact that it involved the subclavian artery ostia (otherwise cause of clinical anisotension) and the aortic isthmus rather than just the descending thoracic aorta.

Aetiologically, heavily calcified plaques grow into the lumen and can cause significant stenosis, leading to upper extremity
hypertension and reduced blood pressure in the lower extremities, intermittent claudication or even heart failure. Although the pathogenesis of CRA remains uncertain, it has been suggested that this unique phenomenon may be attributed to calcification of a fibrin–platelet thrombus. The common risk factors for cardiovascular diseases, such as smoking and arterial hypertension, are not sufficient to explain the pathogenesis of the sclerosis in CRA patients. The sex distribution was found to be nearly equal: 55.3% women and 44.7% men, with a mean age of 59.5 years [2]. The diagnosis of this rare condition can be easily established by routine measurement of blood pressure in the upper and the lower extremities in all hypertensive patients with or without intermittent claudication. In a patient with a proven pressure gradient, total three-dimensional (3D) angiographic visualization of the aorta must be performed.

Coral reef aorta is essentially treated by conventional surgery comprising thromboendarterectomy, resection of the calcified thrombus and graft replacement of the aorta, or placement of a thoracoabdominal bypass graft—in accordance with the localization of the lesion. The endarterectomy is considered the gold standard treatment. These direct aortic surgeries are more invasive, and some authors have described an operative mortality rate of 8.7–11.6% and a rate of postoperative complications requiring corrective surgery of 13.9–15.9% [3]. The treatment of choice is an open transaortic thrombendarterectomy. Usually, a longitudinal aortic incision is described [4]. Our approach was particular, as we employed a partial transverse incision, which facilitated a complete circumferential endarterectomy with good visualization of the subclavian artery ostia and enabled us to avoid an aortic transection. A direct suture repair decreases the operative time and it is well known that a transverse incision reduces the risk of restenosis. The remaining aortic wall was sufficiently solid to avoid aneurysmal evolution. A reinforced suture with Teflon flat or pledgeted knots is recommended. We used the Teflon pledget points of suture. Other less invasive strategies using endovascular devices have been described. However, some defects are associated with these approaches. Stent-grafting might be limited by the lack of self-expanding capacity of the graft in this heavily calcified lesion with a high risk of aortic rupture. In addition, paraplegia due to spinal ischaemia and distal embolization can occur. In our case, spinal ischaemia was avoided by using extracorporeal circulation. With regard to aortic arch lesions, the experience with endovascular treatment modalities in adults is limited, with the mean age in most studies not exceeding 30 years [5]. Our patient was not an appropriate candidate for percutaneous intervention due to the extent of the calcifications and location of the calcification in the subclavian artery. An endograft placement would have been sensible more so if it were covered.

**CONCLUSION**

Coral reef of the aortic arch is rare but must be suspected in every hypertensive patient presenting with blood pressure differences between the upper and lower extremities associated with intermittent claudication, visceral insufficiency and/or cardiac failure.

**Conflict of interest:** none declared.

**REFERENCES**