CLINICAL VIGNETTE

# Coral reef aorta involving the proximal part of the descending aorta

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A 65-year-old man with ischemic cardiomyopathy and acute heart failure due to aortic stenosis was referred to our hospital. Cardiovascular risk factors included mixed hyperlipidemia and hypertension. The patient presented with symptomatic bradycardia. Non–ST-segment elevation myocardial infarction (NSTEMI) was diagnosed.

Coronary angiography indicated multivessel coronary disease. The right coronary artery was totally occluded. The left anterior descending artery and the second marginal branch showed 80% stenosis. No aortic obstruction was noticed during the procedure.

Arteriography of the iliac arteries was performed, which showed 80% stenosis in the left external iliac artery and 60% stenosis in the right external iliac artery. The patient presented symptoms of visceral ischemia.

Echocardiography revealed signs of severe aortic valve stenosis and ischemic cardiomyopathy of the left ventricle with reduced contractility. The end-diastolic and end-systolic dimensions of the left ventricle were 55 mm and 44 mm, respectively, and the left ventricular ejection fraction was 40%.

Chest X-ray did not reveal any abnormalities.

The patient was referred for coronary artery bypass grafting and aortic valve replacement. A 23-mm biological valve was implanted (St. Jude Medical, St. Paul, Minnesota, United States). Three coronary artery bypass grafts were also applied: left internal thoracic artery–left anterior descending artery, aortic root–left obtuse marginal branch, and aortic root–posterior descending artery.

Electrocardiography and echocardiography findings after surgery were normal. The patient reported abdominal pain during hospitalization, which was an indication for computed tomography (CT; dual-source CT scanner SOMATOM Definition Flash, Siemens Heathineers, Forchheim, Germany). It showed massive calcifications with multiple channels in the proximal part of the descending aorta (FIGURE 1A). A similar lesion was found in the region of the aortic bifurcation. The result of CT suggested enteroparesis as the cause of abdominal pain.

The patient died 2 days after the surgical procedure, and an autopsy revealed massive heart revascularization with ischemic cardiomyopathy of the left heart ventricle and arteriosclerosis. Additionally, a calcified thrombus was found in the proximal part of the descending aorta, which was significantly obstructed (FIGURE 1B), along with a similar lesion above the aortic bifurcation. Critical obstruction of the right femoral artery was observed. The cause of death was early visceral ischemia.

Coral reef aorta (CRA) is an uncommon disease with a prevalence between 0.6% and 1.8%.[1] Heavily calcified plaques can cause significant stenosis. Common symptoms of CRA include headache, vertigo, and visual symptoms caused by renovascular hypertension, as well as diarrhea, weight loss, and abdominal pain caused by chronic visceral ischemia.[2] The diagnosis of CRA is based on symptoms and distinctive lesions on CT. Coral reef aorta can sometimes lead to heart failure.[3,4] Surgical treatment involves thromboendarterectomy or a bypass surgery, although the operative mortality ranges between 8.7% and 11.6%.[1]

Coral reef aorta involving the proximal part of the descending aorta is rare as it is usually situated below the renal arteries. In the present case, heart surgery was performed because CRA was

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**FIGURE 1** **A** – computed tomography showing a calcified lesion in the descending aorta; **B** – a picture of the lesion on autopsy; **C** – histological sample with visible channels through which the catheter was guided (asterisks and arrows)
Abbreviations: CRA, coral reef aorta; LCCA, left circumflex coronary artery; LSA, left subclavian artery; RCCA, right circumflex coronary artery

not noticed on coronary angiography. The catheter went through the channels in the pathological structure (FIGURE 1C), and CRA was diagnosed after the surgical procedure, when CT was performed (FIGURE 1A) due to visceral complications.

## SUPPLEMENTARY MATERIAL

Supplementary material is available at www.mp.pl/kardiologiapolska.

## ARTICLE INFORMATION

**CONFLICT OF INTEREST** None declared.

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