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Pulmonary hypertension causing left main coronary artery compression

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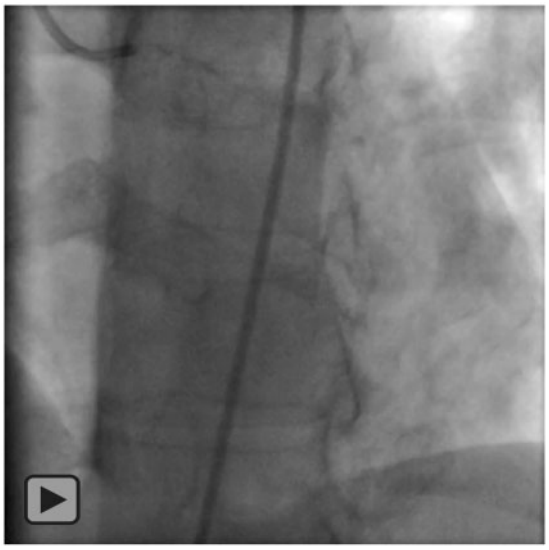
A 31-year-old woman was evaluated due to progressive dyspnoea and anginal complaints on exertion. Echocardiography revealed the presence of a large type II atrial septal defect (ASD) with left-right shunting, a dilated right ventricle with moderately reduced right ventricular systolic function, and a dilated main pulmonary artery with estimated pulmonary arterial pressure of >40 mmHg. A multislice computed tomography coronary angiography showed



Video 1 Diagnostic pre-operative coronary angiography illustrating the proximal lumen reduction of the left main coronary artery, left superior oblique view (LSO 30.3/15.8).

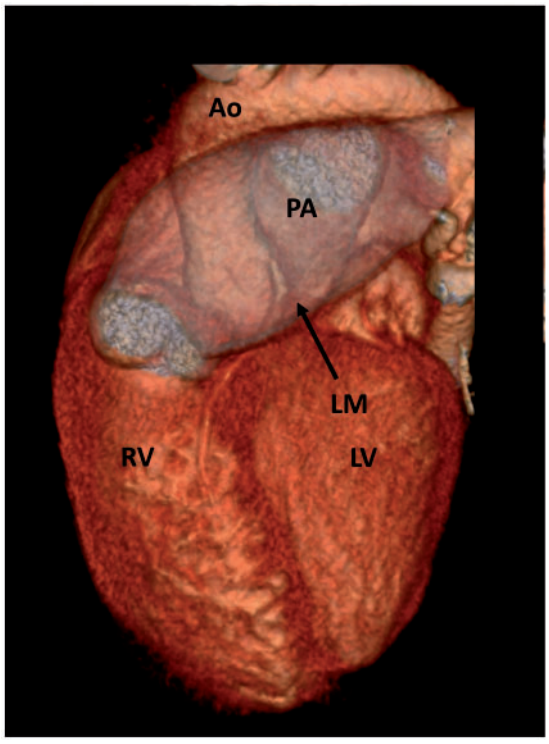


Figure 1 Multislice computed tomography coronary angiography three-dimensional reconstruction of the great vessels and the coronary arteries with an enlarged main pulmonary artery compressing the left main coronary artery. The pulmonary artery has been reconstructed as a semi-transparent vessel to illustrate its spatial relationship with the aortic root and the left main. Ao, aorta; LV, left ventricle; RV, right ventricle.

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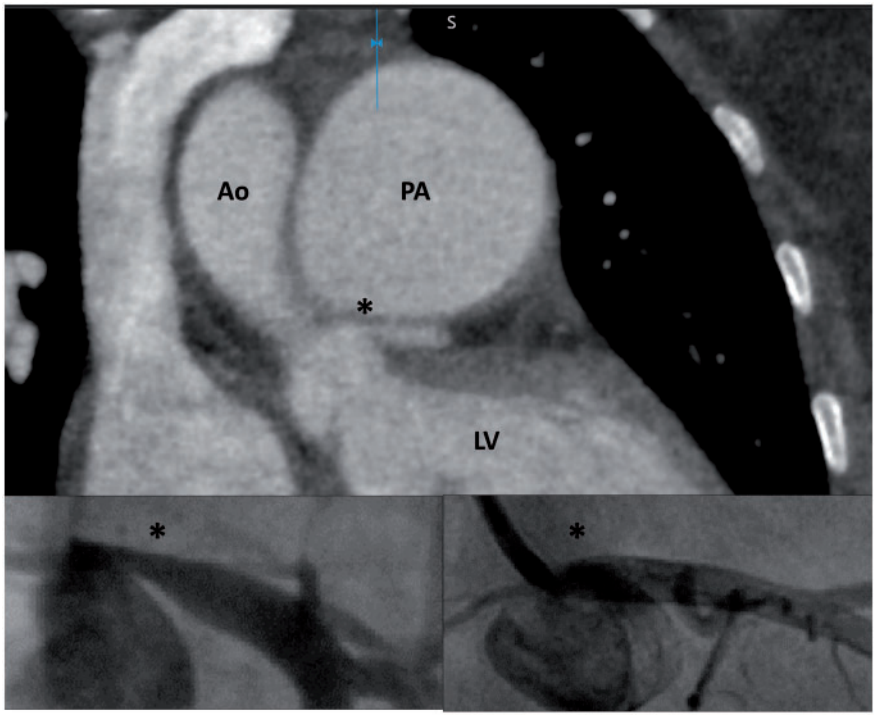
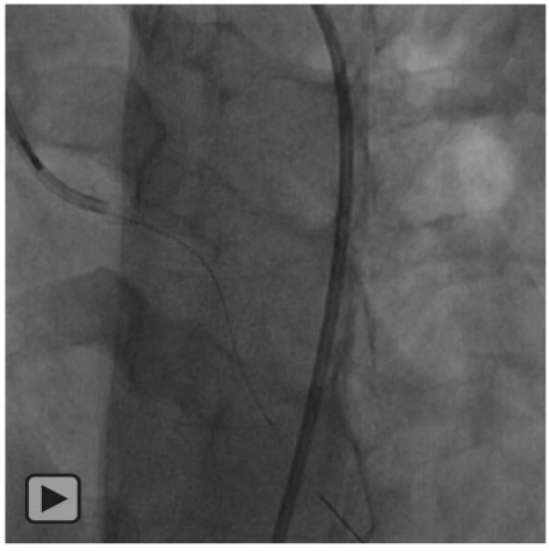


Figure 2 Two-dimensional cross-sectional image of the enlarged pulmonary artery causing extrinsic compression of the left main coronary artery. Ao, aorta; LV, left ventricle. The lower panels shown left main angiography before (left) and after (right) percutaneous coronary intervention.



Video 2 Coronary angiography after stenting of the proximal left main coronary artery showing normalization of the lumen diameter during contrast injection, left superior oblique view (LSO 37.3/20).

the main pulmonary artery to be dilated up to 45 mm and revealed its close spatial relationship with the left main coronary artery (LMCA) suggestive of external ostial compression (*Figures 1* and *2*). Coronary angiography and evaluation using intravascular ultrasound (IVUS) confirmed dynamic ostial compression of the LMCA, a potential cause of angina and ischaemia in patients with dilated pulmonary artery[1] (*Video 1*). Cardiac catheterization established the presence of pulmonary arterial hypertension [PAH, mPAP 52 mmHg pulmonary hypertension (PH) when mPAP ≥ 25 mmHg], left atrial pressure (LAP) 12 mmHg (normal range 6–12 mmHg), transpulmonary pressure gradient (TPG) 40 mmHg (considered significant when > 12 mmHg), pulmonary vascular resistance (PVR): systemic vascular resistance (SVR) of 0.5) with partial reversibility on vasoreactivity testing using inhaled nitric oxide and oxygen (mPAP 40–41 mmHg). There was no atherosclerotic coronary artery disease. The patient was treated with bosentan and sildenafil according to the 'treat and repair' strategy for ASD-associated PAH.[2] Repeated catheterization confirmed significant reduction of the pulmonary pressures and a test balloon occlusion of the ASD was attempted (reduction of TPG to 27 mmHg, PVR: SVR of 0.34). The patient met requirements to pursue a 'repair' strategy consisting of ASD closure with a fenestrated xenopericardial patch. After surgery the patient experienced anginal complaints and showed electrocardiographic signs of pan-ischaemia at heart rates > 90/min (Supplementary material online, *Figure S1*). No ischaemia detection was performed in the postoperative setting. Catheterization and evaluation with IVUS confirmed persistent dynamic ostial compression of the LMCA and a percutaneous coronary intervention (PCI) with placement of a drug-eluting stent in the LMCA was performed with good angiographic and IVUS result (*Figure 2*, *Video 2*). The patient underwent an uncomplicated

recovery and experienced no anginal complaints thereafter. Moreover, exercise testing post-PCI revealed no ischaemia.

Acute pulmonary hypertensive crisis, left and right ventricular (RV) decompensation and residual PAH and RV dysfunction are well recognized risks of ASD closure in patients with ASD-associated PAH. In patients presenting with anginal complaints in the setting of PAH, it is important to be aware of the potential extrinsic compression of the LMCA by the often dilated main pulmonary artery.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Consent : The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidelines.

Conflict of interest: none declared.

References

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