Left main artery compression by pulmonary artery aneurysm and ostial athero-stenosis of left anterior descending artery in a young female with pulmonary arterial hypertension

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A 29-year-old patient (♀) with idiopathic pulmonary arterial hypertension (PAH, WHO functional class IV) treated with sildenafil, treprostinil, and nifedipine suddenly developed recurrent chest pain at rest, accompanied by an acute inversion of T-waves in V3–V6 and a troponin I increase of up to 1.9 ng/mL. Progressive pulmonary trunk dilatation was observed in all her previous echocardiography examinations. Multi-detector computer tomography scan (128 row dual source SOMATOM Definition, Siemens, Forchheim, Germany) revealed huge (~70 mm) aneurysmal dilatation of the pulmonary trunk (PT, Panel A), but no sign of dissection. Coronary CT angiography revealed that the dilated PT displaced and compressed the entire course of the left main coronary artery (LMCA) against the wall of the left sinus of Valsalva, resulting in a critical lumen narrowing (Panel A, ‘1’). Angiography confirmed these findings (Panel B, ‘1’). Intravascular ultrasound (Atlantis SR Pro, Boston Scientific, USA, Panel B, 1–3) revealed more extensive left anterior descending (LAD) artery compression, which was evident in CT or invasive angiography and, in addition to that extrinsic coronary artery compression, atherosclerotic plaque accumulation in the ostium of the LAD (Panel B, 2, asterisk). Because the extrinsic coronary artery compression reduced the size of the LAD, the modest amount of atherosclerotic plaque contributed importantly to the lumen narrowing with a minimal lumen cross-sectional area of only 2.5 mm². Therefore, we stented the LMCA from its origin into the proximal LAD (Panel B, 3) using a Liberte™ 3.5 × 20 mm stent (Panel C, 4–6).

This is the first case report of extrinsic left main compression by aneurysmal dilatation of the pulmonary trunk in a young patient with idiopathic PAH concomitant with atherosclerotic narrowing of the LAD artery ostium. The pathogenesis of this early plaque formation in a young woman without any conventional risk factors for premature atherosclerosis might be attributed to mechanistic external forces affecting the vessel wall related to the enormously dilated pulmonary trunk. These might promote inflammatory cell deposition and disturb the laminar blood flow with the resultant accelerated atherosclerosis. We should be aware of this phenomenon and not forget the diagnostic limitations of angiography, either invasive or non-invasive, before attempting stenting a compressed left main artery.

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