

ST-Segment–Elevation Myocardial Infarction Attributable to Left Main Coronary Artery Compression

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A 44-year-old man with Eisenmenger syndrome in the setting of a subaortic ventricular septal defect under treatment with bosentan presented with angina at rest for 12 hours. He reported effort chest pain and worsened dyspnea in the previous weeks.

The ECG at admission showed transient ST elevation in aVR and V₁ coupled with ST depression in the other leads (Figure A), suggesting ischemia attributable to left main coronary artery (LMCA) obstruction, given the clinical context.

Echocardiography depicted a large subaortic ventricular septal defect with predominant right-to-left shunt and depressed left ventricular ejection fraction attributable to global hypokinesia (Movie I in the online-only Data Supplement).

Troponin I was markedly elevated (78.0 ng/mL; reference range, < 0.07 ng/mL). The patient was referred to urgent coronary angiography that revealed a smooth tapered narrowing at the ostium of the LMCA, without other coronary lesions.

Coronary computed tomographic angiography (Figure B through D) was undertaken to provide optimal detail concerning the mechanism of obstruction. It depicted a significant slit-like narrowing of the ostium and proximal LMCA shaft, with a downward angulation of 30° relative to the left coronary sinus (yellow arrows), secondary to extrinsic compression by a massively enlarged main pulmonary artery (Movie II in the online-only Data Supplement). Moreover, there was no evidence of underlying atherosclerosis.

According to the predictable high surgical risk imposed by severe pulmonary hypertension, a percutaneous coronary approach was attempted. Angiography (Figure E) and intravascular ultrasound (Figure E.I; Movie III in the online-only

Data Supplement) confirmed the LMCA extrinsic compression (green arrow). A bare metal stent 4.5×12 mm (Omega, Boston Scientific Corporation, Marlborough, MA) was deployed with good angiographic result (Figure F), and was also assessed by adequate and symmetrical stent expansion by intravascular ultrasound (Figure F.I; Movie IV in the online-only Data Supplement).

Specific pulmonary hypertension treatment intensification was provided to minimize the compression of adjacent structures by pulmonary artery trunk. At 4-month follow-up, the left ventricular ejection fraction had normalized, and the patient remained free of angina.

Extrinsic compression of the LMCA by a dilated pulmonary artery is a rare condition usually associated with long-standing pulmonary hypertension.¹ An integrated multimodality imaging approach provides incremental value in its diagnosis and management. Although debatable, percutaneous coronary intervention appears to be a feasible, safe, and effective therapeutic option, especially regarding the subgroup of patients with a high predictable surgical mortality.²

Disclosures

None.

References

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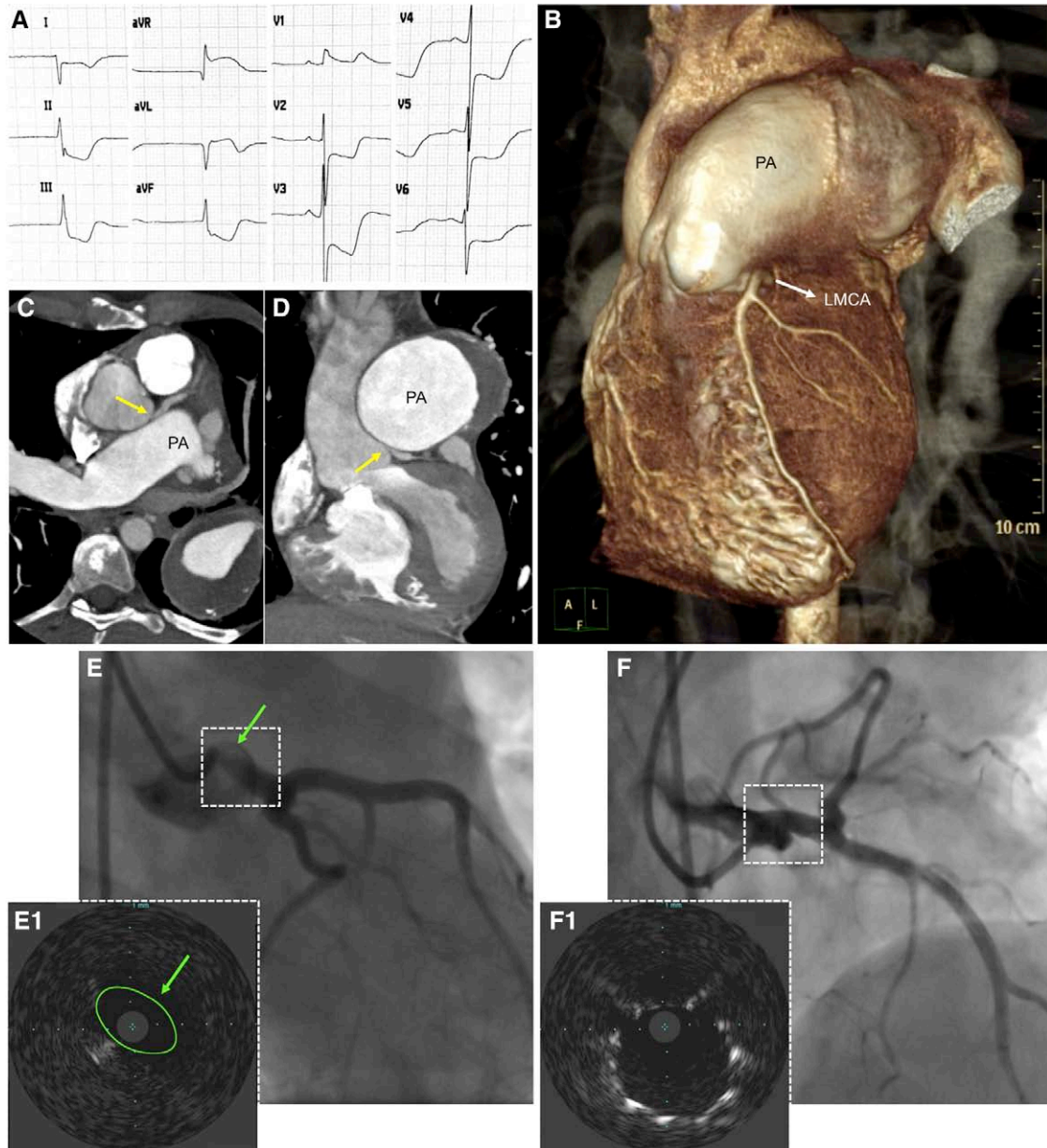


Figure. **A**, Standard 12-lead ECG showing transient ST elevation in aVR and V₁ coupled with ST depression in the other leads. **B**, Cardiac volume rendered computed tomography (CT) showing the spatial relationship between the enlarged pulmonary artery and the left main coronary artery (LMCA). **C** and **D**, Cardiac CT in axial and oblique coronal views, respectively, depicting the high-grade ostial and proximal LMCA stenosis secondary to extrinsic compression by a massively enlarged main pulmonary artery (yellow arrows). **E**, Selective left coronary angiography revealing a smooth tapered narrowing at the ostium of LMCA. **E.1**, Intravascular ultrasound (IVUS) cross-sectional image showing the proximal LMCA luminal reduction attributed to the extrinsic compression (green circle and green arrow). **F**, Final angiogram result after percutaneous coronary intervention. **F.1**, IVUS cross-sectional image demonstrating adequate and symmetrical stent expansion.