Bilateral earlobe creases and left main coronary artery disease

Andreas Yiangou Andreou

Department of Cardiology, Limassol General Hospital, Limassol, Cyprus

A 67-year-old man, cigarette smoker with a history of dyslipidaemia, presented to the emergency department with substernal chest pain and electrocardiographic (ECG) evidence of acute anterior myocardial infarction (MI) (Fig. 1). He denied any other previous medical history and took no medications. Nothing remarkable was noted on physical examination except for bilateral diagonal earlobe creases (DEC) (Fig. 2). The patient was taken emergently to the cardiac catheterisation laboratory for primary percutaneous coronary intervention (PCI). On arrival in the catheterisation laboratory he was pain free with ST-segment elevation resolution suggesting spontaneous reperfusion. Transradial coronary angiography revealed a tight left main coronary artery (LMCA) trifurcation lesion (Fig. 3A) with normal flow in the left anterior descending (LAD) artery but impaired flow in the left circumflex (LCx) and ramus medianus arteries. The right coronary artery was dominant and minimally diseased. The patient was treated successfully with drug-eluting stent-supported PCI using the classic T-stent technique (Fig. 3B). A 3.0 mm × 18 mm stent was implanted in the LCx artery and a 4.0 mm × 20 mm stent was implanted from the LMCA to the LAD artery across the ostium of the LCx artery. The optimal angiographic result achieved was verified with intravascular ultrasound examination. The patient was discharged home with dual antiplatelet therapy, and at 10-month follow-up he is asymptomatic with an echocardiographic left ventricular ejection fraction of 50%. DEC is a fold or wrinkle extending diagonally from the tragus across the lobule to the rear edge of the auricle. It has been associated with the presence, extent, and severity of coronary artery disease (CAD) independently of age and other conventional CAD risk factors also conferring an increased risk for ischaemic heart disease and MI. The aetiology of DEC and its association with CAD is still elusive; however, defect(s) in earlobe collagen that consists of peptide chains with a repeating specific triplet of amino acids and in a chemically similar macrophage scavenger receptor mediating the uptake of modified cholesterol may be a plausible explanation. Our patient demonstrated bilateral DEC in the setting of severe LMCA disease presenting as acute MI. Enhanced awareness of DEC may allow earlier detection of CAD and timely institution of primary preventive measures, thereby preventing cardiovascular morbidity and mortality.