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Angiogenesis between coronary grafts through the aortic wall

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Following a total coronary occlusion, residual perfusion to the myocardium persists through native coronary collateral channels. Provided that they are of adequate size, collateral circulation may protect against myocardial infarction and death [1]. However, there is tremendous individual variability in the function of those neovascularizations among patients with chronic stenosis. Thus, the detailed mechanism of collateral artery growth is still unknown in the human adult.

This report describes a case of a 61-year-old man that was admitted with progressive angina (CCS III on admission) for the last 3 months. He had a previous history of hypertension, dyslipidemia and insulin-dependent diabetes and two surgical myocardial revascularizations performed 14 and 8 years prior to admission. Medications taken on a daily basis included atenolol, amlodipine, trimetazidine, propatylnitrate, atorvastatin, metformin and insulin. ^{99m}Tc-Sestamibi Scintigraphy demonstrated irreversible defect of basal-inferior wall and moderate reversible defect of anterior-apical wall, along with reduced ventricular function during exercise. Coronary angiography showed occlusion of native vessels, pervious left internal mammary graft to a marginal branch and collateral circulation (CC) grade III to right coronary artery. Moreover, there was an ostial occlusion of a free right internal thoracic artery graft (RITA) to left anterior descending artery, which had a

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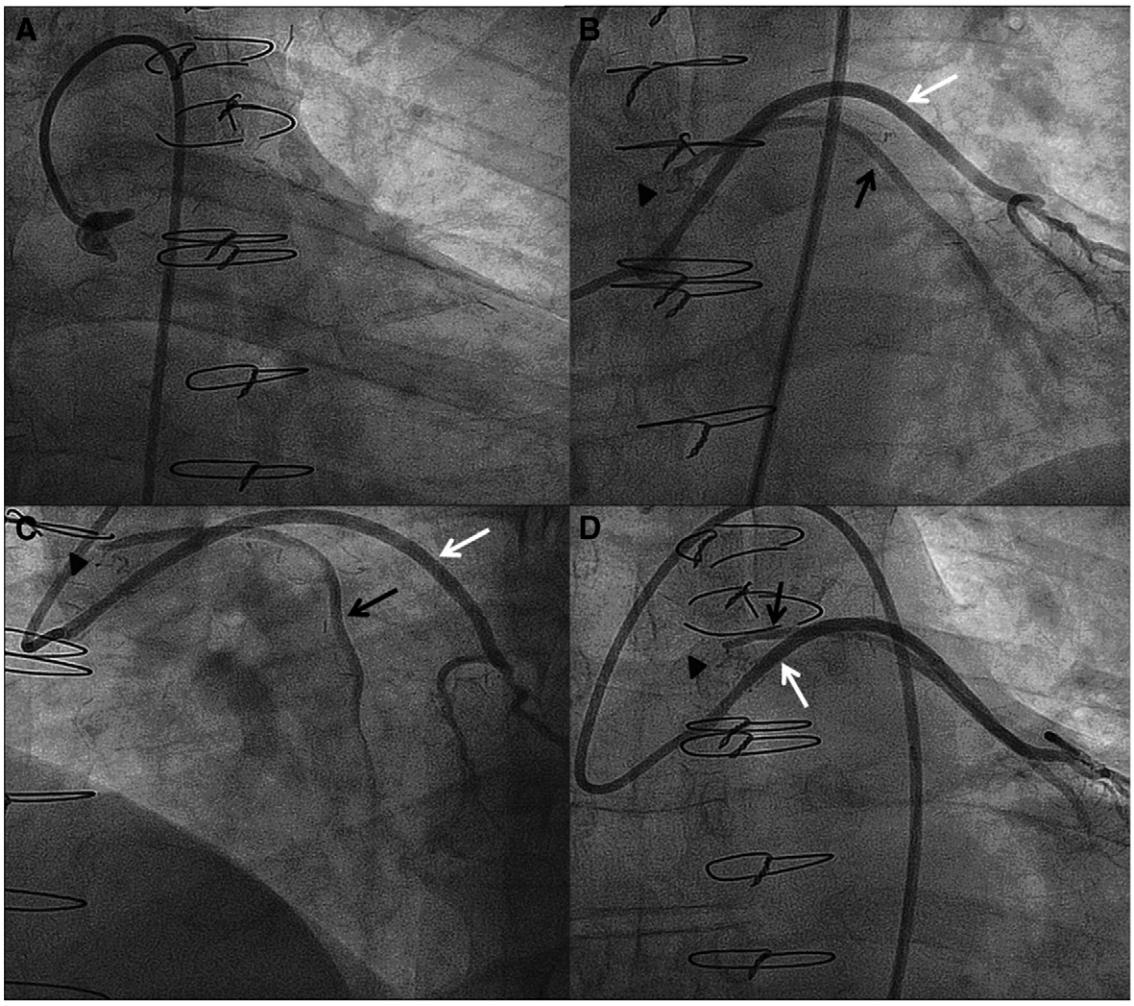

Fig. 1. Coronary angiography in caudal right anterior oblique view (A) showing ostial occlusion of the right internal thoracic artery "free" graft to the left anterior descending artery (RITA-LAD). In cranial right (B), cranial left (C) and caudal right anterior (D) oblique view, the collateral circulation (arrow head) formed by neovascularization can be seen filling the RITA-LAD (black arrow) from a saphenous vein graft to a diagonal branch (white arrow).

delayed filling through CC from a saphenous vein graft (SVG) to a diagonal branch (Fig. 1). This CC adopted a unique trajectory in the aortic wall (Fig. 1). A 320-detector row CT confirmed the angiographic findings, but it was not able to depict the CC between the grafts (Fig. 2). Angioplasty of the RITA, guided by angiography and the 320-detector row CT images, was performed using a drug-eluting stent. Control angiography of the SVG showed the disappearance of the CC between the grafts (Fig. 3). The patient was discharged two days later with normal electrocardiogram and cardiac enzymes and on the same medications taken prior to admission plus clopidogrel. At 12 months of follow-up he remained asymptomatic.

Neovascularization is the process of generating new blood vessels mediated primarily by progenitor and/or endothelial cells leading to tube formation, resulting in a stabilized neovascular channel [2]. Angiogenesis, the predominant form of neovascularization in atherosclerosis, is mediated by endothelial cells sprouting from postcapillary venules [3]. Differently from this case, a study demonstrated that diabetes is associated with decreased expression of the receptor for vascular endothelial growth factor (VEGF) and a downregulation of VEGF signal transduction, which may contribute to impaired angiogenesis [4]. Unfortunately, none of the studies that evaluated neovascularization and CC assessed them between grafts [1–5]. Another possible explanation for the origin of this CC may be the existing vasa vasorum [6].

To our knowledge this is the first report of CC connecting two grafts through the aortic wall. These conduits between both grafts seemed to be developed by angiogenesis, since both were harvested elsewhere and there were no vessels communicating those two aortic anastomosis sites. We emphasize with this case the lack of information on predictors and mechanisms of this unique type of neovascularization.

The authors of this manuscript have certified that they comply with the Principles of Ethical Publishing in the International Journal of Cardiology (Shewan and Coats 2010;144:1–2).

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