CLINICAL VIGNETTE

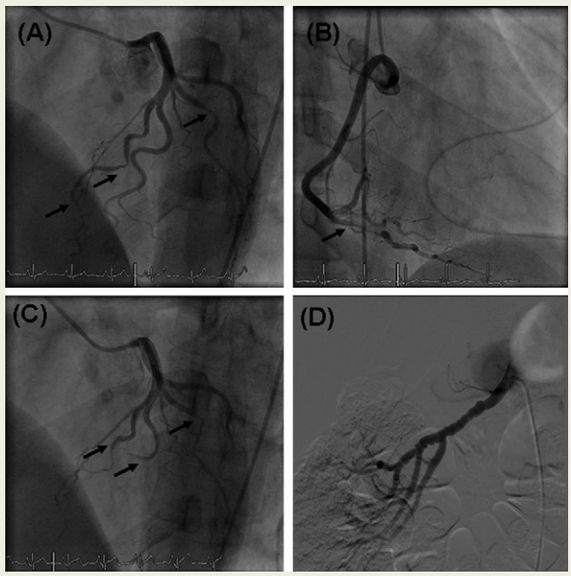
doi:10.1093/eurheartj/ehn241
Online publish-ahead-of-print 2 June 2008

Multiple postpartal coronary artery dissections and fibromuscular renal artery dysplasia

Pia K. Schuler, Thomas F. Lüscher, and Nils Kucher*

Cardiovascular Division, University Hospital Zurich, Raemistrasse 100, Zurich 8091, Switzerland

* Corresponding author. Tel: +41 44 255 8762, Fax: +41 44 255 4401, Email: nils.kucher@usz.ch



A 42-year-old postpartal woman presented 5 months after delivery of her second child with sudden onset of chest pain following exercise. The clinical investigation was unremarkable except for a blood pressure of 185/110 mmHg and a positive troponin test. Her past medical history included controlled arterial hypertension and two episodes of a troponin-positive acute coronary syndrome due to spontaneous coronary artery dissection (SCAD) that were treated conservatively. The first episode was not pregnancy associated and was caused by single SCAD of the posterior descending coronary artery. The second episode occurred 2 months after delivery of her first child and involved a single SCAD of the intermediate branch. During the second episode, a right renal artery stenosis due to fibromuscular dysplasia (FMD) was treated by percutaneous transluminal angioplasty (PTA).

At present, angiography showed multiple SCAD of the left anterior descending artery, first diagonal branch, intermediate branch (*Panel A*), and the right posterior descending artery (*Panel B*). In addition, there was restenosis of the right renal artery (*Panel D*). Despite medical therapy, including aspirin, clopidogrel, unfractionated heparin, transdermal nitrates, and calcium channel blockers, the patient developed anterior ST-elevation myocardial infarction during the next 24 h. Re-angiography revealed progression of the multiple dissections in the left coronary artery (*Panel C*). After insertion of an intra-aortic balloon pump, intravenous nitrates, ablactation, and renal artery PTA, the patient recovered and remained event-free during clinical follow-up of 12 months.

Spontaneous coronary artery dissection is often pregnancy associated and occurs particularly during the early postpartum period (78%); the LAD is involved in approximately 80% of patients. SCAD is thought to be caused by an altered endocrine status and an increased haemodynamic strain during pregnancy and the postpartum period. Whether SCAD in our patient was triggered by secondary hypertension from renal FMD remains unclear. Alternatively, SCAD may be related to FMD; indeed, carotid dissection has been reported in such patients. Urgent coronary angiography is indicated to establish the diagnosis and determine the appropriate therapeutic approach. Conservative medical therapy may be preferred in stable conditions and can lead to complete healing of the dissected coronary artery. In unstable conditions involving proximal coronary dissections with absent coronary flow, percutaneous or surgical revascularization should be considered.

Panel A. Left anterior cranial view showing multiple, rather distally located spontaneous coronary artery dissections of the left anterior descending artery (left arrow), first diagonal branch (middle arrow), and intermediate branch (right arrow).

Panel B. Right anterior oblique view showing dissection of the right posterior descending artery. A dissection membrane is clearly visible (arrow).

Panel C. Left anterior cranial view showing progression of the dissections of the left anterior descending artery (left arrow), first diagonal branch (middle arrow), and intermediate branch (right arrow) during anterior ST-elevation myocardial infarction.

Panel D. Digital subtraction angiography showing the typical bead-string changes of fibromuscular renal artery dysplasia. Haemodynamic significance of the lesion was confirmed by ultrasound.