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Structural and functional characterization of an intermediate stenosis with intracoronary ultrasound and Doppler: A case of "reverse Glagovian modeling"

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A compensatory enlargement of the coronary arterial wall as plaque increases has been described by Glagov et al.,[1] whereas a paradoxical shrinkage of the arterial wall at the site of vascular stenoses was observed by Pasterkamp et al.[2] in femoral arteries. We report a case of a "reverse Glagovian modeling" in a coronary artery detected during the comprehensive assessment of an intermediate lesion with intracoronary ultrasound and Doppler.

A 60-year-old woman complaining of chest pain at rest and during exercise for 1 year, despite treatment with a beta blocker, displayed a slowly ascending depression of the ST segment of 1.5 mm in leads V_5 and V_6 at maximum effort during a bicycle stress test. Coronary angiography revealed no luminal obstruction of the right and left circumflex coronary arteries. The left anterior descending coronary artery (LAD) had a stenosis in the proximal segment and minor luminal irregularities in the midsegment. After intracoronary injection of nitrates, quantitative coronary angiography of the proximal lesion was performed online (CAAS, PieMedical, Maastricht, The Netherlands) and revealed a minimal luminal diameter of 1.92 mm with a 42% diameter stenosis (Fig. 1). The value of this quantitative angiographic analysis, however, was in all projections limited by the superimposition of an intermediate and a septal branch. Subsequently, a 0.014-inch 15-MHz Doppler guide wire (FloWire, Cardiometrics, Mountain View, Calif.) was advanced into the LAD distal to the lesion. Coronary flow velocity was measured at baseline and after intracoronary injection of a bolus of 18 μg of adenosine. Coronary flow reserve, calculated as the ratio between hyperemic and baseline average peak velocity, was 3.3 (Fig. 1). After a new intracoronary injection of 3 mg isosorbide dinitrate, a 2.9F 30-MHz mechanical intracoronary ultrasound (ICUS) catheter (Microview, CVIS, Sunnyvale, Calif.) was advanced distal to the lesion, and a slow, continuous withdrawal of the ICUS catheter was performed with a motorized pullback device at a speed of 0.5 mm/sec. The reference site distal to the plaque showed a mild concentric intimal thickening with a 21% cross-sectional area stenosis. At the site of the angiographic luminal narrowing, a focal noncalcified highly eccentric plaque was found showing a 58% cross-sectional area stenosis and a minimal luminal diameter of 2.0 mm (Fig. 2). A custom-designed analysis system, developed at the Thoraxcenter, was used to obtain a computerized assessment of luminal and plaque dimensions of the entire vascular segment visualized with ICUS.[3, 4] Reduction of the total vessel area from the distal reference segment to the proximal LAD stenosis was observed (Fig. 2). On the basis of the information provided by ICUS imaging and Doppler, no coronary intervention was performed. The absence of inducible ischemia in the myocardial territory subtended by the LAD was confirmed by a dobutamine stress-echocardiography test, which demonstrated a normal thickening and motion of the anterior wall and the interventricular septum at a maximum level of dobutamine (40 μg/kg/min plus 2 mg intravenous atropine).

New intracoronary ultrasound techniques permit a morphologic and functional assessment of coronary stenoses, which may be particularly helpful in patients with angiographically intermediate stenoses[5, 6] when the presence of myocardial ischemia has not been unequivocally demonstrated with noninvasive tests before the cardiac catheterization. Measurement of coronary flow reserve with the

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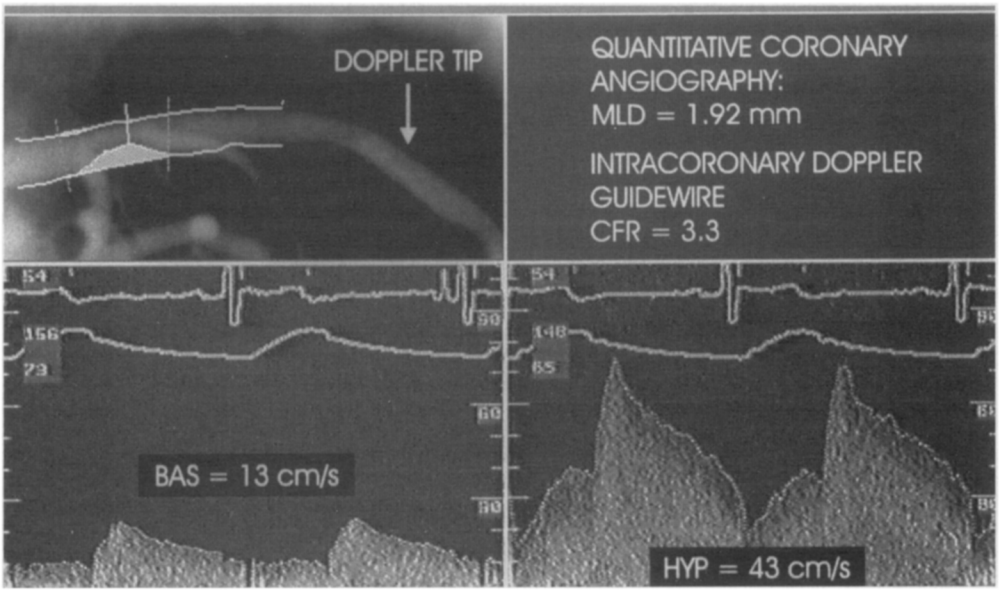

Fig. 1. Quantitative coronary angiography *(left upper panel)* of intermediate lesion in proximal segment of left anterior descending coronary artery. A 42% diameter stenosis was found but angiographic analysis was limited by sidebranches and overlapping vessels in all projections. Assessment of coronary flow velocity with Doppler guide wire distal to stenosis revealed normal coronary flow reserve of 3.3, calculated as ratio between average peak flow velocity at baseline *(left lower panel)* and hyperemia *(right lower panel). Arrowhead* indicates position of Doppler tip during flow velocity measurement. *BAS*, Baseline; *CFR*, coronary flow reserve; *HYP*, hyperemia; *MLD*, minimal luminal diameter.

Doppler guide wire is well correlated with the results of radioisotope perfusion tests and permits a reliable assessment of the hemodynamic significance of coronary stenoses. The safety of deferring coronary interventions in nonsignificant lesions on the basis of results of flow velocity measurements has recently been demonstrated.[6] In this case a normal coronary flow reserve was found in the LAD distal to the lesion, excluding a significant hemodynamic impairment. ICUS permits direct inspection and measurement of the plaque burden and is not affected by vessel overlapping or other causes of insufficient angiographic visualization. The feasibility and clinical value of ICUS for decision making in intermediate coronary lesions have recently been reported.[5] In this case the lesion fulfilled the ICUS criteria for deferral of coronary interventions (minimal luminal diameter <2 mm) proposed by Mintz et al.[5] on the basis of their clinical experience.

Enlargement of the total vessel dimension is a well known mechanism of the vascular wall to preserve the dimension of the lumen during the early stage of atherosclerosis.[1, 7] This mechanism can compensate for an increase of plaque burden until it occupies 40% of the internal elastic membrane area.[1] However, the vessel wall does not uniformly respond to progressive plaque accumulation because the mechanism depends on the integrity of the endothelium-mediated response to the increase in shear stress as luminal narrowing starts to develop. When this compensatory mechanism fails, a "reverse modeling" may occur as described in atherosclerotic human femoral arteries showing a paradoxical shrinkage of the total vessel area at the site of the target stenosis.[2] The ICUS findings of this case suggest that a reverse modeling can also occur in coronary lesions. The complementary information obtained with ICUS imaging and Doppler can be valuable in the process of clinical decision making in patients with intermediate lesions. The question whether coronary lesions with "reverse Glagovian modeling" have a specific prognosis or a different response to treatment is of interest but requires further clinical investigation.

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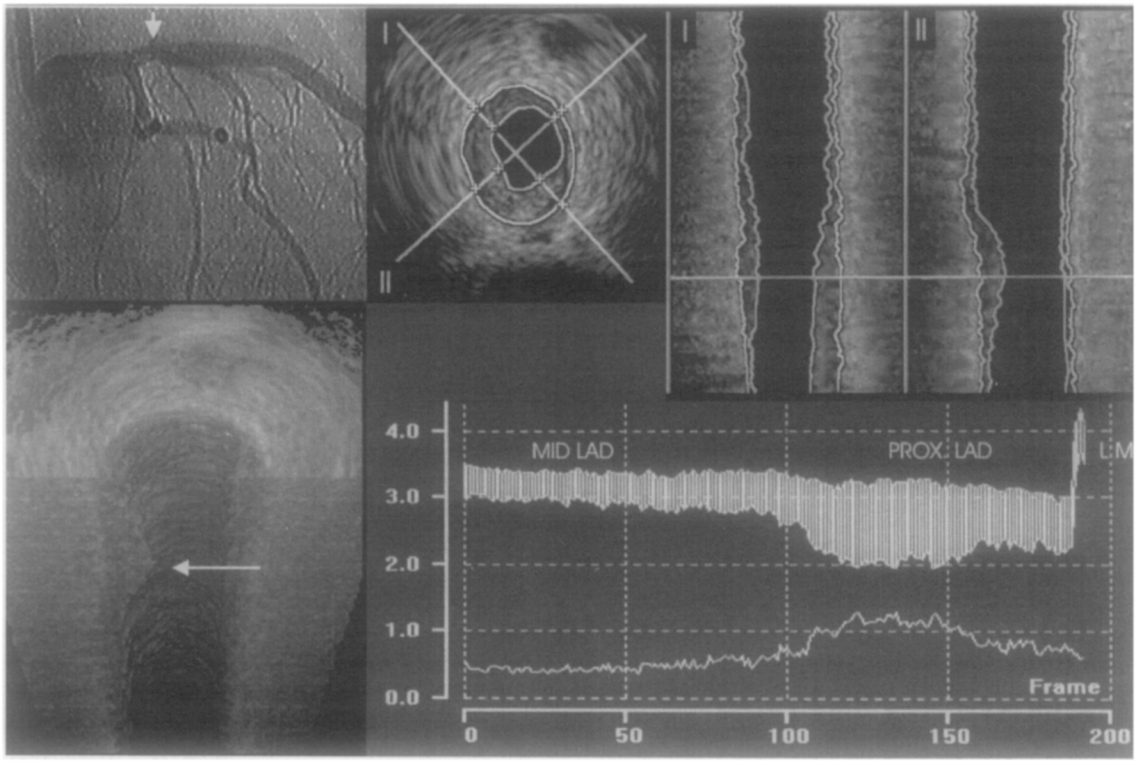

Fig. 2. Proximal stenosis of left anterior descending coronary artery (LAD) *(left upper panel)* assessed by new system for quantitative analysis of three-dimensional intracoronary ultrasound images.[3, 4] Cross-sectional ultrasound images were reconstructed in two perpendicular longitudinal sections (I and II, *right upper panels*) and computerized contour detection was performed providing a three-dimensional view *(left lower panel)* and dimensions of lumen, plaque, and total vessel, calculated from area measurements. Mean diameter measurements (mm) of 200 consecutive ultrasound frames *(right lower panel)* are displayed, with plaque shown as white area between total vessel and lumen diameter. Note reduction of total vessel diameter from distal reference (mid-LAD) to target stenosis. *Arrowheads* in three-dimensional view and angiogram indicate site of stenosis, which is also displayed in cross-sectional ultrasound view *(mid-left upper panel)*. *LM*, Left main coronary artery; *PROX*, proximal.

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Spontaneous coronary artery dissection in a young man with inferior wall myocardial infarction

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Spontaneous coronary artery dissection is a rare cause of myocardial infarction. Its precise incidence, cause, pathogenesis, and treatment have not been clearly established. We report a young man who had an inferior wall myocardial infarction caused by spontaneous dissection of right coronary artery.

A 25-year-old man was referred to our unit for coronary angiography for his postinfarct angina. Patient had an inferior wall myocardial infarction 4 weeks earlier. He had no history of angina before infarction or risk factors for coronary artery disease. He had not had any chest trauma or viral infection preceding this infarction. He had no drug addiction or family history of premature coronary disease or sudden death. Patient had received nitrates, aspirin, atenolol, and heparin for 48 hours at the time of infarction. On clinical examination, his body habitus was normal and he had a blood pressure of 110/70 mm Hg. He did not have any stigmata of hyperlipidemia or collagen vascular disease. The electrocardiogram was consistent with recent inferior wall myocardial infarction. Two-dimensional echocardiography revealed posterobasal and diaphragmatic hypokinesia and left ventricular ejection fraction of 45%.