A coronary fistula in the apical part of the left ventricle as a rare cause of stenocardia

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Short title: A coronary-ventricular fistula in the apical part of the left ventricle

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We report 63-year-old female admitted to Cardiology Department due to recurrent, exercise-related angina. Medical history revealed arterial hypertension, diabetes, hypercholesterolemia, obesity without previous data for coronary-artery disease (CAD), other coronary risk factors such as, smoking, family history of CAD also were negative. Laboratory examinations showed: total cholesterol 108 mg/dl [130 - 200], cholesterol LDL 41.6 mg/dl [45 - 130], NT-Pro BNP 157.6 pg/ml [0 - 125 pg/ml], troponin level was not elevated. There wasn’t significant abnormalities in physical examination and in ECG recorded at rest. Treadmill exercise test revealed ventricular extrasystoles induced by exercise and was interrupted because of fatigue at 4.6 METS, without ST segment abnormalities.

Single-photon emission tomography showed exercise induced, reversible, perfusion defect in the antero-lateral wall encompassing 10% of the left ventricular myocardium. Echocardiography revealed good contractility with left ventricle (LV) ejection fraction 58%, but during color Doppler examination untypical turbulent diastolic flow was observed in apical lateral segment indicating the blood leak into LV chamber, see Figure 1 panels: A and B.

Coronary angiography revealed fistulas connecting second diagonal branch with LV chamber in the apical region. (see Figure 1, panel C). Then, computer tomography angiography with contrast was performed to visualize the details of the fistulas anatomy. Group of thin vessels crossing through cardiac wall connecting coronary artery and LV chamber were observed (see Figure 1 panel D). The localization of the reversible perfusion defect area in SPECT corresponded with the segment of coronary artery, where the fistulae was observed. The pathophysiologic explanation can be coronary stealing phenomenon (reduction of blood flow through artery, distal to the fistulae). While staying on medical therapy comprising nitrates, patient presented symptoms in third CCS class. Nitrate therapy had been stopped. Other CAD
pharmacology treatment was continued with bisoprolol, ramipril and atorvastatin. Exercise tolerance improved to first CCS class after few days of follow-up.

Coronary – ventricle fistulae are observed in 0-2% angiographies [1]. The stealing phenomenon and subsequent ischemic mechanism is related to the pressure gradient between high-pressure coronary artery to a low-pressure LV during diastole. Some studies postulated connection with myocardial hypertrophy, especially apical hypertrophy [2]. Some embryology studies suggested the relationship between fistulae and Thebesian vein system development. According to pathophysiology, symptoms of coronary ischemia are revealed during nitrate therapy because of leakage increase [3]. Fistulaes drains not only to LV, other destinations are well documented, for example vena cava superior [4].

Proposed treatment in the literature include: surgical ligation of fistulae, percutaneous intervention, pharmacology (betablokers, ranolazine). As it was described previously nitrate therapy is not recommended) [3].

In our patient pharmacology intervention resulted in significant stenocardia reduction and since the satisfactory clinical effect was observed, patient was not qualified to invasive treatment. If, in the future, symptoms return, fractional flow reserve assessment will be worth to consider for ischemia diagnostic process extension [5].
References:


Figure 1

A: Apical view with color Doppler displaying narrow inflow (jet) into left ventricle (arrow).

B: Pulsed wave Doppler sampling of the jet, confirming diastolic flow into left ventricle (white arrows).

C: Coronary angiography showing contrast flow into left ventricle (red ellipse).

D: Computer tomography angiography, fistulas connecting coronary artery with left ventricle (red ellipse).