Recurrent Myocardial Infarction in a Patient with an Arteriovenous Coronary Fistula and No Coronary Artery Disease

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Int J Angiol 2016;25:131–133.

Abstract

Keywords

- myocardial infarction
- coronary fistula
- coronary spasm
- coagulation

Myocardial infarction in the absence of coronary artery disease is a rare finding. Mechanisms leading to infarction include paradoxical embolism, coronary dissection, coronary spasm, hypercoagulable states, vasculitis, or—in presence of a coronary fistula—a steal phenomenon. We report for the first time a case of a patient with an arteriovenous coronary fistula and no coronary artery disease, suffering from three incidents of myocardial infarction in three different coronary regions—of which only one was located in the area supplied by the coronary artery connected to the fistula.

Case Report

A 61-year old female patient presented with acute onset of typical chest pain, which had started 2 hours before. She had multiple cardiovascular risk factors including smoking, arterial hypertension, dyslipidemia, obesity, and diabetes. Her medication consisted of aspirin, amlodipine, and atenolol. The electrocardiogram was normal and showed no signs of myocardial ischemia. In contrast, cardiac biomarkers were significantly elevated (high sensitivity troponin T 1.36 µg/L, creatin kinase 805 U/L, and myoglobin 139 µg/L). Suspecting acute non-ST elevation myocardial infarction (MI), heparin was started and urgent coronary angiography was performed, displaying no signs of coronary artery disease (CAD). However, angiography revealed a large arteriovenous fistula between the proximal left anterior descending artery and the pulmonary artery (► **Fig. 1**; ► **Videos 1** and **2**). Left ventricular enddiastolic pressure was elevated at 17 mm Hg whereas pulmonary pressure (mean pulmonary pressure 19 mm Hg) and mixed venous saturation (SvO₂ 70%) were normal.

Video 1

Coronary angiography of the left coronary artery with a coronary fistula connected to the main pulmonary artery. Absence of coronary artery disease. LAO 50-degree/CAUD 20-degree projection. CAUD, caudal; LAO, left anterior oblique.

Online content including video sequences viewable at: www.thieme-connect.com/products/ejournals/html/10-1055-s-0034-1396342-ija-14-0099-v1

Video 2

Coronary angiography of the right coronary artery showing no signs of coronary artery disease. LAO 90-degree/0-degree projection. LAO, left anterior oblique. Online content including video sequences viewable at: www.thieme-connect.com/products/ejournals/html/10-1055-s-0034-1396342-ija-14-0099-v2

Fig. 1 Angiographic representation of the coronary fistula (arrow) in LAO 0-degree/0-degree projection. LAO, left anterior oblique.

Echocardiography revealed preserved systolic function, posterior hypokinesia, and no persistent foramen ovale with bubble contrast. A cardiac magnetic resonance imaging (CMR) study was performed and exhibited late gadolinium enhancement in the inferolateral basal and midcavity segments of the left ventricle with microvascular obstruction (**Fig. 2B**) and edema in the T2-weighted images (**Fig. 2C**)—in line with an acute infarction. In addition, two scar areas (anterior [**Fig. 2D**] and inferior segments [**Fig. 2E**]) representing earlier infarctions were depicted. Stress-induced ischemia was not apparent. CMR showed no relevant shunt volume. Seeking the cause of the recurrent MI, broad laboratory studies were performed: antinuclear antibodies, anti-neutrophil cytoplasmic antibodies, protein C, protein S, activated protein C resistance, lupus anticoagulant, factor V Leiden polymerase chain reaction (PCR), and prothrombin PCR. All results were normal, not referring to a hypercoagulable state or an autoimmune disease.

Fig. 2 CMR images showing akinesia in the inferolateral basal segment (A, white arrow) with a transmurals scar (B, white arrow) and microvascular obstruction on late gadolinium enhancement images and corresponding edema on T2-wighted images (C). (D) and (E) (white arrow) show older subendocardial infarctions on late gadolinium enhancement images. CMR, cardiac magnetic resonance imaging.

In line with the myocardial scar in the inferior region, the patient had already suffered from an ST-elevation MI 14 years earlier. Also then, coronary angiography displayed the fistula and mild inferior hypokinesia but no CAD or signs of coronary embolism. Between the two incidents the patient was asymptomatic and could not remember a symptomatic episode possibly accounting for the third area of infarction.

Suggesting that coronary spasm could account for the symptoms and findings, amlodipine 10 mg once daily (OD) and nitroglycerine as needed were initiated. The β blocker was stopped and aspirin 100 mg OD was continued.

Discussion

MI in the absence of CAD is a rare incident.¹ In general, it can be triggered by paradoxical embolism, coronary dissection, vasculitis, thrombophilia or coronary spasm.² Except for the last one, all these mechanisms could be excluded in this patient. Previously, cases of MI in patients with a coronary fistula have been published.^{3,4} However, these reports describe MI in the region of the vessel connected to the fistula—suggesting a steal phenomenon being responsible for the myocardial ischemia.⁵ In the present case, the very small shunt volume through the fistula as well as the involvement of different coronary regions make this mechanism rather implausible. In view of these findings and given the fact that the patient is female and a current smoker with a history of migraine, the most likely diagnosis seems a spontaneous coronary spasm.

Focusing on a possible connection of these two very rare findings in a single patient, it could be speculated that impaired coronary flow due to the fistula—even though not sufficient for a steal phenomenon—leads to an altered vascular physiology, thus promoting vascular spasm or local thrombus formation.⁶

We decided to treat the patient for coronary spasm and arterial thrombosis. In addition, invasive options—such as closure of the fistula by ligation or percutaneous catheter techniques (coiling, balloons, polyvinyl alcohol foam, double umbrella, and duct occluder)—were discussed.⁵ However, regarding the small shunt volume drug treatment was chosen as first line therapy.

In summary, we report for the first time a case of a patient with an arteriovenous coronary fistula and no CAD, suffering from three incidents of MI in three different coronary regions of which only one was located in the area supplied by the coronary artery connected to the fistula. Further studies will be needed to elucidate possible mechanisms linking MI, increased susceptibility to vasospasm, and the presence of a coronary fistula.

Disclosure
None.

Funding
None.

Acknowledgment
None.

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