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CLINICAL IMAGE

# Three vessel coronary artery-left ventricular multiple micro-fistulas: a rare angiographic finding

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#### **Abstract**

A 54-year-old woman presents with a long history exertional chest pain and was found to have left ventricular systolic dysfunction on trans-thoracic echocardiogram. Coronary angiography revealed no evidence of atherosclerotic coronary artery disease and showed multiple micro-fistulae draining from all three major coronary arteries to the left ventricle. This rare abnormality is the result of failure of obliteration of intra-trabecular embryonic sinusoids and may cause myocardial ischemia through the coronary steal mechanism.

A 54-year-old female presents with a long history of exertional chest pain radiating to the jaw, associated with progressive shortness of breath. Her vital signs and physical exam were unremarkable. Electrocardiogram showed non-specific ST-segment changes along with occasional premature ventricular ectopic beats. Complete blood count, electrolyte panel and Troponin I levels were within normal limits. Transthoracic echocardiogram revealed a left ventricular ejection fraction (LVEF) of 30-35% with severe global hypokinesis. Given her symptoms and depressed LV function, coronary angiography was performed and revealed no evidence of coronary artery disease but showed extensive multiple micro-fistulas (MMFs) draining from all three major coronary vessels to the cavity of the left ventricle (LV) (Fig. 1A and B). She was started on aspirin, metoprolol succinate and Lisinopril given her LV dysfunction. At 6 months follow-up, she described significant improvement in her chest pain and dyspnea.

Coronary artery fistulas (CAFs) are found in 0.3–0.8% of patients undergoing coronary angiography [1], and MMFs draining blood from all three major coronary arteries to the LV is an

exceptionally rare entity with a reported incidence of 0.001% [2]. The congenital abnormality is a result of the failure of obliteration of large intra-trabecular embryonic sinusoids. During fetal development, these endothelial lined spaces act as communications between epicardial vessels and cardiac cavities and serve in the provision of nutrients to the heart during intra-uterine life [3]. They are usually reduced to the size of capillaries with myocardial growth. Failure of their obliteration is thought to be the origin of MMFs [3]. The pathophysiology of MMFs is a consequence of reduced resistance to blood flow in the MMFs in comparison to the normal circulation. This facilitates flow from the coronary arteries to the ventricular cavities through the fistulising vessels, reducing blood supply to the myocardium distal to the MMFs. This phenomenon is known as coronary steal [2]. While surgical correction has been reported [4], the diffuse nature of the anomaly limits the utility of this modality, and conservative medical management is the treatment of choice [2]. Similarly, catheter closure techniques such as micro-coil embolization can be used to manage CAFs, but their use in MMFs is not feasible [5]. Our patient responded

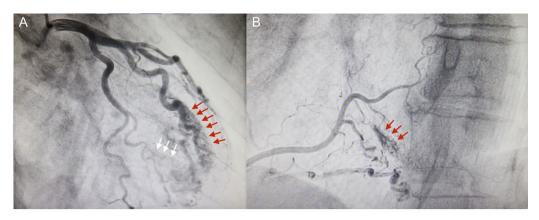


Figure 1: (A) Angiogram of the left coronary vessels showing arterio-luminal micro-fistulas connecting branches of the left anterior descending artery (red arrows) and the left circumflex artery (white arrows) to the lumen of the LV indicated by leakage of the contrast agent on injection, delineating the endocardial border of the left ventricular cavity. (B) Angiogram of the right coronary artery (RCA) showing arterio-luminal micro-fistulas connecting branches of the RCA to the left ventricular cavity (red arrows).

well to medical management as evidenced by improvement in her symptoms in her follow-up visit.

## CONFLICT OF INTEREST STATEMENT

No conflicts of interest.

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## **ETHICAL APPROVAL**

Not required.

## CONSENT

Written patient consent to publication was obtained.

# **GUARANTOR**

Hossam Abubakar and Ahmed Ahmed are both the guarantors of this article

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