

A Case Report of Perioperative Myocardial Ischemia-A Rare Etiology

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Abstract

Coronary artery fistulas are a rare finding and multiple coronary artery-fistulae arising from more than one coronary and draining into the left ventricle are exceptionally rare. We report a case of perioperative myocardial ischemia secondary to multiple coronary artery to left ventricle fistulae. It has been described that this likely occurs due to coronary steal phenomenon. Treatment either encompasses transcatheter closure or conservative management with beta-blockers and ivabradine being previously utilized.

Keywords: Coronary artery; Coronary steal phenomenon; Fistula; Fistulae; Myocardial ischemia; Perioperative

Introduction

Coronary artery fistulas are found in 0.3-0.8% of patients who undergo coronary angiography [1]. These fistulas are defined as single or multiple direct communications that arise from one or more coronary arteries and enter a cardiac chamber, great artery, or vena cava [1]. Nearly half of all patients with coronary artery fistulae remain asymptomatic and the other half experience varying levels of complications--myocardial ischemia, congestive heart failure, or infective endocarditis [2]. Multiple coronary artery-fistulae arising from more than one coronary and draining into the left ventricle are exceptionally rare. We herein report a case of perioperative myocardial ischemia secondary to multiple coronary artery to left ventricle fistulae and discuss the anesthetic implications.

Case Description

The patient is a 55-year-old male who presented for panendoscopy for pharyngeal cancer. He had a history of tobacco and alcohol abuse, glaucoma, and posttraumatic stress disorder. He denied a history of heart disease, hypertension, and diabetes mellitus. Preoperative medications included only psychotropic agents for depression and anxiety. Cardiac auscultation was

unremarkable and the patient denied a history of chest pain or shortness of breath. The preoperative ECG showed normal sinus rhythm at a rate of 80 bpm with non-specific T-wave abnormalities. Chest radiograph revealed clear lung fields with an unremarkable cardiac silhouette. The preoperative hematocrit was 35.8%.

The surgery was cancelled after failed endotracheal intubation. Mask ventilation was adequate with no significant drop in patient oxygen saturation, but intubation was unsuccessful in relation to the pharyngeal cancer. During recovery from anesthesia, the patient reported no chest pain, but developed T-wave inversions in ECG leads V2-6. He was admitted to the intensive care unit and by the following day the ECG abnormality had resolved. Serum markers for myocardial ischemia were not elevated on serial measurements. Echocardiography revealed a dilated left ventricle without significant hypertrophy and a left ventricular ejection fraction estimated at 30%. Cardiac catheterization, performed due to unanticipated T-wave inversions and newly found dilated cardiomyopathy, revealed large ectatic coronary arteries without occlusive disease. A surprising finding was the presence of multiple arterioventricular fistulae between the left anterior descending artery, left circumflex artery, and left ventricle with contrast staining of the periphery of the left ventricle (Figure 1). Subsequent persantine-thallium stress testing showed subtle, but distinct reversibility in the septal distribution, indicating myocardial ischemia.

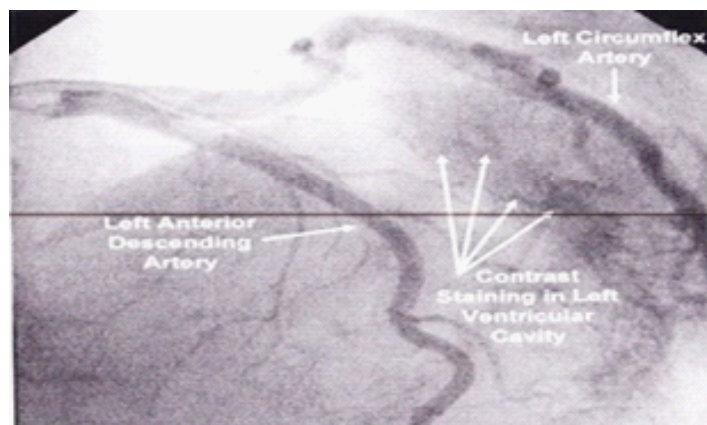


Figure 1: Coronary angiography showing multiple arterioventricular fistulae between the left anterior descending, left circumflex, and left ventricle with contrast staining of the periphery of the left ventricle.

Discussion

Fistulae between the coronary arteries and chambers of the heart are rarely considered as a possible etiology of myocardial ischemia, although their occurrence in patients undergoing coronary angiography has been previously described [3]. Of particular interest are the effects that multiple, extensive fistulae involving the left ventricle have on the heart given the fact that fistulous communication to the left-sided chambers is only about 10% of all reported coronary artery fistulae [4,5]. In general, the etiology of such fistulae is unknown. It has been suggested that these networks are, in fact, a prominent thebesian system, possibly due to the persistence of embryonic vessels or in occurrence with congenital hypoplasia of the coronary sinus [6]. It has also been described that 20% of people with congenital coronary artery fistulas have other concomitant cardiac anomalies [7].

Early reports identified an association between multiple coronary artery-left ventricular fistulae and myocardial ischemia by stress testing [8]. In a more recent study, the association with ischemia was documented by coronary sinus lactate analysis in 6/7 patients with multiple coronary artery-myocardial sinusoid fistulae [9]. It has long been discussed in the literature that the likely mechanism of ischemia is related to the coronary steal phenomenon in the absence of atherosclerotic disease or left ventricular hypertrophy causing oxygen demand-supply imbalance. In this model, an increase in flow through low resistance sinusoidal fistulae results in a decrease in pressure at the origin of the subendocardial microvasculature. At rest, autoregulation is likely able to compensate for this decrease in perfusion pressure. However, in the setting of increased myocardial demand, flow reserve is insufficient and ischemia results. Another possibility is

that coronary-ventricular fistulae are a marker for, or coexist with, more generalized microvascular disease.

In our patient, left heart catheterization revealed no occlusive disease and the concept of coronary steal seems plausible in this case. The follow up transthoracic echocardiogram did show dilated cardiomyopathy and heart failure with reduced ejection fraction. This could be idiopathic or related to alcohol abuse; however, it has been speculated that there could be a left-to-left shunt into the left ventricle resulting in diastolic volume overload and ultimately left ventricular dilation [9,10]. Clinical heart failure has been an uncommon association in multiple case reports on coronary artery fistulae, but has been described as a potential complication depending on the level of shunt fraction and coronary steal that has been experienced over time [10,11]. Treatment of singular coronary artery fistula is surgical or transcatheter closure, but this is not possible for multiple coronary artery-left ventricular fistulae as seen in our patient. Conservative management is recommended with the use of beta-blockers or ivabradine has also been described. Both of these agents will increase diastolic coronary flow and myocardial perfusion in hopes of decreasing myocardial ischemia [8,10,12].

In summary, we describe a patient with multiple coronary arterioventricular fistulae, a rare cause of myocardial ischemia in the absence of coronary artery disease. We conclude that the most likely mechanism is coronary steal phenomenon resulting in an outstripping of coronary flow reserve in the setting of increased myocardial demand likely experienced during induction of general anesthesia. Treatment is medical management with agents that will increase diastolic coronary flow with beta-blockers and ivabradine being utilized in other cases.

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Conflicts of Interest

None for any of the listed authors.

Individual Author Contributions

Shane Durkin: This is the corresponding author who performed the updated literature review and writing of the manuscript.

Eric Thorn: This author aided in initial development of the project after encountering the case and providing essential knowledge from a cardiologist perspective.

Catherine Marcucci: This author performed the initial literature review and provided overall guidance and editing of the manuscript.

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