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A case of type I dual left anterior descending coronary to pulmonary artery fistula

Case Report

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Abstract: We report a case of Type I dual left anterior descending coronary artery (LAD) giving rise to a pulmonary artery fistula via a short LAD branch, causing symptoms of typical angina. This rare coronary artery anomaly is importance to classify; it shows features of both intrinsic coronary artery anatomy anomaly and coronary drainage anomaly.

Keywords: *Dual left anterior descending coronary artery • Coronary artery fistula*

1. Introduction

The left anterior descending coronary artery (LAD) has the most constant origin, course and distribution in human heart. An anomaly of this artery is rare, even though anomalies are fairly common in right coronary artery [1]. Dual LAD is a very rare anomaly that consists of two LADs: a short LAD, which courses and terminates in anterior interventricular sulcus (AIVS) and does not reach to the apex; and a long LAD, which originates either from LAD proper or from the right coronary artery, then enters the distal AIVS and courses to the apex [2]. The incidence of this anomaly in otherwise normal hearts has been reported to range from 0.13% to about 1% [1]. Coronary artery fistulas are relatively common congenital anomalies that have the potential to alter myocardial perfusion, although more than half of the patients with a fistula are entirely asymtomatic. Small coronary fistulas are quite common, and are seen in 0.1% to 0.2% of all patients undergoing to coronary angiography [3]. The majority of small fistulas originate from the LAD and drain into the pulmanory artery [4]. The clinical course of a small asymptomatic coronary fistula is usually benign and does not necessitate intervention. In this report, we present an unusual case of dual LAD with small branch fistulizing to the pulmonary artery found in a man complaining of typical chest pain.

2. Case Report

A 55-year–old man was admitted to our hospital with a history of typical angina pectoris (CCS Class-II). He had been asymptomatic for a long time, but he presented with exercise angina of gradual onset for 6 months duration. As risk factors for coronary artery disease, a history of smoking and systemic hypertension were present. Physical examination did not reveal any abnormal findings. His arterial blood pressure and pulse rate were 140/90 mmHg and 82 beats/min, respectively. Cardiac and lung auscultations were normal. A resting electrocardiogram showed normal sinus rhythm with no evidence of ischemia. For further evaluation, he underwent treadmill exercise testing, and developed chest pain after walking 9 minutes with 0.5 mm upsloping ST depression at 80 ms past the J point through lateral leads. Since the treadmill test was interpreted as borderline, coronary angiographic evaluation was planned. The patient underwent coronary angiography and left ventriculography using standard right femoral Judkins technique. No abnormality in the left ventricular wall motion and left ventricular size was detected in ventriculography. Selective injection of contrast media into the left main coronary artery revealed a normal left main and left circumflex artery. Interestingly, we noticed that a short left anterior decending artery originated

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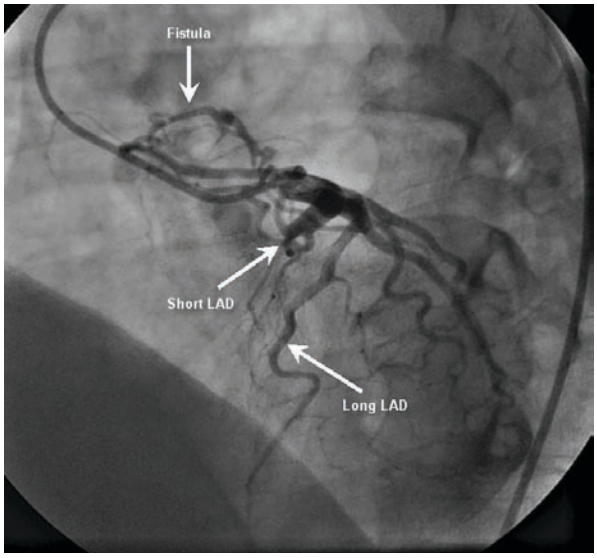
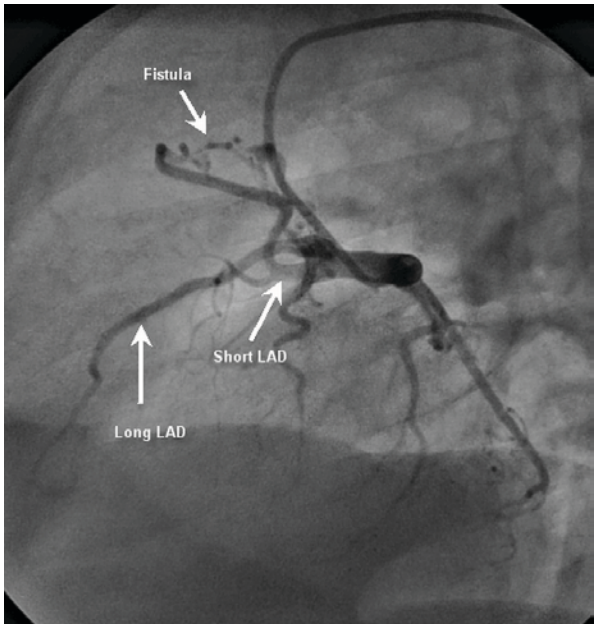
Figure 1. Left anterior oblique view with cranial angulation showing short LAD originated from LAD proper, runing in the AIVS and terminated after giving off first septal branch and more distally a fistular branch to pulmonary artery. Long LAD descended on the left ventricular side of AIVS. The area that is normally perfused by mid to distal portion of LAD was found to be avascular and free of collateral circulation.



LAD, Left anterior descending artery; AIVS, Anterior interventricular sulcus.

Figure 2. Left lateral view showing the take off of fistular branch from the stem of short LAD. Fistula terminated in pulmonary artery.



LAD, Left anterior descending artery.

from the LAD proper and, running normally, terminated after giving off first a septal branch and more distally a fistular branch, at the proximal segment of the anterior interventricular sulcus (Figures 1 and 2). The short LAD ended in the proximal segment of the AIVS and the long LAD descended along the left ventricular side of the AIVS and reentered the AIVS in its distal part. The area that is normally perfused by mid to distal portions of LAD was found to be avascular (Figure 1). Selective right coronary angiography revealed proximal luminal irregularities without any critical obstructive lesion. These coronary angiographic findings were considered to be consistent with Type 1 dual LAD with a short LAD branch fistulizing to pulmonary artery. Stress nuclear scintigraphy was then performed to evaluate the functional significance the of coronary fistula. Since the scintigraphic study revealed no evidence of ischemia, the patient was then started medical therapy without any intervention. He did well after initiation of an angiotensin-converting enzyme inhibitor, acetylsalicylic acid, and a calcium channel blocker at 6-month follow-up. At the 6-month control his functional capacity improved to CCS Class-I and no ischemic changes was observed on resting ECG. On exercise testing he completed 12 minutes without any symptoms and ST changes; stress myocardial perfusion images did not demostrate irreversible ischemia. Moreover transthoracic echocardiography revealed no regional wall motion abnormality and a normal left ventricular systolic function with ejection fraction of 60%.

3. Discussion

The widespread application of coronary angiography has revealed distinct anomalies of the coronary arteries, varying in number, course, distribution, and termination. They are generally discovered incidentally during routine coronary angiography with an incidence of 0.6% to 1.3% [5]. Angelini et al. have provided an anatomical classification of coronary artery anomalies as follows: (1) anomalies of the origin and course of the vessels; (2) anomalies of intirinsic coronary arterial anatomy; (3) anomalies of coronary drainage; and (4) anomalous collateral vessels. The descriptive value of this classification together with its clinical implications have already been reported [6]. Another proposed classification depends mainly on the functional importance of the anomaly. Coronary anomalies may be the cause of myocardial ischemia and even sudden death [7]. For this reason, it is clinically important to recognise malignant and non-malignant forms of coronary anomalies, depending on whether or not they are able to produce myocardial ischemia.

The dual LAD, in which two LADs course and supply different parts of the anterior septum, is classified as a

congenital anomaly of intinsic coronary arterial anatomy. Spindola-Franco and coworkers [2] defined dual LAD, and classified it into 4 subtypes according to the origin and course of the long LAD, as follows:

Type I: The long LAD descends on the left ventricular side of the AIVS and enters the AIVS distally.

Type II: The long LAD descends on right ventricular side of the AIVS and enters the AIVS distally.

Type III: The long LAD courses deep within the interventricular septum proximally and appears on the epicardial surface in the distal part of the AIVS.

Type IV: The long LAD originates from the right coronary artery, traverses the right ventricular outflow tract, and enters the AIVS.

Coronary artery fistulae are defined as abnormal communications between a coronary artery and a cardiac chamber or major vessel, such as the vena cava, right or left ventricle, pulmonary vein, or pulmonary artery, and are classified as drainage anomalies of coronary arteries [8,9]. The clinical presentation associated with coronary artery fistulae is dependent upon the type of fistula, shunt volume, site of the shunt, and presence of other cardiac conditions, although patients often remain asymptomatic. Common clinical presentations include fatigue, dyspnea, orthopnea, angina, endocarditis, arrhythmias, stroke, myocardial ischemia, or myocardial infarction. Coronary fistulae can cause ischemia by two possible mechanisms: first, there can be a diversion of nutrient blood flow to the fistulous tract from the normal coronary branches; and second, there can be stenosis of nutrient side branches secondary to thrombus associated with fistulous tracts, ulcerations, and atherosclerosis [8]. The main indications for closure are clinical symptoms, most particularly heart failure and myocardial ischemia, and in asymptomatic patients with high-flow shunting, to prevent occurrence of symptoms or complications, especially in the pediatric population [9]. Currently, percutaneous treatment is proposed as the first choice because it is less radical and entails a shorter period of hospitalization; surgery is reserved for cases of multiple fistulae, those affecting large branches during embolization of coils, or when the fistulous connection is narrow, restrictive and draining into a cardiac chamber [10,11].

In our patient, we recognized a complex coronary artery anomaly of a dual LAD, with a short LAD giving rise to a fistula draining into pulmonary artery, and therefore showing features consistent with both classes of coronary artery anomaly: an intrinsic coronary artery anatomy anomaly and a coronary drainage anomaly. On extensive review of the medical literature, we were not able to find any reported case showing similar anatomical features. Although this complex anatomical variant poses a sort of classification ambiguity, it is of no clinical importance because no evidence of ischemia was detected on myocardial perfusion scintigraphy; that finally led us to think of it as a clinically non-malignant anomaly. We believe that the patient would probably not suffer from myocardial ischemia because the fistula is too small to result in significant shunting and, fortunately, originates from the short LAD, which has relatively less contribution to global myocardial perfusion and is compansated by a disease-free, normal functioning, long LAD. Finally, for this patient, we proceeded using medical therapy with close outpatient follow-up.

In conclusion, we report here an unusual type of coronary artery anomaly, a dual left anterior descending artery giving rise to short LAD to pulmonary artery fistula, presenting clinically by angina.

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