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Case Report

CASE REPORT

One down, one to go: coronary anomaly dual LAD blood supply with worsening chronic stable angina and 100% occluded left short LAD type IV variant

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Abstract

Although rare, usually asymptomatic, and without concurrent disease, dual left anterior descending arteries (LAD) poses great challenges. We describe a 55-year-old male with no history of coronary disease, who presented with worsening substernal chest pain with exertion, and was ruled out for myocardial infarction. On left heart catheterization and subsequent computed tomography angiogram, he was determined to have a dual LAD with a long LAD emerging from the right coronary artery. Moreover, this long LAD gave collaterals to a native long diagonal that ran parallel to this vessel from the left system and was chronically occluded. The long LAD, consistent with type IV classification, traveled in the anterior intraventricular groove to supply left ventricular myocardium; the chronically occluded long left native diagonal supplying lateral walls and apex is a unique variant. It is important to be aware of these anomalies to establish correct diagnoses and determine treatment options.

INTRODUCTION

Anomalies in coronary artery anatomy can be incidentally discovered during cardiac catheterization performed for other indications. Alternative high-resolution modalities, such as computed tomography (CT) angiogram or magnetic resonance angiography (sensitivity 88%, specificity 100%), can further help to delineate the course of the vessels. A rare variation described herein is an anomalous left anterior descending artery (LAD); this vessel emerges from the right coronary artery but courses between the thoracic aorta and pulmonary outflow tract while finally transcending into the anterior intraventricular sulcus (AIVS). Spindola-Franco *et al* originally described dual LAD physiology by defining it as an early bifurcation of the LAD into two vessels or a second LAD that emerges from the right coronary artery. One vessel remains in the AIVS and does not extend to the apex and the other continues distally in the AIVS towards the apex. Although this variation is not immediately pathologic, it complicates management decisions for revascularization when disease is found.

CASE REPORT

A 55-year-old male with a past medical history of chronic obstructive pulmonary disease, hypertension, hyperlipidemia, and nicotine dependence presented with worsening substernal chest pain with exertion. Initial work up showed a non-contributory electrocardiogram and troponin levels within normal limits. Patient already

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had a recent stress test myocardial perfusion imaging scan performed at outside hospital that was negative for ischemia or scar or high-risk features. The patient's persistent atypical pain and risk factors for coronary artery disease (CAD) soon resulted in cardiac catheterization for possible acute coronary syndrome. Left heart catheterization via the right radial artery showed a large left main coronary artery that bifurcates into left circumflex artery with minimal disease and short LAD that gave a small diagonal artery which was occluded proximally (Fig. 1). Upon engaging the right sinus of valsalva, an anomalous course was discovered. The right coronary artery was small and nondominant. The long LAD emerging from the right coronary artery gave collaterals to the previously mentioned long diagonal system on the left (Fig. 2). Ventriculogram showed normal ejection fraction with normal intracardiac and transvalvular pressures.

His unique anatomy was observed in greater detail during a CT angiogram the following day which helped to further establish the course and to ensure the LAD did not course between aorta and

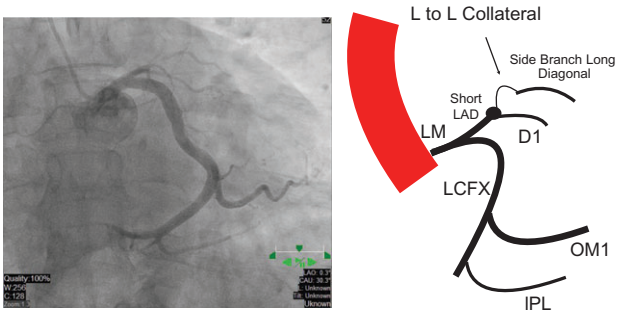

Figure 1: Catheter engagement of the left sinus of valsalva demonstrating angiographically significant vessels left main, large left circumflex with medium size OM and lPL branches. Native LAD is occluded at proximal segment 100% and gives one diagonal prior to occlusion. There is left to left collateralization with microcirculation that is able to fill what was later found to be a side branch of the native long diagonal that courses parallel to the anomalous LAD originating from the right sinus of valsalva.

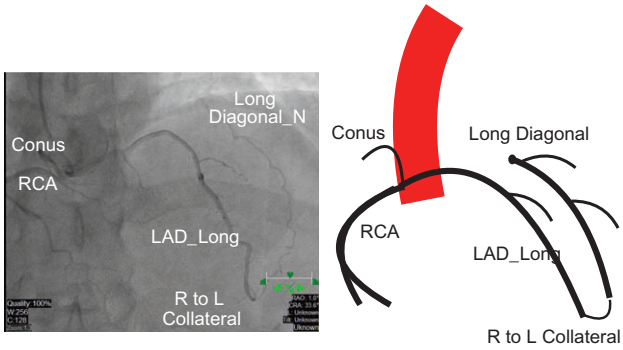

Figure 2: Catheter engagement of the right sinus of valsalva demonstrating angiographically significant vessels RCA, conus branch, anomalous LAD that comes from the right sinus of valsalva and then courses into the left anterior interventricular groove. This branch gives right to Left collateralization at the apex filling the native long diagonal that courses in a parallel distriubtion alongside the anomalous LAD

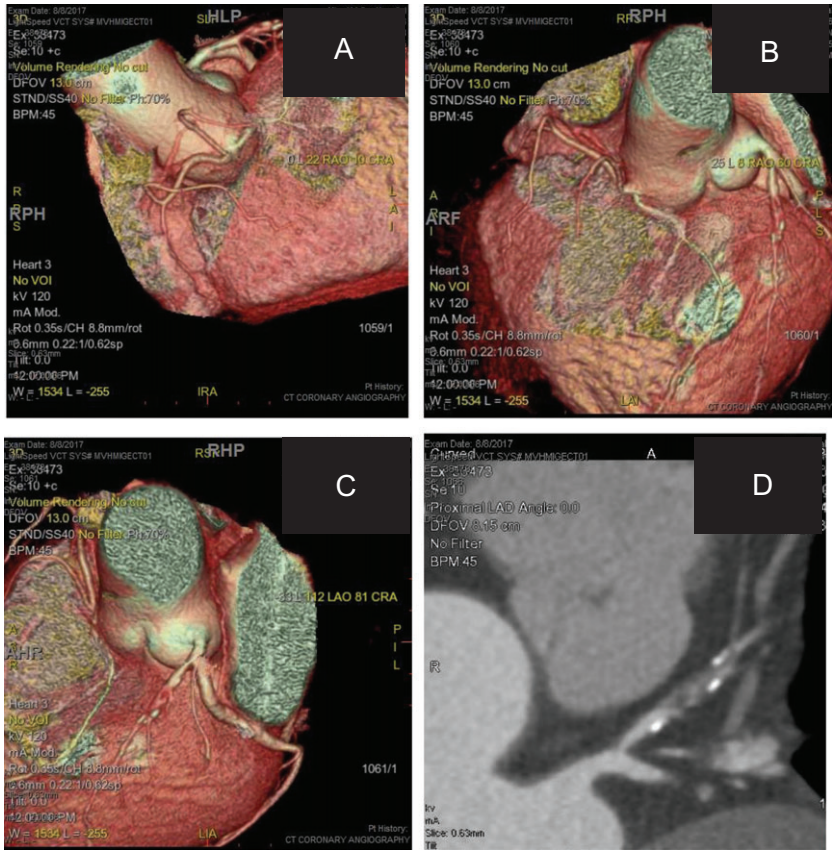

Figure 3: (**A**) Right sinus of valslava with RCA and conus branches. (**B**, **C**) Anomalous LAD visualized coursing in anterior interventricular space and native LAD occlusion proximally followed by a continuation parallel to the anomalous LAD in a diagonal distribution that reaches the apex (**D**). Proximal occlusion of native LAD with calcification.

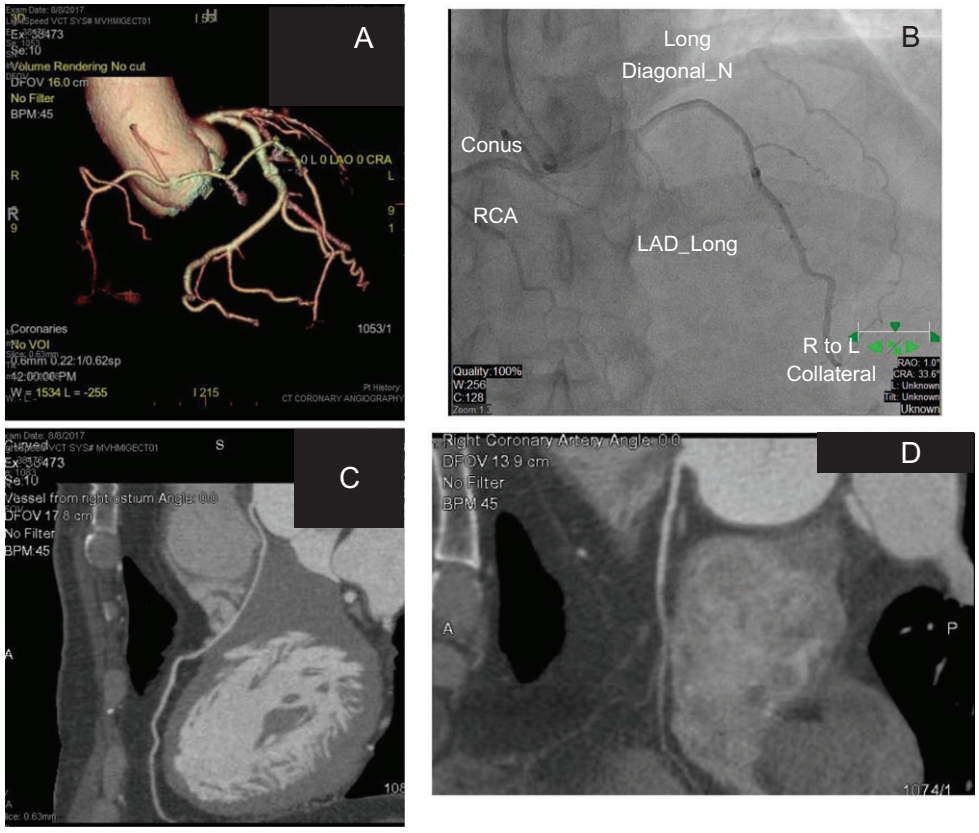

Figure 4: (**A**) CT angio view of the left and right systems superimposed not capturing the long diagonal that was captured on left heart cath angiography (**B**). (**C**) CT angiography course of the anomalous LAD originating from the right sinus of valsalva. (**D**) CT angiography course of the anomalous long LAD originating from the right sinus of valsalva that courses adjacent to the RCA origin between the thoracic aorta and the pulmonary outflow tract into the anterior intraventricular groove, a typical course for the LAD.

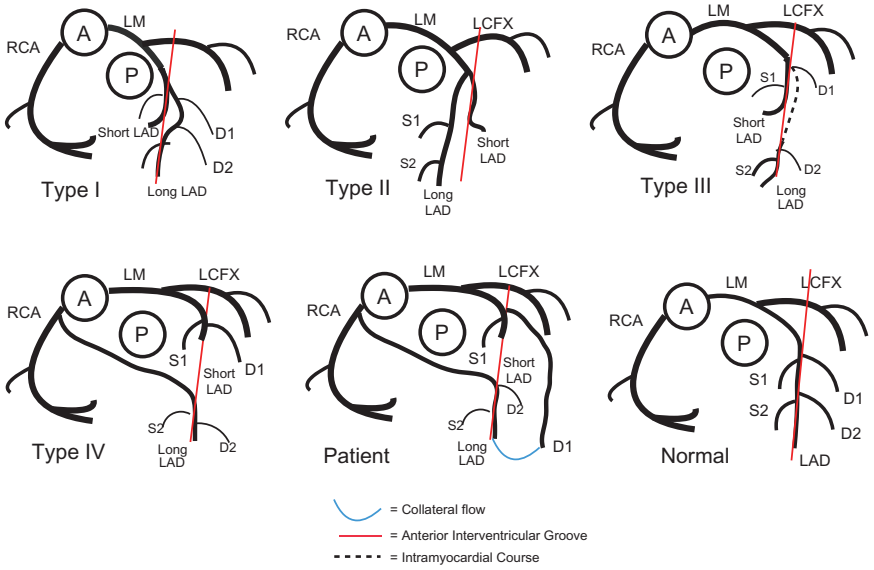

Figure 5: Normal type—Normal line diagram demonstrating normal coronary anatomy distribution. Normal LAD originates from the left main bifurcation and travels in the anterior interventricular groove giving rise to septal and diagonal branches. Type I—Short and long LAD originate from common LAD trunk. Short LAD courses along proximal anterior interventricular groove. Long LAD after proximal course runs along left side of the interventricular territory into the distal interventricular groove. Type II—Short and long LAD originate from common LAD trunk. Short LAD courses along proximal anterior interventricular groove. Long LAD after proximal course runs along right side of the interventricular territory into the distal interventricular groove. Type III—Short and long LAD originate from common LAD trunk. Short LAD courses along proximal anterior interventricular groove. Long LAD after proximal course gives a intra-myocardial course and then finishes in the distal interventricular groove. Type IV—Short LAD originates as branch of LMCA and Long LAD originates from the right side and is labeled in accordance to how it courses with respect to the great vessels. Type IV is prepulmonic, or in front of the pulmonary artery.

pulmonary arteries. It was confirmed that the right coronary artery traveled in the anterior intraventricular groove to supply left ventricular myocardium, consistent with a type IV dual LAD per Spindola-Franco classification (Figs 3 and 4). Moreover, the normal MPI was reassuring that there was not compression involved; since that was the case, intravascular ultrasound was not performed in order to further assess this point. Cardiovascular surgery was not pursued since anomalous long LAD supply of anterior and septal regions was adequate without disease. The chronically occluded native long diagonal had adequate collateralization from the right long anomalous LAD at the apex. This was effectively single vessel disease with chronic total occlusion and negative low risk stress test on intermediate medical therapy. By appropriate use criteria for revascularization, it was determined the best option would be for further optimal medical therapy. The patient was discharged on optimal medical management including multiple antianginals: isosorbide mononitrate 30 mg, ranolazine 500 mg po bid, atenolol 25 mg po daily. High-risk percutaneous intervention of the ostial D1 would be pursued if symptoms could not be properly controlled.

DISCUSSION

The embryologic development of the coronary blood supply is complex with primitive sinusoids that evolve into a network of arteries as endothelial buds arise from the early truncus arteriosus. A lot can go awry in this complex process resulting in a range of variations from subclinical to sudden cardiac death. Prevalence of anomalies has been described from 0.64 to 1.3% [1, 2], with 81% considered benign according to a 28-year study at the Cleveland Clinic [3]. A dual LAD is significantly less frequent with prevalence ranging from 0.01 to 0.03% [1]. Spindola-Franco *et al* classified dual LAD into 4 original different types though up to 10 have since been described (Source 9): (see Fig. 5). This unique variant with long diagonal reaching the apex on the left system has not been described to the best of our knowledge.

Normal type—LAD originates from left coronary artery and travels in the anterior interventricular groove giving rise to septal and diagonal branches until it reaches the apex.

Type I—Most common, short and long LAD both from LAD proper, the shorter travels in AIVS terminating before apex while the longer travels on the anterior surface of the left ventricle with the distal third entering the AIVS continuing to the apex.

Type II—Short and long LAD as in type I, but the long LAD traveled on the anterior wall of the right ventricle; anterior surface of the left ventricle was supplied by branches of the diagonal vessel.

Type III—Short and long LAD as in type I, short LAD gave off branches supplying the anterior interventricular septum while long LAD had intra-myocardial course again ending in the distal interventricular groove

Type IV—Long LAD originates from right coronary artery, descends on AIVS giving off septal and diagonal branches, the short LAD gave rise to septal and diagonal branches [4].

Although the case we describe here is a type IV classification, there are two notable distinctions that make this case even more unique. Firstly, the RCA was dominant in all four cases described by Spindola-Franco *et al*. which contrasts with the dominant LCA we describe here. Secondly, the long branch seen on the left of this patient did not ever course in the interventricular septum but rather parallel to it, making it more specifically a diagonal branch (Figs 2 and 3).

As illustrated in this case, dual LAD anomalies complicate intervention in patients with diseased vessels. It can be difficult to determine anomalous courses if the vessel runs through the myocardium or inter-arterially [3]. A short LAD (i.e. Types I–III) should not be interpreted as a completely occluded vessel. To help differentiate the two one should look for an avascular area near the left coronary artery without collaterals [4]. Although not demonstrated in this case, type IV dual LAD that travels between the aorta and pulmonary trunk has been implicated in sudden cardiac death associated with physical activity in young patients [5].

It is theoretically impossible for any two patients to have the same coronary artery anatomy given small septal branches or distribution of vessels [3]. However, there can be large variation in the typical anatomy that one would expect. Therefore, it is imperative for cardiologists to be fully aware of the wide spectrum of possibilities to correctly evaluate and intervene when necessary. Intervention may be based on appropriate use criteria for revascularization and clinical judgment utilizing knowledge of distribution of epicardial blood supply. Moreover, surgeons must be aware of the anatomy to prevent ligation of anomalous vessels or inappropriate arteriotomy placement. Although it is possible to discover anomalies on coronary angiography it may be difficult to know the origin of some vessels without a 3D image [4].

In conclusion, we report a rare coronary artery anomaly of a dual LAD. This and other anomalies present challenges to cardiologists and surgeon in particular defining the distribution of myocardium supplied by these arteries and translating this information into clinically directed medical therapy.

CONSENT

Written consent was obtained from the patient for publication of this article.

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