# Myocardial Infarction in a Patient with a Rare Coronary Anomaly

# Arindam Basu<sup>1</sup>, Angshumitra Bandyopadhyay<sup>2</sup>, Santanu De<sup>3</sup>, Kajal Ganguly<sup>4</sup>

#### **Abstract**

Congenital anomalies of the coronary arteries include anomalies in the origin, distribution and termination of the coronary arteries. Common anomalies included under this heading includes abnormal origin of a coronary artery from a different sinus, or from another coronary artery, passage in between two great arteries, or drainage into a cardiac chamber. Dual origin of a coronary artery constitutes an extremely rare form of such congenital coronary anomalies. Although such anomalies are common with the right coronary artery (RCA), those with the left anterior descending coronary artery (LAD) are rarely reported.

## Introduction

ongenital anomalies of the coronary arteries have an incidence of < 1.3%. Of them, anomalies involving the RCA are although encountered often, those involving the left system, particularly the LAD are rarely, if ever, reported. Different researchers have put the incidence of Dual origin of LAD at different figures, mostly < 1%.<sup>1,2</sup> Also, this rare anomaly has been subclassified based on their angiographic characteristics. Reports of acute coronary syndrome in these patients are very rarely reported.

### **Case Report**

A 75 year old male, non-diabetic and non-hypertensive, presented to the Cardiology emergency at 2 AM with the complaints of acute onset of chest pain of 2 hours duration. Initial ECG done in the emergency revealed Inferior wall ST elevation myocardial infarction. The resident on duty admitted the patient to the ICCU. His vitals revealed bradycardia with a pulse rate of 52/ min, a blood pressure of 92/60 mm Hg, and a SpO2 of 87%. A central venous access was secured with the possibility of development of advanced AV block in this patient and need for temporary pacemaker insertion. Next, he was thrombolysed with Injection Streptokinase. Concurrently he was given loading doses of aspirin, clopidogrel and atorvastatin and was also put on supplemental oxygen delivery @3L/min to maintain a SpO2 of > 90%.

Post thrombolysis, his vitals improved with a pulse rate of 62/min and a blood pressure of 110/70 mm Hg. There was significant residual angina but no dyspnoea, and the ECG showed a < 50% ST segment resolution. 2D- Echo showed hypokinesia of the infero-posterior wall with borderline depressed LV systolic function, with an LVEF of 50%. Blood biochemistry revealed an Hb level of 11.2gm/L and a creatinine level of 1.3 mg/dl, with an eGFR of 41.67ml/min. Patient was taken up for coronary angiography the next day.

Coronary angiography revealed the LMCA originating from the left sinus and branching into a small left anterior descending branch and a relatively larger circumflex branch. The LAD coursed normally till about the middle of the anterior inter-ventricular groove, giving off three diagonals and a large septal branch, and then the LAD suddenly seemed to disappear. The right coronary artery showed a tight lesion in the proximal part and an intermediate lesion in the mid part. The surprising part was that an aberrant branch of the RCA originating from the proximal part coursed back towards the anterior inter-ventricular groove and followed the course of a normal LAD from the middle of the groove till the apex and wrapped around towards

the inferior surface of the LAD. This aberrant vessel was of a similar calibre as that of the native LAD, was longer and gave rise to a few septal branches.

The RCA proximal lesion was identified as the culprit lesion and this was dilated and a DES of size 3.5mm x 32mm placed and inflated at 14 atmospheric pressure. The procedure was uneventful.

The patient was initially observed in the ICCU for a day more and later, shifted to the general ward. He was released on the 5<sup>th</sup> post operative day and was found to be doing reasonably well on his first follow up visit 1 week later. Considering the aberrant distribution of his coronaries he was asked to undergo a CT coronary angiography, to rule out any further obstruction to the course of LAD. The CT coronary angiography confirmed the diagnosis of Dual LAD (Figure 1).

# Discussion

Congenital coronary anomalies are rare and reported to occur in  $0.64 - 1.3\%^3$  of patients undergoing angiography. They may or may not be associated with underlying coronary artery disease. Particularly, anomalies concerning LAD (its origin, course and distribution) are rare, although such anomalies are common with RCA.

Dual LAD (also known as dual anterior interventricular artery) had been reported to occur with an incidence of 1% by Morettin as well as Spinaldo-Franco et al.<sup>1,2</sup> The term Dual LAD is an unusual coronary artery anomaly proposed by Spindola in 1983, who also classified these into four angiographic types <sup>2</sup> (recently, the number of types of Dual LAD has been increased to six) (Table 1).<sup>4</sup>

Type I: Running in the anterior interventricular sulcus (AIVS), the

<sup>1</sup>Interventional Cardiologist, Nightingale Hospital, Kolkata, West Bengal; <sup>2</sup>RMO-cum-Clinical Tutor, RG Kar Medical College and Hospital, Kolkata, West Bengal; <sup>3</sup>RMO-cum-Clinical Tutor, <sup>4</sup>Professor and HOD, Nil Ratan Sircar Medical College and Hospital, Kolkata, West Bengal

Received: 12.07.2017; Accepted: 29.10.2018

Table 1: Spinaldo-franco classification of dual LAD

Type	S-LAD			L-LAD	Origin of major
	Origin	Course	Origin	Course	Diagonal vessels
I	Proximal LAD	Proximal AIVG	Proximal LAD	Epicardial course on the left ventricular side of the proximal AIVG, reentering the distal AIVG	Proximal LAD and/or L-IAD
II	Proximal LAD	Proximal ATVG	Proximal LAD	Epicardial course on the right ventricular side of the proximal AIVG, reentering the distal ATVG	Proximal LAD
III	Proximal LAD	Proximal AIVG	Proximal LAD	Intramyocardial course in the proximal septum, then either emerging epicardially in the distal AIVG, or terminating intramyocardially as septal perforator arteries	Proximal LAD or S-IAD
IV	LMCA	Proximal AIVG	RCA	Epicardial free wall course anterior to the infundibulum of the right ventricle traversing to the distal AIVG, or intramyocardial course within the septal crest emerging epicardially in the distal AIVG	S-LAD
V	LCS	Proximal AIVG	RCS	Intramyocardial course within the septal crest emerging epicardially in the distal AIVG	S-LAD
VI	LMCA	Proximal AIVG	RCA	Underneath the RVOT in the area of the interventricular septum	S-LAD

short LAD is generally the source of all major septal perforators. The long LAD also runs in the AIVS, descending on the left ventricular side of the AIVS, and then re-entering the AIVS in order to reach the apex.

Type II: The short LAD is the same as in Type I. The long LAD descends on the right ventricular side before reentering the AIVS.

Type III: The short LAD is consistent with that described in Types I and II. The long LAD travels intra-myocardially in the ventricular septum.

Type IV: High in the AIVS, a very short vessel is formed by the LAD proper and the short LAD. From this vessel, the major septal perforators, as well as the diagonal branches, originate. The long LAD is unusual in its origin, arising from the RCA.

The essential criteria to diagnose this anomaly are –

- Two LADs should be identified.
- One would be large and another small.
- Both should give a diagonal branch.

Apart from the surprise element, the second LAD has little impact on the interventional protocol. It may, however, confer an ischemic protection as the critical anterior wall has twin blood supply. Second LAD may act as an additional collateral channel. In our case, the LAD was identified as a Type IV Dual LAD as per Spinaldo's classification scheme.



Fig. 1: 3D reconstructed image of the CT coronary angiography of this patient

#### Conclusion

Dual LAD is an extremely rare variety of coronary artery anomaly; however, it has very little clinical significance in the absence of stenosis. When affected by obstructive disease, it can be subjected to revascularization by surgery. Familiarity with the types can help the surgeon avoid an incorrectly placed arteriotomy.<sup>5</sup> Even though it confers an additional ischemic protection to the anterior wall, patients may still be exposed to the danger of an ischemic event affecting the other territories of the heart, as occurred in this case, or rarely, even the anterior wall may be affected by a myocardial infarction.

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