



# Dual LAD Anatomy, Classification and Clinical Significance: A Review

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## ABSTRACT

Dual Left Anterior Descending (LAD) anatomy is a rare coronary artery anomaly characterized by presence of two LADs in the anterior inter ventricular groove. Since its first description almost three decades ago, multiple variants of the entity have been reported. This article aims to provide a comprehensive review of the dual LAD variants, their classification and clinical significance.

**Keywords:** Coronary artery anomalies; Dual LAD anatomy; Long LAD; Short LAD

## INTRODUCTION

The prevalence of Coronary Artery Anomalies (CAA) varies depending on the definition and imaging modality used. According to the literature, CAA is identified in 0.2%-1.3% of patients undergoing coronary angiography and in 0.3% of autopsy series [1,2]. Classification of CAA anomalies based on anatomical considerations into (a) anomalies of origin and course (b) intrinsic CA anomalies (c) anomalies of termination is widely accepted. A modified embryological-anatomical classification has also been proposed [3].

Dual LAD is a rare congenital coronary anomaly, with its first description almost three decades ago. With increasing number of diagnostic and therapeutic coronary procedures, a higher prevalence of the anomaly is being reported. The use of Computed Tomography Coronary Angiography (CT CAG) allows recognition of the entity and description of variants, in addition to providing prognostic information. With thirteen variants reported to date, few of these variants have important therapeutic implications and hence are important to identify. This article aims to review the definitions, embryologic basis, classification and recognition of clinically significant dual LAD variants.

## DUAL LAD: ANATOMY AND DEFINITION

The Left Coronary Artery (LCA) arises from the left sinus of Valsalva, courses behind the pulmonary trunk and bifurcates into the Left Anterior Descending Artery (LAD) and Left Circumflex Artery (LCX). The LAD courses in the Anterior Interventricular Groove (AIG) and gives rise to the diagonal branches (supplying the anterior and lateral wall of LV) and septal perforating branches (runs in the musculature of the interventricular septum). The

LAD terminates near the cardiac apex and communicates with ramifications of the posterior descending artery which runs in the posterior interventricular groove.

Dual LAD anatomy, also described as duplication of LAD is defined as presence of two LAD arteries in the AIG. The short LAD usually arises from the left coronary system (in rare instances from right coronary system) and terminates in the proximal portion of AIG without reaching the cardiac apex. The long LAD has a variable origin (either from left or right coronary system), enters the distal portion of AIG reaching up to the cardiac apex. When the long LAD originates from the opposite sinus, it courses pre pulmonary, retro aortic, inter arterial or transeptal to reach the AIG. Transeptal course is the most common, whereas an inter arterial course can be malignant. Septal and diagonal branches may arise from both vessels and is highly variable. Even though this anomaly is seen more often with congenital malformations such as tetralogy of Fallot or transposition of great arteries, its incidence in normal hearts ranges from 0.13%-1.38% [4-7].

## DUAL LAD: EMBRYOLOGIC BASIS

Knowledge of embryologic basis of coronary anomalies is largely derived from animal data. The coronary ostia develop after separation of the common truncus into aorta and pulmonary trunk after 42 days of embryo formation. Animal and human models have demonstrated that cells derived from Peritruncal Capillary Plexus (PCP) penetrate the aortic wall (aided by local apoptosis) resulting in the formation of coronary ostia. Coronary ostia establish connections with capillary plexus (derived from sinus venosus endocardium, ventricular endocardium and epicardial cells) and initiate recruitment of Vascular Smooth Cells (VSMCs) thus establishing coronary circulation. Abnormalities

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involving Vascular Endothelial Growth Factor (VEGF), Neural Crest Cells (NCCs) and Tbx1 expression (gene implicated in arterial pole development) can result in anomalous connections between coronary ostia and capillary plexus [3,8]. Dual LAD may result from an abnormal development of coronary ostia or due to development of anomalous connections between the ostia and peripheral capillary plexus.

## DUAL LAD: MORPHOLOGICAL CLASSIFICATION

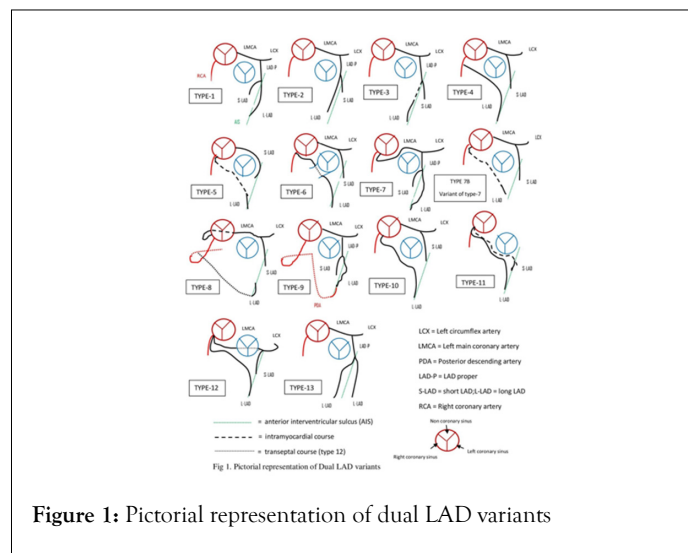
Classification of dual LAD is based on the origin and course of short and long LADs. An early attempt to classify dual LAD

anomalies based on angiography was made by Franco et al. [4] in 1983 that lead to the identification of first four types of dual LAD anomalies. Of this type 1 is the most common type while type 4 is the least common. Further studies lead to the addition of several other types based on the origin and course of the two LAD arteries [9-16]. Currently, there are 13 types. Of dual LAD anomalies identified till date. Anatomical classification of dual LAD is depicted in Table 1 (Adapted and modified from [16]) (Figure 1). Though the reported variants are bound to increase with time, the short LAD is more consistent in its origin and course. On the other hand, the long LAD has a variable origin and course and determines the hemodynamic significance in most instances.

**Table 1:** Anatomical classification of Dual LAD variants.

Type	Short-LAD origin	Short-LAD course	Long-LAD origin	Long-LAD course
1	LCS(LAD)	Terminates in the proximal portion of AIG	LAD-Proper	Runs along the LV side of proximal AIG (epicardial) and enters the distal AIG
2	LCS(LAD)	Terminates in the proximal portion of AIG	LAD-Proper	Runs along the RV side of proximal AIG(epicardial) and enters the distal AIG
3	LCS(LAD)	Terminates in the proximal portion of AIG	LAD-Proper	Runs intramyocardial course in the emerges epicardial in the distal AIG.
4	LCS(LMCA)	Terminates in the proximal portion of AIG	Proximal RCA	Anomalous prepulmonic (epicardial) course anterior to RVOT and enters the distal AIG
5	LCS	Terminates in the proximal portion of AIG	RCS	Anomalous intramyocardial course and then emerges epicardial in the distal AIG
6	LCS(LMCA)	Terminates in the proximal portion of AIG	Proximal RCA	Anomalous epicardial course between RVOT and aortic root, (Interarterial) then enters distal AIG
7	LMCA that originates in the RCS and courses inter-arterially	Terminates in the proximal portion of AIG	LMCA that originates in the RCS and courses inter-arterially	Enters distal AIG
7b (Variant of 7)	LCS(LMCA)	Terminates in the proximal portion of AIG	RCS (separate ostium)	Intramyocardial course and emerges in the distal AIG
8	LMCA (originates from RCS and shows retro-aortic course)	Terminates in the proximal portion of AIG	Mid RCA	Courses the inferior surface of the RV (epicardial), turns around the apex to reach the distal AIG
9 (Triple LAD)	LCS(LAD)	Terminates in the mid portion of AIG	LCS(LAD)	Runs on LV side of mid AIG(epicardial) then enters the distal AIG, terminating proximal to the apex-
10	LCS(LMCA)	Terminates in the proximal portion of AIG	RCS(separate ostium)	Epicardial prepulmonic course anterior to RVOT and enters the distal AIG
11	RCS (common ostium with RCA)	Intramyocardial course within proximal septum and emerges in the proximal AIG(where it terminates as the first diagonal branch)	RCS ( common ostium with RCA)	Takes an anomalous prepulmonic course anterior to RVOT and enters the distal AIG
12	LMCA from RCS- same ostium with RCA	Terminates in the Proximal portion of AIG/LCX arises from LMCA	RCS-common ostium with RCA	Epicardial prepulmonic course to reach distal AIG
13	No short-LAD	N/A	LCS(LAD) LCS(LAD)	Two long LADs move out of the AIG and courses to the cardiac apex, one laterally and the other medially to the AIG

**Abbreviations:** LAD: Left Anterior Descending Artery; RCS: Right Coronary Sinus; LMCA: Left Main Coronary Artery; RCA: Right Coronary Artery; LCS: Left Coronary Sinus; AIG: Anterior Interventricular Groove; LV: Left Ventricle; RV: Right Ventricle; RVOT: Right Ventricular Outflow Tract.



**Figure 1:** Pictorial representation of dual LAD variants

## DUAL LAD VARIANTS AND THEIR CLINICAL SIGNIFICANCE

Dual LAD is commonly an incidental finding during coronary angiography. Most of the dual LAD anomalies are hemodynamically insignificant, except those with inter-arterial or intra-myocardial course. The presence of a short LAD may confer an ischemic protection due to a twin blood supply and potential to form collaterals.

Failure to identify dual LAD anatomy can result in adverse therapeutic implications during bypass artery grafting. Knowledge of anatomy is essential to avoid incorrect placement of arteriotomy. Failure to recognize and graft the short LAD may result in incomplete revascularization. Grafting the short LAD may warrant a more proximal approach, due to its termination in the proximal AIG. A short LAD can be misinterpreted as a Type I LAD, especially when the long LAD arises from the opposite sinus [17]. A short LAD can also mimic a mid-LAD occlusion. On the other hand, occlusion of a short LAD may be overlooked and a potential revascularization benefit may be missed. Detection of hypoplastic or small LAD during angiography and paucity of vessel opacification in the LAD territory should raise the suspicion of a dual LAD anomaly. The long LAD may have an epicardial or intramyocardial course. Type 3 and Type 5 variants of dual LAD are identified by presence of intramyocardial course of the long LAD, which is important to recognize while planning revascularization procedures [18]. Type 6 and 7 variants are characterized by a malignant inter-arterial course and are associated with an elevated risk of myocardial ischemia and sudden cardiac death (SCD). In Type 6 variant, the long LAD has an inter arterial course whereas in type 7 variant, the LMCA arises anomalously from right sinus and courses inter arterially, to divide and give rise to the short and long LAD, along with LCX. Type 10, 11 and 12 variants are more likely to be injured during median sternotomy due to their pre pulmonic course and close proximity with the sternum. Type 11 variant carries additional significance due to intra-myocardial course of short LAD. Dual LAD is also reported to be associated with anomalies involving other coronaries (LCX and RCA) [19].

Though more variants may be added to the classification with time, prompt recognition and treatment of malignant variants carries priority. Long term prognosis of dual LAD variants and

their association to increased risk of atherosclerosis have not been established.

## ROLE OF CT CAG IN DIAGNOSIS

Conventional angiography is considered the gold standard for evaluation of coronary anatomy. But however, multislice CT CAG with 3 dimensional (3D) construction is considered superior for identification and characterization of coronary anomalies. CT CAG has additional advantage of being noninvasive. With respect to dual LAD variants, CT CAG yields valuable information regarding the variable origin (especially of the long LAD) and course of the branches. Conventional angiography may suffice to establish a diagnosis when both short and long LAD arises from common LAD (LAD proper). But however, when the long LAD arises from the opposite sinus, CT CAG is needed to delineate the origin and course. The presence of long LAD arising from opposite sinus can be missed during conventional angiography, if not suspected and searched for. CT CAG studies suggest that prevalence of dual LAD may be much higher than reported using conventional angiography [11]. Intramyocardial or epicardial course is important to recognize in need for revascularization. Identification by CT CAG an inter arterial course between the RVOT and aorta in symptomatic patients demand immediate intervention. In asymptomatic patients with inter arterial course, further evaluation to demonstrate physiological consequences and ischemia is warranted. The risk benefit ratio of surgical procedure in such patients has to be considered. Rare instances where the long LAD was seen arising from the pulmonary artery have also been reported and was identifiable only by CT CAG.

## CONCLUSION

Dual LAD anatomy characterized by presence of short and long LAD is most often a benign coronary anomaly. CT CAG is superior in delineating the origin and the course of short and long LAD compared to conventional angiography and should be considered as the choice of investigation. Failure to identify a dual LAD anatomy may significantly affect the outcome of revascularization procedures. A higher risk of ischemia and sudden cardiac death is associated with few malignant variants and is important to recognize. A high degree of suspicion and judicious use of CT CAG will unmask more dual LADs and their variants.

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