

A case of variant of dual left descending artery in patient with acute myocardial infarction

Abstract

Coronary artery anomalies are sporadic, most consist of minor variations and are benign. In contrast, some coronary artery anomalies are well described as a cause of cardiovascular morbidity and mortality. Dual left anterior descending artery (LAD) is an extremely rare coronary anomaly. In the present article, we describe a case of type 6 dual LAD in a patient with acute ST elevation myocardial infarction. The aim of this paper is to review the incidence and classification regarding of dual LAD systems.

Keywords: coronary anomaly, percutaneous coronary intervention, acute myocardial infarction/stemi

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Abbreviations: LMCA, left main coronary artery; LAD, left anterior descending artery; Lcx, left circumflex artery; RC, right coronary artery; AIVS, anterior intraventricular sulcus; RVOT, right ventricle outflow tract; RSV, right valsalva sinus

Introduction

Coronary artery anomalies arise sporadically and are found in 0.3 to 1.3% of patients undergoing coronary angiography, most consist of minor variations in the location of the coronary arterial origin and are commonly benign. In contrast, some coronary artery anomalies are well described as a cause of cardiovascular morbidity and mortality due to a specifically abnormal course, traditionally called inter-arterial, or “between the aorta and pulmonary artery,” and associated with a severe prognosis.²

Dual left anterior descending artery (LAD) is an extremely rare coronary anomaly traditionally classified into one of four types described by Spindola-Franco.³ As described by these authors, Dual LAD consists of a short LAD that ends high in the anterior interventricular sulcus (AIVS) and a long LAD that most commonly originates as an early branch of the LAD proper (types 1–3) and rarely originates anomalously from the right coronary artery (type 4). Subsequently, other variants of this anomaly were described, called type 5 by Manchanda et al.,⁴ and type 6, by Maroney et al.,⁵ when the course of “long LAD” is between the right ventricle outflow tract (RVOT) and the aorta root. Other variants have been proposed in recent studies using CT angiography by Bolzar et al.,⁶ (Table 2-4). In the present article, a case of Type 6 double LAD is described in a patient with acute ST elevation myocardial infarction. The aim of this paper is to review the incidence and classification regarding double LAD system.

Case history

67-year-old male, former smoker and with history of hypertension is admitted to coronary care unit due to acute myocardial infarction with posterolateral ST-segment elevation, treated with thrombolytic and positive reperfusion criteria, referred to our institution for coronary angiography. The echocardiogram showed preserved ventricular function and moderate aortic valve stenosis. Selective left coronary angiography through a trans-radial access revealed normal left main coronary artery (LMCA) divided in Left Circumflex artery

(LCx) having in its posterolateral branch an eccentric lesion with stenosis of 80% (Figure 1A) and LAD which emerges a large diagonal branch and the first great septal branch and runs in the top of the AIVS where it ends. We initially interpreted this as a completely occluded LAD in the proximal segment.

Selective right coronary angiography revealed a dominant right coronary artery (RCA), without lesions (Figure 1B), during the injection, contrast media dyes unselectively an artery that comes very close to the origin of the RCA. Selective angiography of this vessel reveals that it is the middle - distal segment of the LAD which is directed right to left and return to its position on the AIVS giving a septal branch near its origin and a second diagonal branch (Figure 1D) establishing the diagnosis of a dual LAD system (Figure 1C), the proximal segment of the long LAD was narrower than the middle segment without an arteriosclerotic lesion and no compression was observed during systole. CT angiography identified the anomalous origin of long LAD from the Right Valsalva Sinus (RVS) near the origin of the RCA, and passing between the aortic root and RVOT providing a second diagonal branch without atherosclerotic lesions (Figure 2A&2B), and ending in the distal AIVS. The lesion in the posterolateral branch was successfully treated with the implant of 3.0 x 28mm everolimus eluting stent.

Discussion

As stated in the paper of Angelini et al.⁷, the primary assumption is that the term “normal” must apply to 99% of the population, which automatically implies that the prevalence of artery anomalies must be below 1%. Anomalies of and variations in the coronary arteries are important in pathophysiology and the treatment of cardiovascular diseases. The LAD coronary artery has the most consistent origin, course, and distribution in the human heart, and anomalies are rare. The dual LAD system is a very rare congenital anomaly and reports estimate a prevalence around 1%. Awareness of dual LAD anomalies could be very important for coronary surgery and interventional procedures. Subtype 4 LADs have a prevalence of ~ 0.021% in large series of patients referred for coronary angiography,⁸⁻¹³ (Table 1).

Spindola – Franco³ divided this anomaly into four types (Table 2) in series of twenty-three patients, subtype 1 and 2 are considered normal variants and are the most common and have benign prognosis. The origin of the LAD in the opposite sinus, has drawn attention

for its association to sudden death or life-threatening ventricular arrhythmias. Further case reports identified two additional variations, called subtype 5 and subtype 6 (Table 3) due to certain minor anatomical variations, which added to the traditional classification of Spindola-Franco. These new subtypes appear in the first case, due to the separate source in the RVS (type 5) or on the way to take the LAD to resume its normal position in the AIVS, for example, between the aortic root and RVOT (subtype 6).

A study of prevalence of this anomaly by CT angiography⁶ in 1337 patients found incidence of dual LAD to be 4%, four times greater than the frequency described in the catheter angiography studies, the most frequent was the type 1 (86%), three case of type 3 and two case of type 4 (0.03%). Type 2, 5 and 6 were not detected and the authors propose three new subtypes suggesting to call them type 7 to 9 (Table 4). However, in two of these cases (type 7 and 8), the

LMCA emerged from the opposite coronary sinus: in the former, both short and long LAD are born on the LAD properly. In our opinion this anomaly, would be a source of LMCA combined with a LAD 1 type variant, this type of anatomy was presented by Tuncer et al as a variant (called type 5) double LAD.¹⁴ This shows that through various case reports, many authors have been changing and adding new types, generating confusion in the classification. In the second type proposed by the authors, together with the anomalous origin of LMCA, long LAD merges directly from a marginal branch of RCA. We consider that the only anomaly in the rise of LMCA is the opposite coronary sinus carrying a prognosis that depends exclusively on the course that takes the LMCA to return to its normal position, and therefore should not be considered as new variants of dual LAD. Type 9, corresponds, on type 1 with poorly development of the long LAD and prominent development of the posterior descending artery to cover the area of the apex.

Table 1 Prevalence Values of the Anomalous Left Anterior Descending Artery in Several Angiographic Studies

	Patient/Sample Size	Prevalence of the anomalous LAD	Origin from the RCA	Origin from the RSV	Origin from the PSV	Initial course of the LAD
Yamanaka et al., ¹	38/126595	0.03	-	-	-	-
Spindola-Franco ³	Feb-40	0.009	2	-	-	Anterior (2)
Tuncer et al., ⁸	12/70850	0.017	4	7	1	Interarterial (2) Septal (2) Anterior (8)
Gol MK et al., ⁹	6/58023	0.01	2	4	-	-
Villa RB et al., ¹⁰	2/13500	0.014	-	2	-	Anterior (2)
Wilkins et al., ¹¹	2/10661	0.018	2	-	-	Anterior (2)
Harikrishnan et al., ¹²	Jan-00	0.013	-	1	-	Anterior (1)
Kimberis et al., ¹³	Feb-00	0.028	-	2	-	Anterior (1) Septal (2)
Total	65/296169	0.02%				

LAD: Left Descending Artery; RCA: Right Coronary Artery; RVS: Right Valsalva Sinus; PVS: Posterior Valsalva Sinus

Table 2 The original dual LAD classification made by Spindola-Franco et al.,³

	LMCA	LAD proper	Origin		Course		Location of long LAD	
			Short LAD	Long LAD	Short LAD	Long LAD	Proximal long LAD	Distal long LAD
Type 1	Present	Present	LAD proper	LAD proper	Originates from LAD proper terminates in the proximal AIVS	Originates from LAD proper; course on LV side of the proximal AIVS, reenters the distal AIVS	Epicardial (LV)	Distal AIVS
Type 2	Present	Present	LAD proper	LAD proper	Originates from LAD proper terminates in the proximal AIVS	Originates from LAD proper; course on RV side of the proximal AIVS, reenters the distal AIVS	Epicardial (RV)	Distal AIVS
Type 3	Present	Present	LAD proper	LAD proper	Originates from LAD proper terminates in the proximal AIVS	Originates from LAD proper follows an intramyocardial course in the septum proximally, and emerges epicardially in the distal AIVS (or does not emerge in the AIVS and terminated, intramyocardially)	Intramyocardial (intraventricular septum)	Distal AIVS
Type 4	Present	Absent	LMCA	Proximal RCA	Originates from the LMCA, terminate in AIVS	Originates from the proximal RCA follows an anomalous prepulmonic course anterior to the RVOT, and enters the distal AIVS	Epicardial (prepulmonic)	Distal AIVS

LMCA: Left Main Coronary Artery; LAD: Left Anterior Descending Artery; AIVS: Anterior Intraventricular Sulcus; LV: Left Ventricle; RV: Right Ventricle; ROVT: Right Outflow Ventricle Tract

Table 3 Other dual LAD types published in the literature

	LMCA	LAD proper	Origin		Course		Location of long LAD	
			Short LAD	Long LAD	Short LAD	Long LAD	Proximal long LAD	Distal long LAD
Type 5 (4)	Absent	Absent	LVS	RSV	Originates from LVS proper, terminates in the proximal AIVS	Originates from proximal RVS, follows an anomalous intramyocardial course within the septal crest, emerges epicardially, and enters the distal AIVS	Intramyocardial (septal crest)	Distal AIVS
Type 6 (5)	Present	Absent	LMCA	Proximal RCA	Originates from LMCA, terminates in the proximal AIVS	Originates from proximal RCA, follows an anomalous course between the RVOT and the aortic root, and enters the distal AIVS	Epicardial (retropulmonic)	Distal AIVS

LMCA: Left Main Coronary Artery; LAD: Left Anterior Descending Artery; LVS: Left Valsalva Sinus; RVS: Right Valsalva Sinus; AIVS: Anterior Intraventricular Sulcus; RCA: Right Coronary Artery; RVOT: Right Ventricular Outflow Tract.

Table 4 Morphologic features of novel dual LAD subtypes described in this series.⁶

	LMCA	LAD proper	Origin		Course		Location of long LAD	
			Short LAD	Long LAD	Short LAD	Long LAD	Proximal long LAD	Distal long LAD
Type 7	Present (LMCA originates from RCA and follows interarterial course)	Present	LAD proper	LAD proper	Originates from LAD proper, terminates in the proximal AIVS	Originates from LAD proper, course on LV side of the proximal AIVS, reenters the distal AIVS	Epicardial (LV)	Distal AIVS
Type 8	Present (LMCA originates from RCA and follows retroaortic course)	Absent	LMCA	Mild RCA (branch right marginal artery)	Originates from LMCA, terminates in the proximal AIVS	Originates from mid RCA, course inferior wall surface of the RV turns around of apex the and reaches to the distal AIVS	Epicardial (RV inferior side)	Distal AIVS
Type 9 (triple LAD)	Present	Present	La proper	LAD proper	Originates from LAD proper, terminates in the mild AIVS	Originates from LAD proper course on LV side of the mid AIVS, reenters the distal AIVS, and terminates before reaching to the apex	Epicardial (LV)	Distal AIVS

AIVS: Anterior Intraventricular Sulcus; LAD: Left Anterior Descending Artery; LVS: Left Valsalva Sinus; LMCA: Left Main Coronary Artery; LV: Left Ventricle; RCA: Right Coronary Artery; RVS: Right Valsalva Sinus; RV: Right Ventricle

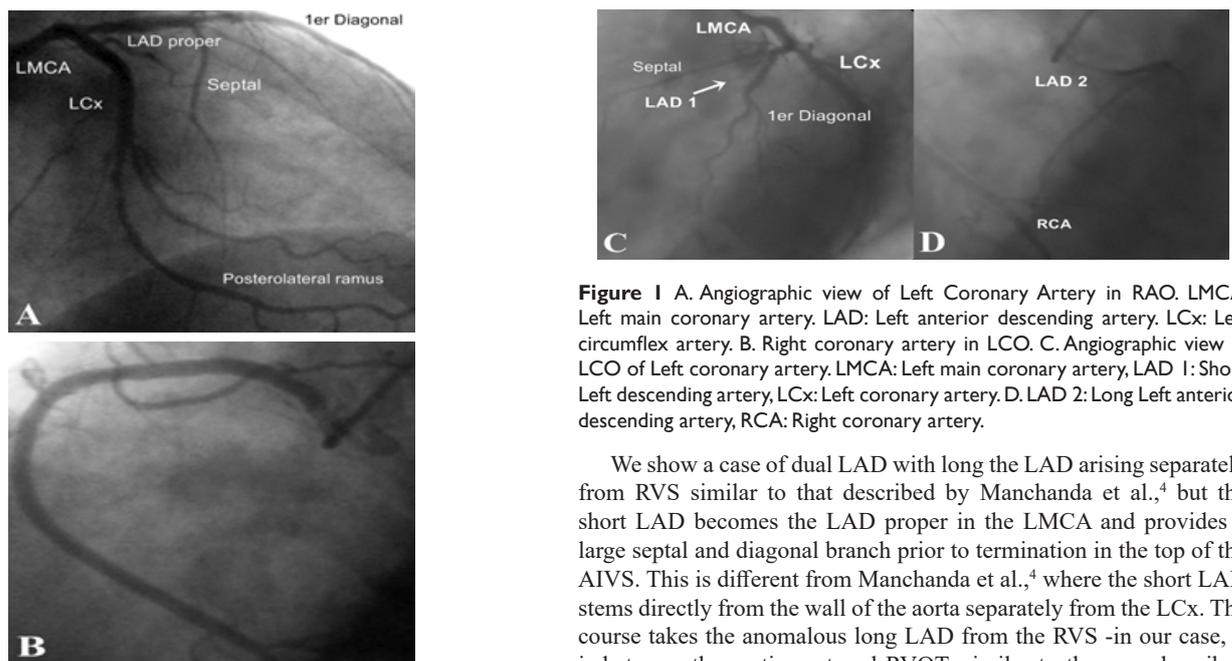


Figure 1 A. Angiographic view of Left Coronary Artery in RAO. LMCA: Left main coronary artery. LAD: Left anterior descending artery. LCx: Left circumflex artery. B. Right coronary artery in LCO. C. Angiographic view in LCO of Left coronary artery. LMCA: Left main coronary artery, LAD 1: Short Left descending artery, LCx: Left coronary artery. D. LAD 2: Long Left anterior descending artery, RCA: Right coronary artery.

We show a case of dual LAD with long the LAD arising separately from RVS similar to that described by Manchanda et al.,⁴ but the short LAD becomes the LAD proper in the LMCA and provides a large septal and diagonal branch prior to termination in the top of the AIVS. This is different from Manchanda et al.,⁴ where the short LAD stems directly from the wall of the aorta separately from the LCx. The course takes the anomalous long LAD from the RVS -in our case, it is between the aortic root and RVOT, similar to the case described

by Maroney et al⁵, this being the type of path which carries worse prognosis. The confusion in the classification is, in the first case, of the low incidence of this anomaly. This is more important when the subtype 4 is involved, secondly, because when these anomalies are present at birth with slight variations. For example: in termination or course, different authors propose new types. In our opinion, the source in the opposite sinus of the LMCA, should not be considered as dual LAD.

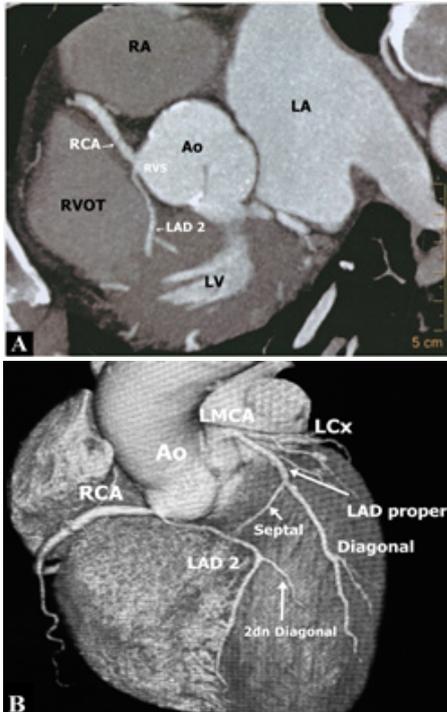


Figure 2 A. CT angiography axial section. RA: Right atrial. LA: Left atrial. Ao: Aortic root. RVS: Right Valsalva sinus. RVOT: Right ventricle outflow tract. LV: Left ventricle. RCA: Right coronary artery. LAD 2: Long left anterior descending artery. B. 3D volume CT angiography. LMCA: Left main coronary artery. LAD: Left anterior descending artery. LCx: Left circumflex artery.

Conclusion

Abnormalities in the LAD are highly rare, natural history is unknown, we present a case of type 6 double LAD, where the long LAD arises directly from the RVS and takes a course between the aortic root and RVOT (interarterial) in a patient with MI with ST elevation successfully treated with thrombolytics and subsequent stenting.

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Conflicts of interest

Author declares there are no conflicts of interest.

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