

CHALLENGES OF ANOMALOUS CORONARY ARTERY INTERVENTIONS – A CASE SERIES

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ABSTRACT

Background: The Dual left anterior descending (LAD) artery is a rarely reported coronary anomaly, consisting of two branches supplying the usual distribution of the LAD. Type 4 and type 10 dual LAD anomalies in which a short LAD arises from the left main coronary artery and a long LAD arise from the right coronary artery or right coronary artery sinus respectively are remarkably rare. The s-LAD (Short LAD) should not be misdiagnosed as total occlusion of the distal segment and the l-LAD (Long LAD) arising from proximal RCA or right coronary sinus, should not be misidentified as a conus branch. Recognition of the dual LAD is of critical importance before planning any revascularization procedures, and all novice cardiologists should be aware of this rare congenital coronary anomaly.

Case Presentation: We describe a total of four cases of dual LAD coronary anomalies consisting of two cases each of type 4 and type 10 anomalies, which are rarely encountered in clinical practice detected by conventional coronary angiogram (CAG) and subsequently underwent successful Percutaneous Coronary Intervention (PCI) of each anomaly, highlighting the importance of knowledge of these rare anatomical variants and application of this knowledge in clinical practice contributing to the success of interventional procedure translating into improved patient outcomes.

Conclusions: This case series demonstrates the importance of knowledge regarding the presence of rare variants of dual LAD coronary anomalies which aid in the diagnosis and management of these patients ensuring successful revascularisation, with either PCI or CABG, translating into symptomatic relief and significant cardiovascular benefit.

Keywords: Left Anterior Descending Coronary Artery (LAD), Coronary Anomalies, Coronary Angiography, Percutaneous Coronary Intervention.

Introduction

Dual LAD (left anterior descending coronary artery) is a rare coronary anomaly. In this rare congenital anomaly, there are two distinct segments of the vessel, one short LAD (S LAD) that ends high up in the Anterior Inter Ventricular Sulcus (AIVS), and another long LAD (L LAD) that enters the distal AIVS and supplies the apex. The s-LAD should not be misidentified as total occlusion of distal segment and the l-LAD arising from proximal RCA

or right coronary sinus, should not be misidentified as a conus branch. A paucity of distribution of vessels in the apical LAD territory with a small LAD proper during coronary angiography (CAG) should alert the cardiologist to dual LAD as one of the likely possibilities.^[1] Recognition of dual LAD is of critical importance before planning any revascularization procedures, and all novice cardiologists should be aware of this rare congenital coronary anomaly. Herein we report a case series of a total of four cases of dual LAD anomalies consisting of two cases of each of type 4 and type 10 variants respectively who underwent a coronary angiogram and successful PCI of each anomaly followed by a short review of the literature of these anomalies.

Case Series

Case 1

A 50-year-old female who is a known case of diabetes presented with exertional angina of 4 months duration to our outpatient department. On evaluation, routine blood tests and Electrocardiogram (ECG) were within normal limits. 2D-Echocardiography (2 dimensional - Echo) showed mild Left Ventricular Hypertrophy (LVH) with normal biventricular function and normal valves. Exercise Treadmill testing (TMT) was positive for inducible ischemia with the patient developing angina during the second stage, without significant ECG changes. The conventional CAG was done on the patient, where The left coronary artery (LCA) angiogram showed the left main coronary artery (LMCA) to be normal giving rise to a short LAD which ends prematurely in proximal AIVS [Fig 1A] and a dominant left circumflex (LCX) artery, which was normal. The short LAD gave a small septal before termination. The right coronary artery (RCA) angiogram showed a non-dominant, normal RCA, Posterior descending artery (PDA) and Posterior left ventricular Vessel (PLV) and another vessel originating from proximal RCA and coursing leftward entering the AIVS and giving diagonals suggestive of a long LAD [Fig 1B]. According to **Spindola-Franco angiographic classification**, this was consistent with type 4 dual LAD (2). The angiogram did not show any lesion causing significant luminal narrowing, requiring no intervention, and the patient was discharged on appropriate medications.

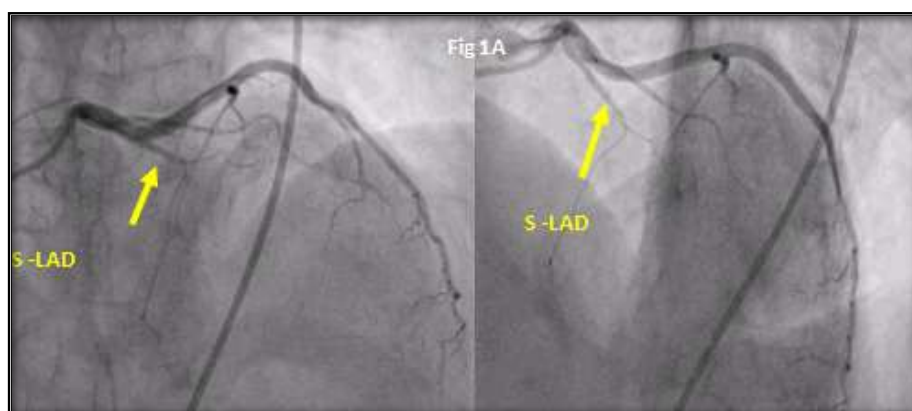


Figure 1A: Left coronary angiography in AP cranial view and LAO cranial view showing short LAD giving a small septal before termination and left circumflex artery.

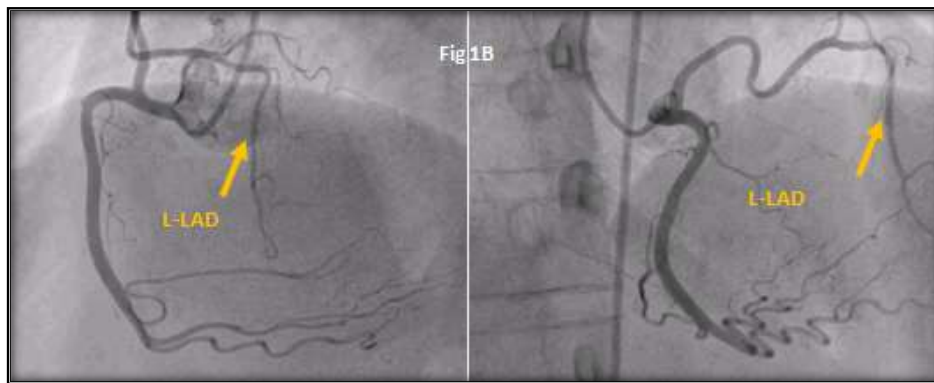


Figure 1B: Right coronary artery in LAO view and RAO view showing normal RCA with TIMI III flow and the long LAD arises from the proximal RCA and gives off-diagonal branches.

Case 2

A 45 years old male, with a history of long-standing type 2 diabetes and smoking presented with h/o recurrent rest angina of 4 hours duration, with ECG showing ST elevations in V 1 – V4 with reciprocal ST depressions in inferior leads along with elevated cardiac enzymes and was diagnosed to have acute anterior wall myocardial infarction (AWMI). 2 D echocardiography showed hypokinetic anterior IVS and apical segments with preserved wall thickness and Left Ventricular Ejection Fraction (LVEF) of 42%. The patient was taken up immediately for conventional CAG and possible revascularization. Left Coronary Angiogram showed normal LMCA with proximal LAD giving a small diagonal before ending abruptly and giving an impression of mid-total occlusion with LCX being non-dominant and major Obtuse Marginal (OM) having proximal 50 % discrete stenosis. The patient was planned for Percutaneous Transluminal Coronary Angioplasty (PTCA) stenting to LAD and the same was attempted unsuccessfully. On the careful evaluation of the LCA angiogram, it was noted that LAD was short and ended abruptly and prematurely in the proximal AIVS (S LAD) [Fig 2A]. Right, Coronary angiogram showed dominant and normal RCA and its branches, with LAD arising from proximal RCA and traversing across Right Ventricular Outflow Tract (RVOT) to the left and continuing in distal AIVS (L - LAD). L - LAD gave rise to septal and diagonal branches and had a distal 95% discrete stenosis with TIMI II flow[Fig 2B]. The patient was diagnosed to have type 4 dual LAD and the culprit lesion was in the L- LAD arising from proximal RCA. The patient was promptly revascularized through stenting to L LAD resulting in TIMI III flow, followed by resolution of symptoms and improved hemodynamics. The patient was discharged in stable condition on appropriate medications.

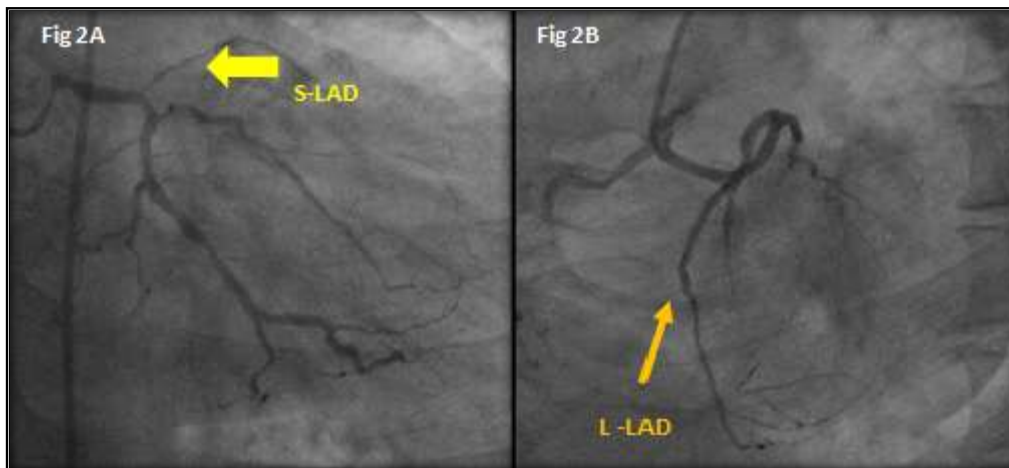


Figure 2A: LCA angiography in AP caudal view LAD giving a small diagonal before termination and left circumflex artery.

Figure 2B: RCA angiography in LAO cranial view showing long LAD with diagonals and septal arising from Proximal RCA.

Case 3

A 60-year-old male with complaints of effort angina for the past 2 days, He had a longstanding history of smoking with no other risk factors for CAD. ECG showed ST elevation with T wave inversions in inferior leads along with elevated cardiac troponin levels and was diagnosed to have evolved inferior wall myocardial infarction. 2D echocardiography showed hypokinetic segments in inferobasal and inferior segments with near-normal left ventricular function. Due to ongoing chest pain, the patient was taken up for CAG. The LCA angiogram showed normal LMCA and dividing into LAD and LCX. This LAD was the short (S-LAD) and ended prematurely in the proximal AIVS creating an illusion of complete occlusion of the mid-LAD artery. This S-LAD also gave rise to the first diagonal and septal branch [Fig 3A]. The LCX coronary artery was non-dominant and normal. Selective right coronary artery (RCA) angiography showed mid-RCA total occlusion and the long LAD (L-LAD) originating from the right coronary sinus separately from the right coronary artery [Fig 3B]. This long vessel traversed to the left side, giving diagonal branches and re-entering the distal AIVS to reach the apex of the heart and was consistent with type 10 dual LAD (1,4). The patient underwent PTCA + stenting to mid-RCA and was discharged on appropriate medications. The patient was asymptomatic on subsequent follow-ups.

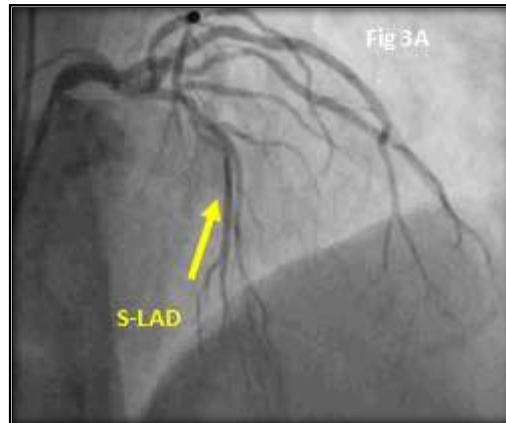


Figure 3A: Left coronary angiogram (LCA) showed the LAD coronary artery arising from the left main coronary artery short LAD (S-LAD).

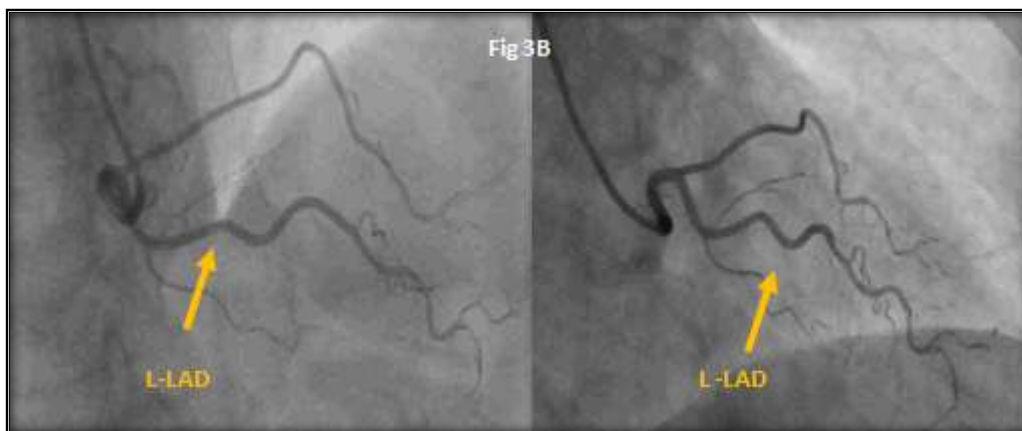


Figure 3B: Right coronary angiogram showed the long LAD (L-LAD) originated from the right coronary sinus separately from the right coronary artery.

Case 4

A 52 year old male with a long-standing history of diabetes and hypertension presented with a history of recurrent rest angina 8 years back. Cardiac troponins were elevated with ECG showing ST elevation in V 1 to V 4 anterior leads and echocardiography showed hypokinetic distal IVS and apical segments with LVEF of 45 %. The patient was diagnosed to have acute AWTMI and was taken up promptly for CAG with possible revascularisation. The LCA angiogram showed the LAD and LCX arising from the normal LMCA with LAD being short and ending prematurely in the proximal AIVS (Short LAD) after giving the first diagonal and septal branches giving an impression of totally occluded mid-LAD segment. The LCX coronary artery was non-dominant and had proximal 70 % discrete stenosis [Fig 4A]. Selective RCA angiography showed mid-RCA 80 % discrete stenosis with normal PDA and PLV branches. On careful review of the RCA angiogram, it was observed that the long LAD originated from the right coronary sinus separately from RCA origin traversing to the left side, giving diagonal branches and re-entering the distal AIVS to reach the apex of the heart and was diagnosed to have type 10 dual LAD coronary anomaly [Fig 4B]. The L - LAD arising from the right coronary sinus had mid-segment, discrete 95 % stenosis with grade 3 thrombus and TIMI II flow, resulting in acute AWTMI. The long LAD was revascularized

with PTCA and stenting promptly resulting in TIMI III flow, followed by resolution of symptoms and hemodynamic stability. The patient underwent PTCA and stenting to proximal LCX and mid-RCA during the same hospitalization as a staged procedure and was discharged in stable condition on appropriate medications.

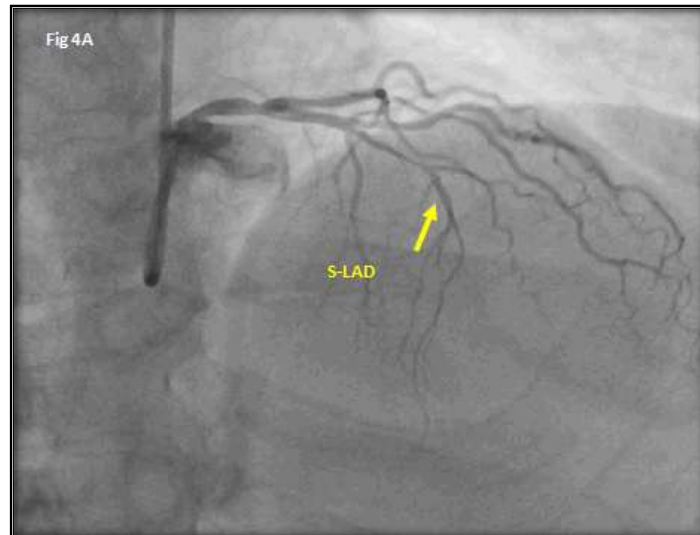


Figure 4A: Left coronary angiogram (LCA) in AP cranial view showed the LAD coronary artery arising from the left main coronary artery short LAD (S-LAD).

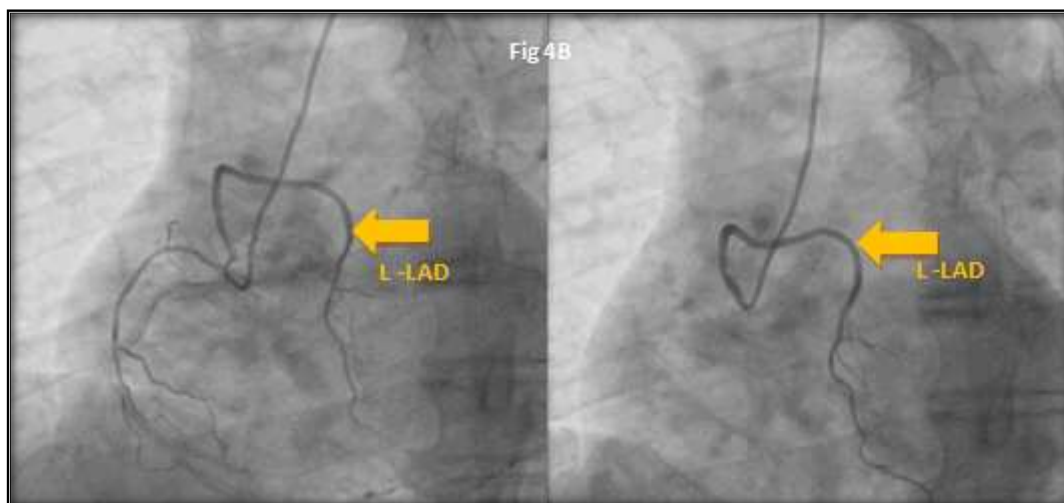


Figure 4B: Right coronary angiogram showed the long LAD (L-LAD) originated from the right coronary sinus separately from the right coronary artery.

Discussion

Congenital coronary anomalies are reported to occur in 0.64–1.3% of patients undergoing coronary angiography (2, 3) which are rare. Coronary anomalies involving their origin, distribution, and course are common with right coronary artery (RCA) circulation but are considerably rare with the LAD artery. Spindola –Franco et al. gave the first angiographic classification of dual LAD anomalies.^[2] According to Spindola-Franco et al., the incidence of Dual LAD is seen in <1% of patients undergoing coronary angiography. The dual LAD coronary anomaly consists of two branches, short and long that supply the usual distribution

of the LAD. According to this classification, LAD consists of a short LAD that ends high in 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 RCA(Type 4).

Angiographic classification of dual LAD (Spindola-Franco et al):

Type 1: Short LAD runs in the AIVS, long LAD also runs in AIVS, descends on the left ventricular side of AIVS, then re-enters the distal AIVS to reach the apex.

Type 2: Short LAD is the same as in type I, long LAD runs on the right ventricular side of AIVS to re-enter AIVS.

Type 3: Short LAD is the same as in types I and II. Long LAD travels intramyocardial in the ventricular septum.

Type 4: Short LAD forms a very short vessel, travels in AIVS, and gives off septal perforators and diagonal branches. Long LAD arises from the RCA, courses anteriorly down to the infundibulum of the right ventricle, and turns towards AIVS sharply, to give septal and diagonal branches.

According to the above classification, the analysis of origin and course of LAD after CAG of cases 1 and 2 of the case series are consistent with the Type 4 type of dual LAD coronary anomaly.

Based on Computed Tomography – CAG (CT-CAG), until now, additional 6 types of dual LAD variants have been described in the literature (Table 1) (4).

According to the above CT -CAG classification, cases 3 and 4, showed that short LAD arises from LCA and long LAD arises from the right sinus of Valsalva from separate ostia, courses anteriorly down the to the infundibulum of the right ventricle through a prepulmonic course, and turns towards AIVS sharply, to give septal and diagonal branches. The origin and course were consistent with the type 10 variety of dual LAD and thus considered a rare type of coronary anomaly (Table 1).

Table 1: Morphologic features of dual LAD subtypes

TYPE	ORIGIN		COURSE	
	Short LAD	Long LAD	Short LAD	Long LAD
Type 1	Proper LAD	Proper LAD	Proximal AIS	LV side of the proximal AIS, and reenters the distal AIS
Type 2	Proper LAD	Proper LAD	Proximal AIS	RV side of the proximal AIS, and reenters the distal AIS
Type 3	Proper LAD	Proper LAD	Proximal AIS	Intramyocardial course in the septum proximally, and emerges epicardially in the distal AIS
Type 4 (Presented cases 1 & 2)	LMCA	RCA	Proximal AIS	Prepulmonic course anterior to the RVOT, and enters the distal AIS
Type 5	LCS	RCS	Proximal AIS	Intramyocardial course within the septal crest emerges epicardially and enters the distal AIS

Type 6	LMCA	RCA	Proximal AIS	Between the RVOT and the aortic root and enters the distal AIS
Type 7	Proper LAD	Proper LAD	Proximal AIS	LV side of the proximal AIS, and reenters the distal AIS (*LMCA originates from the RCS and shows inter-arterial malignant course)
A new variant of Type 7 (Saglam et al. – recently published)	LMCA	RCS	Proximal AIS	Intramyocardial course within the septal crest emerging epicardially in the distal AIS
Type 8	LMCA	Mid-RCA	Proximal AIS	The inferior wall of the RV turns around the apex and reaches the distal AIS (*LMCA originates from the RCS and shows retro-aortic course)
Type 9	Proper LAD	Proper LAD	Mid AIS	LV side of the mid-AIS reenters the distal AIS and terminates before reaching the apex (*Posterior descending coronary artery extends distal AIS)
Type 10 (Presented Cases 3 & 4)	LMCA	RCS	Proximal AIS	Prepulmonic course anterior to the RVOT, and enters the distal AIS
AIS: anterior interventricular sulcus; LAD: left anterior descending artery; LCS: left coronary sinus; LMCA: left main coronary artery; LV: left ventricle; RCA: right coronary artery; RCS: right coronary sinus; RV: right ventricle.				

The first description of a type IV LAD was given in 1939 by Waterson et al. in the case of Sir James Mackenzie, who had this coronary anomaly in addition to ischemic heart disease(5). Since then, many similar cases of dual LAD arising from proximal RCA have been reported in the literature (6-7) including a novel variant of type 10 dual LAD by Celik T et al in 2015.[4] In this case series, we describe a total of 4 cases of dual LAD coronary anomalies rarely observed in clinical practice consisting of 2 cases of type 4 and 2 cases of type 10 LAD variants with different presentations and therapeutic implications (Table 2). In the present case series described, cases 2 and 4 were detected to have type 4 and type 10 dual LAD anomalies on conventional CAG, with each case giving an impression of mid LAD occlusion (short LAD) and on further careful evaluation found to have Long LAD arising from proximal RCA and Right coronary sinus from separate ostia respectively, which had flow-limiting significant stenosis resulting in acute AWTMI, the knowledge of which resulted in successful PCI and symptom resolution. Cases 1 and 3 were detected to have type 4 and type 10 dual LAD anomalies respectively, with no flow-limiting stenosis in LAD and were managed medically. In cases of PCI or coronary artery bypass grafting surgery (CABG), a lack of knowledge regarding the presence of dual LAD may result in stenting or grafting

stenosing of only one of those LADs, leading to incomplete revascularization of the anterolateral wall, interventricular septum, or apex. Familiarity with the variants of dual LAD is a critical step in complete revascularization either with surgery or PCI. If short and long LADs are severely stenosed, grafts to both the vessels may be needed in surgery because supply to the septum and the anterior left ventricular wall may be from two separate vessels. More importantly, it is critical to understand exact coronary anatomy when considering anomalous origin and course of anomalous LAD since long LAD could be injured during median sternotomy due to the very close location of the enlarged right ventricle to the sternum.

Table 2: Demographic Profile of the patients

	Age (years)	Sex	Presentation	ECG	2 D- Echo	Type of Dual LAD anomaly on CAG	Management
Case 1	50	F	Effort angina, Exercise ECG testing positive	Normal	LVEF=60%	Type 4 Dual LAD	Medical management
Case 2	46	M	Acute AWTMI	ST elevations in V 1 – V4 with reciprocal ST depressions in inferior leads,avF	LVEF=42%	Type 4 Dual LAD	L - LAD revascularized with PCI
Case 3	60	M	Evolved IWTMI	ST elevations in inferior leads with T wave inversions	LVEF=50%	Type 10 Dual LAD	RCA revascularized with PCI
Case 4	52	M	Old AWTMI, Recent UA	QS complexes in V1, and V2 anterior leads	LVEF = 48%	Type 10 Dual LAD	L - LAD revascularized with PCI during previous AWTMI Medical management during present hospitalization

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Recognition of dual LAD is important as numerous variants are also associated with congenital heart diseases like tetralogy of Fallot, and transposition of great vessels, where the exact anatomy of the arteries facilitates an appropriate surgical approach during corrective surgery.

Conclusion

We describe a case series of total 4 cases, consisting of 2 rare types of dual LAD with unique type 4 and type 10 variations, in which CAG shows a short LAD arising from LCA and a separate origin of long LAD originates from proximal RCA or Right coronary sinus respectively, wherein successful PCI of these rare anatomic variants ensured complete revascularization. This case series demonstrates the importance of knowledge regarding the presence of rare variants of dual LAD coronary anomalies which aid in the diagnosis and management of these patients ensuring successful revascularisation, with either PCI or CABG, translating into symptomatic relief and significant cardiovascular benefit.

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