

Dual left anterior descending artery with anomalous origin of long LAD from pulmonary artery - rare coronary anomaly detected on computed tomography coronary angiography

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Abstract

Dual left anterior descending artery is a rare coronary artery anomaly showing two left anterior descending arteries. Short anterior descending artery usually arises from the left coronary artery, while long anterior descending artery has anomalous origin and course. Dual left anterior descending artery with origin of long anterior descending artery from the pulmonary artery (ALCAPA) is a very rare coronary artery anomaly which has not been reported previously in the literature. We present the computed tomography coronary angiographic findings of this rare case in a young female patient who presented with atypical chest pain.

Key words: Anomalous origin of left coronary artery from the pulmonary artery; coronary anomaly; dual left anterior descending artery; pulmonary artery

Introduction

Coronary artery anomalies are rare with a reported incidence of 0.64–1.3% in various coronary angiographic studies.^[1,2] Coronary anomalies are one of the most common cardiovascular causes of sudden death in young patients.^[3]

Dual left anterior descending artery (LAD) is a very rare coronary artery anomaly with an incidence of 0.13-1% reported in various studies.^[4]

Anomalous origin of left coronary artery from the pulmonary artery (ALCAPA syndrome) is a rare

congenital coronary artery anomaly. It is usually detected in infancy and is often fatal if left untreated, due to left ventricular ischemia.^[5] Anomalous origin of left anterior descending artery from the pulmonary artery is even rarer.^[6,7]

But presence of dual LAD with ALCAPA has not been reported previously in the literature to our best knowledge. We present these rare findings in a young patient who was referred to our department for computed tomography (CT) coronary angiography.

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Case Report

A 23-year-old young female patient was referred to our department for CT coronary angiography study from some private hospital. Patient presented with atypical chest pain since few months. History revealed features of exertional angina. Physical examination revealed the following findings: Blood pressure- 140/80 mm Hg and pulse- 78/min. Her chest was clear with normal respiratory rate and heart sounds. There was marked sinus arrhythmia. Electrocardiography (ECG) showed anterolateral marked ST segment abnormality with prolonged QT interval, indicating ischemia of the anterior wall of the left ventricle, possibly subendocardial injury. Patient was, therefore, advised coronary angiography.

Conventional coronary angiography study was done in some other hospital. Imaging findings revealed on conventional angiography were dilated and tortuous right coronary artery (RCA) and left circumflex artery (LCX). Only the proximal portion of LAD was visualized, so mid LAD occlusion was reported. In addition, multiple fine tortuous vessels were arising from RCA draining into the pulmonary artery through a dilated vessel. So, suspicion of fistulous communication between the coronary artery and the pulmonary artery was suspected on angiography. Patient was referred to our department for CT coronary angiography for confirmation of diagnosis.

CT coronary angiography was performed on 128-slice scanner (Siemens Somatom Perspective, 128 slice). Patient was premedicated with oral metoprolol (25 mg) one night before the study and half an hour before the study. Sublingual nitroglycerine single spray (0.4 mg) was given just before the scan to induce coronary vasodilatation.

Initially plain scan of the cardiac area was done from the level of carina upto the cardiac apex for calcium scoring of coronary arteries. Then CT angiography for coronary arteries was done. Scan parameters were as follows: Slice thickness 3 mm, rotation time 0.48 s, pitch 0.27, tube voltage 130 kV, and tube current 204 mA with scan time of 8 s. CT angiography was done using automatic bolus tracking. Prescan was taken at the level of aortic root with the region of interest (ROI) placed on descending aorta. Helical scanning was performed in a single breath hold from 1 cm below the carina to the bottom of the heart using biphasic saline chasing technique, keeping the threshold in ascending aorta at 120 HU with scan delay of 3 s. Sixty milliliters of non-ionic contrast medium (iohexol 300 mg I/ml) at a flow rate of 5.5 ml/s was given followed by 50 ml of saline at the same rate to wash out the contrast from the right ventricle. ECG was recorded simultaneously and stored on computer workstation. Retrospective ECG gating was done for CT coronary angiography. Raw helical CT data and ECG trace were used retrospectively

to reconstruct the images. Postprocessing was done by using a combination of various postprocessing techniques including multiplanar reformations (MPRs), curved MPR image, maximum intensity projections (MIPs), and volume-rendered images (VR) for evaluation of coronary arteries.

Plain CT scan showed no calcium burden in the coronary arteries. Agatson calcium score was 0.

CT coronary angiography demonstrated anomalous origin of LAD with combination of dual LAD and ALCAPA.

Left main coronary artery (LMCA) showed normal origin from the left coronary cusp. It bifurcated into short LAD and LCX. Short LAD gave one septal branch and then terminated into septal and long diagonal branches which were tortuous in their course. Long LAD showed anomalous origin from the main pulmonary artery, coursing in the anterior interventricular groove and reaching up to the cardiac apex. Long LAD gave origin to multiple septal and diagonal branches which supplied the interventricular septum and the anterior wall of the left ventricle [Figures 1A, B and 2A, B].

LCX was dilated and showed normal course in the left atrioventricular groove. Its obtuse marginal branches were normal. RCA showed normal origin from the right coronary cusp. RCA and its acute marginal artery were also dilated and tortuous [Figure 3A].

Right-sided dominance was seen.

In addition, RCA and LCX supplied extensive collaterals to LAD territory [Figure 3B and C]. Multiple collaterals were also seen arising from internal mammary artery.

Concentric hypertrophy of walls of the left ventricle with mild dilatation of left ventricle and left atrium was also seen as a consequence of long-standing ischemia.

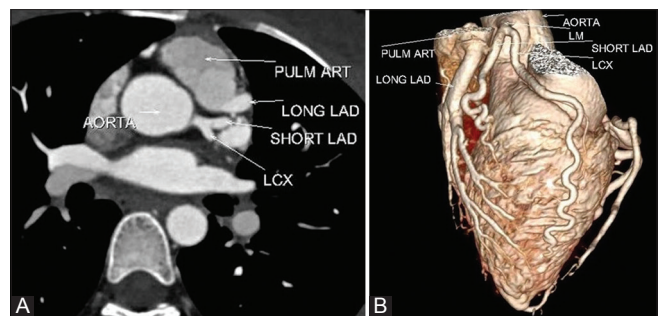


Figure 1 (A and B): (A) Axial CT coronary angiogram image showing origin of long LAD from the pulmonary trunk and of left main coronary artery from the aorta, with its bifurcation into short LAD and circumflex artery (B) 3D volume-rendered CT angiogram image showing origin of long LAD from the pulmonary trunk with the origin of short LAD from the left main coronary artery

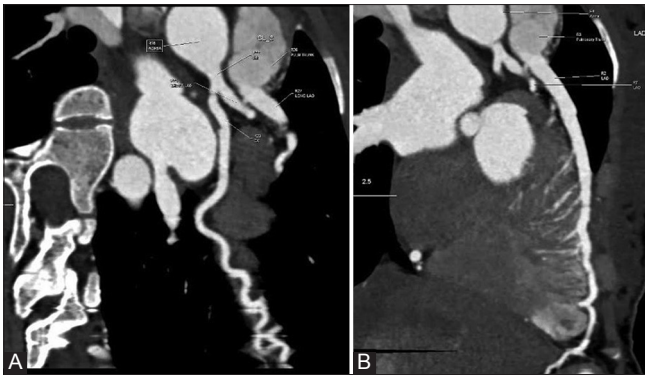


Figure 2 (A and B): (A) Curved MPR images showing origin of left main coronary artery from the aorta with bifurcation into short LAD and circumflex, with the latter coursing in the left atrioventricular groove and short LAD and terminating in proximal interventricular groove (B) MPR image showing long LAD originating from the pulmonary trunk and coursing in anterior interventricular groove

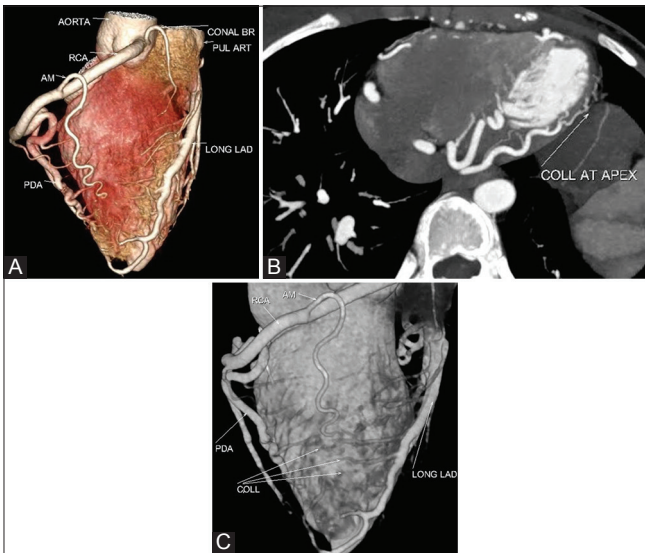


Figure 3 (A-C): (A) Volume-rendered image showing dilated, tortuous right coronary artery, acute marginal branch and terminal branch PDA (B) Axial image shows multiple tortuous collaterals along the epicardial surface (C) Volume-rendered CT angiogram images showing bridging collaterals between PDA and long LAD

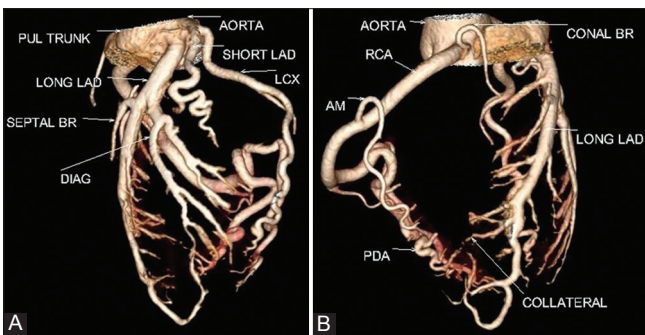


Figure 4 (A and B): (A and B) Segmented 3D CT image demonstrates the origin of RCA and LCA from the respective coronary sinuses, LCA terminating into short LAD and circumflex arteries and long LAD arising from the pulmonary trunk

CT coronary angiographic findings were consistent with dual LAD, with long vessel arising from the main pulmonary trunk (ALCAPA) and short vessel arising from LMCA [Figure 4A and B].

Conventional angiographic findings were misleading as they raised the suspicion of fistulous communication between the coronary artery and the pulmonary artery due to multiple tortuous vessels seen, which were actually tortuous intercoronary collaterals. There was actually retrograde filling of LAD from RCA with reversed coronary flow from LAD to pulmonary artery due to low pressure in the pulmonary artery (coronary steal phenomena). Also, short LAD was misinterpreted as mid LAD occlusion on conventional angiography.

Presence of dual LAD in ALCAPA patient and extensive collateralization contributed to lesser degree of ventricular ischemia and delayed presentation.

Discussion

Dual LAD is a rare congenital coronary artery anomaly with two LADs. Short LAD usually arises from the LMCA and terminates in the proximal part of the interventricular septum and does not reach the cardiac apex. Long LAD arises from the right or left coronary system, and takes a variable course and enters the distal part of the anterior interventricular septum and reaches up to the cardiac apex.^[8]

Spindola-Franco *et al.* classified dual LAD into four subtypes according to the origin and course of LAD on conventional angiography [Table 1].^[4]

Manchanda *et al.* described a novel variant of dual LAD, which they named as type V in which short LAD arises directly from the left coronary sinus and long LAD arises directly from the right coronary sinus.^[9]

Maroney and Klein described another variant of dual LAD in which short LAD arises from LMCA while long LAD arises from proximal RCA. Long LAD then courses between right ventricular outflow tract (RVOT) and aortic root on the epicardial surface and distally enters mid or distal anterior inter-ventricular septum (AIVS). It was named as type VI dual LAD.^[10] This is the malignant interarterial course of LAD and can result in sudden death due to compression of anomalous coronary artery between two vessels.^[11]

In our case, dual LAD was seen with short LAD arising from LMCA and long LAD from the pulmonary artery. This is an unusual case with a combination of dual LAD and ALCAPA. It does not fit into the classification system of dual LAD and has not been reported previously in the literature to our best knowledge. This can be considered another variant of dual

Table 1: Classification of dual LAD^[4]

Type	Origin		Course of long LAD
	Short LAD	Long LAD	
I	Proximal LAD	Proximal LAD	Descends epicardially on the left ventricular aspect of the anterior interventricular groove and re-enters the groove distally
II	Proximal LAD	Proximal LAD	Descends epicardially on the right ventricular aspect of the anterior interventricular groove and re-enters the groove distally
III	Proximal LAD	Proximal LAD	LAD runs intramyocardially in the IV septum proximally and then appears on the epicardial surface in the distal part of IV septum
IV	LMCA	Proximal RCA	Runs anterior to right ventricular outflow tract on the epicardial surface and then enters distal anterior interventricular groove

LAD: Left anterior descending, LMCA: Left main coronary artery

LAD and we propose to include this anomaly as type VII in the above-mentioned classification.

There are few case reports of anomalous origin of LAD from the pulmonary artery which is a rare coronary anomaly, but in none of these cases, dual LAD has been reported.

Anomalous origin of left coronary artery (LCA) from the pulmonary artery, also referred to as Bland-White-Garland syndrome, is a rare congenital cardiac anomaly with a reported incidence of 0.25-0.5% of all congenital cardiac anomalies.^[5] ALCAPA syndrome is classified into adult and infantile types depending on the tendency to develop collaterals between RCA and LCA. Patients with well-established collaterals are classified as “adult” type and those with no collaterals as “infantile” type.^[12,13] Due to reduced pressure in LCA originating from low-pressure pulmonary artery system, reversed coronary flow develops leading to myocardial ischemia. This left to right shunt is known as coronary steal phenomena. It is one of the most common causes of myocardial infarction and cardiac death in children.

Origin of LAD from the pulmonary artery has better prognosis as compared to anomalous origin of LMCA from the pulmonary artery because less myocardium is in jeopardy from retrograde myocardial perfusion.^[14] Depending upon the development of collaterals and pulmonary artery resistance, some patients remain asymptomatic until adulthood, as occurred in our case. Due to the presence of dual LAD and extensive collaterals in our case, patient remained asymptomatic till this age.

Treatment includes surgical repair. Surgery usually includes ligation of anomalous LAD at its pulmonary origin with re-implantation to aorta or by arterial grafts.^[15]

Takeuchi procedure is considered when direct coronary re-implantation is not feasible for treatment of ALCAPA.^[16]

Conventional angiography is the gold standard for diagnosis of coronary artery disease. But due to lack of 3D information, it provides limited information regarding the course and sometimes origin of anomalous coronary artery. Multi-slice CT allows the 3D analysis of coronary artery anatomy and is extremely useful to identify coronary artery anomalies and the anomalous course of artery which can induce myocardial ischemia and sudden death.^[13] Also, it is sometimes difficult to differentiate total occlusion of LAD from the anomalous origin of LAD as occurred in our case. Presence of extensive tortuous vessels gave suspicion of fistulous communication between the coronary artery and the pulmonary artery on conventional angiography in our case. But CT angiography helped in direct visualization of dual LAD with the origin of long LAD from the pulmonary artery (ALCAPA) and extensive interarterial collaterals. CT angiography is also useful to evaluate postoperative complications.

Conclusion

We conclude that coronary artery anomalies are rare. Both dual LAD and anomalous origin of left anterior descending artery from the pulmonary artery (ALCAPA) are very rare anomalies. But combination of dual LAD with ALCAPA is a very rare anomaly which has not been reported previously. Multi-slice CT provides accurate diagnosis of coronary artery anomalies regarding their origin and course with good anatomical detail non-invasively. Conventional angiography can sometimes be misleading due to lack of 3D orientation. CT angiography can be considered as the modality of choice in diagnosis of coronary artery anomalies. Recognition of these anomalies is important as they can lead to ventricular arrhythmias and sudden death in young patients.

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Conflicts of interest

There are no conflicts of interest.

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