Case Reports

A new anomaly of the left anterior descending artery: Type X dual LAD

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Abstract

Dual left anterior descending (LAD) coronary artery anomaly is traditionally classified as four types anomaly by classical coronary angiogram. Nowadays, coronary computed tomographic angiography (CCTA) allows clinicians to understand other variants of dual LAD anomaly. Up to date, 9 types of dual LAD variants detected from not only classical coronary angiogram but also CCTA imaging have been reported.

In the present case, we aimed to show a novel dual LAD anomaly, which is demonstrated by CCTA during preoperative evaluation and it has not been previously reported.

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1. Introduction

Dual left anterior descending (LAD) artery anomaly is a rarely observed congenital coronary artery anomaly. It may affect reperfusion strategy, especially in patients with congenital heart disease.1 In this congenital anomaly, there are two distinct segments of the vessel. It is feeding the anterior interventricular sulcus (AIS) of the heart and generally shows a short LAD terminating in the proximal AIS and a long LAD (which proximally courses outside the AIS), terminating in the distal AIS.

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Dual LAD anomaly is traditionally classified into four types based on classical coronary angiogram. However, coronary computed tomographic angiography (CCTA) imaging allows clinicians to notice other variants of dual LAD anomaly in current era. Uptil date, 9 types of dual LAD variants detected from not only classical coronary angiogram but also CCTA imaging have been reported.3-6

In the current case, we tried to present a novel case of dual LAD anomaly demonstrated by CCTA during preoperative evaluation which has not been previously reported.

2. Case

A 39-year-old woman presented with complaints of palpitation and dyspnea on exertion (NYHA Class-III). She had a history of acute rheumatic fever at the age of 12. During the physical examination, she was diaphoretic and tachyynepic. Her arterial blood pressure was 110/60 mmHg and heart rate was 90 ppm (irregular). On inspection, pectus excavatum deformity was observed. Cardiac auscultation revealed a loud first heart sound and an opening snap in early diastole followed by a holodiastolic decrescendo rumbling murmur and loud holosystolic murmur. Moreover, second pulmonary sound was found to be louder. Also, fine crackles were heard on basal segments of both lungs. Resting ECG revealed atrial fibrillation accompanied with bialtrial abnormality and mild right axis deviation. Transthoracic echocardiography showed moderate to severe mitral stenosis, severe mitral, and tricuspid insufficiency combined with moderate pulmonary hypertension. After administration of medications for her clinic condition, it was consulted with cardiothoracic surgeons regarding mitral valve replacement. Classical coronary angiography demonstrated that aberrant LAD was originating from the right coronary sinus (RCS) without evidence of epicardial coronary artery disease (Fig. 1a and b). It was thought that, this anomaly may cause surgical complication during sternotomy because of pectus excavatum deformity, and preoperative CCTA should be carried out.

Colored 3D volume rendered CCTA image showed both the long LAD and right coronary artery (RCA) originating from the RCS with different ostia on 320-row MDCT scanner (Toshiba Aquilion One, Toshiba Medical System, Japan) as shown in Fig. 2a. Short LAD originated from the left main coronary artery (LMCA) and terminated in the proximal AIS. However, it was noticed that long LAD originated from the RCS with separate ostium and followed an anomalous prepluralmonic course anterior to the right ventricle outflow tract (RVOT), and entered mid to distal AIS (Fig. 2b). Moreover, it was found that proximal part of long LAD coursed in very close proximity to the sternum due to excavatum deformity (Fig. 2c).

The patient underwent successful robotically assisted mechanical mitral valve replacement (St. Jude, 29 mm) and she was uneventfully discharged from hospital 5 days later.

3. Discussion

In healthy people, LAD originates from the LMCA, courses in the AIS towards the cardiac apex, and gives diagonal and septal branches. While septal branches extend to interventricular septum, diagonal branches extend to left ventricle (LV) anterior wall and sometimes to right ventricle (RV) anterior wall.1,2 Dual LAD is a rare congenital coronary anomaly traditionally classified into 4 types. In that anomaly, the functional LAD is divided into a short and a long segment.1,2 Although the short LAD typically arises from the LAD proper and terminates high in the interventricular groove,1,2 the long LAD takes a more variable course around the short segment and returns to the interventricular groove distally.1,2

In spite of the fact that four subtypes of dual LAD had initially been described by Spindola-Franco based on morphoanatomical features of the coronary arteries, five additional subtypes including new variant of type 7 were later published as shown in Table 1.2,5-6 Diagonal and septal branching patterns may be different among the dual LAD anomalies. In general, major septal branches arise from the short and proper LADs, while diagonal branches arise from the long and proper LADs.
Although current case looked like type IV dual LAD, RCA and long LAD originated from the RCS with different ostia. Short LAD originated from the LMCA and terminated in the proximal AIS. But it was noticed that long LAD originated from the RCS with separate ostium and followed an anomalous prepubmonic course anterior to the RVOT, and entered distal AIS (Fig. 1a and b).

In type VI dual LAD variant, long LAD originates from proximal RCS, follows an anomalous course between RVOT and aortic root, and enters distal AIS. In type VII dual LAD

| Table 1 – Morphologic features of dual LAD subtypes. |
|---|---|---|---|
| | 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 | LMCA | RCA | Proximal AIS | Prepubmonic 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 interarterial malignant course) |
| 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 | Inferior wall of the RV, turns around the apex and reaches to the distal AIS (*LMCA originates from the RCS and shows retroaortic course) |
| Type 9 | Proper LAD | Proper LAD | Mid AIS | LV side of the mid AIS, reenters the distal AIS, and terminates before reaching to the apex (*Posterior descending coronary artery extends distal AIS) |
| Type 10 (presented case) | LMCA | RCS | Proximal AIS | Prepubmonic 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.
variant, long LAD originates from LAD proper, courses on LV side of the proximal AIS, and reenters distal AIS. In the new variant of type VII dual LAD described by Saglam et al., long LAD originates from proximal RCS with a separate ostium like our case but it follows intramyocardial course within the septal crest emerging epicardially in the distal AIS.

It is essential to know presence of dual LAD before any cardiothoracic surgical intervention. Lack of this knowledge may result in coronary bypass graft surgery covering only one of those LADs, which in turn may cause wrong or deficient revascularization of the anterolateral wall, interventricular septum, or apex. On the other hand, conventional coronary angiography may not demonstrate entire coronary vasculature as three-dimensional, particularly in the identification of coronary vessels with aberrant origin. In fact, in cases with long LAD originating from the RCS, only short LAD can be visualized during angiography, which in turn can be easily misinterpreted as mid-LAD occlusion.

Familiarity with the variants of dual LAD is a critical step for avoiding incorrect placement of an arteriotomy and for revascularization of the correct vessel during surgery. If short and long LADs are severely stenosed, grafts to both the vessels may be needed in surgery because the major supply to the septum and the anterior left ventricular wall may come from the two separate vessels. More importantly, it was very 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 very close location of enlarged right ventricle to sternum as in the current case.

In conclusion, we demonstrated a novel type of dual LAD anomaly in the present case. It should be kept in mind that coronary anatomy should be extensively evaluated by required method for diagnosis and management of those patients.

Conflicts of interest

The authors have none to declare.

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