

Dual Left Anterior Descending Artery

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Abstract

We report a rare case of Type IV dual left anterior descending artery (LAD) in 65 year old male nondiabetic, nonhypertensive and nonsmoker, with effort dyspnea NYHA class II presenting to OPD of Dept of Cardiology, AIIMS, Bhubaneswar for presurgical evaluation. Dual left anterior descending artery constitutes 1% of all abnormal coronary artery morphogenesis described by Moretini [1] and Spindola-Franco et.al [2]. Ours case which is one of the six variants of Dual LAD i.e. Type IV was first described in 1939 by Waterson et.al [3] in Sir James Mackenzie who had this type of distribution. Concept of this rare anomaly should be there in the mind of interventional cardiologists and cardiac surgeons when this anomaly develops atherothrombosis and limiting angina and needs a balloon or graft landing. Single LAD usually supplies 54% of the ventricular myocardium in normal persons but in this rare anomaly the same job is carried out by two LADs.

Keywords: Dual; Coronary; Angiogram.

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Introduction

Dual LAD is a rare incidental finding in Cath lab. Although many times it harbors a benign course in rarest of its events it results in atheroinfarction in LAD territory and sudden cardiac death. Till now literature describes six variants of dual LAD where as four variants i.e. Type I to IV was described by Spindola-Franco et.al and Type V and Type VI was described by Machanda et.al [4] and Maroney J and Klein LW et.al [5] respectively. Type I to IV in some earlier series was described as A, B, C & D types respectively. This anomaly has some unique characteristics i.e. the proximal part is short and the distal part is long; usually the proximal or short LAD gives rise to main septal perforator (S₁) and main diagonal (D₁).

Type I: Short LAD gives off major septals and Long LAD runs on left ventricular site.

Type II: Short LAD same as Type I but Long LAD runs on right ventricular site.

Type III: Short LAD same in Type I but Long LAD runs intramyocardially in ventricular septum, neither on left side nor on right side.

Type IV: Short LAD very high in interventricular sulcus and long LAD arises from right coronary artery.

Type V: Short LAD arises from left coronary sinus and Long LAD arises separately from right coronary sinus, runs an intramyocardial course before reaching distal interventricular groove.

Type VI: Short LAD arises from left sinus and Long LAD arises from RCA, runs beneath RVOT in interventricular septum to reach interventricular sulcus. This variety is more prone to ischemic sudden cardiac death due to compression between aorta and pulmonary artery in conditions of increased pulmonary blood flow.

Recently a Type VII Dual LAD [6] has been described where long LAD runs a long intramyocardial septal course before emerging in interventricular groove. Ours case is a rare type of

Type IV LAD having long LAD arising from RCA whose proximal part was interarterial i.e. between aorta and pulmonary artery leading to obligatory ischemia.

Case

We report a case of 65 year old male nonsmoker, nondiabetic and nonhypertensive presenting to the OPD of Dept of Cardiology for presurgical evaluation of benign prostatic hypertrophy with effort dyspnea NYHA Class II and ECG revealed anterior wall ischemia. ECHO revealed no RWMA with fair LVEF and out of all routine blood chemistries TC was elevated to be 242mg/dl with LDL being 142mg/dl. In view of resting ST-T changes with effort dyspnea we subjected the patient to invasive coronary angiogram to delineate the coronary tree. Coronary angiogram revealed Left coronary artery arising from left coronary sinus dividing into a short (proximal) LAD and LCX. Short proximal LAD was giving rise to first septal and first diagonal and LCX revealed 20% focal occlusion in its proximal segment. RCA was originating from right coronary sinus and it was

giving rise to long distal LAD which was traversing to apex forming classical moustache sign.

We thought to delineate the cause of effort dyspnea in aforesaid patient and proceeded for 256 slice coronary CT angiogram under tailored heart rate modulation and gained the 3D CT reconstruction route of Dual LAD which revealed that long LAD which was originating from RCA was traversing being sandwiched between pulmonary artery and aorta in proximal part just after origin; whose compression in exertion may be the explanation of effort dyspnea in this patient. We treated the patient with antiplatelet, highest dose statin, beta blocker and nitrate with clearance for surgery under moderate perioperative CV risk with restriction of

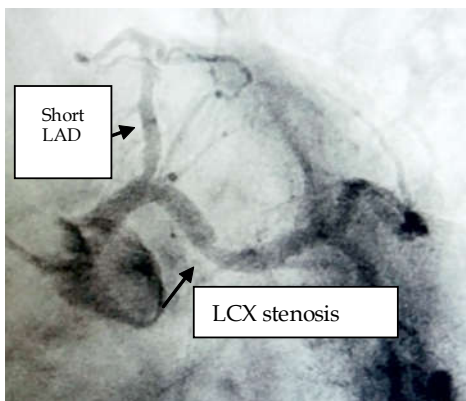


Fig. 1: Short LAD arising from LCA

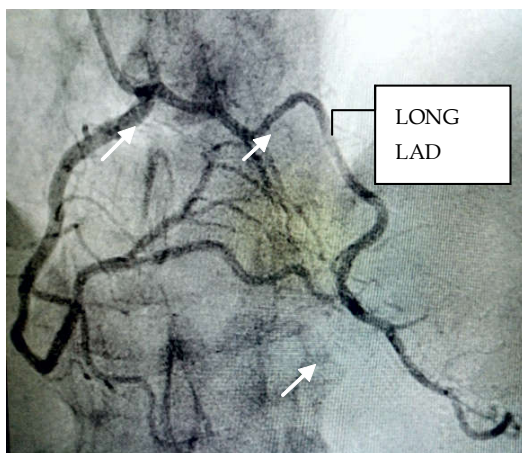


Fig. 2: Long LAD arising from RCA

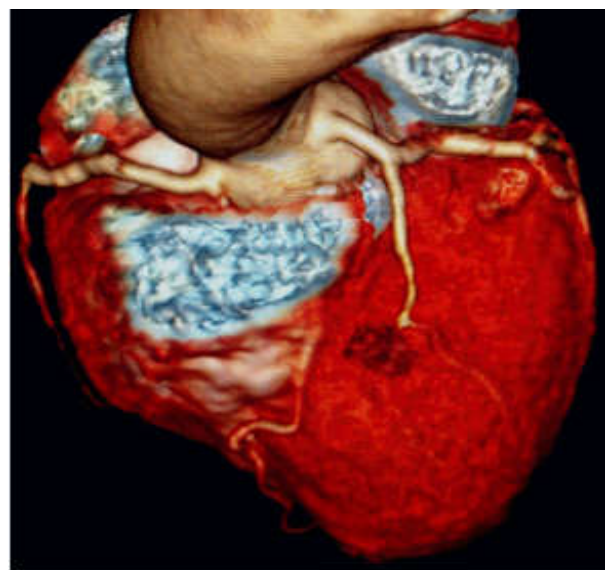


Fig. 3: Dual LAD (short and long) and long LAD arising from RCA in 256 slice CT coronary angiogram

strenuous exercise in future so that sandwiching compression of LAD will not occur.

Discussion

The incidence of Dual LAD although varies from 0.13-1% among normal hearts, in our cath lab we encountered it singly among 400 routine angiogram cases. It may be found in congenital heart diseases especially in Tetralogy of Fallot and Transposition of Great Arteries (TGA). Whereas Dual LAD increases the risk of atherosclerosis lacks ample of evidence; still it holds a clear consensus that by increasing the number of branches it predisposes to increased atherosclerosis as bifurcation and turbulence increase endothelial injury resulting in atherosclerosis. There exist case series reports with Dual LAD presenting with massive myocardial

necrosis when both the left anterior descending artery are atherothrombosed perpetually. Although they often run a benign course, on rarest of occasions they may land up in myocardial infarction or sudden cardiac death. In today's era CT angiogram plays a promising role in delineating the course of those anomalous arteries which is quite difficult to ascertain in conventional invasive coronary angiogram.

There is more concern when the long LAD gets compressed between RVOT and aorta in Type VI which may lead to sudden cardiac death [7]. Length disparity of both LADs is not a rule; Dual LAD can happen with equal length of both arteries. Ours case a rare case report of type IV Dual LAD (till now up to 10 cases described) without atheroma although significant plaque requiring revascularization in the form of PTCA or CABG has been described in literature.

It is difficult for the cardiac surgeon to trace the intramyocardial course or graft a short LAD. Cannulating those anomalous arteries sometimes become difficult especially with long LAD from separate ostia in right coronary sinus. Type VIII and Type IX variant of dual LAD also exists; in Type VIII, long LAD originates from acute marginal artery which is a branch of RCA and in Type IX long LAD originates itself from native LAD and terminates in mid interventricular septum. Type IX also called Triple LAD as you can find three arteries in IVS (Interventricular Septum) as also posterior descending artery in this anomaly curves around the apex and reaches IVS. More variation in Dual LAD is yet to be decorated in literature [8]. *The most important feedback this anomaly gives that whenever you would come across a very short LAD in left injection, always think and search for dual LAD.* For cardiac surgeons it is important to remember that whenever you are planning to anastomose short LAD in Dual LAD scenario always search in high interventricular septum. Baseline ECG changes has driven us to proceed for coronary angio in this case; if it would be harboring a type VI Dual LAD presurgical TMT before angio would have landed in catastrophe.

Conclusion

Dual left anterior descending artery is a rare anomaly where myocardial perfusion is blessed with two left anterior descending arteries; inspite it bears the risk of artherothrombosis, syncope and sudden cardiac death. Ours case dictates to search for dual LAD always when someone encounters very short LAD falling short even that of type II LAD in routine coronary angiogram.

Rare to encounter this type of anomalous coronary where duplicacy may be harboring a catastrophe.

References

1. Morretin L. Coronary arteriography: uncommon observations. *Radiol Clin North Am* 1976; 14(2):189-208.
2. Spindola-Franco H, Grose R, Solomon N. Dual left anterior descending artery: Angiographic description of important variants and surgical implications. *Am Heart J* 1983; 105(3):445-455.
3. Waterson D, Orr J, Capell DF. Sir James Mackenzie's heart. *Br Heart J* 1939; 1(3):237-248.
4. Machanda A, Qureshi A, Brofferio A et. al. Novel variant of dual left anterior descending coronary artery. *J Cardiovasc Comput Tomogr* 2010; 4(2):139-141.
5. Maroney J, Klein LW. Report of a new anomaly of the left anterior descending artery: type VI dual LAD. *Catheter Cardiovasc Interv* 2010; 80(4):626-629.
6. Lee Y, Lim YH, Shin J et.al. A case report of type VII dual left anterior descending coronary artery anomaly presenting with non ST segment elevation myocardial infarction. *BMC Cardiovasc Disord* 2012; 12:101.
7. Angelini P. Coronary artery anomalies an entity in search of an identity: *Circulation* 2007; 115(10):1296-1305.
8. Abbasov E, Manafov S, Abdullayev F. Dual LAD-Contemporary Review. *J Cardiol Curr Res* 2015; 3(2):95.