CASE REPORT

Hyper-dominant left anterior descending coronary artery with continuation as a posterior descending artery—An extended empire

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Hyper-dominant left anterior descending artery (LAD) is a rare coronary anomaly where LAD continues as a posterior descending artery. It is a rare coronary anomaly and there are only 19 cases reported so far in 17 case reports in the literature. Its involvement during acute coronary syndrome can be fatal as it leads to ischemia/infarction of a larger area of left and/or right ventricular myocardium. Its early recognition and management is essential with a high index of clinical suspicion.

Keywords: Congenital coronary anomalies, Hyper-dominant left anterior descending artery, Primary percutaneous coronary intervention

Introduction

Coronary artery anomalies are rare anatomical variations of their origin, course, and supply associated with or without other congenital abnormalities. Overall, they are observed in ~1–2% of the general population in those who undergo conventional or computed tomographic coronary angiography, as reported in a different series [1]. Most of the coronary anomalies are incidental findings, as in our case during conventional coronary angiography and interventions.

We report a case of a patient who presented with acute anterior wall myocardial infarction, underwent primary angioplasty of the left anterior descending artery (LAD), which continued as a posterior descending artery (PDA) in the posterior interventricular groove upon the restoration of Thrombolysis in Myocardial Infarction (TIMI) 3 flow. This is a rare coronary anomaly with only 19 cases reported so far in 17 case reports in the

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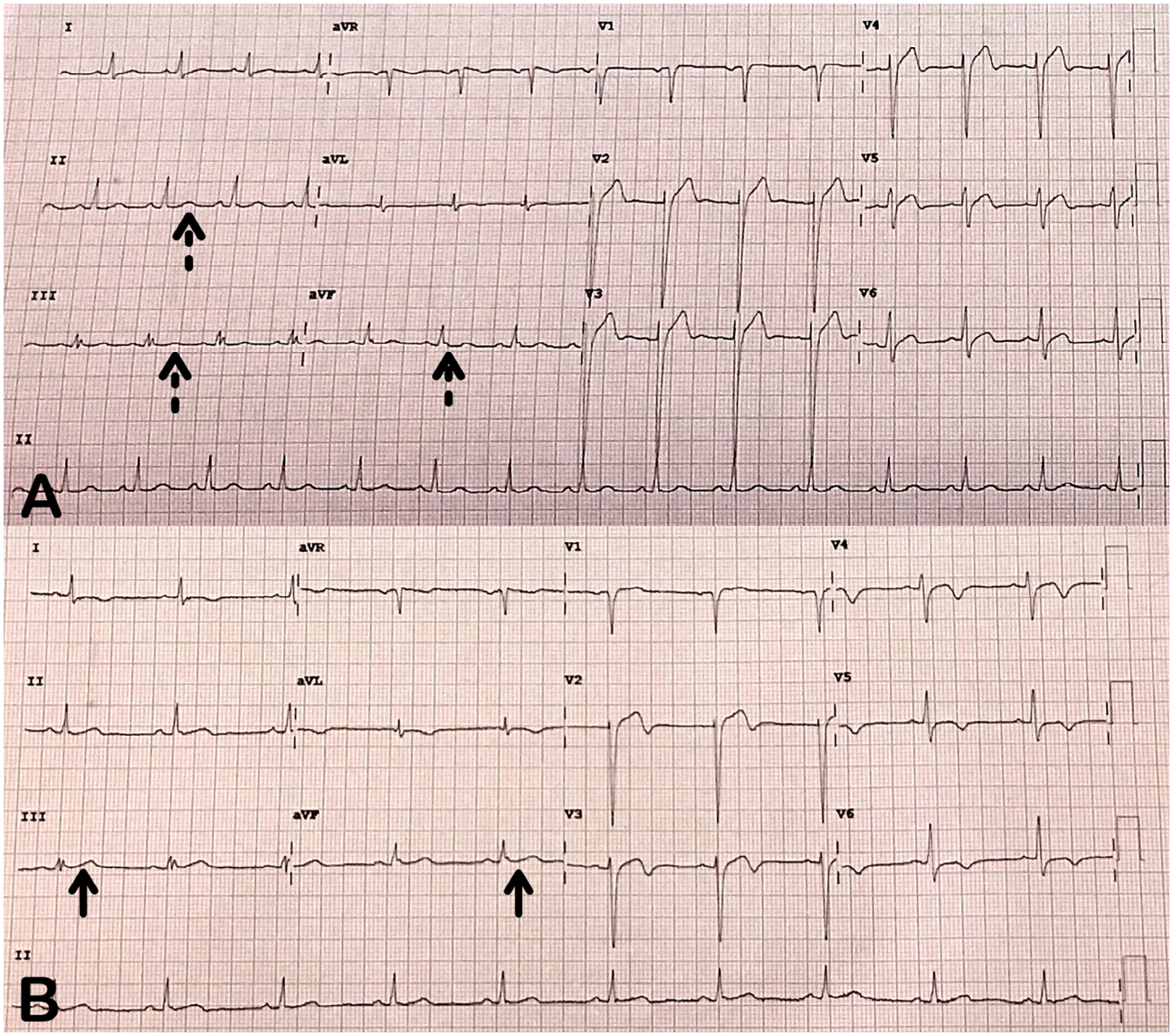

Figure 1. (A) Electrocardiography showing ST-segment elevation in leads V1–V6 with absence of the reciprocal ST-segment depression (Dashed black arrows) of inferior leads (II, III, aVF). (B) Post PCI of the LAD, Electrocardiography showed discrete ST-segment elevation in leads V1–V6 with T-wave inversions with absence of the reciprocal changes (Solid black arrows) in inferior leads (II, III, aVF). LAD = left anterior descending; PCI = percutaneous coronary intervention.

literature of the anomalous origin of the branches of the right coronary artery, mainly of the posterior descending artery from the left anterior descending artery or its branches.

Case report

A 56-year-old male, hypertensive, presented with complaints of retrosternal chest discomfort for 8 hours. Electrocardiography (ECG) showed ST-segment elevation in leads V1-V6, with the conspicuous absence of the reciprocal changes in II, III, and aVF (Fig. 1A). Echocardiography showed hypokinesia of anterior and inferior wall (Ejection Fraction, 42%). Cardiac markers were elevated [high sensitivity (HS)-troponin I, 302 ng/L; normal range, 8–28 ng/L]. Coronary angiography (CAG) revealed 99% stenosis of the proximal segment of the LAD with TIMI I flow (Fig. 2A). The patient underwent primary percutaneous coronary intervention (PCI) of LAD using 3.0 × 38 m m drug-eluting stent (DES) and final CAG, upon restoration of the flow, showed the hyper-dominant LAD wrapping around left ventricular apex, did not show any tapering and ran along the posterior interventricular groove as a posterior descending artery (PDA) up to the crux (Fig. 2B). The right coronary artery (RCA) was nondomi-

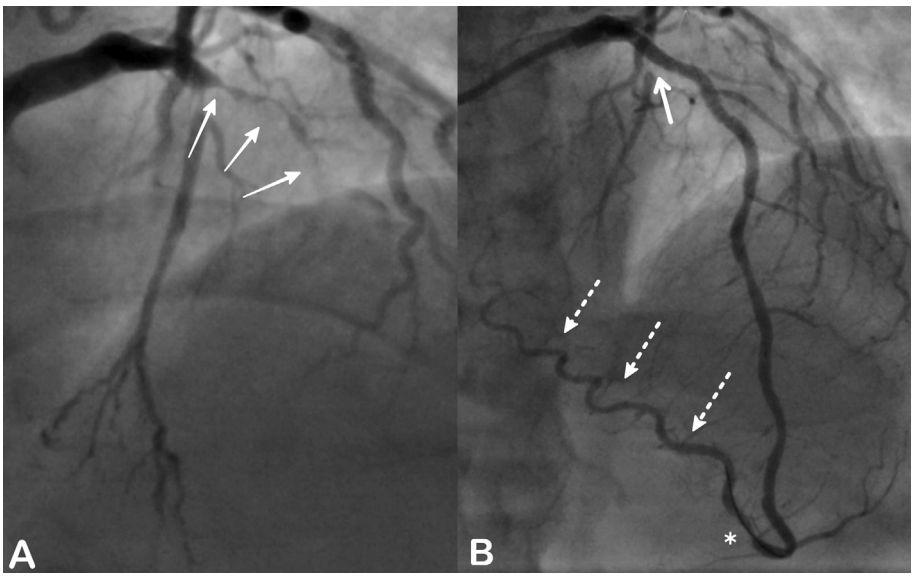

Figure 2. (A) CAG revealed critical stenosis of the proximal LAD with TIMI I flow (solid arrows). (B) After PCI and the deployment of the DES leads to Thrombolysis in Myocardial Infarction (TIMI) III flow of the LAD. Final CAG revealed the entire LAD, which continued as the PDA along the posterior interventricular septum, reached up to the crux (dashed arrows). There was significant 80% stenosis of the proximal segment of the PDA (asterisk) after its continuation with the LAD near the Left Ventricular apex. CAG = coronary angiography; DES = drug-eluting stent; LAD = left anterior descending; PCI = percutaneous coronary intervention; PDA = posterior descending artery.

nant and small, diminutive without any communication with the PDA or overlap of their course. Post PCI, ECG showed T wave inversions and reduction of the ST segment elevations and voltages of the S wave in the V1–V6; I, aVL with mild subtle ST–segment elevations with upward concavity of inferior leads (Fig. 1B). The patient was discharged in a stable condition with the advice of optimal medical management in the form of the dual antiplatelet agent, statin, beta-blocker, and angiotensin converting enzyme (ACE) inhibitor.

The patient did well during follow up without any further symptoms.

Discussion

The origin of PDA from RCA, Left circumflex (LCX) artery, or either decides the dominance of the coronary circulation. Type III LAD is a common finding but its continuation as a PDA is described, as ''hyper-dominant LAD'', ''super-dominant LAD'', or ''type IV LAD'' [2–4]. It can also be called as anomalous origin of the PDA from the LAD. Though it is an incidental finding during coronary angiography its clinical implication is that its involvement during acute coronary syndrome leads to a large left ventricular ischemia or infarction of both anterior and inferior territories (>50%). The overall subtle changes in the ECG may be due to cancellation of electrical forces by an infarction in another area. This may present as a cardiogenic shock and may lead to other mechanical complications of acute myocardial infarction-like ventricular septal rupture, free wall rupture, etc. [2,4].

This coronary anomaly should not be confused with coronary collateral or intercoronary anastomosis, as they are distinctively different entities [5]. Basically, congenital anomalies are defects in the embryogenesis leading to various anatomical variations in their origin, course, and supply with varied prognosis [1]. The interventional cardiologist, cardiac surgeons and physicians should know these variations as they pose difficulties during the percutaneous coronary intervention and coronary artery bypass grafting.

We searched the literature for similar coronary anomalies and found 17 such case reports with 19 cases so far, as Clark et al. [6] reported a cluster of three cases (Table 1).

In this spectrum of coronary anomalies, there is an abnormal origin of the PDA and/or other terminal branches of the RCA from the LAD or its branches like first septal perforator [7].

It either reaches up to the crux or crosses beyond it and may or may not run into the anterior atrio-ventricular groove, partially parallel to the native RCA [8]. Terminally, it may supply the left atrium or left ventricle or may divide into the RCA branches [3,9].

The clinical suspicion should arise in cases with severe hemodynamic compromise in the presence of minor ECG changes, absence of signs of

Table 1. List of the cases of the origin of the posterior descending artery from the left anterior descending artery either as a direct continuation or its origin from the branches of LAD.

Serial no.	Case no.	Author	Yr	Age/sex	Clinical presentation with ECG changes	Treatment & outcome	Unique features
1	1	Baroldi et al. [10]	1967	–	Autopsy study	-	1st case reported in the literature
2	2	Clark et al. [6]	1985	63/male	Old MI with flattening of T wave in infero-lateral leads. Positive TMT with ST-segment depressions in anterior and inferior leads.	CABG with uneventful postoperative course.	Though uncommon, it is the only reported cluster of 3 cases with this anomaly
	3			77/male	Post-PPI with first-degree AV block, left anterior hemi block, & increased R wave amplitude in the anterior precordial leads. Old posterior wall MI without reciprocal changes	CABG with uneventful postoperative course.	
	4			74/ female	Dilated cardiomyopathy. Left ventricular hypertrophy with nonspecific ST-T changes.	Stabilized with medical management in the form of diuretics & ACE inhibitor.	
3	5	Musselman et al. [18]	1992	54/male	STEMI – ST-segment elevation with q waves & T wave inversion in inferior leads.	CABG with uneventful postoperative course.	At the crux, the PDA bifurcated giving rise to branches traversing both AV grooves. The branch to the left AV groove in turn gave rise to postero-lateral branches.
4	6	Singh et al. [1]	1994	40/male	Atypical chest pain, nonspecific ST-T changes in infero-lateral leads.	Medical management, outcome not described.	LAD continued as a PDA, which did not give any branches including septal perforators.
5	7	John [11]	2002	54/male	Unstable angina. ECG changes not described.	CABG. Outcome not described.	Aberrant vessel passed anterior to the root of the main pulmonary artery & the right ventricle to reach the acute margin of the heart before passing onto the inferior surface and terminating as the main PDA.
6	8	Hamodraka et al. [12]	2005	44/ female	Unstable angina. ECG changes not described.	Medical management. Outcome not described.	LAD continued as a PDA along the posterior interventricular septum.
7	9	Javangula et al. [2]	2007	61/male	Exertional angina. Left ventricular hypertrophy with strain pattern.	Patient had associated moderate aortic stenosis with triple vessel disease, underwent CABG with AVR with uneventful postoperative course.	PDA gave left ventricular branch to the inferior surface of left ventricle and thereafter continued as the distal RCA without establishing any communication with the atretic proximal RCA.
8	10	Tehrai et al. [9]	2011	50/ female	Chest pain for evaluation. ECG changes not described.	Medical Management. Outcome not described.	CT coronary angiography did not show any significant lesion. PDA terminally gave rise to two branches to the left ventricle.

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Table 1 (*continued*)

Serial no.	Case no.	Author	Yr	Age/sex	Clinical presentation with ECG changes	Treatment & outcome	Unique features
9	11	Kim et al. [13]	2011	67/male	STEMI – ST- segment elevation myocardial infarction involving leads V1—4, II, III, & aVF (AWMI + IWMI)	Primary PTCA with uneventful post-procedure course.	In addition origin of PDA as a continuation of LAD, RCA originated from the proximal segment of the LAD.
10	12	Patra et al. [14]	2013	65/male	New onset effort angina with positive TMT ECG normal at rest.	Patient refused treatment.	LAD artery had a large first septal branch which divides into large posterior left ventricular branch (PLV)) & a small PDA.
11	13	Roy et al. [15]	2013	41/male	STEMI – ST-segment elevation in leads II, III, AVF, V5, & V6 with reciprocal ST depression in lead AVL (IWMI + LWMI)	PTCA. Asymptomatic for >2 y follow up.	LAD continued as a PDA, which supplied the inferior septum and the inferior wall. Distal LAD at the apex had 75% narrowing with a thrombus.
12	14	Mannuva et al. [8]	2013	66/male	STEMI – ST-segment elevations in leads V1-V4, II, III & aVF (AWMI + IWMI)	Primary angioplasty. Discharged postprocedure on Day 5.	Patient had cardiogenic shock. LAD continued as the PDA beyond the crux into the left posterior atrioventricular groove with a small RCA.
13	15	Uçar et al. [7]	2013	43/ female	Unstable angina. Nonspecific ST-segment & T-wave abnormalities.	Medical management. Discharged & prescribed with beta blocker to relieve symptoms.	1st septal continued as a PDA crossing the interventricular septum into the posterior interventricular groove as a PDA.
14	16	Ramesh Babu et al. [3]	2015	22/male	Chest pain for evaluation, ECG changes not described.	Medical management, outcome not described.	CT coronary angiography did not show any significant lesion of coronaries. LAD continued as a PDA up to the crux of the heart. LAD had anomalous left atrial branch
15	17	Khan et al. [16]	2016	66/male	NSTEMI-deep T-wave inversions in the precordial leads.	FFR 0.90, Medical management. Uneventful postprocedure course.	70% lesion in the mid segment of LAD which continued as a PDA.
16	18	Udupa et al. [4]	2016	56/ female	STEMI – ST-segment elevations in anterior leads (AWMI), Cardiogenic shock.	Primary PTCA, Outcome not described.	After predilatation of the proximal 99% stenosis of the LAD, authors could visualize continuation of the LAD as a PDA as in our case.
17	19	Dubey et al. [17]	2016	51/male	STEMI – ST-segment elevations in leads V1–V4 (ASMI).	Primary PTCA, uneventful postprocedure course.	After stenting of the 100% occluded LAD, authors could visualize continuation of LAD as a PDA as in our case. The patient had cardiogenic shock.

ACE = angiotensin converting enzyme; AV = atrio-ventricular; ASMI = antero-septal myocardial infarction; AVR = aortic valve replacement; AWMI = anterior wall myocardial infarction; CABG = coronary artery bypass grafting; CT = computed tomography; ECG = electrocardiogram; FFR = fractional flow reserve; IWMI = inferior wall myocardial infarction; LAD = left anterior descending; MI = myocardial infarction; NSTEMI = non-ST-segment elevation myocardial infarction; PDA = posterior descending artery; PPI = permanent pacemaker implantation; PTCA = percutaneous transluminal coronary angioplasty; RCA = right coronary artery; STEMI = ST-segment elevation myocardial infarction; TMT = treadmill test.

prior (old) myocardial infarction but poor left ventricular function involving anterior and inferior walls.

Conclusion

Hyper-dominant LAD or anomalous origin of PDA from RCA is a single epicardial vessel supplying both the territories of the LAD and the PDA. Our case was one of the rare cases where the PDA arises from the LAD making it hyper-dominant, and reported among the 19 cases in 17 case reports in the world literature so far.

The early intervention can be lifesaving with a high degree of suspicion particularly when there is a combined ST elevation of anterior and inferior segments, conspicuous absence of reciprocal changes in inferior leads with ST segment elevation of anterior leads or combined ischemia of both, anterior and inferior territories during the stress test. The shift of the dominance entirely to the LAD territory from either RCA or LCX can alter the clinical presentation. Our case report and review of literature will help in its early diagnosis and planning better management strategies.

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