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

Pankaj Jariwala a,*, Edla Arjun Padma Kumar a

a Department of Cardiology, Maxcure-Mediciti Hospitals, Hyderabad, Telangana

a India

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.

© 2018 The Authors. Production and hosting by Elsevier B.V. on behalf of King Saud University. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

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...
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 mm 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 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.
nant and small, diminutive without any communi-
cation with the PDA or overlap of their course.
Post PCI, ECG showed T wave inversions and
reduction of the ST segment elevations and volt-
ages of the S wave in the V1–V6; I, aVL with mild
subtle ST–segment elevations with upward con-
cavity 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 com-
mon 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 implica-
tion is that its involvement during acute coronary
syndrome leads to a large left ventricular ischemia
or infarction of both anterior and inferior territo-
ries (>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 anasto-
mosis, 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 cardiolo-
gist, cardiac surgeons and physicians should know
these variations as they pose difficulties during
the percutaneous coronary intervention and coro-
nary 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 ter-
inal 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 ante-
ier 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. Though uncommon, it is the only reported cluster of 3 cases with this anomaly. |
| 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. |                  |
| 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. STEMI – ST-segment elevation with q waves & T wave inversion in inferior leads. | Stabilized with medical management in the form of diuretics & ACE inhibitor. |                  |
| 5         | 5        | Musselman et al. [18] | 1992 | 54/male | Unstable angina. ECG changes not described. | CABG with uneventful postoperative course. |                  |
| 6         | 6        | Singh et al. [1]    | 1994 | 40/male | Atypical chest pain, nonspecific ST-T changes in infero-lateral leads. | Medical management, outcome not described. |                  |
| 7         | 7        | John [11]           | 2002 | 54/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. |                  |
| 8         | 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. 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 atritic proximal RCA. CT coronary angiography did not show any significant lesion. PDA terminally gave rise to two branches to the left ventricle. |
| 9         | 9        | Javangula et al. [2] | 2007 | 61/male | Chest pain for evaluation. ECG changes not described. | Medical Management. Outcome not described. |                  |
| 10        | 10       | Tehrai et al. [9]   | 2011 | 50/female |                        |                  |                  |

(continued on next page)
Table 1 (continued)

| Serial no. | Case no. | Author(s) | 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. LAD artery had a large first septal branch which divides into large posterior left ventricular branch (PLV) & a small PDA. |
| 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. 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. | Patient had cardiogenic shock. LAD continued as the PDA beyond the crux into the left posterior atrioventricular groove with a small RCA. |
| 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.

References

[1] Singh SP, Soto B, Nath H. Anomalous origin of posterior descending artery from left anterior descending artery with unusual intraseptal course. J Thorac Imaging 1994;9:255–7.
[2] Javangula K, Kaul P. Hyperdominant left anterior descending artery continuing across left ventricular apex as posterior descending artery coexistent with aortic stenosis. J Cardiothorac Surg 2007;2:42.
[3] Ramesh Babu CS, Khare S, Asthana AK, Saxena S, Gupta OP. Posterior descending artery arising as a continuation of hyperdominant left anterior descending artery. Int J Anatomy Radiol Surg 2015;4:16–9.
[4] Udupa A, Goyal BK, Pagad S. Hyperdominant left anterior descending artery (LAD): a rare coronary anomaly. Indian Heart J 2016;68:S151–2.
[5] Padma Kumar EA, Jariwala P. Intercoronary communication or anastomosis?—A collateral without obstructive coronary artery disease. IHJ Cardiovasc Case Reports 2017;1:37–8.
[6] Clark VL, Brymer JF, Lakier JB. Posterior descending artery origin from the left anterior descending: an unusual coronary artery variant. Cath Cardiovasc Diagn 1985;11:167–71.
[7] Uçar FM, Gül M, Ötkent RS, Topaloğlu S, Gücük E. A rare coronary artery anomaly: posterior descending artery arising from septal perforator artery. Turk Kardiyol Dern Ars 2013;41:668.
[8] Mannuva BB, Durgaprasad RVV. Hyperdominant left anterior descending artery continuing as posterior descending artery. Cath Lab Dig 2013;21(1):2. https://www.cathlabdigest.com/articles/Hyperdominant-Left-Anterior-Descending-Artery-Continuing-Posterior-Descending-Artery-Rare-C.
[9] Tehrani M, Saidi B, Goodarzi M, Baharjoo H, Roshanali F, Davoodi M. Anomalous origin of posterior descending artery from left anterior descending in the presence of a diminutive right coronary artery: diagnosed by ECG gated multi-detector CT. Heart Lung Circ 2011;20:734–5.
[10] Baroldi GSG. Coronary circulation in the normal and pathological heart. 2nd ed. Washington, D.C.: Office of the Surgeon General, Department of the Army; 1967, p. 10–13.
[11] John LCH. Anomalous origin of the posterior descending artery from the left anterior descending coronary artery: cardiac surgeons beware. Heart 2002;161.
[12] Hamodraka ES, Paravolidakis K, Apostolou T. Posterior descending artery as a continuity from the left anterior descending artery. J Invasive Cardiol 2005;17:343.
[13] Kim JH, Cha KS, Park SY, Park TH, Kim MH, Kim YD. Anomalous origins of the right and posterior descending coronary arteries from the left anterior descending coronary artery: unusual pattern of the single coronary artery. J Cardiol Cases 2011;3:26–8.
[14] Patra S, Srinivasa BC, Agrawal N, Manjunath CN. Super dominant left anterior descending artery with the origin of both posterior descending artery and posterior left ventricular artery from the septal branch. BMJ Case Rep 2013;2013:1–3. https://doi.org/10.1136/bcr-2013-010303.
[15] Roy STN, Nagham JS, Anil Kumar R. Acute inferior wall myocardial infarction due to occlusion of the wrapped left anterior descending coronary artery. Case Rep Cardiol 2013;2013:983943.
[16] Khan HS, Iftikhar I, Kayani AM, Gul U. Hyperdominant left anterior descending artery (LAD): a rare coronary anomaly. J Coll Physicians Surg Pak 2016;26:52–3.
[17] Dubey L, Adhikari R, Kc PJ, Panjiyar R, Gurung TB, Subramanyam G. Primary angioplasty of a super dominant left anterior descending coronary artery. J Coll Med Sci-Nepal 2016;12(2):81–2.
[18] Musselman DR, Tate DA. Left coronary dominance due to direct continuation of the left anterior descending to form the posterior descending coronary artery. Chest 1992;102:319–20. Available from: http://linkinghub.elsevier.com/retrieve/pii/S0012369216359013.