

Extremely Hyperdominant LAD Supplying the Anterior, Inferior, and Lateral Walls of the Left Ventricle

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ABSTRACT: Hyperdominant left anterior descending artery (LAD) is a rare anomaly in which the LAD gives rise to the posterior descending artery. Our case report describes an extreme case of hyperdominant LAD supplying the anterior, inferior, and partially the lateral walls of the left ventricle. It is crucial that physicians be aware of the possibility of extreme LAD dominance. A proximal occlusion of such a vessel might have catastrophic consequences with atypical presentation on electrocardiogram.

CASE PRESENTATION

A 45-year-old male smoker presented to an outside facility after acute onset of substernal chest pain. His past medical history included essential hypertension and diabetes mellitus type 2, with family history being significant for coronary artery bypass grafting (CABG) due to acute myocardial infarction (AMI) in his mother, who was in her 40s at the time. His chest pain, which radiated to both shoulders and to the jaw, was pressure-like in character, 7/10 in intensity, exertional, and associated with diaphoresis and shortness of breath. It lasted for a total of 30 minutes. He described having similar episodes of chest pain on exertion a few times in the past, which would last only a few seconds.

On physical examination, the patient had a body mass index of 36, regular heart rate of 70 beats per minute, and blood pressure of 159/103 mm Hg. On auscultation, S1 and S2 were normal, there was no appreciable murmur, and lung sounds were clear. No jugular venous distension or lower extremity edema were present. A chest radiogram was within normal limits, and the electrocardiogram (EKG) was unremarkable with no signs of acute ischemia. Laboratory studies showed initial troponin elevation of 1.89 ng/mL, with peak troponin at 7.019 ng/mL after 6 hours. A coronary angiogram revealed significant stenosis of obtuse marginal-2 artery.

On the basis of his symptoms and elevated troponin, the patient was diagnosed with an acute coronary syndrome, and appropriate guideline-directed medical therapy of non-ST-elevation myocardial infarction was started with dual antiplatelet therapy, beta blocker, ACE inhibitor, and statin. The patient underwent percutaneous coronary intervention (PCI) with placement of two drug-eluting stents to the obtuse marginal-2 artery. The right coronary artery (RCA) had chronic

total occlusion (CTO) with left-to-right collaterals from the left anterior descending artery (LAD). After PCI, the patient was referred to our center for RCA-CTO intervention.

In our facility, detailed review of angiographic images revealed an extreme form of LAD dominance, with a large-caliber LAD wrapping around the apex and continuing in the posterior interventricular groove as the posterior descending artery (PDA). After reaching the crux, the vessel made a 180° turn and continued in apical direction as the posterolateral artery (PLA), traveling through the whole longitudinal dimension of the left ventricle (LV), reaching the apex (Figures 1, 2). In light of the very small, nondominant RCA (Figure 3) and medium-sized left circumflex artery (LCX), it appeared that the overwhelming majority of the inferior wall was supplied by the LAD. Confirming these findings, a computed tomography coronary angiogram showed the large LAD supplying nearly the whole inferior wall of the LV, giving rise to the PDA and PLA, although the contrast material was unable to reach the distal PLA for adequate visualization (Figure 4).

DISCUSSION

Coronary artery anatomy shows a high level of interindividual variability, ensuring that no two coronary artery anatomic patterns are exactly alike. If a specific anatomical variation is present in more than 1% of the population, it is considered a variant rather than a congenital anomaly. According to this 1% rule, most variations involving take-off and course of coronary arteries, source of blood supply to sinoatrial and atrioventricular nodes, and the presence of separate conus branch or ramus intermedius are considered normal.¹ A notable area with considerable variation is the blood supply of the inferior and posterior myocardium, which determines coronary dominance.

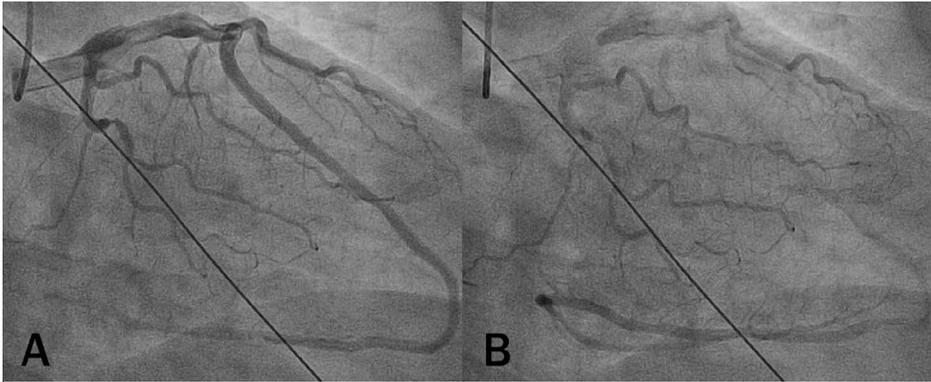


Figure 1.

Right anterior oblique (A) caudal and (B) distal views showing gradual opacification of left anterior descending artery as it turns around the apex, continues in the posterior interventricular groove, then makes another 180° turn at the crux to continue back to the apex.

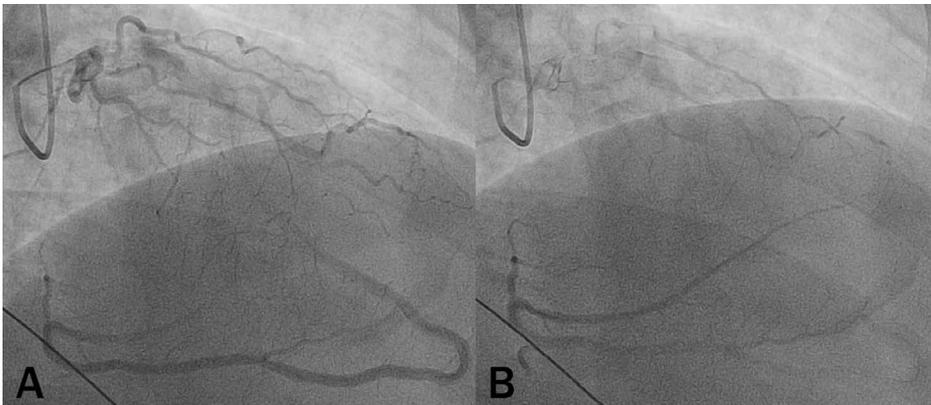


Figure 2.

Right anterior oblique (A) proximal and (B) distal cranial images allowing better visualization of distal left anterior descending artery running in the posterior interventricular groove, then turning around at the crux to continue back to the apex.

Coronary artery anomalies represent marked deviations from the normal pattern and are present in < 1% of the population.^{1,2} Although usually asymptomatic and discovered incidentally during coronary angiography, they have gathered considerable interest in recent years because they represent the second most common cause of sudden cardiac death in young competitive athletes.¹⁻³ Clinically, coronary artery anomalies are classified as either benign or

malignant depending on their potential to cause myocardial ischemia. Even in the case of anomalies generally considered benign, knowledge of a coronary anomaly in a particular patient facilitates rapid, accurate assessment via coronary angiography in emergency situations and helps in planning suitable follow-up and treatment. This gives the angiographer key information to perform accurate evaluations and avoid errors in management. For patients undergoing open heart surgery, awareness of such

anomalies helps the surgeon avoid life-threatening complications from accidental transection or ligation of an anomalous coronary artery.¹

Hyperdominant/superdominant LAD is a rare coronary anomaly in which the PDA arises from the LAD instead of the LCX or RCA. Multiple cases of superdominant LAD have been published in the past. For instance, Clark et al. and Baroldi and Scomazzoni described patients with an LAD that formed the PDA and terminated at or before the crux.^{5,6} Musselman et al. described AMI in a patient whose LAD wrapped around the apex of the LV and formed the entire PDA, then at the crux gave rise to branches traversing both atrioventricular grooves.⁷ Shakhil Sattar et al. described a case of hyperdominant LAD that continued as PDA beyond the crux into the posterior interventricular sulcus.⁸ To our knowledge, we report the first case of an extreme level of LAD-dominant circulation, with the LAD not only giving rise to the PDA but making a 180° turn at the crux and continuing apically as the PLA. In this sense, the LAD was truly hyperdominant, supplying the anterior wall, the overwhelming majority of the inferior wall, and even significant areas of the lateral wall via a large diagonal branch.

In our opinion, a proximal lesion of an extremely dominant LAD should be viewed as the equivalent of at least a two-vessel disease with a proximal LAD lesion, warranting evaluation by the heart team and consideration of coronary artery bypass grafting (CABG). If the patient has a prohibitively high risk for surgical revascularization, protected PCI may be feasible. Of note, in case of AMI in a patient like the one described in our report, the EKG may not show the usual pattern of anatomically localized ST elevations with reciprocal ST depressions but instead may show diffuse ST elevations without any reciprocal changes, superficially resembling pericarditis.

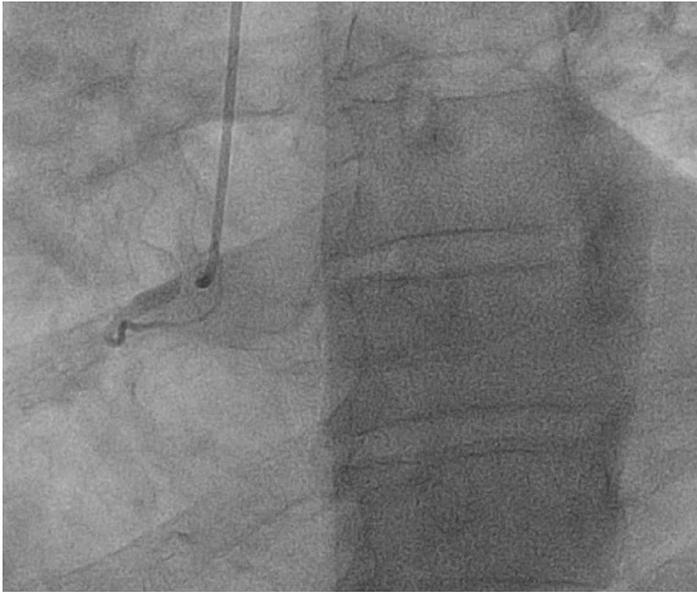


Figure 3.
Right coronary artery (RCA) in left anterior oblique (LAO) caudal plane; only rudimentary RCA is visualized in the LAO plane.



Figure 4.
Three-dimensional reconstruction of left anterior descending artery (LAD) on cardiac computed tomography angiography, which allows us to better appreciate the spatial orientation of the vessel. Unfortunately, the long course of the LAD did not allow adequate contrast passage to the most distal parts of the vessel.

It is crucial that physicians be aware of the possibility of extreme LAD dominance. A proximal occlusion of such a vessel may have catastrophic consequences such as rapid development of cardiogenic shock requiring immediate intervention. Internal medicine physicians, family medicine practitioners, cardiologists, and surgeons should be aware of the possibility of such a rare anomaly since rapid recognition of an atypical presentation of an acute coronary syndrome might significantly improve the clinical outcome.

CONCLUSION

Our report is, to our knowledge, the first described case of this extreme level of LAD dominance, with the LAD giving rise not only to the PDA but also to a full-length PLA. A proximal occlusion of such a vessel may have catastrophic consequences and might require emergent CABG or high-risk PCI. Physicians need to be aware that in this clinical scenario, the EKG may not show the usual pattern of localized ST elevations with reciprocal ST depressions but instead may show more diffuse ST elevations, superficially resembling pericarditis.

Conflict of Interest Disclosure:

The authors have completed and submitted the *Methodist DeBakey Cardiovascular Journal* Conflict of Interest Statement and none were reported.

Keywords:

hyperdominant LAD, superdominant LAD, coronary artery anomaly, acute coronary syndrome

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