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Anomalous Origin of the Posterior Descending Artery Causing Inferolateral Myocardial Ischemia in an Octogenarian

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Coronary artery anomalies can affect the origin, path, and supply of the vessels either alone or in combination with other inherent defects, with a prevalence of less than 1.5%. [1] Normally, the posterior descending artery (PDA) arises from the right coronary artery (in a right dominance or co-dominance pattern) or the left circumflex artery (in a left dominance pattern). [2] Rarely, the posteroinferior septum is supplied by a hyperdominant left anterior descending artery (LAD), continuing as the posterior descending artery (PDA). [3] The unique feature of this report is that an octogenarian sustained an inferolateral ST-segment elevation myocardial infarction (STEMI) that was not due to an occlusion of the left circumflex artery, but due to a lesion in a hyperdominant LAD continuing as the PDA. The patient underwent a successful percutaneous intervention

An 81-year-old woman presented to a tertiary care unit in Oman with a history of exertional angina, chest pain, and dyspnea of one week's duration in 2023. She was known to have diabetes and hypertension and was receiving medical treatment for these conditions. She mentioned that the chest pain was central and was not radiating. However, at the time of indexed admission, she was dyspneic and hypertensive [systolic/diastolic: 200/80 mmHg] but had no angina. The electrocardiogram showed 'ST' segment elevation with 'q' waves in the limb, augmented vector,

and chest leads [LII, III, aVF, and V4-6] [Figure 1A]. High-sensitivity cardiac troponin (hs-cTn) was 30 pg/mL. The patient presented late with a missed inferior lateral ST-elevation myocardial infarction. The transthoracic echocardiographic assessment revealed a normal left ventricle size with preserved systolic function (left ventricular ejection fraction: approximately 45-50%). There was evidence of Grade II diastolic dysfunction, indicated by an E/A ratio of 0.5 and an E/E' value of 15. An aneurysmal apex, characterized by an akinetic apical cap, was also observed. The right ventricle appeared normal in size with preserved systolic function, as demonstrated by a tricuspid annular plane systolic excursion of 20 mm and an S' velocity of 10 cm/s. Trace tricuspid regurgitation was noted, but no signs of pulmonary hypertension were present, with a right ventricular systolic pressure estimated at approximately 25 mmHg. An urgent coronary angiogram revealed that the right coronary artery was diminutive in size. The left main coronary artery appeared normal. The proximal segment of the LAD was normal. The LAD wrapped around the apex and continued as the PDA with no lesion [Figure 2A,B,C and Supplementary Videoclips 1,2,3]. In the LAD, there was a 70-80% stenosis after the first diagonal branch and a 30-40% stenosis just before the third diagonal branch. The third diagonal branch was large and normal. The left circumflex artery (LCX) was co-dominant and normal, with both the obtuse marginal (OM) branch and the left posterior lateral branch (LPLB) also appearing normal. The LPLB originated from the LCX, while the left posterior descending artery (LPDA) arose from the LAD, indicating a rare coronary anomaly. These findings suggested significant stenosis in the LAD and highlighted an unusual anatomical configuration. The left main coronary artery was accessed using a 6F JL4 guiding catheter. A 0.014-inch BMW wire was used to cross the vessel, followed by predilation with an NC TREK 3.0 x 15 mm balloon [Supplementary Videoclip 4]. A drug-eluting stent with a biodegradable polymer measuring 4.00 mm in diameter and 18 mm in length was deployed, and post-dilation was performed with an NC TREK 4.5 x 12 mm balloon. A repeat coronary angiogram showed satisfactory results [Figure 2D, Supplementary Videoclip 5]. The patient was discharged on the third day after the procedure. The electrocardiograms were taken immediately after stent placement and at discharge; both showed improvement in the ST-segment changes [Figure 1B,C]. A written informed consent from the patient and the ethical committee of the Ministry of Health, Muscat, Oman, provided approval [MOH/CSR/CR/24/10] to publish this report.

Comment

In this report, we present an octogenarian who suffered a type-1 inferolateral STEMI caused by a plaque in the mid-LAD artery, which was hyper-dominant and continued as the PDA. There have been several reported unusual variations in the origin of PDA.[4] It is uncommon for the PDA to originate from the LAD artery. However, it is common to observe a continuation of the LAD artery around the apex, typically referred to as the "wrap around" LAD artery.[5] Javangula et al. described the LAD continuing as the PDA up to the crux of the heart across the left ventricular apex for the first time.[6] Musselman et al. described a PDA originating from the LAD, extending beyond the crux, and branching into two, with each branch traveling on either side of the posterior interventricular sulcus.[7] In our case, the LAD was hyperdominant in the true sense that it did travel for a considerable distance into the posterior interventricular sulcus beyond the crux as the PDA. The right coronary artery in our patient was non-dominant. To the best of our knowledge, it is an extremely rare case in which the hyperdominant LAD artery continued as PDA beyond the crux into the posterior interventricular sulcus.

The conservative management of a middle-aged man who presented with chest discomfort and in whom a coronary angiography showed a lesion-free LAD looping around the left ventricular apex and running through the posterior interventricular groove as a PDA beyond the crux, by adjustment of the patient's diabetic and hypertensive medications was reported recently.[8] Similar to our management, a case of acute inferior wall myocardial ischemia due to occlusion of LAD continuing as PDA, which was successfully managed by stent deployment, was also reported earlier.[9]

The clinical significance of this anomaly is profound, as occlusion of a hyperdominant LAD artery can jeopardize a significant portion of the myocardium. Clinicians must maintain a vigilant approach, particularly in cases presenting with combined ST elevation in both anterior and inferior leads, absence of reciprocal changes in inferior leads during anterior lead ST elevation, or concurrent anterior and inferior ischemia during stress testing. The shift in coronary dominance to the LAD from either the right coronary or left circumflex artery can dramatically alter the clinical presentation, necessitating careful assessment and timely intervention. Patient

consent and institutional ethical committee approval were obtained for publishing this manuscript.

Authors' Contribution

ARB conceptualized and designed the work. ARB and AAJ acquired the data. All authors analyzed and interpreted the data, drafted and edited the manuscript. All authors approved the final version of the manuscript.

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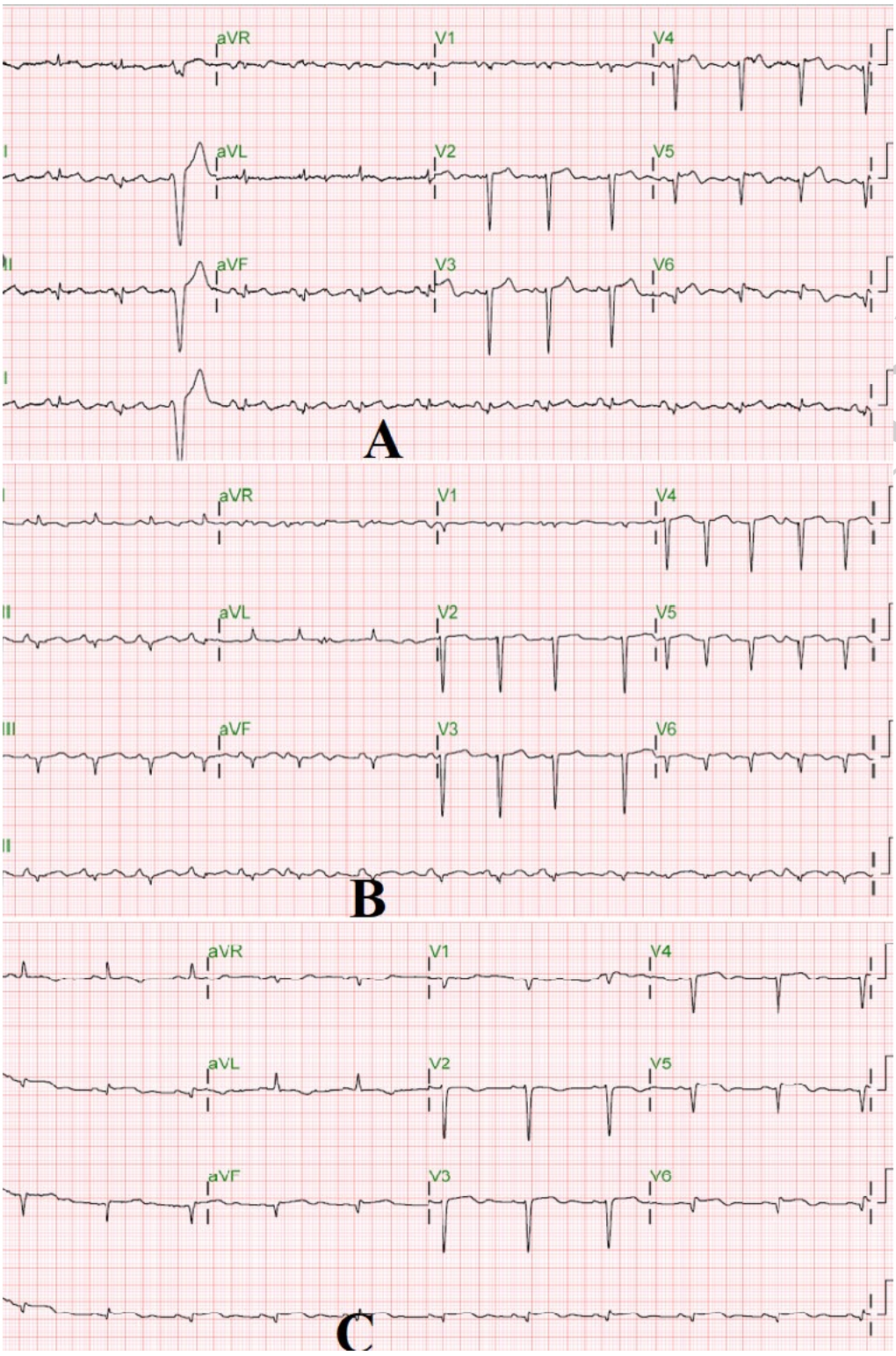

Figure 1: Electrocardiogram panel showing the inferolateral myocardial ischemia on admission [A], changes immediately following the coronary intervention [C], and at the time of discharge [C].

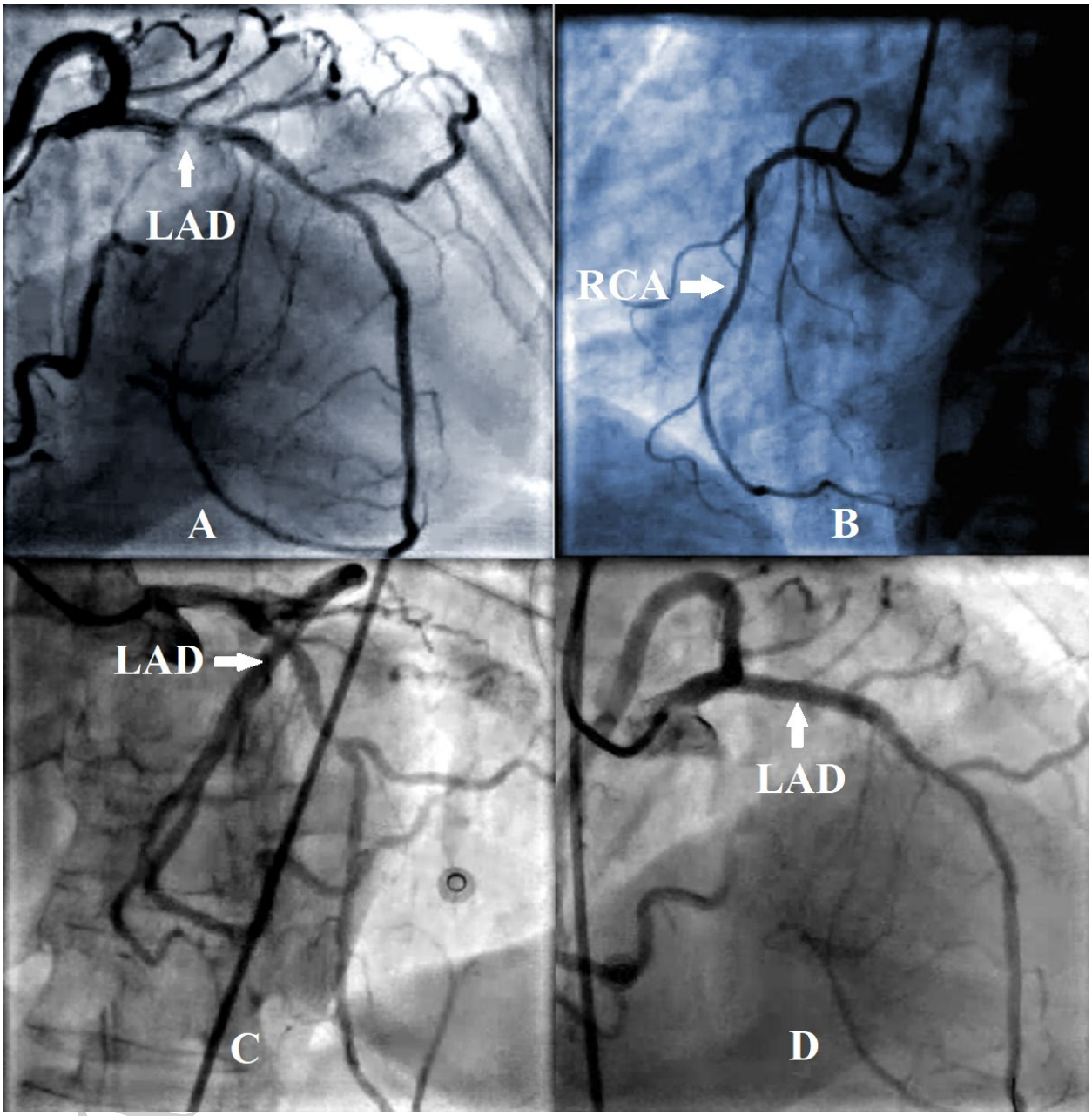

Figure 2: Coronary angiography images of the patient. Anteroposterior cranial view showing the 90% lesion [white arrow] in the proximal left anterior descending artery [A], the left anterior oblique cranial view showing the normal right coronary artery [white arrow] [B], left anterior oblique cranial view showing the lesion in the left anterior descending artery [white arrow] [C] and the right anterior oblique cranial view showing the stent in the left anterior descending artery [D].[LAD= left anterior descending artery; PDA= posterior descending artery].

Supplementary Videoclip 1: Coronary angiogram showing the left anterior descending artery continuing as the posterior descending artery in the right anterior oblique cranial view.

Supplementary Videoclip 2: Coronary angiogram showing the left anterior descending artery continuing as the posterior descending artery in the left anterior oblique cranial view.

Supplementary Videoclip 3: Coronary angiogram showing the left anterior descending artery continuing as the posterior descending artery in the anteroposterior cranial view.

Supplementary Videoclip 4: Coronary angiogram showing the balloon angioplasty of the left anterior descending artery.

Supplementary Videoclip 5: Left coronary angiogram showing the successful outcome after deployment of the drug-eluting stent.