An extremely rare coronary artery anomaly: Monster left anterior descending coronary artery

A 48-year-old male patient with known coronary artery disease and chest pain was admitted to the cardiology department. Two years ago, he underwent percutaneous coronary intervention for stable angina pectoris at another hospital. His physical examination was normal. Likewise, his electrocardiogram and laboratory findings were also in the normal limits. The treadmill exercise stress test revealed horizontal ST segment depression measuring 1 mm in the inferior leads. Coronary angiogram scans demonstrated that the left anterior descending (LAD) coronary artery continued to stretch into the posterior interventricular groove as posterior descending coronary artery (PDA) and was further extended beyond the crux. After the crux, it continued to extend into the posterior left ventricular branch. The right coronary artery was nondominant, diminutive, and terminated on the lateral wall of the right ventricle. The circumflex coronary artery (Cx) was within its normal anatomic course. The stent on the obtuse margin vessel was easily recognizable (Fig. 1, Panel a-c, Videos 1-3). We found that it was challenging to opacify the whole LAD during the opaque injection because of its length. There were nonsignificant plaques both on Cx and proximal LAD along with significant stenosis on the PDA. Similarly, a significant stenosis was also present on the nondominant right coronary artery (RCA). However, the diameter of the vessel was less than 2 mm. Hence, successful percutaneous coronary intervention was performed with drug-eluting stents for PDA (Fig. 2, Video 4), and the patient was discharged on optimized medical treatment.

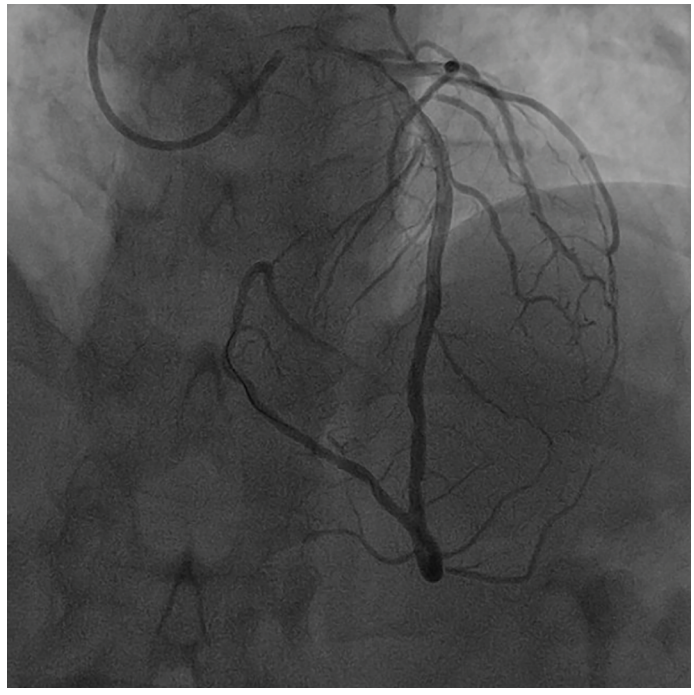

Figure 2. Drug-eluting stents were deployed into the posterior descending coronary artery (PDA). The final left anterior oblique cranial projection shows the easily visible superdominant left anterior descending (LAD) coronary artery

The reported incidence of coronary artery anomalies was 0.3%–1% in an autopsy series (1). Most of these anomalies are benign and asymptomatic. About 85% of PDA originates from RCA, whereas the rest of it originates from CX. PDA originating from LAD is a rare coronary anomaly. However, the case of a superdominant LAD continuing through PDA and reaching beyond the crux is extremely rare. Only a few patients have re-

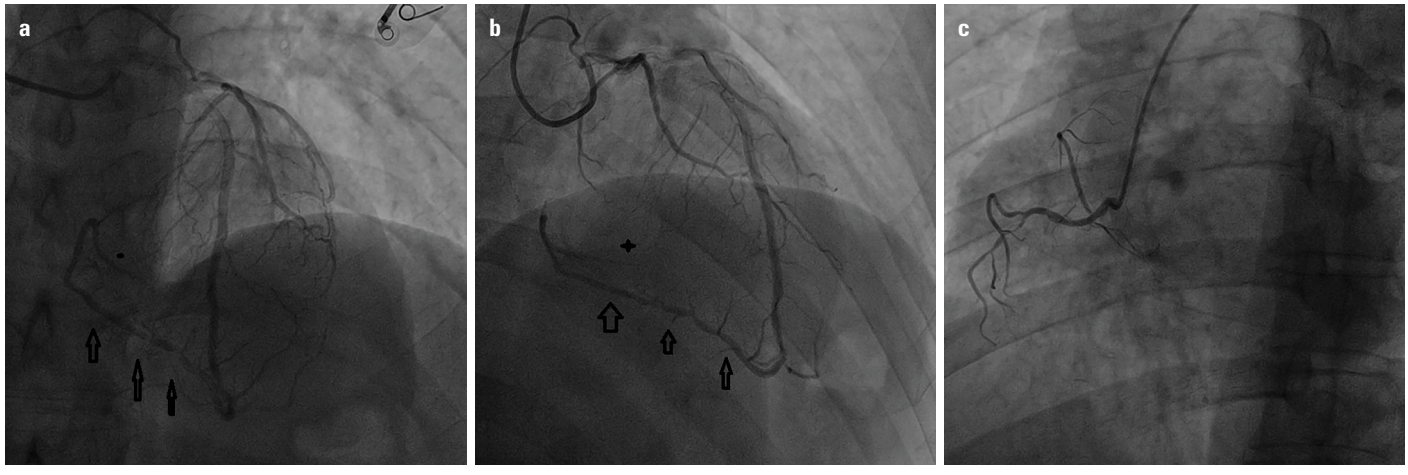

Figure 1. Coronary angiogram views. Panel a: The anteroposterior cranial projection shows the superdominant left anterior descending (LAD) coronary artery reaching the apex and stretching into the posterior groove (arrows) and the crux. After coursing through the crux, the LAD continues to extend into the posterior left ventricle branch (dot), Panel b: The right cranial oblique cranial projections shows the course of superdominant LAD beyond the crux (dot and arrows), Panel c: The left anterior oblique cranial projection shows the nondominant and diminutive right coronary artery (RCA)

ported this anomaly in the literature (2, 3). This anomaly is clinically significant because the LAD provides a large area of the ventricles and affects the normal coronary perfusion because of the LAD length.

Informed consent: Written informed consent was acquired from the patient for the publication.

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Videos 1-3. Angiograms showing the superdominant left anterior descending (LAD) coronary artery.

Video 4. The final angiogram scan after percutaneous coronary intervention

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Weird pacing spikes

A 74-year-old man with a history of severe mitral stenosis had received bioprosthetic mitral valve replacement. Ten years later, we implanted an epicardial pacemaker with VVIR mode because of sick sinus syndrome. Three years later, we suspected the development of a junctional rhythm and epicardial lead fracture, based on high lead impedance (Fig. 1a). Moreover, we implanted a dual-chamber rate-modulated (DDDR) pacemaker through the left subclavian vein, while leaving the epicardial pacemaker in place, and shifting to OVO mode (Fig. 1b, Red circle). The 12-lead electrocardiogram revealed atrial and ventricular dual-paced rhythm (Fig. 1c). Five years after implantation of the DDDR-mode permanent pacemaker, we noted a pacing-induced cardiomyopathy, with left ventricle (LV) ejection fraction of 34%, despite optimized medical therapy. The cardiac resynchronization therapy pacemaker (CRT-P) was upgraded (Fig. 1d). Although biventricular pacing initially functioned well (Fig. 1e), high LV pacing threshold prompted us to adjust the LV lead from bipolar to unipolar (LV lead to can) pacing (Fig. 1f). Symptoms were relieved after implanting CRT-P. However, we noted weird pacing spikes, with regular rhythm at 65 bpm without interference of the biventricular pacing. These spikes were unaffected by adjusting the CRT-P mode from DDD to VVI (Fig. 1f). Thereafter, we learned the pacing spike was generated by the unremoved epicardial pacemaker. The pacemaker reached elective replacement indication and started to pace at VVI mode of 65 bpm. Thus, the epicardial pacemaker had no influence on the CRT-P, but continued to produce spikes on the electrocardiogram due to sensing and capture failure.

Informed consent: Informed consent was obtained from the patient.

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