Surgical repair of an aerialized coronary arterial segment causing myocardial ischemia

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CLINICAL COURSE

A 66-year-old woman with a previously diagnosed myocardial bridge of the left anterior descending artery (LAD) and progressively worsening shortness of breath, chest pain, and fatigue refractory to medical management was referred for surgery. Medical history included Raynaud’s disease, hemochromatosis, and hypertension. Current medications included ranolazine and carvedilol. She had previously been trialed on amiodipine and isosorbide mononitrate, but did not tolerate the medications due to adverse reactions. Transthoracic echocardiography revealed normal biventricular function and normal valves. Chest computed tomography (CT) scan was unremarkable. Left heart catheterization revealed 30% stenosis of the mid-LAD (resting full-cycle ratio, 0.91), with a distal myocardial bridge and kinking of the apical LAD (Figure 1, A). Stress positron-emission tomography (PET) CT scan revealed a medium sized, moderate, reversible apical perfusion defect, consistent with ischemia in the distal LAD territory (Figure 1, B and C). Due to the refractory and progressive nature of her symptoms, the decision was made to proceed with unroofing of the myocardial bridge. Of note, resting full-cycle ratio was believed to not accurately reflect the potential for ischemia in this coronary segment because the dynamic kink appeared to straighten after passing the wire.

Following sternotomy, the abnormal segment of LAD was easily identified (Video 1). There was no myocardial bridge. In fact, the distal half of the LAD was markedly superficial on the epicardium. The segment corresponding to the area in question on left heart catheterization was aerialized to a greater degree than proximal and distal segments and was contained in a cylinder of epicardial fat, which created a small cavity of a few millimeters depth between the LAD and the myocardium immediately underneath (Figure 2,
The hypermobile LAD segment was stabilized using the off-pump coronary artery bypass grafting Octopus retractor (Medtronic, Minneapolis, Minn). Two 5–0 Prolene mattress sutures with felt pledgets were placed from left to right under the segment to plicate it. The pledgets were used to partially obliterate the cavity underneath the aerialized LAD and were angled in order to straighten the mobile segment (Figure 2, B). The sutures were tied firmly enough to achieve gross improvement in the mobility and kinking of the segment. Intraoperative transesophageal echocardiography did not show any wall motion abnormalities and the electrocardiogram was normal.

The patient progressed well postoperatively and was discharged home after 5 days. Her preoperative symptoms had completely resolved by 1 month following surgery, and a follow-up stress positron-emission tomography–CT showed no areas of ischemia (Figure 1, D and E). Institutional review board review is not required at our institution for case reports. Consent was obtained from the patient for publication of this report.

DISCUSSION

CAAs are a diverse group of conditions that impair the ability of the coronary system to effectively deliver blood
to the myocardium. The normal course of a coronary artery is in the subepicardial position at the edge of the epicardial fat. There are 3 abnormal positions of a coronary artery: aerial, intramuscular, and intracavitary. Intramuscular coronary arteries are a well-known phenomenon and are encountered in clinical practice on a regular basis. However, to our knowledge, this is the first report of an aerial coronary artery with physiologic effect.

Our patient was initially diagnosed with a myocardial bridge, a type of CAA where a segment of the artery courses within the myocardium, instead of in the epicardial position. The intramuscular portion of the artery is compressed during systolic contraction of the myocardium. Myocardial bridge is relatively common, with a reported angiographic incidence of 0.15% to 25%, but rarely results in clinical symptoms. Treatment of a symptomatic myocardial bridge typically involves unroofing the intramuscular segment by cutting the overlying muscle fibers, or bypass grafting for longer segments. Of note, a repair technique similar to that utilized in our patient has been recently described to fix a ventricular perforation while unroofing a coronary segment associated with myocardial bridge.

We expected to encounter a myocardial bridge in our patient, but instead found an abnormally elevated segment of LAD in an aerial position above the epicardium. Before this segment appeared to be kinking with systolic contraction due to excessive mobility, plication and immobilization was performed with pledgeted suture. This highlights the potential consequences of any deviation from the normal coronary arterial course in the epicardial position. Although myocardial bridge can cause coronary ischemia due to the contraction of overlying muscle fibers, ischemia can result with an aerial coronary artery due to excessive mobility and kinking with underlying myocardial contraction. Medical management would likely alleviate symptoms in many patients, but our particular patient failed medical therapy with progressively worsening symptoms, necessitating surgical intervention.

CONCLUSIONS
An aerial coronary artery segment is an extremely rare cause of myocardial ischemia. Immobilization of the kinking aerial segment will restore normal coronary blood flow and can be achieved with pledgeted suture repair.

References
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