Multimodality imaging to clarify an atypical presentation of branch vessel coronary occlusion
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Clinical History
A 45-year-old female with a history of hypertension, dyslipidemia and morbid obesity managed with gastric bypass resulting in 120 pound weight loss, presented to an outside hospital with chest pain and anterolateral ST-segment elevations on electrocardiogram. She was in her usual state of health until the day prior to presentation when she woke up with left chest, shoulder, and jaw pain associated with diaphoresis. Her chest pain spontaneously resolved in 30 minutes but recurred the next day. Emergent invasive coronary angiography (ICA) revealed a dual-left anterior descending (LAD) system with sluggish flow in the smaller, septal LAD, but no culprit vessel was apparent. The patient was presumed to have focal myocarditis involving the anterolateral wall.

Subsequent outpatient cardiac magnetic resonance imaging (MRI) demonstrated mild left ventricular dysfunction and anterolateral akinesis with transmural abnormal late gadolinium enhancement in the anterolateral wall. Stress and rest nuclear myocardial perfusion imaging (SPECT-MPI) revealed a medium sized, severe, partially fixed perfusion defect in the mid to distal anterior wall.

Upon referral from the outside hospital, conflicting information was present. The patient’s presentation included stuttering chest pain and territorial, transmural myocardial involvement by cardiac MRI and SPECT-MPI, each of which was suggestive of myocardial infarction. However, the lack of a culprit vessel on diagnostic coronary angiography led to a conflicting diagnosis of focal myocarditis. Coronary CT Angiography (CCTA) was consequently performed to assess for the possibility of a missed coronary occlusion.

Findings
CCTA confirmed “double LAD” configuration, with a smaller, septal LAD shallow bridged segment. The LAD did not contain plaque or stenosis. A small D1 branch was well opacified for approximately 15mm beyond its origin,
at which point an abrupt cutoff and diminished distal contrast opacification was noted. The branch supplied the hypokinetic/akinetic segment of the left ventricle.

**Discussion**

In this case, invasive angiography was not diagnostic of the small branch coronary occlusion which resulted in an anterolateral ST elevation myocardial infarction (STEMI). Cardiac MRI and SPECT findings raised the possibility of ischemic insult, and coronary CT angiography depicted the arterial findings, and again confirmed corresponding, territorial ischemic myocardial findings. Together, these multimodality findings allowed a definitive diagnosis of anterolateral STEMI and helped set goals for secondary prevention in this young female patient.

**Figure 1:** ICA demonstrates a dual-LAD system initially interpreted as sluggish flow in the smaller, septal LAD without stenosis or occlusion. In retrospect a septal perforator (arrow) is suspicious for a small branch occlusion.

**Figure 2:** Maximum intensity projection CCTA image demonstrates a well-opacified first diagonal (D1) branch, with an abrupt caliber change 15 mm beyond its origin. This vessel is of small caliber (0.2 -0.3 mm), at the limit of spatial resolution of CT. The D1 branch supplies the hypokinetic/akinetic segment of the left ventricle.

**Figure 3:** 3-dimensional volume-rendered CCTA image allows an epicardial view of the abrupt transition in caliber of D1 with tapered distal vessel.

**Figure 4:** **(A)** Double oblique short axis inversion-recovery prepared MRI image of the mid left ventricle demonstrates transmural abnormal late gadolinium enhancement in the anterior and anterolateral wall consistent with an ischemic insult (infarction). **(B)** Thickened slab (8 mm) multiplanar reformatted short axis CCTA images demonstrates a matching thin, hypointense anterior wall infarction.

**REFERENCES**

