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Clinical Image

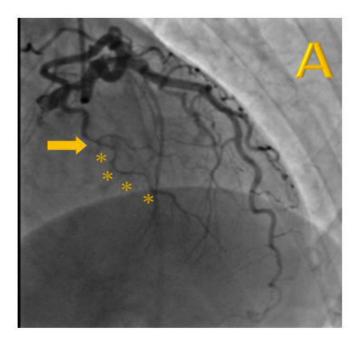
Female Runner with An Elevated Troponin

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45-year-old female runner with a past medical history of hypertension presented to the Emergency Department with sudden onset substernal chest pain and diaphoresis after one of her usual running sessions. EKG showed normal sinus rhythm with nonspecific T-wave changes. Initial troponin I was 1.16 ng/mL, she was admitted to the Coronary Care Unit and started on a heparin drip. Her troponin I peaked at 38 ng/mL. Coronary angiography was performed the following day which suggested Type II spontaneous coronary artery dissection (SCAD) involving the obtuse marginal artery. Cardiac MRI was done which confirmed the findings of non-transmural infarct in the area of distribution of affected artery. Patient was managed conservatively, started on dual antiplatelet, and beta block blocker prior to discharge home and continues to do well. SCAD accounts for approximately 25% of cases of Acute Coronary Syndrome in women ≤50 years old [1] and 90% of patients presenting with SCAD are women between 47-53 years of age. SCAD results from separation of the layers of an epicardial coronary-artery wall by intramural hemorrhage, with or without an intimal tear [2]. Majority of cases can be managed medically as angiographic studies have shown improvement in stenosis and healing at follow up.



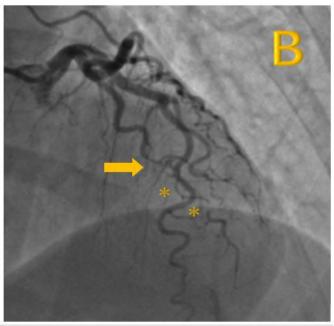


Figure 1: Coronary angiography shown in the RAO cranial (A) and AP cranial (B) projections, revealing a mid-distal subtotal coronary occlusion in the Obtuse Marginal Artery. Abrupt change in vessel caliber (yellow arrow), followed by a linear stenosis and lack of contrast staining, consistent with type II SCAD (yellow asterisks).

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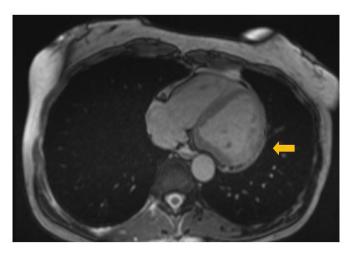


Figure 2: Cardiac MRI showing non transmural scar of the mid anterolateral wall of left ventricle (yellow arrow) consistent with myocardial infarction in the distribution of Obtuse Marginal Artery with SCAD.

Reference

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