NSTEMI Progressing to STEMI in a Healthy Young Female

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Introduction

Spontaneous Coronary Artery Dissection (SCAD) is a rare cause of acute coronary syndrome (ACS) in healthy individuals. There are no risk stratification tools for the prevention of SCAD and no clear guidelines for the acute management. The use of traditional ACS therapies, including antiplatelets agents, antithrombotic, beta-blockers and statins remains controversial.

Case Presentation

• A 40-year-old female with hypertension and distant history of postpartum pre-eclampsia presented with sudden onset, substernal chest pain radiating to her left arm and jaw. Her pain began while cooking and was associated with dizziness, fatigue and diaphoresis.

• In the Emergency Department, she was given sublingual nitroglycerin and aspirin 325mg with immediate improvement in her symptoms. An EKG demonstrated normal sinus rhythm with T-wave inversion in V1-V5. Troponin-I levels were initially normal at 40 ng/L (ref range: <50 ng/L), elevating to a peak of 22,620 ng/L after 12 hours. She was treated with unfractionated heparin, clopidogrel 300mg, atorvastatin 80mg, and aspirin 81mg for the management of NSTEMI and admitted to the telemetry unit.

• Left heart catheterization revealed an ejection fraction of 55-60% with apical hypokinesis, and 95% obstruction of the mid LAD with diffuse tapering to the distal segment consistent with Type 2 SCAD. TIMI-2 flow was maintained and her symptoms had resolved so no additional intravascular imaging or percutaneous intervention was performed.

• Unfractionated heparin was stopped and atorvastatin was discontinued given her 10-year ASCVD risk of less than 7.5%, and no evidence of atherosclerotic epicardial coronary artery disease. Four hours post-catheterization she experienced recurrent chest pain while resting in bed. Repeat EKG demonstrated new ST-segment elevations in leads V2 through V5 consistent with STEMI. Troponin-I levels rose to 5270 ng/L after previously trending to 4920 ng/L. Repeat catheterization was deferred given known diagnosis of SCAD and prompt resolution of chest pain with further medical management.

• Unfractionated heparin was not restarted given concern for worsening intraluminal hematoma. Serial EKGs demonstrated resolution of ST segment elevations.

• She did not experience any additional episodes of chest pain and remained hemodynamically stable for the rest of her hospital stay. She was discharged home on hospital day 5 on metoprolol tartrate 25mg twice a day, aspirin 81mg daily, and 1 year of clopidogrel 75mg daily. She reported no new or worsening symptoms on post-discharge days 10 and 23 and is tolerating cardiac rehabilitation.

Discussion

• This is a case of a young woman with NSTEMI progressing to STEMI due to Type 2 SCAD. The risks of catheter-induced progression of luminal dissection versus progression of intramural hematoma with unfractionated heparin must be considered based on the angiographic appearance of the affected artery and the patient’s clinical course. Although there are no societal guidelines for the acute management of SCAD, expert consensus has found a role for dual-antiplatelet and beta-blocker therapy in minimizing major adverse cardiac events post-SCAD. More investigation is needed, however, on the use of standard ACS therapies and risk prediction tools for the occurrence and recurrence of SCAD.

References


