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Introduction:

There are multiple reports in the medical literature on acute myocardial infarction (MI) following cannabis use. Research is limited and controversial on the causative pathophysiology, however, reports highlight patients with significant cannabis use history with subsequent development of a coronary thrombus. Further research is needed to investigate this correlation.

Case Report:

A 52-year-old male with a past medical history of paroxysmal atrial fibrillation, daily marijuana use via inhalation, hypertension, and benign prostatic hyperplasia presented after he was in a motor vehicle collision. He was in his usual state of health until the morning of admission, when he was awoken with sudden onset epigastric pain associated with nausea, vomiting, and dyspnea. He initially went to an outside hospital, where he was diagnosed with viral gastroenteritis versus cannabinoid hyperemesis syndrome and discharged with anti-emetics. On his way home from the hospital, the patient got into a motor vehicle collision where his vehicle was flipped upside down. He reported loss of consciousness when his vehicle was hit. He presented via ambulance to the emergency department and underwent CT imaging of his head, cervical spine, chest, abdomen, and pelvis that were unrevealing for any acute abnormality. He was hemodynamically stable with an unremarkable physical exam. On interview, he endorsed intermittent chest pain and shortness of breath.

An EKG revealed ST segment elevations in leads II, III, and aVF with T wave inversions in V4-V6. Initial high-sensitivity troponin was 6500 ng/L. The patient was immediately STEMI-activated, loaded with aspirin, and taken to the cath lab. Diagnostic right and left coronary cineangiography revealed a large clot in the left main (LM) coronary artery adherent to the inferior wall and a distal left anterior descending (LAD) embolus in the small-vessel at the apex. The LM thrombus was noted to be partially occlusive and was subsequently aspirated. Sequential angiography showed reduction and then elimination of the thrombus. After the successful LM thromboaspiration, the patient was admitted to the medical ICU for post-STEMI care. A transthoracic echocardiogram after the event showed a left ventricular ejection fraction of 30-35%, inferior wall and apical akinesis, and lateral wall hypokinesis. The remainder of the patient's hospital course was uneventful. Troponins peaked at 45,000 ng/L and then trended down. He no longer endorsed chest pain. He was transitioned from aspirin to clopidogrel and from a heparin drip to apixaban after completion of 72 hours on the heparin drip. On discharge, patient was sent home with cardiology follow-up, clopidogrel, high-intensity statin, apixaban, sacubitril-valsartan, and metoprolol succinate, with plans to continue to optimize his heart failure medication regimen outpatient.

Discussion:

In a 52-year-old male with a past medical history of well-controlled hypertension, atrial fibrillation noted in a hospitalization for cannabinoid hyperemesis syndrome which did not require anticoagulation given his low risk, and no family history of coronary artery disease, there was question as to the etiology of his left main coronary artery thrombus. Upon further questioning, he uses marijuana daily and has been for many years. We believe that his heavy marijuana use may have been his most significant contributing factor, as this has been previously hypothesized in literature. Additionally, this patient had an interesting case of left main and LAD lesions with ST elevation in the inferior leads. This was due to his anatomical variation of a left-dominant coronary artery system with a "wrap-around" LAD, in which his LAD extended past the apex and supplied the inferior wall. His nausea and vomiting were anginal equivalents.