**AN UNUSUAL CASE OF ACUTE MYOCARDIAL INFARCTION WITH NORMAL CORONARY ARTERIES**

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Myocardial infarction with nonobstructive coronary arteries (MINC) is a clinical syndrome characterized by presence of an acute myocardial infarction (AMI) in the absence of obstructive coronary artery disease (≥50% stenosis). We present a case of a young patient with Factor V Leiden mutation and multiple strokes, who presented with a MINC despite anticoagulation.

**Case :** A 27-year-old male with bipolar disorder, Factor V Leiden mutation, four strokes, and patent foramen ovale closure presented with typical chest pain accompanied by shortness of breath. Upon arrival, vitals signs were: blood pressure 135/74 mmHg, heart rate 84 bpm, O2 saturation 100 %. Physical exam was unremarkable. Workup revealed up trending troponins (0.01,0.18,0.33 ng/l). Electrocardiogram showed S1Q3T3 pattern concerning for right heart strain. Chest CT angiogram excluded pulmonary emboli and aortic disease. Transthoracic echocardiography showed normal ejection fraction, moderate aortic regurgitation, and regional wall motion abnormalities without apical ballooning. Thus, a non ST-elevation myocardial infarction (NSTEMI) was diagnosed. Aspirin, statin, beta-blocker, and heparin drip were initiated. Cardiac catheterization revealed normal coronary arteries.

**Discussion:** MINC occurs in 5%-10% of all patients with AMI and is associated with a 12-month all-cause mortality of 4.7%. Etiologies include coronary artery spasm, spontaneous coronary thrombosis/emboli, and coronary dissection, takotsubo cardiomyopathy, auto-immunes and hematologic disorders.

This is an unusual case of MINC in a young patient with Factor V Leiden mutation. Hypercoagulability is a common risk factor for venous thrombosis and to a lesser extent, arterial thrombosis. Likely, his NSTEMI was caused by a thrombus partially occluding a coronary artery. We hypothesize that this thrombus was dissolved by heparin. Early treatment of an AMI secondary to a hypercoagulable state using dual anti-platelet therapy and anticoagulation (heparin) may be sufficient to revascularize the culprit artery without the need for invasive interventions. Nevertheless, a left heart catherization would still be required to confirm the diagnosis of MINC.