A Report of Non-ST Elevation Myocardial Infarction in a Transgender Woman

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Background

Recent surveys estimate that transgender men and women comprise approximately 1.4 million of the nearly 11 million members of the United States' adult LGBTQ community (1,2). National cross-sectional reports illuminate the disproportionately elevated risk of MI and cardiovascular disease associated mortality among this group relative to their cisgender peers (1-3). Comparative analysis of socioeconomic, behavioral, and psychosocial factors demonstrate s that despite being younger than their cisgender peers, transgender patients exhibit higher rates of obesity, stress, sedentary lifestyle, alcohol consumption, smoking, HIV infection, lack of insurance, and atherosclerosis promoting comorbidities such as hypertension, renal disease, and diabetes mellitus (2,4).

Case Presentation

A 37-year-old transgender woman off of gender-affirming hormonal therapy for the last 2 years with a history of hypertension, end-stage renal disease, active tobacco use, right hip silicone-induced granulomatosis, and prior hemorrhagic stroke presented with 36 hours of substernal chest pain radiating to the left arm. Pain was worsened with exertion, relieved by rest and associated with dyspnea on exertion. On admission, she was afebrile, hypertensive to 178/138 mmHg, tachycardic at 100 BPM, saturating 97% on room air. Her exam was not significant for diaphoresis, jugular venous distention, adventitious heart sounds, murmurs, and/or lower extremity swelling. Troponin peaked at 1.48 ng/mL. Electrocardiogram revealed hyperacute peaked T waves in V1-V2 without pathologic Q waves or ST elevations. Transthoracic echocardiogram showed preserved ejection fraction with basal inferior, inferolateral, anterolateral and mid-anterolateral hypokinesis. Given concern for acute myocardial infarction, patient was loaded with aspirin, started on heparin drip, high-intensity statin, and carvedilol. Patient was taken for emergent left heart catheterization with attempted thrombectomy with aspiration catheter yielding no thrombus. She subsequently underwent percutaneous coronary intervention with deployment of two overlapping drug-eluting stents in the proximal and mid-RCA with restoration of flow.

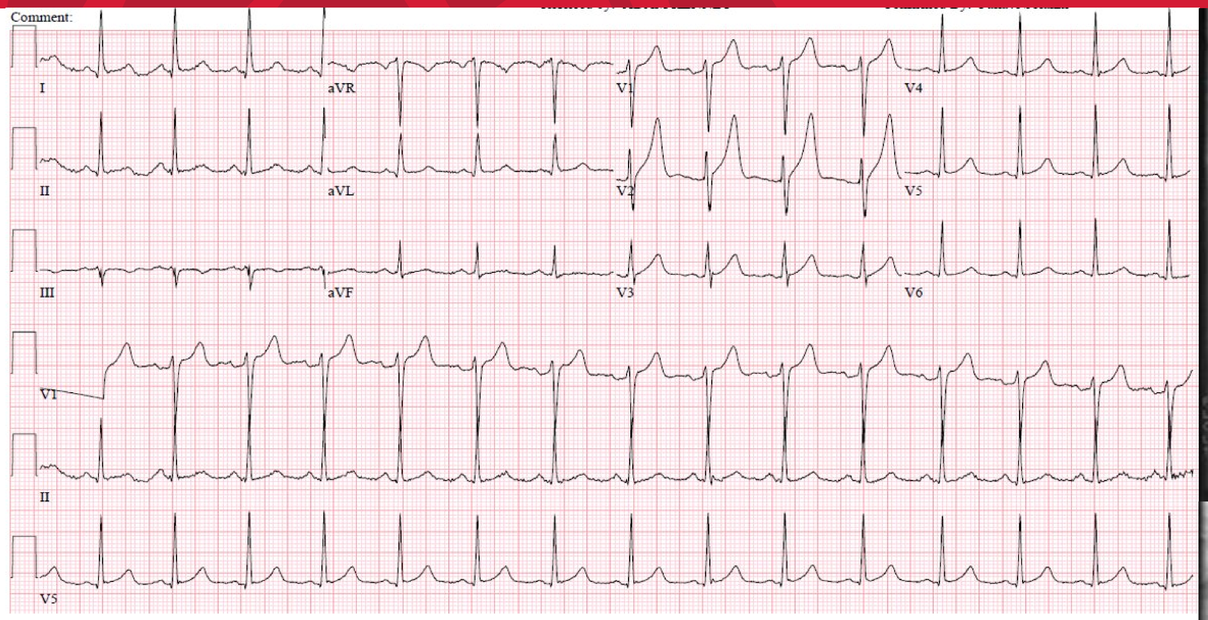

Figure 1: Electrocardiogram on admission showing hyperacute T waves in anterior leads (V1 and V2) without ST elevation and non-pathologic septal Q waves in leads I, aVL, V5, and V6.

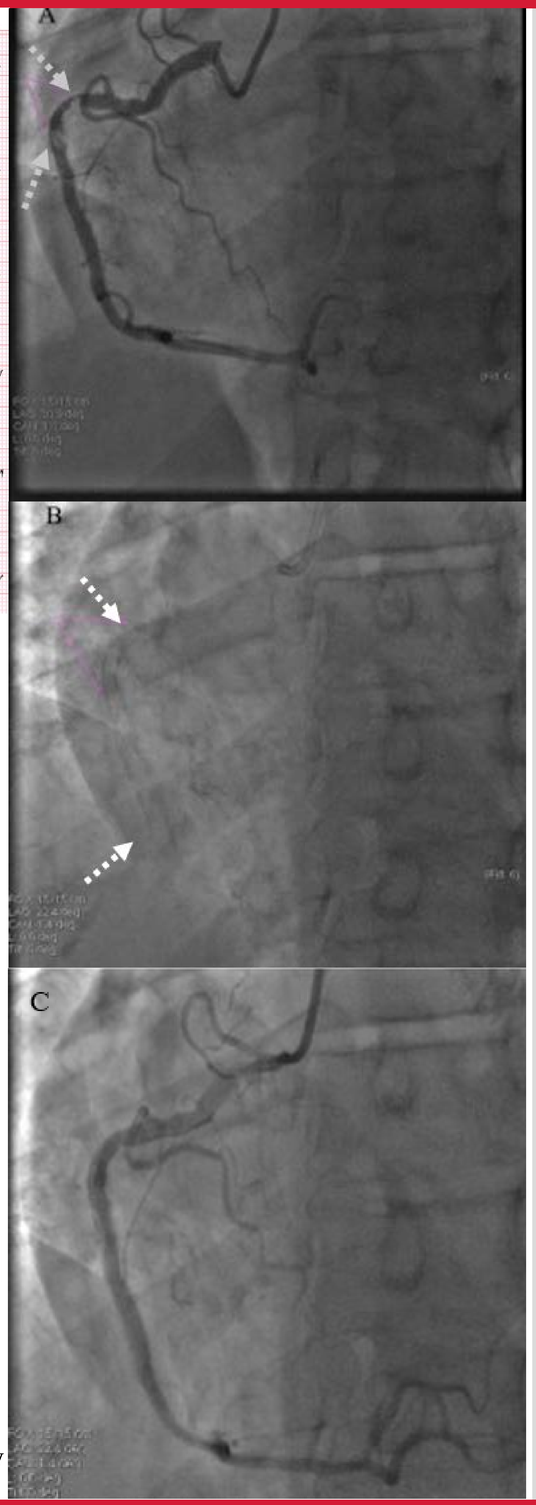

Figure 2: **A)** Long thrombus appearing culprit lesion (arrows) in proximal to mid-RCA. Unsuccessful aspiration thrombectomy performed, suggestive of calcified plaque. **B)** Therefore, two overlapping stents (arrows) in proximal and mid-RCA were deployed with patency. **C**) Restoration of TIMI 3 flow achieved through prox-mid RCA.

Discussion

Our case highlights the intricacies of cardiovascular health in transgender men and women. This population has unique and complex cardiovascular risk profiles, distinct from their cisgender counterparts. After adjusting for age, comorbidities, smoking, and exercise, transgender men and woman are at increased risk for myocardial infarction compared to cisgender men and women. Additionally, this risk increases for those on gender-affirming hormonal therapy, as well as those who underwent gender-affirming surgery (3,4). Moreover, this disparity is multifactorial, including psychosocial stressors, effects of long-term hormone use, and chronic inflammation. Estrogen causes a dose-dependent increase in risk for thrombosis. Androgen therapy increases oxidative stress, accelerating deposition of atherosclerotic plaque (5,6). This complex intersection between oxidative stress, inflammation, and increased risk of thrombosis in transgender men and women requires further investigation for adequate management.

Conclusion

Our case highlights a transgender woman off of gender affirming hormonal therapy presenting with a non-ST elevation myocardial infarction and angiographic single-vessel coronary disease. Specifically, stress, inflammation, dyslipidemia, and thromboembolism predispose this understudied cohort to coronary artery disease and increased cardiovascular mortality. As the transgender and larger LGBTQ population continues to grow, dedicated research in both retrospective and prospective clinical trials are needed to further elucidate disease pathways and devise strategies for both primary and secondary prevention of coronary artery disease in these populations.

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