

Introduction

Life threatening right coronary artery ischemia can be challenging to diagnose, especially, when it is presented in females with nonspecific or atypical symptoms without changes in cardiac biomarkers. Multiple confounding pathologies such as congestive heart failure, chronic kidney disease, and COPD add an additional layer of complexity to this situation. In this case, we will discuss a patient who presented with symptoms of congestive heart failure exacerbation without chest pain or elevation in troponin. She was found to be in junctional rhythm and later discovered to have 99% RCA occlusion on left heart catheterization.

Case Presentation

A 79-year-old obese female with insulin-dependent type 2 diabetes mellitus, stage 4 chronic kidney disease, heart failure with preserved ejection fraction, obstructive sleep apnea on CPAP, and hypertension presents to ED with chief complaint of shortness of breath. She stated, she had gained 7 lbs in the last 2 days and complained of no urine output for 14 hours. Vitals were notable for HR 49 bpm, RR 22, BP 121/87 mmHg SpO2 98% on 3L O2 via nasal cannula (no oxygen at baseline). EKG (Figure 1A) was notable for junctional rhythm, and CXR was notable for trace right pleural effusion, mild interstitial edema, and cardiomegaly. Labs were notable for Hb of 9.1g/dL, lactate 2.3, Na 132 mmol/L, K 4.7 mmol/L, creatinine 3.97 mg/dL, eGFR 10 ml/min/SA, Mg 2.1mg/dL troponin-I less than 0.3 ng/mL, and pro-BNP of 3315 pg/mL (10 months ago which was 636 pg/mL). Previous echocardiogram was done on 5/24/2022 and it was notable for EF 65-70 %, RVSP 35-40 mmHg, and grade II diastolic dysfunction. Patient was admitted for treatment of acute on chronic exacerbation of heart failure with preserved ejection fraction and was started on diuretics. Cardiology and nephrology services were consulted. Nephrology recommended avoiding nephrotoxic medication and adjusted diuretic dosing with I&O monitoring with Foley catheterization. Cardiology considered overall decompensated status of patient and recent change in dosing of carvedilol to be the cause of junctional bradycardia. They recommended holding carvedilol and avoiding QT prolonging agent while waiting for Echocardiography with plan of future stress testing once patient's acute decompensation improved.

On second day of hospital stay, patient's ABG was notable for pH 7.42, pCO2 36.1 mmHg, pO2 82mmHg, HCO3 23.5mmol/L on 3L via nasal cannula, labs notable for Na 134mmol/l, K 4.4mmol/L, creatinine 4.22mg/dL, eGFR 9 ml/min/SA, with an improvement in urine output. Patient continued to stay in junctional rhythm with rate in the 50s, and Cardiology considered monitoring for another day with plan of temporary pacing if bradycardia did not improve.

On third day of hospitalization vitals were notable for BP 114/64, heart rate 53 bpm, RR 18, SpO2 98%. Unfortunately, patient's heart rate continued to stay in accelerated junctional rhythm in the rate 50s to low 60s. She continued to stay relatively weak and short of breath. She was off carvedilol for 48 hours and continued to stay in junctional rhythm. New development of sick sinus syndrome was considered as primary differential, and patient underwent uneventful temporary atrial pacemaker placement in hopes of improving cardiac output, with set heart rate of 80bpm. Initially she did well but in the Cath lab recovery area had a sudden obtundation, pallor, and hypotensive with BP with 62/50 mm Hg. EKG (figure 1B) at that time was notable for ventricular tachycardia with captured beats notable for ST elevation in inferior leads. She subsequently lost her pulse and resuscitation was begun immediately. Intermittent cardiopulmonary resuscitation with eventual spontaneous return of circulation. EKG (figure 1C) at that time was notable for atrial paced rhythm with ST elevations in inferior leads. Urgent echo performed during CPR was notable for severe left ventricular systolic dysfunction with EF of less than 15% severe global hypokinesis. Interventional cardiologist was consulted, urgent left heart catheterization was performed (Figure 2a-c), which was notable for 99.999% occlusion of ostial region of right coronary artery. Salvage percutaneous intervention of acute ostial right coronary artery occlusion with angioplasty and drug-eluting stent placement was performed. Maximum troponin-I reached after the event was 1.42 ng/mL. Echo post procedure was notable for a recovery of EF 60-65% and grade 3 diastolic dysfunction with stability at 2 month repeat Echo.

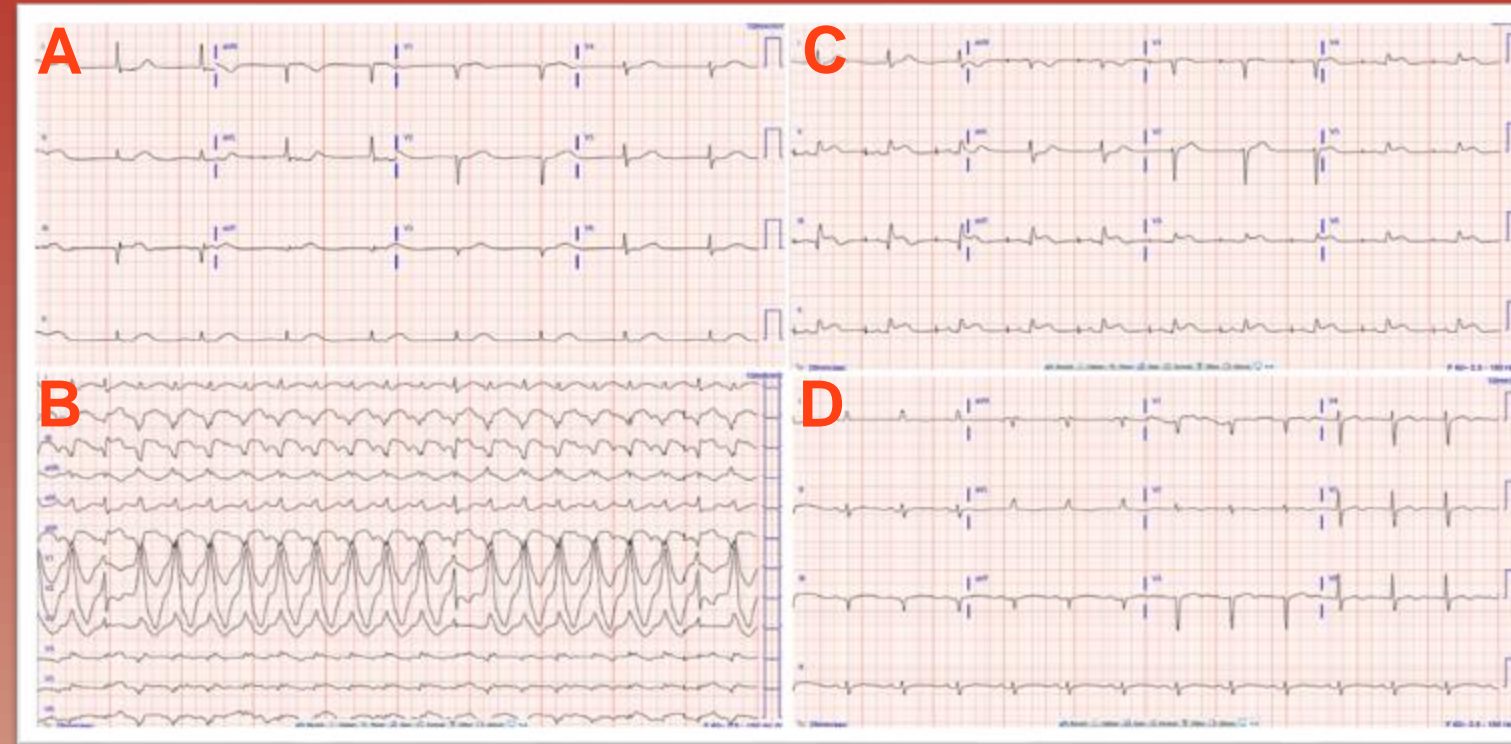


Figure 1: A. presenting ECG notable for junctional rhythm. B. ECG post temporary atrial pacemaker placement notable for ventricular tachycardia with capture beats notable for ST elevations in Leads II, III, aVF, V3, V4, V5, V6. Upward deflections of qRs complexes in pericardial leads and downward deflection of qRs complexes in inferior leads point to a ventricular rhythm originating from inferior wall of left ventricle (the ischemic area). C. ECG after return to spontaneous circulation notable for atrial paced rhythm with ST elevations in Leads II, III, aVF, V3, V4, V5, V6 and ST depressions in leads I and aVL. D. ECG post removal of temporary pacer notable for sinus rhythm with nonspecific ST and T wave abnormalities.

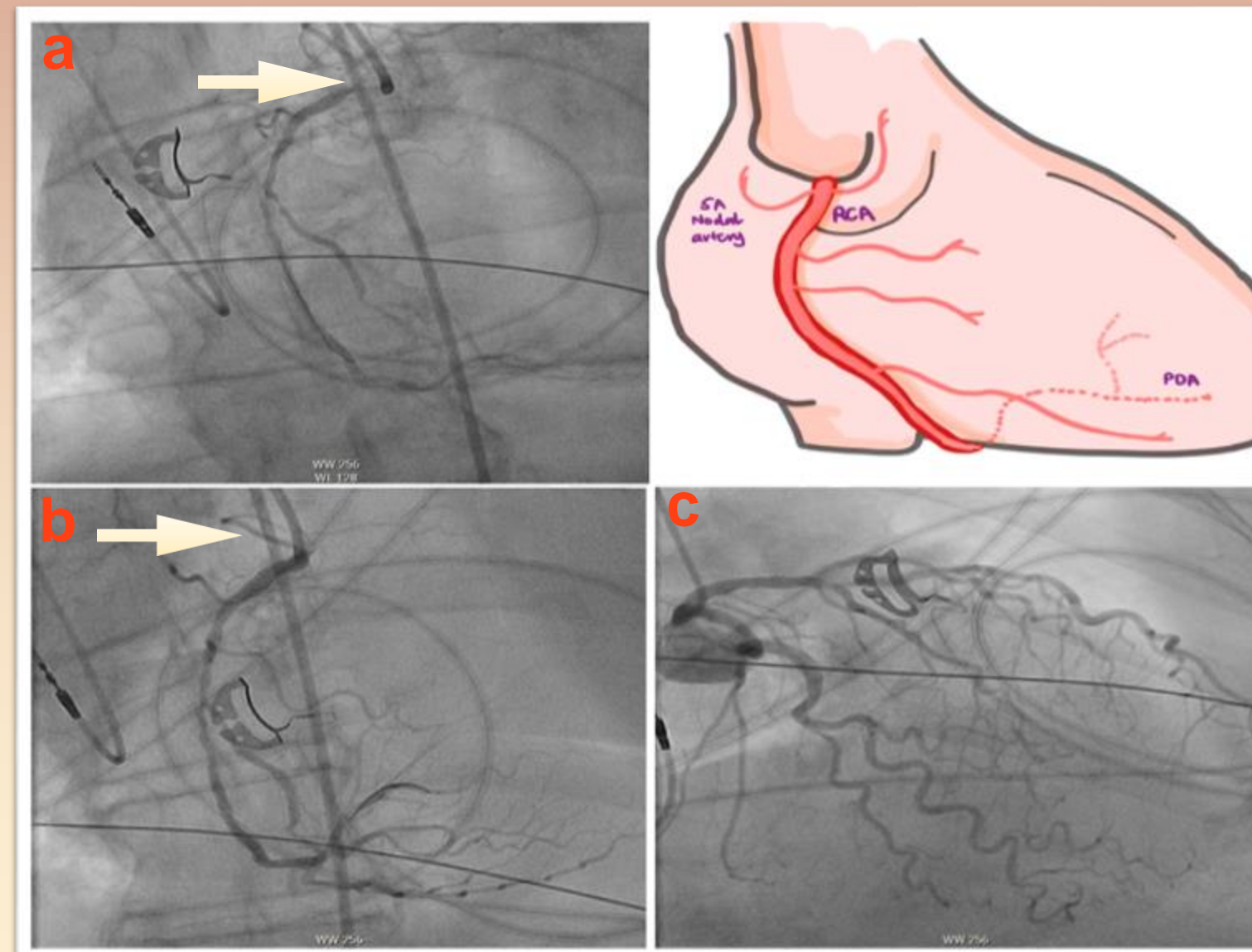


Figure 2: a. Right coronary artery notable for severe ostial occlusion (arrow). b. right coronary artery post PCI notable for reperfusion of sinoatrial nodal artery (see arrow). c. Non pathological perfusion of left coronary artery, left anterior descending coronary artery and left circumflex artery (LAO).

Discussion

Heart disease is the leading cause of death in females ("Women and heart disease," 2022). According to CDC in 2020 heart disease was responsible for 314,186 (1 in every 5) deaths in females ("Women and heart disease," 2022). Diagnosis of acute coronary syndrome in female continues to be a challenge given the nonspecific or atypical presenting symptoms. In a study published in 2018 authors reported a lower number of women compared to men who presented with classic symptoms of chest pain (31% compared with 42% men) this was particularly true in younger women (Aggarwal et al., 2018). According to a study published in 2003 most common prodromal symptom of acute myocardial infarction in females were unusual fatigue (70.7%), sleep disturbance (47.8%), shortness of breath (42.1%), indigestion (39.4%), and anxiety (35.5%) (McSweeney et al., 2004, p.9), which may be considered nonspecific, and is highlighted in our case.

Our patient presented with shortness of breath, fatigue, and fluid overload with EKG finding of junctional rhythm. Given her comorbidities the initial differentials considered in this setting included heart failure exacerbation, fluid retention secondary to worsening kidney failure, and medication side effects. Given confounding comorbidities, patient received pacer which worked as a stress test and presumably caused precipitation of critical ischemia. Thankfully, swift recognition of the event resulted in complete cardiac recovery but unfortunately this came at expense of loss of kidney function. As it is seen in the case above, critical ischemia should be considered in female presenting with nonspecific symptoms.

References

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