Heart: An Unusual Case of Acute Beta Blocker Withdrawal

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Introduction

Stress induced (Takotsubo’s) cardiomyopathy is a relatively rare cause of acute coronary syndrome and acute decompensated heart failure. Historically it has been associated with episodes of severe stress, however there have been case reports of Takotsubo’s being associated with acute beta blocker withdrawal and adrenergic overload.

Case Description

HPI: The patient is a 73yo female with a PMHx of paroxysmal Afib, COPD and OSD on nocturnal O2 who presented to the hospital from pre-operative clinic with AFib with rapid ventricular rate (RVR). Three months prior to admission she had been admitted for a ground level fall resulting in a femur fracture and Afib with RVR. Echocardiogram at that time was within normal limits. She was discharged to a rehab facility on metoprolol succinate 150mg daily with a plan for a right total hip replacement in the coming months. 5 days prior to her planned procedure she stopped all of her medications including the metoprolol. She was seen in pre-op clinic where she was found to be in Afib with rates in the 160’s as well as lower extremity swelling and exertional dyspnea.

Past Medical History

Paroxysmal Afib
OSA
HTN
Atrial regurgitation

Medications

Abuterol
ASA 325 mg
Fluticasone-salmeterol
HCTZ
Lisinopril
Metoprolol succinate
Sildenafil

Social History

Former smoker
EtOH – 1-2 a night
No illicit drugs

Physical Exam

BP 157/83 | Pulse 72 | Temp 36.9 °C (98.4 °F) | RR 28 | SpO2 98% | BMI 35.04 kg/(m²)

HEENT: JVP ~10cm at 45 degrees
CV: irregularly irregular, 3/6 high pitched systolic murmur
CHEST: Mildly tachypneic, bibasilar crackles in the mid-lung fields
ABD: soft, non-tender, non-distended
EXT: 2+ edema to the knee b/l L>R
SKIN: warm

Lab Interpretation

Na 128 - chronic baseline
K 4.6, Mg 1.9
Cr 0.88 - baseline
LFTs = AST 179/ALT 130, ALP 178, TBili 1.6, INR 1.45
Trop 0.25
BNP 42035 – no priors available for comparison
WBC 11.81, Het 43, Plts 414
TSH 1.5

EKG: Afib with rates in the 100’s. New Q waves in V2-5. No ST elevations or T wave inversions.

Echocardiogram 3/5/2015
1. The left ventricular cavity size is normal.
2. The LV systolic function is severely decreased.
   - The entire apex, mid and apical anterior wall, mid and apical inferior septum, and mid and apical inferior wall are akinetic.
3. Severely reduced RV systolic function.
4. Compared to the most recent exam dated,12/29/2014,there has been an immense decrease in the LVEF.

Echocardiogram 3/10/2015
1. The left ventricular cavity size is normal.
2. The LV systolic function is normal.
3. Visually estimated left ventricular ejection fraction is 55 - 60%.
4. Left ventricular systolic thickening is normal in all segments.
5. Compared to the most recent exam dated,03/05/2015,the LVEF has improved.

Imaging

Figure 1. Coronary angiogram without significant coronary obstruction. No ventriculogram available

Figure 2. Echocardiogram showing LV apex ballooning concerning for Takotsubo’s stress cardiomyopathy

Figure 3. Echocardiogram showing LV apex ballooning concerning for Takotsubo’s stress cardiomyopathy

Hospital Course and Follow-up

- Work-up for new diagnosis of decompensated heart failure was initiated.
- Coronary angiogram was negative for obstructive coronary disease
- Out of concern that recent abrupt cessation of beta blockers may have precipitated the event she was restarted on beta blockade on day 1 of admission.
- On day 5 of admission her symptoms had improved dramatically.
- Repeat thoracic aortic echocardiogram just 5 days after admission showed complete normalization of her LV and RV systolic function.

Teaching Points

Stress cardiomyopathy is an increasingly reported condition that is usually characterized by transient systolic dysfunction, typically lasting 7 days. Its pathogenesis is not entirely known but is thought to be partially due to catecholamine surge resulting in microvascular spasm and myocardial stunning.

This patient’s troponin was not significantly elevated nor did she have ischemic changes on EKG which is typical of Takotsubo’s. However given the rapid resolution of her systolic dysfunction only 5 days after re-initiation of beta blockade her presentation was felt to be most consistent with stress cardiomyopathy from acute beta blocker withdrawal rather than tachycardia mediated which typically takes longer to resolve.

While most cases have been associated with emotional stressors there are increasing case reports of stress cardiomyopathy associated with beta-blocker withdrawal thought to be due to acute catecholamine surge.

Considered to be a condition that largely involves the left ventricle. R ventricular involvement is common and has been associated with more severe LV dysfunction.

It is a class I recommendation to continue beta-blockers pre-operatively but our case indicates that abrupt withdrawal of beta-blockers by have detrimental effects for patients and delay necessary surgeries. There is little data on pre-operative planning after a patient is diagnosed with stress cardiomyopathy. Ultimately this patient was advised to hold surgery for at least 1 month after this event.

She tolerated her surgery well without difficulty.

References