To be or not to be a Type II NSTEMI

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Introduction

- Myocardial ischemia is characterized by rise and/or fall of cardiac biomarkers (i.e troponin) plus one of the following: symptoms of ischemia, new ischemic ECG changes, pathological Q waves, or imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
- Type 2 Non-ST elevation myocardial infarction (NSTEMI) is a common diagnosis in hospitalized patients. Type 2 has been reported up to 25% of cases of MI depending on the population studied. Type 2 NSTEMI is defined as myocardial ischemia resulting from mismatched myocardial oxygen supply and demand that is not related to unstable coronary artery disease (CAD).

Reduced Myocardial Perfusion

- Coronary artery spasm, microvascular dysfunction
- Coronary embolism
- Coronary artery dissection
- Sustained bradyarrhythmia
- Hypotension or shock
- Respiratory failure
- severe anemia

Increased Myocardial Oxygen Demand

- Sustained tachyarrhythmia
- Severe hypertension with or without LVH

Table 1: Etiologies of Type 2 NSTEMI

- Type I NSTEMI is due to unstable CAD with atherosclerotic plaque disruption resulting in a coronary thrombus & subsequent ischemia.
- Differentiation between Type I and Type 2 NSTEMI can be critical as it will guide management. Type I NSTEMI employs anti-platelet and antithrombotic therapies i.e percutaneous coronary intervention. Treatment of Type II NSTEMI is directed at managing the underlying condition.

Case Description

- A 63-year-old-man with history of ischemic cardiomyopathy with 2 prior stents presents to the ED with 1 week of progressive weight gain, lower extremity and abdominal edema, dyspnea, and subacute substernal chest discomfort at rest. Workup reveals significant volume overload with bilateral pleural effusions, pulmonary edema, and anasarca. Also found to have acute renal failure necessitating admission to the ICU for possible line placement and urgent dialysis for decompensated heart failure.
- **Past medical history:**
  - 3 vessel CAD s/p DES x2
  - Renal cell carcinoma s/p left nephrectomy
  - stage 4 CKD
  - COPD
  - Type 2 diabetes mellitus.

Clinical Course

ICU Admission

- Labs: troponin T 0.43ng/L, NT-pro BNP 40,559pg/mL, K 5.5, Cr 4.2, BUN 87
- Electrocardiogram demonstrated ST flattening in V1, V2, AVL, AVF.
- Chest radiograph: stable cardiomegaly, pulmonary edema, and small bilateral pleural effusions
- Received 325mg ASA and IV 80mg furosemide in the ED.
- NSTEMI believed Type II physiology due to volume overload and decreased troponin clearance in the setting of acute renal failure.

Hospital Course

Hospital Day 1:

- Labs: Troponin T 0.35ng/L, K 5.3, Cr 3.9, BUN 85
- Comparison with ECG 7 months prior to admission found anteroseptal Q waves and loss of R-wave progression in precordial leads.

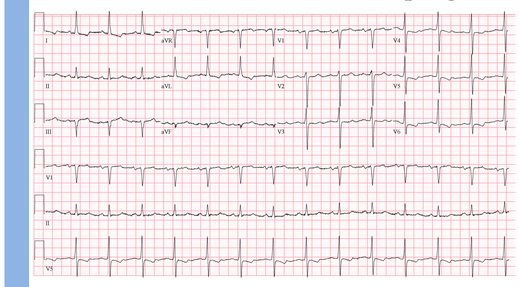

Figure 1: ECG 7 months prior to admission demonstrating normal R-wave progression

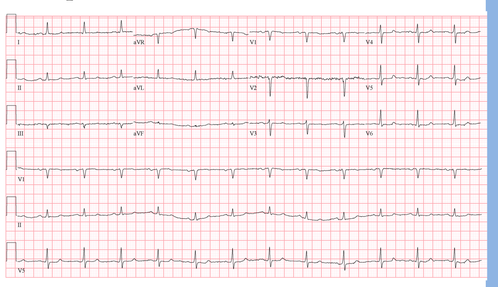

Figure 2: Admission ECG with loss of R-wave progression in precordial leads.

- Chlorothiazide augmentation to furosemide drip resulted in robust diuresis; decreasing concern for urgent HD.

Hospital Day 2:

- Transthoracic echocardiogram showed unchanged left ventricular systolic function with ejection fraction 45-50% and hypokinetic inferior and inferolateral LV wall segments, unchanged from prior.

Hospital Days 3-20:

- Patient was subsequently diuresed with IV furosemide, beta-blocker up-titration, and nitrate initiated for optimal medical management of his three vessel CAD and systolic heart failure.

Hospital day 21:

- Discharged home at which time Troponin T was 0.33ng/L and Cr 2.3 (near patient's baseline).

Discussion

- It is a challenge to differentiate between Type II NSTEMI and subacute Type I NSTEMI in the setting of decompensated CHF and acute renal failure with persistently elevated cardiac biomarkers.
- Per the CURE study, dual-antiplatelet therapy does reduce CV mortality, but increases major bleeding risk.
- It was determined that the risks outweigh the benefits thus Clopidogrel, heparin, and cardiac catherization were held given the possible need for hemodialysis. Also, ACS-NSTEMI management would have little benefit as the infarct would have been days old.

Discussion (cont.)

- Ultimately, this case was treated as a Type II NSTEMI by correcting volume overload. It was suspected that the decompensated heart failure was not due to a new ischemic event, but rather gross volume overload secondary to acute renal failure and medication nonadherence.

Take Home Points

- Patients can present with a mixed picture in regards to Type I vs type II NSTEMI, and treatment strategies are frequently limited by other co-morbidities.
- EKG and TTE changes from prior studies can aid in discerning the etiology. Coronary angiography is more definitive to establish the diagnosis, but not always clinically indicated.
- Studies have shown that invasive treatment strategies and cardioprotective medications are less frequently used in Type 2 NSTEMI, while maintaining similar clinical outcomes compared to Type-1 NSTEMI.
- While ACS-NSTEMI type I have clear guidelines for treatment, there is still a need for evidence-based diagnostic and therapeutic strategies for Type 2 NSTEMIs.

References

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