Just A Broken Heart?

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INTRODUCTION

Transthoracic echocardiogram (TTE) is an easily available and rapid tool for identification and risk stratification of occlusive myocardial infarction (OMI) and acute heart failure. While an EKG meeting STEMI criteria triggers immediate evaluation and intervention, there can be significant delay in diagnosis and care of patients with inconclusive EKGs or no ST elevations. Studies have found regional wall motion abnormalities (RWMA) present in > 50% of OMIs presenting as NSTEMIs. Diagnosis of MI by RWMA identification occurred about 45 minutes prior to troponin results, and led to significantly decreased arrival-to-revascularization time. (1) When trained appropriately, ED physicians can reliably and accurately identify RWMA. When STEMI cases are included in retrospective analysis, POCUS identified 87% of cath-proven OMIs. (2) The ED physician must pay attention to patterns of coronary artery supply and be aware of conditions that mimic RWMA from acute coronary ischemia, a differential discussed in this case.

CASE

A 70-year-old female with a history of hypertension, diabetes, and hyperlipidemia presented to the ED with 1 week of new onset shortness of breath. She denied any current or previous chest pain, and had never had symptoms like this before. She returned from her home country about 3 weeks ago after visiting for her mother's funeral, and she has had stress, fatigue, and decreased appetite since that time.

Physical Exam:

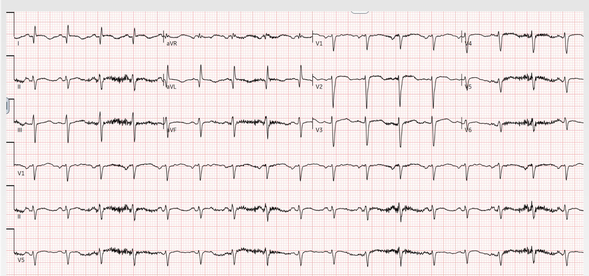
Vital Signs: Temp 97.9F, BP 147/93, **HR 108, RR 22**, O2 sat 98% on RA

General: Alert, awake, oriented x3. Patient is not in distress but is **tachypneic** to low 20s and has slight **increased work of breathing**

Pulmonary: Crackles throughout and decreased breath sounds at the bases

CV: No JVD, no lower extremity edema. Heart tachycardic and regular. No murmurs or gallop

EKG: **Sinus Tachycardia** at 108 bpm, left anterior fascicular block, T-wave inversion in I and aVL, Poor R-wave progression, **subtle and dynamic 0.5-1 mm STE in V2 and V3**. No prior available.



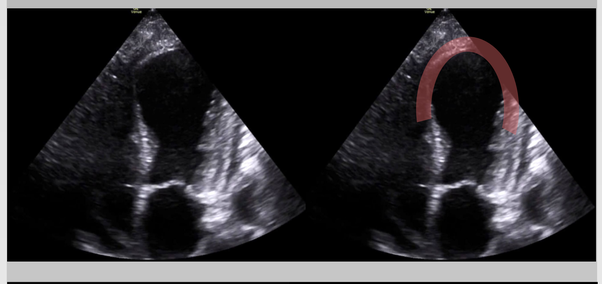
POC TTE was performed within minutes of initial evaluation (see QR Video)

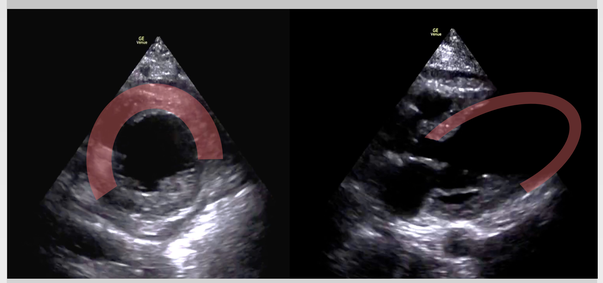
Troponin 0.322 (reference <= 0.010)

BNP: 11,856

CXR: Enlarged cardiac silhouette, pulmonary edema, bilateral pleural effusions

TTE FINDINGS





CLINICAL QUESTIONS

Q: What are 3 differential diagnoses for this echo?

A:
Acute heart failure caused/complicated by:
a. Takotsubo (stress) cardiomyopathy (TCM)
b. Acute MI/ischemic cardiomyopathy
c. LV aneurysm

Q: How does prognosis and management differ for each of these diagnoses?

A:
TCM: Supportive treatment and avoidance of catecholamine medications; good candidate for early LVAD or ECMO if unstable; good prognosis (all patients require cath for ischemic rule out)
Acute MI: STAT cardiac catheterization, anticoagulation; prognosis tenuous
LV aneurysm: anticoagulation, consider surgical evaluation

CASE RESOLUTION

- Patient was transferred to a catheterization-capable center and found to have 100% occlusion of the proximal LAD
- Due to thinning of the ventricular wall and timing of symptoms, ischemia was determined to be subacute and PCI was not performed
- Follow up official TTE 3 months after initial ED visit showed slight improvement of EF from 10% to 25% and an ICD was placed several months ago

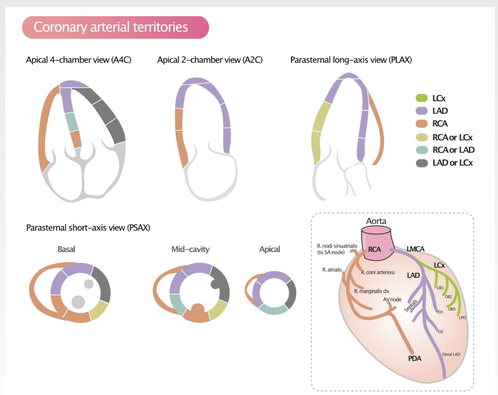
DISCUSSION

Proximal LAD (pLAD) occlusion, Takotsubo cardiomyopathy (TCM), and left ventricular (LV) aneurysms can share TTE findings of apical dilation and circumferential apical hypokinesis. (3, 4) pLAD occlusion impacts the anteroseptal and occasionally lateral and inferior walls. TCM commonly affects the apex, but can cause akinesis of any nonspecific distribution. pLAD occlusion and TCM can lead to similar initial presentations of chest pain, dyspnea, troponin elevations, and ST segment elevations on EKG (5). Being familiar with coronary artery distribution correlates on TTE can help differentiate between occlusive MI and other nonspecific cardiomyopathic conditions. Pathology that crosses over multiple coronary supply distributions is less likely to be caused by OMI. However, this case demonstrates how extensive pLAD occlusion ischemia can be.

TCM is theorized to be a temporary myocardial injury due to catecholamine surges, and thus may occur in the absence of underlying coronary artery disease. (5) While cardiac catheterization is recommended in suspected TCM cases to definitively rule out OMI, the prognosis and management of immediate complications can differ greatly from pLAD occlusions. TCM carries an annual 3.2% mortality rate and most patients will recover to full baseline cardiac function within about 3 weeks. (9, 10, 11) In contrast, pLAD occlusions hold a short-term in-hospital mortality rate of 6.7-8% and will more emergently require immediate cardiac catheterization and/or thrombolytic administration. (12, 13)

REGIONAL WALL MOTION ABNORMALITIES

- Official cardiology TTE RWMA evaluation involves assessment of 17 segments of the LV wall - this exam is too extensive for ED usage
- POCUS assessment will include
  - PLAX
  - PSAX in at level of mitral valve, mid-papillary, and apical levels
  - A4C +/- addition of A2C
- Look for equal **wall excursion** to the center of the ventricular cavity and appropriate **wall thickening**
- Correlate your findings with EKG changes



https://ecgwaves.com/topic/the-coronary-arteries/

TAKE HOME POINTS

1) In patients presenting with new cardiac symptoms, POC TTE can identify global or regional structural abnormalities and can provide rapid diagnosis of OMI.
2) TTE with apical ballooning and hypokinesis may be due to either TCM or pLAD occlusion. It is important to consider both diagnoses, as they present similarly on history, EKG, and TTE, but have different prognosis, rates of complications, and managements.
3) Know your coronary artery distribution patterns on TTE, as OMIs are more likely to follow these patterns, while TCM, aneurysms, and other nonischemic cardiomyopathies may cross standard artery distribution lines.

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ACKNOWLEDGEMENTS

Thank you to Dr. Allison Lee for support and guidance on this case report and to the Ultrasound Department at Kings County Emergency Department for continuous encouragement to expand the use of POCUS in our department.