College of Human Medicine
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A Case of COVID-19–Induced Takotsubo Cardiomyopathy Mimicking STEMI

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Background

- Takotsubo cardiomyopathy (stress cardiomyopathy) is a transient systolic dysfunction of the left ventricle, typically triggered by emotional or physical stress[5]
- Mechanism: Caused by excessive catecholamine release causes myocardial stunning without infarction[5]
- Relevance:
  - COVID-19 infection can act as both a physiological and emotional stressor, precipitating stress cardiomyopathy[5]
  - A study of 1.65 million hospitalized COVID-19 patients found a 0.1% incidence of stress cardiomyopathy[1]
- Clinical importance: often mimics acute coronary syndrome (STEMI) which can lead to unnecessary interventions if not recognized[4]

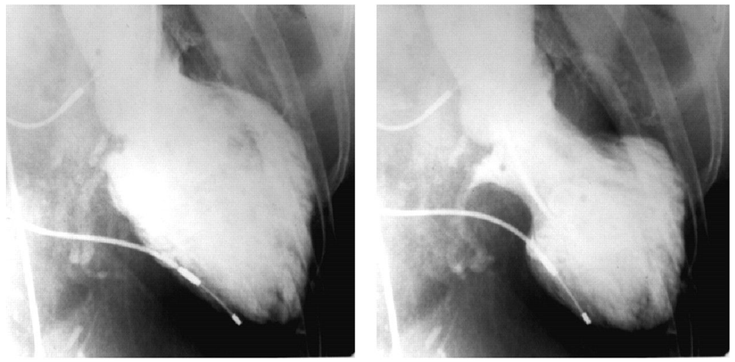

Figure 1. Typical findings for stress cardiomyopathy with apical ballooning
Adapted from Prasad A et al., Circulation. 2008;118(25):2754–2762.

Case Presentation

- Patient: 82-year-old woman with no significant cardiac history
- Chief Complaint: Dyspnea on exertion lasting ~8 minutes and cough for 3 days
- Recent exposure: sick contact with husband; COVID-19 positive
- Initial findings:
  - Troponin uptrending from 63 to 3779 ng/L
  - BNP: normal
  - CXR: small pleural effusion
  - EKG: ST elevations in I/aVL (lateral leads) and ST depressions in III/aVF (inferior leads)
- Initial management
  - Started on heparin for presumed STEMI
  - Urgent cardiac catheterization demonstrated mild non-obstructive CAD
- Echocardiogram: reduced left ventricular ejection fraction with apical akinesis, consistent with stress cardiomyopathy

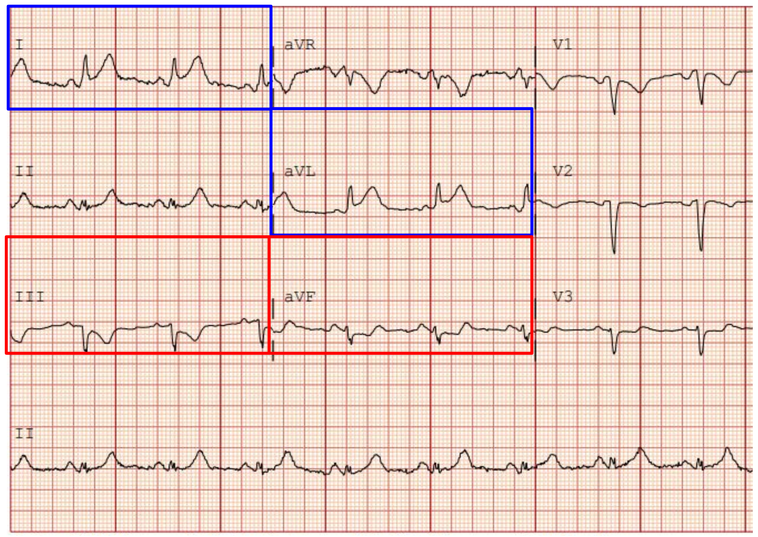

Figure 2. EKG findings from the patient's stay during the emergency department
Blue = ST elevations
Red = ST depressions

Intervention

- COVID-19 treatment:
  - Treated with Remdesivir and prednisone 6mg IV
  - Required 1L nasal cannula O2, and was later weaned to room air
- Cardiac management:
  - Initially started on heparin for presumed STEMI, discontinued after catheterization results
  - Began Cozaar 50mg and Toprol-XL 25mg, but later held due to hypotension
  - Continued aspirin 81mg and statin 40mg
  - LifeVest initiated for arrhythmia protection
  - Planned for outpatient guideline-directed medical therapy (GDMT) during follow-up with cardiology for monitoring for heart failure or thromboembolic complications

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Discussion

- Pathophysiology:
  - Catecholamine surge due to a large emotional/physiological stress.
  - COVID-19 virus also causes direct viral myocardial injury via ACE2 receptors.[2]
  - This leads to systemic inflammation and cytokine-mediated myocardial stunning.
- Clinical significance:
  - Presentation can mimic STEMI with similar EKG changes, a rise in troponin, shortness of breath, and chest discomfort[4]
  - Distinguishing features of stress cardiomyopathy include non-obstructive coronary arteries, apical ballooning pattern of ventricles on echocardiogram, and a modest troponin elevation relative to the degree of wall-motion abnormality[5]
- Hypotension in this case limited GDMT protocol but demonstrates the reduced contractility due to transient left ventricular akinesis[1]

Conclusion

- COVID-19 induced stress cardiomyopathy can present similarly to STEMI but shows non-obstructive coronary findings on angiography[4]
- Diagnosis relies on combining clinical, EKG, echocardiographic and catheterization findings
- This case reinforces the importance of considering stress cardiomyopathy in COVID-19 patients presenting with acute coronary features
- Early recognition of COVID-19 as a potential trigger can initiate more accurate diagnosis and tailored management, improving patient outcomes[1]

References

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