A Painful Lift

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Disclosures

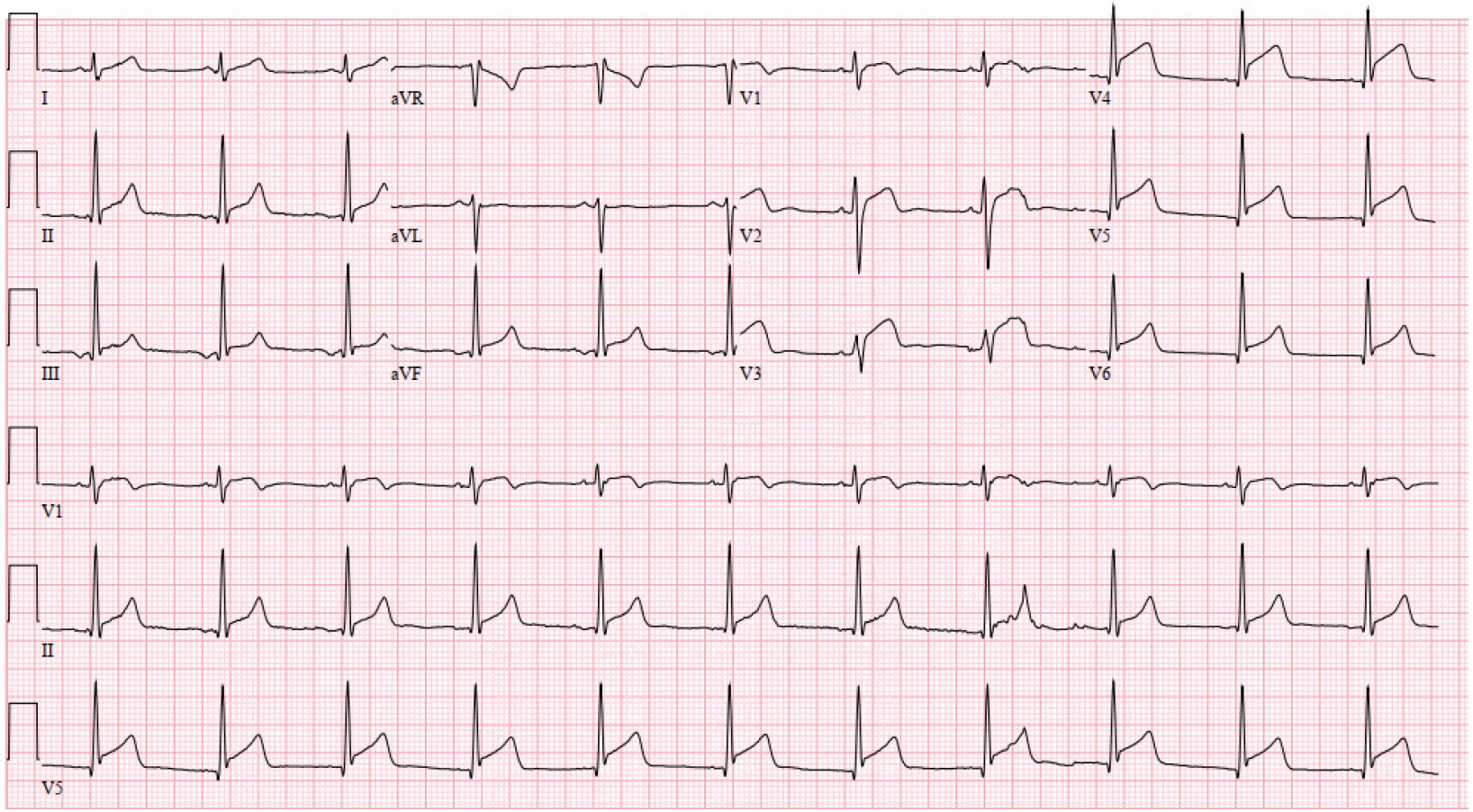
I have no relevant relationships with commercial interests to disclose.

Patient History

- 23-year-old male presented with left sided chest pain of 4 hours duration after lifting weights
- Powerlifter who was doing compound Olympic lifts
- Past medical history of asthma
- Denied any illicit drug use, specifically any anabolic steroid use

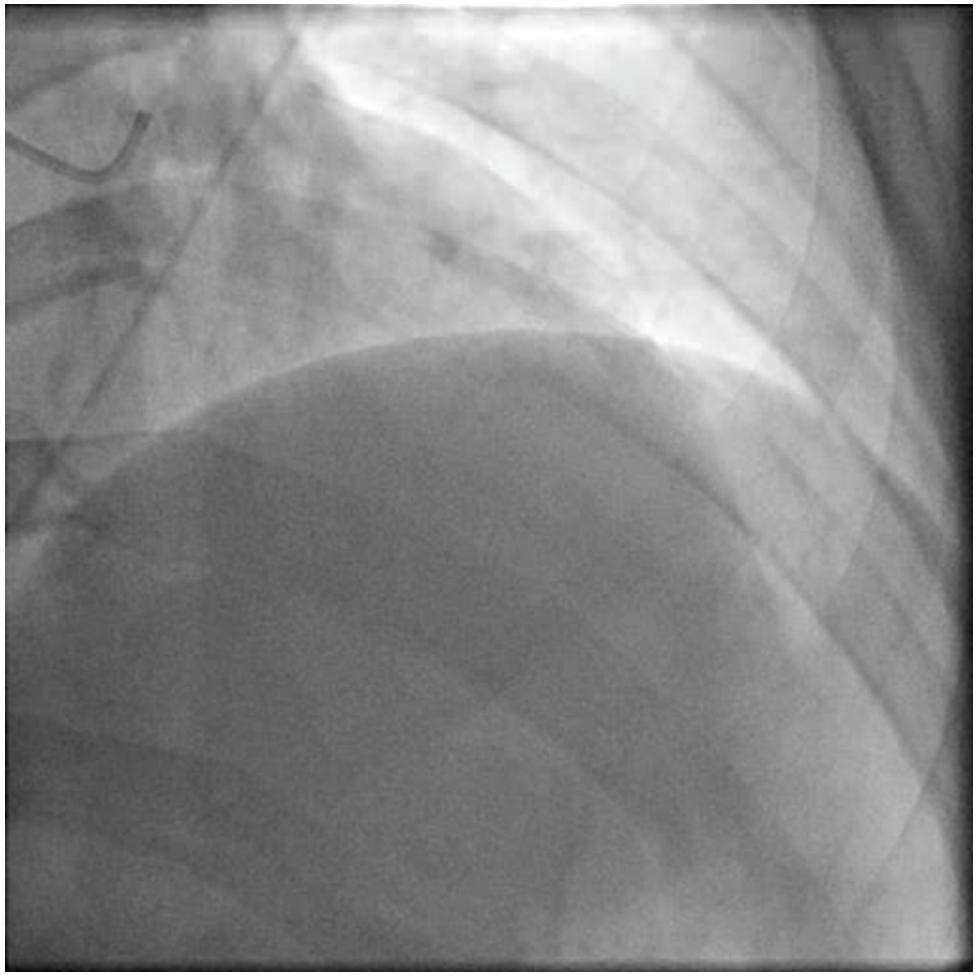
Evaluation

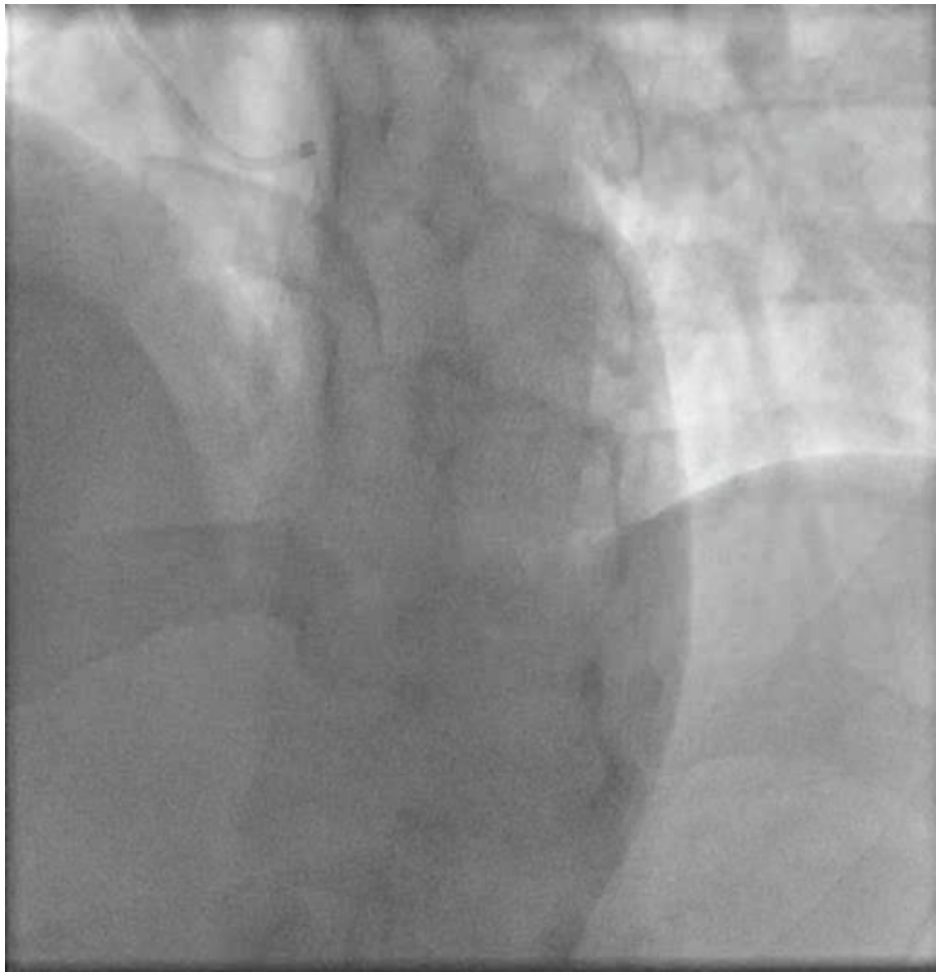
- Initial troponin elevated to 2.3 (nl <0.05 ng/mL)
- ECG revealed sinus rhythm with diffuse anterolateral ST elevation
- Bedside echocardiogram showed apical hypokinesis
- **COVID-19 test positive**



Treatment

- Emergent coronary angiography was performed
- The LAD artery was completely occluded at the mid segment
- Concern for Distal Left main – proximal LAD mural thrombus

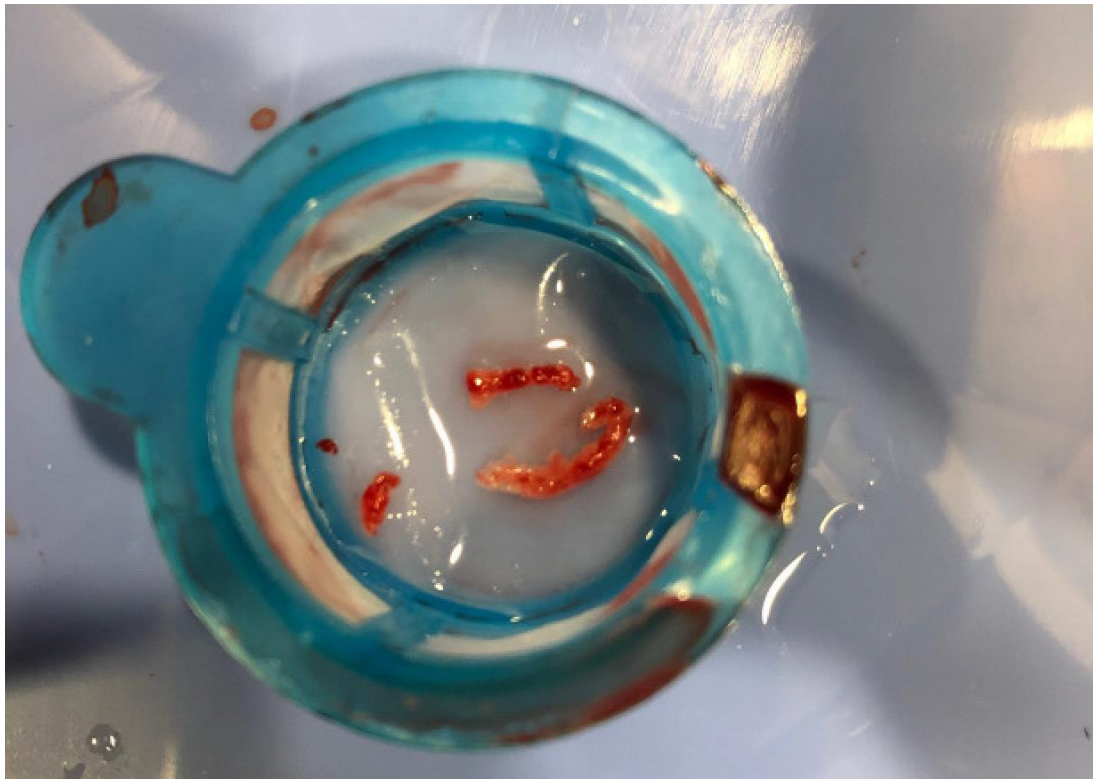


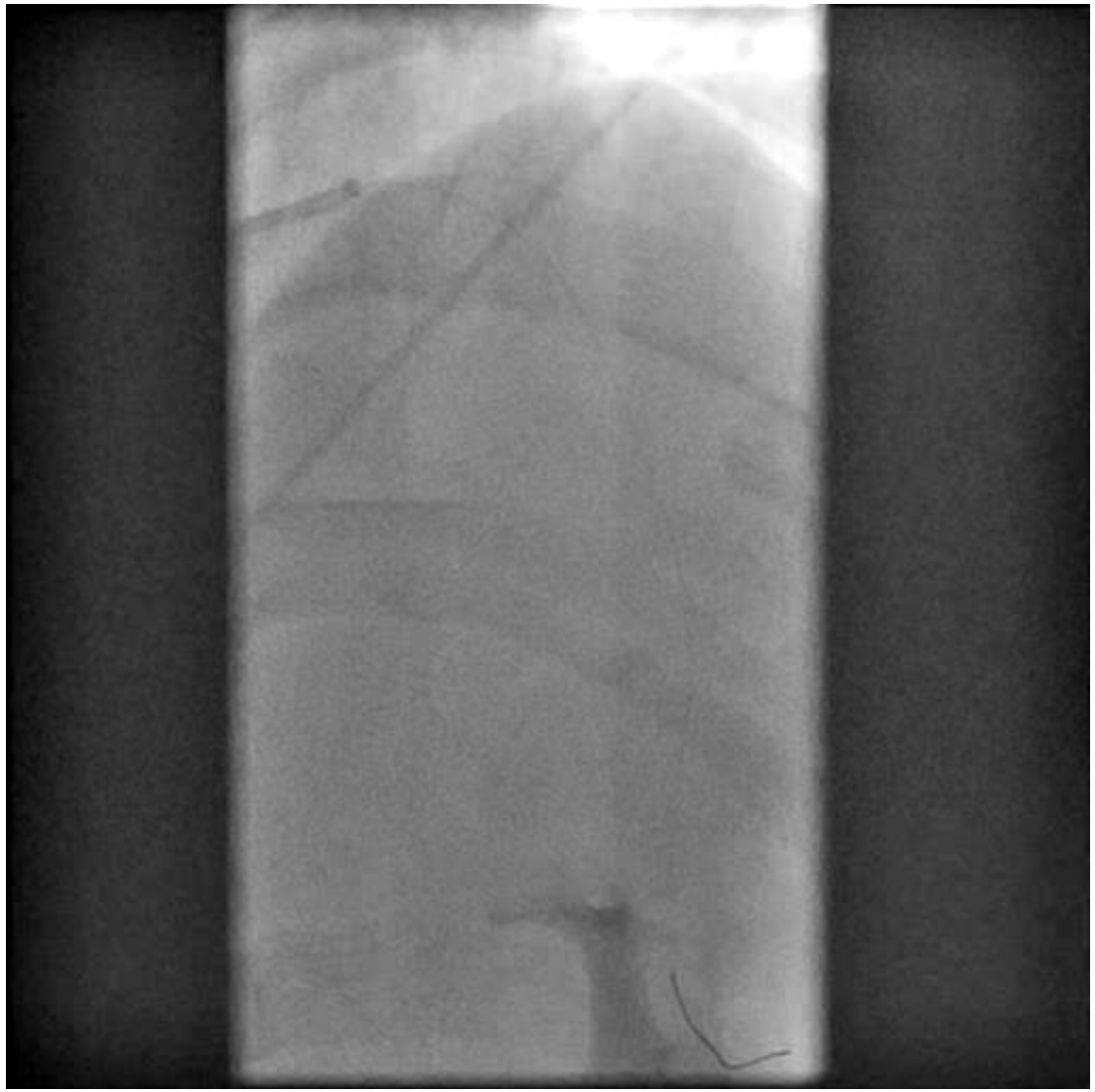


Treatment

- LAD occlusion was easily traversed with Runthrough coronary wire
- Serial inflations with a 2.0 mm X 12.0 mm semi-compliant balloon and several passes of the Export Advance™ aspiration catheter were unable to restore flow
- Mechanical power aspiration with the Indigo® System CAT™ RX (Penumbra, Inc.) was successful in restoring flow with significant thrombus removal (next slide)
- Apical LAD showed evidence of reduced flow, which was treated with multiple intracoronary injections of nitroglycerin, adenosine, and verapamil were performed
- No coronary stent placed as there was no evidence of an underlying lesion after thrombectomy

Treatment





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Hospital Course and Follow-Up

- Eptifibatide continued for 18 hours post thrombectomy
- Transthoracic echocardiogram revealed normal left ventricular ejection fraction with apical wall motion abnormality
- Hypercoagulable work up (Factor V Leiden, Antiphospholipid, Prothrombin) was negative
- Discharged on Aspirin 81 mg, Ticagrelor 90 mg BID and Apixaban 5 mg BID for one month, then Apixaban stopped
- Doing well at one month follow up in clinic

Question

- What is the proposed mechanism behind acute coronary syndrome in COVID?

    a. Direct endothelial disruption
    b. Cytokine storm
    c. Activated macrophages leading to plaque rupture and hypercoagulablity
    d. All of the above

Correct Answer, Rationale, and Reference

D. All of the above

- Histologic studies have shown that there is direct viral infection of the endothelial cells and diffuse inflammation
- Activated macrophages secrete collagenases that degrade collagen, a major constituent of the fibrous cap on atherosclerotic plaques, which can lead to plaque rupture. Macrophages also secrete tissue factor, which is a potent procoagulant
- Cytokine storm leads to systemic inflammation

Conclusions and Learning Points

- Need to have a high index of suspicion for acute thrombotic occlusion in patients with COVID-19 and ECG changes regardless of age or other typical symptoms of viral syndrome
- Multiple proposed mechanisms for hypercoagulability and plaque disruption with COVID-19
- Best management of these patients with intracoronary thrombosis related to viral syndrome is not well-defined but can usually encompass aspiration thrombectomy, therapeutic anticoagulation and dual antiplatelet therapy