Acute Thrombosis of the Circumflex and Right Coronary Arteries in the Setting of a COVID-19 Positive Patient

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BACKGROUND

The relationship between Covid-19 and inflammatory mediators (CRP, IL-1, IL-6, and TNF α) is associated with hypercoagulability.[1] There are few case reports that describe an association between acute coronary syndrome (ACS) and Covid-19 infection. This case highlights the potential cardiovascular impacts of a Covid-19 infection, specifically ACS. Here we illustrate the importance of anticipating possible ACS in those patients who are infected with Covid-19 in order to intervene quickly and improve patient outcomes.

CASE DESCRIPTION

An 84 y/o female with no known past medical history presented with acute nausea, vomiting, and profound weakness. She was hypoxemic and hypotensive requiring emergent intubation and pressors. Covid-19 test resulted positive. Initial EKG was non-diagnostic but later became consistent with posterolateral myocardial infarction. CT chest suggested aspiration pneumonia and was negative for pulmonary embolism. High sensitivity troponin was elevated at 414 pg/mL. Transthoracic echocardiogram confirmed akinesis of the inferior and lateral walls. Cardiac catheterization showed occlusive thrombus of the ostium of the circumflex and multiple filling defects consistent with thrombus in the RCA. Pulmonary capillary wedge pressure was dramatically elevated. Cardiac output was initially preserved.

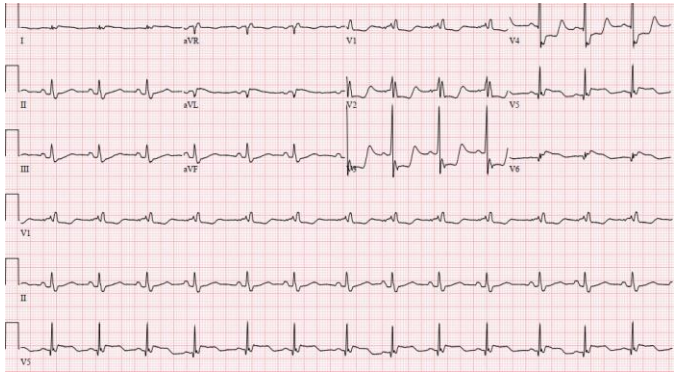

Figure 1: Repeat EKG demonstrating posterior Mi.

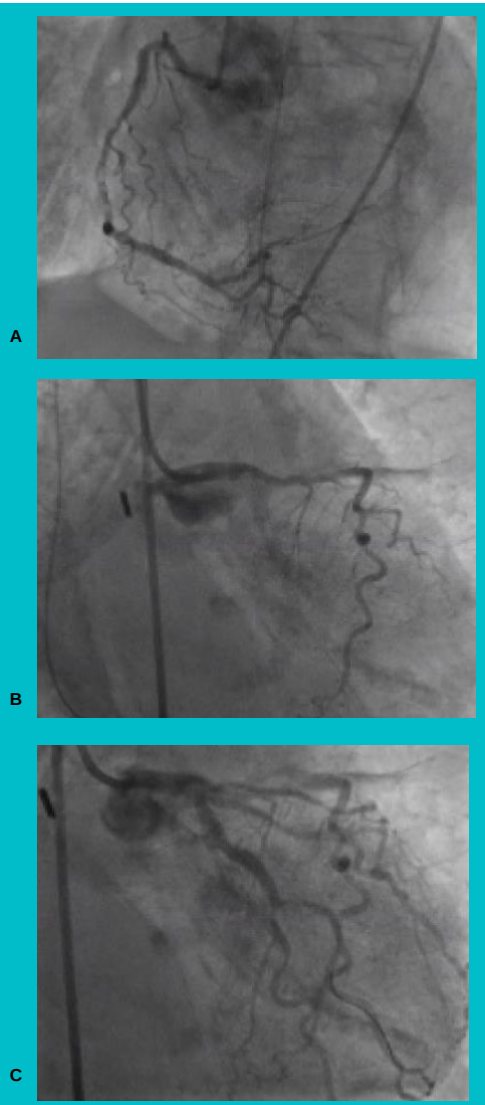

Figure 2: Coronary angiography- **A:** Multiple filling defects of the RCA. **B:** Total occlusion of the ostial left circumflex. **C:** TIMI 3 flow of the left circumflex after DES placement to left main into the circumflex.

DECISION MAKING

Successful PCI and DES placement with restoration of TIMI 3 flow to the left circumflex artery was performed as she was suffering an acute posterolateral STEMI. Despite thrombus burden we opted not to intervene on the right coronary artery as there was TIMI 3 flow. She remained hypotensive on pressors requiring intra-aortic balloon pump placement. Hypoxemia, hypotension, and acidosis continued despite maximal mechanical ventilation and her family transitioned her to comfort measures.

DISCUSSION

Covid hypercoagulability is thought to be driven by inflammatory mediators. A systematic review highlighted the association between Covid and inflammatory mediators (CRP, d-dimer, fibrinogen, IL-1, IL-6, and TNF α) that are known to induce coagulation.[1] More recent data suggests that Covid can increase thrombotic risk by binding to angiotensin-converting enzyme 2 (ACE2).[2] This cascade increases tissue factor expression and endothelial dysfunction [2] This pathophysiology supports the idea that Covid-19 infection may induce acute thrombus triggering acute coronary syndromes as was seen in this patient.

CONCLUSION

This case's high thrombus burden is strongly suggestive of a pro-thrombotic state in setting of COVID infection.

REFERENCES

1 Sara, T., Safah, K., Almasri, M., & Hussein, R., et al (2021). Systemic inflammation in COVID-19 patients may induce various types of venous and arterial thrombosis: A systematic review. *Scandinavian Journal of Immunology*, *94*(5). https://doi.org/10.1111/sji.130972

2 Gorog, D.A., Storey, R.F., Gurbel, P.A. *et al.* Current and novel biomarkers of thrombotic risk in COVID-19: a Consensus Statement from the International COVID-19 Thrombosis Biomarkers Colloquium. *Nat Rev Cardiol* **19**, 475–495 (2022). https://doi.org/10.1038/s41569-021-00665-7