Coronary artery thrombus resulting in ST-elevation myocardial infarction in a patient with COVID-19

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SUMMARY
COVID-19 is a prothrombotic condition that is also associated with raised troponin levels and myocardial damage. We present a case of a 54-year-old man who was admitted with respiratory failure due to COVID-19 and developed a ST-elevation myocardial infarction (STEMI) during his admission. His coronary angiogram did not show any significant coronary artery disease other than a heavily thrombosed right coronary artery. In view of heavy thrombus burden, the right coronary artery was treated with thrombus retrieval using a distal embolic protection device in addition to manual thrombectomy and direct (intracoronary) thrombolysis without the need for implantation of a coronary stent. After successful revascularisation, triple antithrombotic therapy was instituted with an oral anticoagulant in addition to dual antiplatelets. This case illustrates the association of COVID-19 with coronary artery thrombosis, which may require disparate management of a STEMI than that resulting from atherosclerotic coronary artery disease.

BACKGROUND
COVID-19 that is caused by SARS-CoV-2 may cause coagulation abnormalities resulting in increased incidence of venous and arterial thrombotic events. This thrombotic tendency may result from excessive inflammation, endothelial dysfunction and platelet activation. Cardiac injury has also been frequently reported in patients with COVID-19, and high levels of troponin are often recorded in patients with COVID-19 admitted to hospital. A nationwide Danish study of over 5000 patients with COVID-19 identified an increased incidence of acute myocardial infarction (MI) with an incidence ratio of 5.9. Case reports of patients with COVID-19 presenting with ST-elevation MI (STEMI) show a higher proportion of patients without an identifiable culprit coronary lesion. Increased rates of cardiac multivessel thrombosis were also reported by one study in such patients. We present a case of a patient admitted with respiratory failure due to COVID-19, who developed a STEMI as an in-patient with angiographically normal coronary arteries as a result of coronary artery thrombosis.

CASE PRESENTATION
A 54-year-old man was admitted under acute medicine with a 2-week history of breathlessness, chest tightness, fever and dry cough. His wife had tested positive for SARS-CoV-2 15 days prior to admission. He had a history of mild asthma but was not receiving treatment for this and was usually fit and well. On examination, he was tachycardic and tachypnoeic with a respiratory rate of 27 breaths per minute and oxygen saturations of 96% on 8 L of oxygen. He had crackles bilaterally consistent with COVID-19 pneumonitis. He was transferred under the care of respiratory medicine. Haemoglobin A1c (HbA1c) testing suggested undiagnosed type 2 diabetes mellitus. He was started on regular nebulisers due to wheeze to cover an exacerbation of asthma. On day 2 of the admission, he became profoundly hypoxic with oxygen saturations of 89% on 15 L of oxygen. He described central and right-sided chest pain worse on inspiration. An ECG showed ST elevation in the inferior leads and an urgent referral to cardiology was made. He was transferred to the cardiac catheterisation laboratory for primary percutaneous coronary intervention (PCI).

INVESTIGATIONS
PCR swab confirmed that this patient was positive for SARS-CoV-2. Blood tests from admission showed a white cell count of 11.1×10⁹/L, D-dimer of 414 mg/mL and C reactive protein of 176 mg/L. The admission chest X-ray showed bilateral extensive patchy infiltrates consistent with COVID-19 pneumonitis. After treatment with dexamethasone, blood glucose levels were repeatedly greater than 12 and HbA1c was 50 mmol/mol consistent with undiagnosed type 2 diabetes mellitus. On day 2 of the admission, his ECG showed ST elevation in the inferior leads: II, III, aVF (augmented vector foot) with ST depression in aVL (augmented vector left) and V6.

After identification of ST elevation on the ECG, this patient was transferred urgently to the cardiac catheterisation laboratory. His coronary angiogram showed patent left main stem, left anterior descending and circumflex arteries without any significant coronary artery disease. His right coronary artery was dominant and large with an occlusive large thrombus burden mid vessel (video 1).

An echocardiogram performed after his angiography showed an ejection fraction of 45%–50% with inferolateral hypokinesia.

We discussed this case with haematology who did not think a coagulopathy screen was clinically indicated and there was no clinical suspicion of hyperhomocysteinaemia. A lupus screen was requested, which was negative.

TREATMENT
The patient received dexamethasone for severe COVID-19 pneumonitis as per guidelines’ with nebulisers to treat his asthma and humulin I to treat his type 2 diabetes mellitus.
After coronary angiography, a distal embolic protection device was placed in the distal right coronary artery and thrombus was aspirated with a manual thrombectomy device. Thereafter, the residual thrombus was captured by distal embolic protection device followed by intracoronary alteplase delivered directly into the distal right coronary artery with a microcatheter. Thrombosis in myocardial infarction (TIMI)-3 flow (TIMI 0 at baseline, complete perfusion) was established with this treatment with very minimal distal embolisation (video 2). He commenced triple antithrombotic therapy with an anticoagulant in addition to dual antplatelets. He was started on an insulin sliding scale intravenously.

After treatment of his coronary thrombosis, this man was initially transferred to the coronary care unit. However, he had another episode of desaturation, and after a short period of treatment with continuous positive airway pressure, he required intubation and was transferred to the intensive care Unit.

OUTCOME AND FOLLOW-UP
This man was discharged from hospital 14 days after admission. Due to his infiltrates on chest X-ray, a repeated chest X-ray will be performed after 3 months to ensure resolution of his chest X-ray changes. As his STEMI was a result of thrombosis, he will be anticoagulated with apixaban for 3 months with 1 year of aspirin and clopidogrel. A bubble echocardiogram will be performed as an outpatient as it is possible his STEMI resulted from an embolus, and he will be followed up by cardiology.

DISCUSSION
Raised troponin levels suggesting myocardial injury happen relatively frequently in patients hospitalised with COVID-19 occurring in over 30% of patients. One study concluded that patients were at five times increased risk for acute MI during the 14 days after a positive swab for SARS-CoV-2. However, the evidence for STEMI in the context of COVID-19 primarily comes from case reports. These reports suggest that a relatively high proportion (30%-40%) of patients with STEMI and COVID-19 have unobstructed coronary arteries. The patient in this case had angiographically normal coronary arteries other than his coronary artery thrombus. This combined with the evidence that COVID-19 is a prothrombotic state might suggest that the cases of STEMI with unobstructed coronary arteries seen in other studies may have resulted from coronary artery thrombus formation. Supporting this, one study of 39 patients with STEMI and COVID-19 at a coronary intervention centre showed higher levels of D-dimer in these patients in comparison to COVID-19-negative patients with STEMI. The patient in this case also had a raised D-dimer at admission. Similarly to this patient, patients in this study were more likely to have thrombus within coronary vessels if they were positive for SARS-CoV-2.

Similarly, the study found that a greater proportion of patients with COVID-19 presenting with STEMI required thrombus aspiration in comparison with patients with STEMI who were SARS-CoV-2 negative.

STEMI resulting from coronary artery thrombus in comparison with coronary artery obstruction and plaque rupture has implications for the treatment of patients. In this case, thrombus was aspirated, both with conventional catheter-based aspiration and retrieval with the help of distal embolic protection device. In addition, intracoronary alteplase was used for direct thrombolysis delivered via a microcatheter to achieve sustained TIMI 3 flow without need for stenting. Recognising COVID-19 as prothrombotic condition, he was also started on therapeutic anticoagulation, which is not traditionally used in the management of classical STEMI, resulting from coronary artery disease.

In conclusion, when managing patients with STEMI who have COVID-19, it is important to be aware that patients may not have obstructive coronary disease, and coronary artery thrombus may be the underlying precipitant rather than plaque rupture.
STEMI as a result of vessel thrombosis may require different management to STEMI, resulting from coronary artery disease and may require thrombus aspiration, intracoronary thrombolysis and a period of therapeutic anticoagulation.

**Learning points**

► Patients presenting with ST-elevation myocardial infarction who test positive for SARS-CoV-2 may have developed myocardial ischaemia from coronary artery thrombosis rather than plaque rupture.

► Management of patients with coronary artery thrombus may require thrombus aspiration and direct coronary artery thrombolysis rather than stent insertion at percutaneous coronary intervention.

► Coronary artery thrombus is a sign of a thrombotic tendency and a period of therapeutic anticoagulation should be considered in these patients.

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**REFERENCES**


