COVID-19 and ST elevations–keep an open mind: a case report

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Background

Coronavirus disease 2019 (COVID-19) has been associated with a range of cardiovascular manifestations, including myocardial injury and thrombo-embolism. Pulmonary embolism (PE) causing anteroseptal/anterior ST elevations that mimic myocardial infarction have previously been described. This phenomenon is thought to be related to right ventricular injury from large emboli.

Case summary

A 48-year-old woman with history of type 2 diabetes mellitus and hypertension presented to her local hospital with fever, cough, nausea, and dyspnoea. A test for SARS-CoV-2 was taken, and she was discharged with instructions to self-quarantine. She was subsequently notified of a positive SARS-CoV-2 result. Three days later, she represented with worsening dyspnoea and respiratory failure requiring intubation. On hospital Day 6, she became acutely hypoxic and hypotensive. Telemetry was noted to have ST changes, prompting ECG that revealed sinus tachycardia with prominent new ST elevations in her precordial leads. Transthoracic echocardiogram showed normal left ventricular function; however, the right ventricle was moderately dilated with positive McConnell’s sign. Due to her unstable clinical state and high suspicion for PE, she was treated with tenecteplase 50 mg i.v. with complete resolution of her ST elevations and improved oxygenation.

Discussion

Given the high rates of thrombo-embolic events in COVID-19 patients, PE should be in the differential diagnosis of ST elevation, particularly in younger patients with few risk factors for coronary artery disease.

Keywords

COVID-19 • ST elevation • Pulmonary embolism • Thrombolysis • Case report

Introduction

Coronavirus disease 2019 (COVID-19) has been associated with a range of cardiovascular manifestations, including myocardial injury and thrombo-embolism.1,2 Higher incidences of arterial and venous thrombo-embolisms are thought to be due to inflammatory cytokines, hypoxia, immobilization, and diffuse intravascular coagulation.3,4 PEs causing anteroseptal/anterior ST elevations that mimic myocardial infarction have previously been described. This

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phenomenon is thought to be related to right ventricular injury from large emboli.6–9

Here, we describe a case involving a critically ill patient with acute decompensation following new ST elevations secondary to suspected PE, and successfully treated with thrombolitics.

**Timeline**

| Index day of illness | Patient reported first experiencing symptoms of fever, cough, nausea, and dyspnoea |
|----------------------|--------------------------------------------------------------------------------|
| Day 3 of illness     | Presented to community hospital for initial evaluation. Was discharged following COVID-19 testing. Instructed to self-quarantine. |
| Day 8 of illness     | Re-presented to hospital for worsening symptoms. Found previous COVID-19 test positive. |
| Day 9 of illness     | Required intubation for acute respiratory distress syndrome. |
| Day 14 of illness    | Patient became acutely hypoxic and hypotensive. Additionally, ST elevation observed on telemetry and electrocardiogram prompted immediate evaluation. Treated with systemic thrombolysis. + 4 hours, repeat ECG with resolved ST elevations and improved oxygenation. Continued treatment with anticoagulation. |
| Day 42 of illness    | Died from nosocomial respiratory infection following prolonged ventilation. |

**Case presentation**

A 48-year-old woman who was a healthcare worker with history of type 2 diabetes mellitus, hypertension, and obesity [body mass index (BMI) 32] presented to her local hospital with several days of fever, cough, nausea, and dyspnoea. Her home medications were metformin 750 mg daily and lisinopril 40 mg daily. A test for SARS-CoV-2 was taken, and she was discharged with instructions to self-quarantine. She was subsequently notified of a positive SARS-CoV-2 result. Three days later, she presented to our hospital with worsening dyspnoea on mild exertion.

On examination, she was tachycardic, tachypnoeic, and hypoxic with oxygen saturation 79% on room air that improved to 93% with a 4 L nasal cannula. Auscultation revealed no cardiac murmur of significant crickles on lung fields. Initial laboratory evaluation was notable only for mild leucocytosis of 11 700/mL (reference range 4000–11 000/mL), D-dimer 432 ng/mL (normal <500 ng/mL), and estimated glomerular filtration rate (eGFR) of 106 mL/min (normal low >60 mL/min), while chest X-ray showed bilateral patchy airspace opacities. She was initially admitted to a medical ward for treatment of pneumonia. She then developed progressive respiratory failure and was therefore transferred to the intensive care unit (ICU) and intubated for presumed acute respiratory distress syndrome. Her hospital course was further complicated with non-oliguric acute renal failure.

The patient’s clinical status remained unchanged until hospital Day 6, when she became hypotensive requiring vasopressor support with norepinephrine, and developed worsening hypoxia despite 100% FiO₂ and PEEP of 12 mmHg. Telemetry was noted to have ST changes, prompting ECG that revealed sinus tachycardia with prominent new ST elevations in precordial leads V2–V5 (Figure 1A). This was markedly different from her baseline ECG on presentation (Figure 1B). She also had sinus tachycardia and an STQ3T3 pattern on ECG. High-sensitivity troponin T returned at 138 ng/L. Transthoracic echocardiogram (TTE) showed normal left ventricular function with no left ventricular wall motion abnormalities (Supplementary material online, Movie A and B). However, the right ventricle was dilated, with mid diameter measuring 5.1 cm, and severely hypokinetic in the base and mid segments, but with preserved apical wall motion, consistent with McConnell’s sign (Supplementary material online, Movie C). Estimated right ventricular systolic pressure was 45 mmHg. There was no atrial septal defect.

Given her echocardiographic findings without any anterior wall motion abnormalities, relatively modest troponin elevation, as well as clinical presentation of rapidly worsening hypoxia, it was felt that her presentation was more consistent with a large pulmonary embolus (PE), and the ECG findings were due to right ventricular strain and ischaemia. She was hypotensive and had significant desaturations with minimal movement, and was therefore too unstable to be taken to CT scan, and transportation to the catheterization laboratory was felt to be high risk. She was therefore treated with tenecteplase 50 mg i.v. for presumed PE, and heparin drip was resumed. Repeat ECG after thrombolysis showed resolution of ST elevation (Figure 2). Repeat high-sensitivity troponin T was modestly decreased at 136 ng/L. She also underwent prone positioning. Her oxygen requirements improved from FiO₂ 100% down to 50%. Her pressor requirements also improved over the next 24 h. No other imaging was performed at this time. Ultrasound and CT chest 2 weeks later did not show deep venous thrombosis, coronary calcifications, or large vessel PE. The patient remained intubated for a prolonged period and ultimately died from respiratory failure due to hospital-acquired pneumonia.

**Discussion**

Patients with COVID-19 who require hospitalization usually present with significant respiratory symptoms. However, a range of cardiovascular manifestations can also occur. In the first report of 41 patients in Wuhan, China, 5 (12%) had myocardial injury, manifesting primarily as elevated high-sensitivity troponin.1 Another report of 150 patients from Wuhan reported pre-existing cardiovascular disease as a predictor of death, and attributed 7% of deaths to circulatory failure and 33% of deaths to a combination of respiratory and circulatory failure. Fulminant myocarditis as a manifestation of COVID-19 was also described.2

Whereas myocardial injury from myocarditis or haemodynamic alterations may be common, the incidence of plaque rupture acute coronary syndrome in COVID-19 is unclear. ST segment changes can be seen due to other reasons, including pericarditis or focal
myocarditis. Anecdotal cases of ST elevations with normal coronary arteries by catheterization have been described, but large-scale studies have not yet been published.

COVID-19 may also predispose to both venous and arterial thromboembolic events due to inflammation cytokines, hypoxia, immobilization, and diffuse intravascular coagulation. Klok et al. studied 184 patients admitted to Dutch ICUs with COVID pneumonia and reported a 31% incidence of thrombotic complications, with PE being the most frequent thrombotic complication (81%) among these (n = 25, 81%), despite all patients being on at least standard

Figure 1  (A) ECG immediately following decompensation with sinus tachycardia and acute ST elevations in precordial leads V2–V5, as well as S1Q3T3 pattern. (B) Baseline ECG on presentation with no signs of ischaemia.

Figure 2  ECG post-thrombolytic therapy with tenecteplase 50 mg i.v. for presumed pulmonary embolus.
dose thromboprophylaxis. A second study of 81 patients in the ICU reported a 25% venous thrombo-embolism rate as well.

Our patient had ST elevation in the anterior leads. However, the echocardiogram showed normal left ventricular function with no wall motion abnormalities and minor troponin elevation, making acute transmural myocardial infarction highly unlikely. She had a dilated and hypokinetic right ventricle with apical sparing, or McConnell’s sign, which has 77% sensitivity and a 94% specificity for acute PE. Given her haemodynamic instability requiring vasopressors and significant hypoxia, she was treated with i.v. tenecteplase, with resolution of ST elevation on ECG and rapid improvement in haemodynamic and respiratory status.

PE causing anteroseptal/anterior ST elevations that mimic myocardial infarction have previously been described. This phenomenon is thought to be related to right ventricular injury from large emboli.

Given the high rates of thrombo-embolic events in COVID-19 patients, PE should be in the differential diagnosis of ST elevation, particularly in younger patients without coronary artery disease risk factors. Echocardiography can be extremely useful to evaluate for regional wall motion abnormalities, as well as right ventricular function, and can often lead to the correct diagnosis and help avoid additional procedures, such as cardiac catheterization and their attendant risks. Confirmation of diagnosis with other imaging modalities can be considered in stable patients. In unstable patients without contraindications to thrombolysis, thrombolysis provides effective therapy.

Lead author biography

Michael R. Kendall is a cardiology fellow in training based in Tucson, AZ, USA. He completed his medical degree at Keck School of Medicine, USC, in 2014 and medicine residency at Los Angeles County Hospital, USC, in 2017. He is currently completing his cardiology fellowship at the University of Arizona, Tucson. His interests include interventional cardiology, thromboembolism, and the development of biomedical devices.

Supplementary material

Supplementary material is available at European Heart Journal – Case Reports online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that necessary consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE and HIPPA guidance.

Conflict of interest: none declared.

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