Case of right ventricular and aortic thrombi in a patient with severe COVID-19

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SUMMARY

Emerging evidence suggests that novel COVID-19 is associated with increased prothrombotic state and risk of thromboembolic complications, particularly in severe disease. COVID-19 is known to predispose to both venous and arterial thrombotic disease. We describe a case of a 61-year-old woman with history of type II diabetes, hypertension and hyperlipidaemia who presented with dry cough and acute abdominal pain. She was found to have a significantly elevated D-dimer, prompting imaging that showed thrombi in her right ventricle and aorta. She had rapid clinical deterioration and eventually required tissue plasminogen activator with subsequent durable clinical improvement. This case highlights a rare co-occurrence of venous and arterial thrombi in a patient with severe COVID-19. Further studies are needed to clarify the molecular mechanism of COVID-19 coagulopathy, the utility of D-dimer to predict and stratify risk of thrombosis in COVID-19, and the use of fibrinolytic therapy in patients with COVID-19.

BACKGROUND

The novel COVID-19 caused by the SARS-CoV-2 rapidly emerged as a global public health threat, and was declared a pandemic by the WHO on 11 March 2020.[1] Despite mounting efforts to control spread, the prevalence and associated mortality of COVID-19 continue to climb.[2] COVID-19 primarily targets the lungs, but its clinical spectrum is wide-ranging, with increasing evidence pointing to its association with prothrombotic state and increased risk of venous and arterial thromboses, especially in severe cases.[3–5] Several cases have been reported in current literature of patients with COVID-19 presenting with either venous or arterial thromboembolism; rarely do both co-occur and rarely do they involve the right ventricle.[6 7] Furthermore, the role of the D-dimer as a risk-stratification tool is under active investigation,[8] as is the role of fibrinolytic agents in patients with severe COVID-19.[9 10] We present a unique case of a patient with severe COVID-19 with markedly elevated D-dimer, found to have right ventricular (RV) and aortic thrombi, and who eventually required tissue plasminogen activator (tPA).

CASE PRESENTATION

Our patient was a 61-year-old woman with a history of type 2 diabetes mellitus, hypertension, hyperlipidaemia, gastroesophageal reflux disease and bipolar disorder who presented on 11 April 2020 with acute onset 10/10, non-radiating midabdominal pain occurring on morning the of presentation. She also endorsed dry cough in the preceding 2 weeks. She otherwise denied any fevers/chills, rhinorrhoea, anosmia, headache, chest pain or pressure, palpitations, shortness of breath, nausea or vomiting. Her husband was recently diagnosed with COVID-19, and she had been his primary caregiver. She denied any personal or family history of blood clots. No clear history of spontaneous abortions. She had no known history of malignancy. Her medications included baby aspirin, metformin, lisinopril, simvastatin, ranitidine, conjugated oestrogen vaginal cream, calcium/vitamin D and multivitamins. She denied any alcohol, tobacco or recreational drug use.

On initial presentation, she was afebrile, heart rate 112 beats/min, blood pressure 144/83 mm Hg, respiratory rate (RR) 28 breaths/min, SpO_2 95% on room air. Her examination was notable for diffuse abdominal tenderness. She had no evidence of respiratory distress on initial presentation. She was immediately placed on airborne and contact precautions.

INVESTIGATIONS

Reverse-transcription PCR testing via nasopharyngeal swab later returned positive for COVID-19. Additional laboratory analysis revealed a lymphocyte count of 0.74×10^9/L, procalcitonin 0.10 ng/mL, C reactive protein 295.7 mg/L, lactate dehydrogenase 478 U/L, creatine kinase 37 U/L, ferritin 146 μg/L, fibrinogen 682 mg/ dL, prothrombin time 15.5 s, partial prothrombin time 23.7 s, high-sensitivity troponin T of <6 ng/L and an elevated D-dimer level of 8264 ng/mL (table 1).

ECG revealed sinus tachycardia; no evidence of atrial fibrillation or other arrhythmia. Chest X-ray revealed multifocal bilateral patchy opacities with confluent opacities in the right upper lobe (figure 1). Given the elevated D-dimer, CT-pulmonary angiogram (CT-PA) protocol and CT angiogram abdomen/pelvis were performed. CT-PA revealed no evidence of pulmonary embolism (PE); however, it did demonstrate bilateral peripheral ground-glass opacities with intralobular septal lines and areas of dense consolidation (figure 2A). There was an area of hypoattenuating focus within the RV apex concerning for thrombus (figure 2B). CT angiogram abdomen/pelvis revealed multiple filling defects within the thoracic and abdominal aorta likely representing thrombi (figure 3); mesenteric vessels were clear. Urgent bedside point-of-care ultrasound (POCUS) was subsequently obtained to evaluate the RV apex findings, and this showed an echogenic mass measuring 19×14 mm in the RV cavity most consistent with thrombus. RV size and

Table 1 Admission laboratory values and normal ranges for the described case (SI units)

	Patient value on admission	Normal range
Complete blood count, initial		
White blood cell count, $\times 10^9$/L	13.78	4.5–11.0
Lymphocyte count, $\times 10^9$/L	0.74	1.0–4.8
Haemoglobin, g/L	103	120–160
Platelets, $\times 10^9$/L	562	150–400
Chemistries, cardiac markers		
Creatinine, µmol/L	56.42	50–110
Creatine kinase, µkat/L	0.62	0.67–2.51
Albumin, g/dL	38	33–50
High-sensitivity troponin-T, initial, ng/L	<6	<6
High-sensitivity troponin-T, subsequent, ng/L	<6	<6
N-terminal pro-B-type natriuretic peptide, ng/L	1237	0–100
Inflammatory, coagulation markers		
Ferritin, µg/L	146	10–200
Procalcitonin, µg/L	0.10	0.00–0.08
C reactive protein, mg/L	295.7	<8.0
Lactate dehydrogenase, µkat/L	7.98	1–1.67
D-dimer, µg/L	8264	<500
Fibrinogen, g/L	6.82	1.5–4.0
Prothrombin time, s	15.5	11.5–14.5
Activated partial thromboplastin time, s	23.7	22.0–36.0
Interleukin-6, pg/mL	30.6	≤1.8

function were normal (figure 4, online supplemental video 1). Bilateral lower extremity venous ultrasound was negative for deep venous thrombosis (DVT). Hypercoagulable panel was checked and was negative.

TREATMENT

In the emergency room, patient was given intravenous fluids and intravenous morphine. She received empiric intravenous ceftriaxone (1 g every 24 hours) and oral doxycycline (100 mg two times per day) for 5 days, though clinical suspicion for bacterial pneumonia was low given her low procalcitonin level. She also

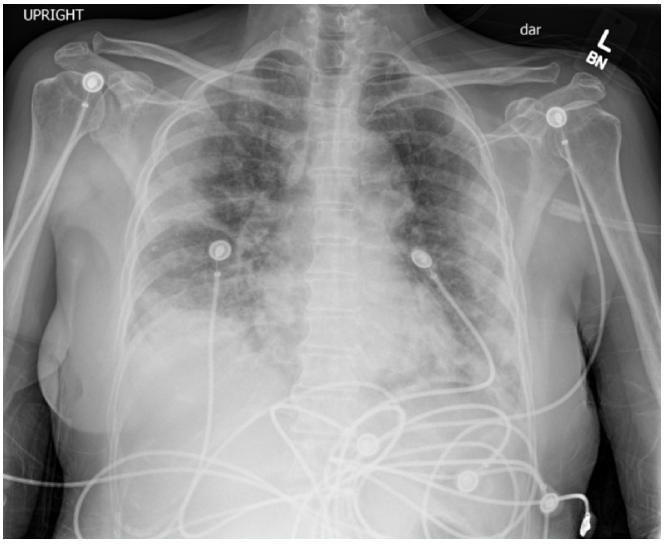

Figure 1 Chest X-ray showing multifocal bilateral patchy opacities with confluent opacities in the right upper lobe.

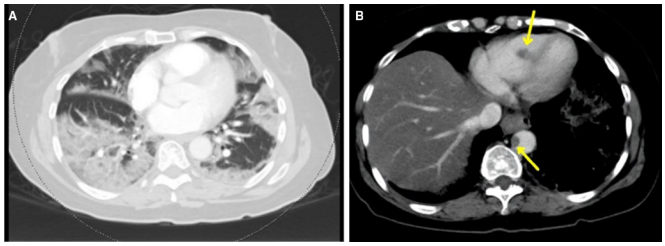

Figure 2 CT chest pulmonary angiogram (axial view) (A) showing bilateral peripheral ground-glass opacities with intralobular septal lines and areas of denser consolidation (B) showing an area of hypoattenuating focus within the right ventricular apex as well as thoracic aorta concerning for thrombi.

received oral hydroxychloroquine (400 mg two times per day on day 1, followed by 400 mg daily on days 2–5), as was routinely used for patients with COVID-19 at the time she presented. She received a heparin bolus (80 units/kg) and was subsequently started on a drip (18 units/kg/hour). Approximately 3 hours after the patient was admitted to the floor, she rapidly declined, with RR 29 breaths/min and SpO_2 91% on a non-rebreather mask. She was proned without clinical improvement. A Pulmonary Embolism Response Team (PERT) consult was called, and a multidisciplinary conversation involving vascular medicine, haematology, pulmonary/critical care and cardiac surgery was conducted. This discussion resulted in a decision to give tPA given little pulmonary reserve with COVID-19 and risk of cardiac arrest if the RV clot were to embolise. Patient received 50 mg intravenous alteplase (25 mg/hour × 2 hours) and tolerated it well. She did not require intubation. Formal trans-thoracic echocardiogram obtained 33 hours following tPA administration showed a mobile echodensity in the RV apex near the moderator band,

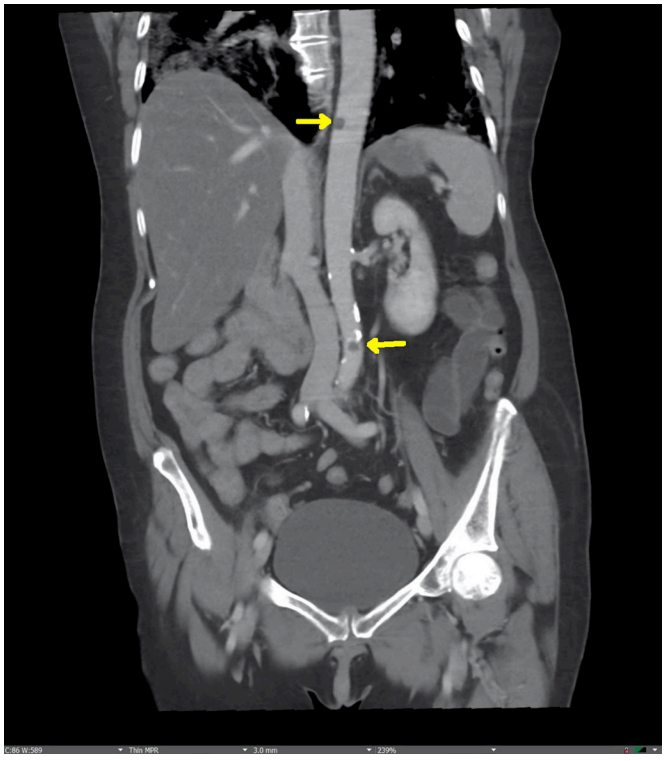

Figure 3 CT angiogram abdomen/pelvis (coronal view) showing filling defects within the thoracic and abdominal aorta (yellow arrows) likely representing thrombi.

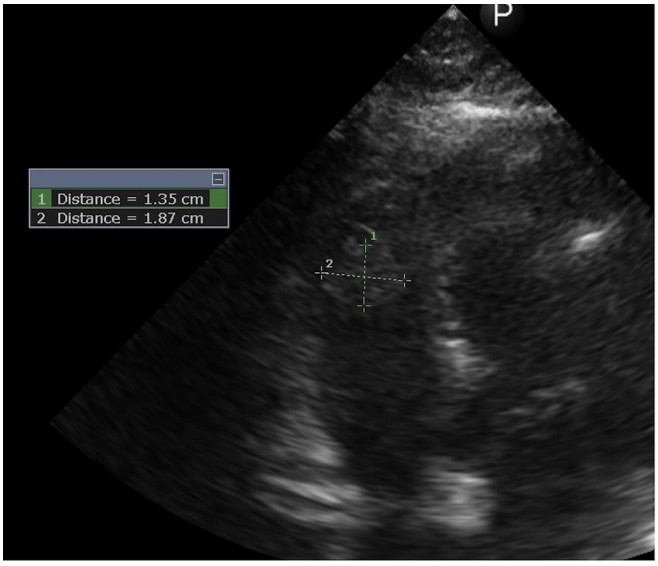

Figure 4 Pre-tissue plasminogen activator: bedside point-of-care ultrasound showing a mass of echoes measuring 19×14 mm in the right ventricular cavity most consistent with thrombus.

most likely thrombus, now decreased in size, measuring 14×5 mm. RV size and function was normal. LV ejection fraction was normal at 54% with no segmental wall motion abnormalities. There was no evidence of patent foramen ovale (figure 5, online supplemental video 2). By hospital day 3, she was weaned to 1-litre nasal cannula. She was transitioned to therapeutic low-molecular weight heparin (LMWH) at 1 mg/kg two times per day. Unfortunately, her health insurance did not cover LMWH use as outpatient. Warfarin was deferred given risk of staff exposure during routine INR checks. She was therefore eventually discharged on rivaroxaban (15 mg two times per day for 21 days, followed by 20 mg once daily). By day of discharge (hospital day 8), she had been weaned to room air.

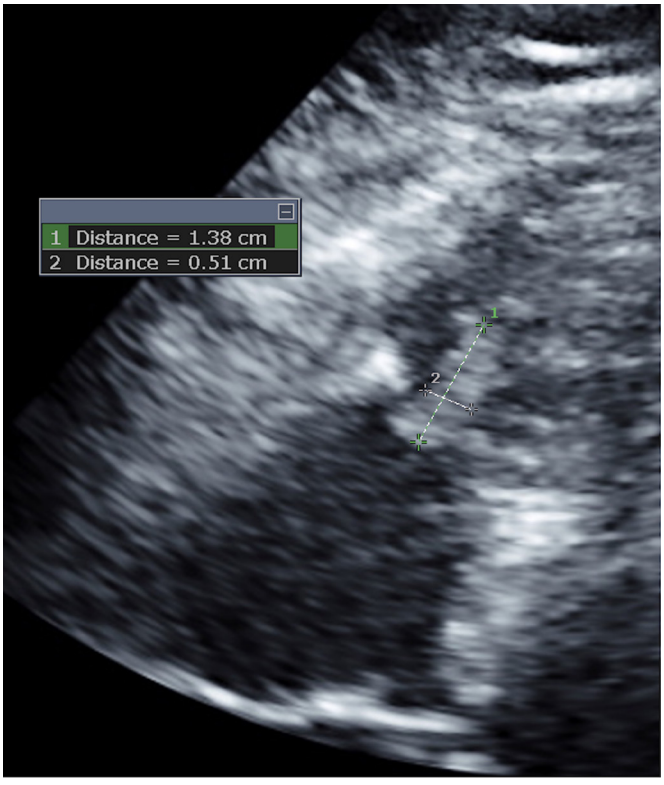

Figure 5 Thirty-three hours post-tissue plasminogen activator: formal trans-thoracic echocardiogram showing a mobile echodensity (14×5 mm) in the right ventricular apex near the moderator band, most likely thrombus.

OUTCOME AND FOLLOW-UP

At 1-month and 5-month follow-up clinic visits, the patient remained stable and asymptomatic. There was a recommendation by PERT for repeat imaging to reassess thrombosis burden, however, the patient was unable to return to have this performed.

DISCUSSION

We describe an unusual case of a patient with COVID-19 presenting with thrombosis involving the right ventricle as well as the thoracic and abdominal aorta. Besides use of conjugated oestrogen vaginal cream which in itself has not been shown to be associated with increased thrombosis risk,[11] this patient had no other preceding risk factors for thrombosis besides COVID-19.

This case highlights the importance of early recognition and management of thrombosis as a complication in patients presenting with COVID-19. Thrombotic complications in patients with severe COVID-19 is prevalent, with an incidence of 31% (95% CI: 20% to 41%) in a cohort of patients admitted to a Dutch intensive care unit (ICU),[12] and 27.9% (95% CI: 22.1% to 34.1%) in a more recent subgroup meta-analysis of 31 studies involving patients in ICU.[13] Venous thromboembolism such as DVT and pulmonary emboli are more commonly described,[3–5] as have RV thrombi.[6 14] Arterial thromboembolism including aortic thrombi are less frequently described.[14 15] Rarely do both co-occur,[16] as did in our patient. The mechanisms involved in the formation of thrombosis in COVID-19 are not yet fully elucidated but appear multifactorial. It is postulated that dysregulated immune responses mediated by inflammatory cytokines, lymphocyte cell-death, hypoxia and endothelial damage are involved.[17] However, it is unclear how the formation of arterial and venous thrombi are related. Evidence from non-COVID-19 literature suggests obesity may be a key epidemiologic link,[18] though the observed association between other cardiovascular risk factors and venous thromboembolism appears weak or non-significant: OR 1.51 for hypertension (95% CI: 1.23 to 1.85), 1.42 for diabetes mellitus (95% CI: 1.12 to 1.77) and 1.18 for smoking (95% CI: 0.95 to 1.46).[19] Associations between cardiovascular risk factors and clot risk in patients with COVID-19 remain yet to be explored.

The development of thrombosis in patients with COVID-19 who are already compromised with respiratory conditions can further contribute to worsening respiratory failure and rapid clinical deterioration. Early detection and management is therefore key, however, there is a paucity of data on tools to predict and risk stratify thrombotic events in patients with COVID-19. In a study of 199 patients with COVID-19, a D-dimer value above 1μg/mL was associated with an adjusted HR of 18.4 (95% CI: 2.6 to 128.6) for in-hospital mortality.[20] In addition, in a series of 400 patients with COVID-19, a D-dimer >2500 ng/mL was predictive of not only increased risk of thrombosis (adjusted OR of 6.79 (95% CI: 2.39 to 19.30)), but also increased risk of bleeding (adjusted OR of 3.56 (95% CI: 1.01 to 12.66)).[21]

Drawing on such early evidence, the Internal Society on Thrombosis and Hemostasis recommends that in patients with markedly elevated D-dimers (arbitrary cut-off of threefold to fourfold increase above upper limit of normal), hospital

admission should be considered even in absence of other symptoms suggestive of disease severity.[22] This guideline was applied in early clinical decision-making in our patient, prompting imaging that revealed her extensive thrombotic burden. The COVID-19 pandemic has left in its wake significant staffing, infection control and other resource constraints which have limited access to recommended diagnostic imaging techniques. For patients with COVID-19 in whom there is a high clinical suspicion for PE but for whom CT or ventilation-perfusion scanning cannot be obtained for these reasons, other modalities may be used to help in the decision of whether to start therapeutic-dose anticoagulation. Diagnostic surrogates such as echocardiography (including POCUS) and lower extremity ultrasounds have been used and incorporated into algorithms to aid in diagnosis and treatment of thrombotic complications of COVID-19.[14] The recently published PERT consortium position paper on the diagnosis and treatment of PE in patients with COVID-19 can significantly aid in the care of these vulnerable and complicated patients.[23] By using a team-based approach for decision-making and coordination of care, PERT was able define the best course of treatment for this patient.

Data are currently lacking on the optimal choice of anticoagulation for haemodynamically stable patients with COVID-19 with proven thrombosis. The preferred anticoagulant based on consensus guidelines is LMWH. Warfarin may also be considered, though all efforts should be taken to minimise exposure risk to staff during INR checks.[24 25] In our patient, neither LMWH nor warfarin were tenable options, resulting in our eventual use of rivaroxaban, a direct oral anticoagulant (DOAC). In non-COVID-19 literature, DOACs are well-studied for use in venous thrombosis, but less so for arterial thrombosis.[26] Two phase III randomised trials are currently assessing dabigatran (RE-SPECT ESUS, NCT: 02239120)[27] and rivaroxaban (NAVIGATE ESUS, NCT: 02313909)[28] in patients with embolic stroke of undetermined source (ESUS). The results of these large trials should hopefully inform future studies investigating treatment choices in patients with aortic thrombosis, including patients with COVID-19. To our knowledge, there are are no known studies investigating the role of COVID-19 therapeutics such as remdesivir or interleukin-6 (IL-6) inhibitors in preventing or treating thrombotic events in COVID-19 infections, though the potential role of IL-6 inhibitors is especially noteworthy given prior research in non-COVID-19 literature suggesting increased IL-6 expression in patients with thrombotic disease.[29]

Of increasing interest is the use of fibrinolytic agents in thrombotic disease in patients with COVID-19.[30 31] A class I indication for fibrinolytic therapy is PE with associated haemodynamic instability.[32] The use of thrombolysis in haemodynamically stable cases of PE (ie, submassive PE) is less well-defined, as clinical trials have not been sufficiently powered to show a clear survival benefit.[33] Use of thrombolysis in cases of RV thrombosis tends to occur in the setting of RV clot in transit,[14 34 35] and use of systemic thrombolysis in aortic thrombosis is rare,[36] largely limited to neonatal settings.[37 38] Our report raises potential questions surrounding the expanded use of systemic thrombolysis, including and especially in patients with COVID-19. In a case series of three critical patients with acute respiratory distress syndrome due to severe COVID-19, intravenous tPA was used with temporary improvement in all three patients and sustained improvement in one.[10] Evolving evidence suggests timing is key, speculating that early (rather than late) administration of tPA in the course of COVID-19 infection may lead to better outcomes.[31] Such early findings have influenced ongoing trials investigating use of fibrinolytic agents in patients with COVID-19.

Learning points

- COVID-19 appears to be associated with an increased risk for thrombosis formation and propagation.
- A high level of clinical suspicion is necessary to detect thrombotic events and treat patients expeditiously.
- Future studies should further elucidate the molecular mechanism of COVID-19 coagulopathy, the utility of D-dimer or other markers in predicting and stratifying risk of thrombosis in, and the use of advanced therapies such as fibrinolytic therapy in patients with COVID-19.

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