Dear Sir,

A 66-year-old man with a medical history of diabetes, hypertension and coronary artery disease presented to the emergency department with acute abdominal pain. The pain was diffuse, continuous type associated with non-bloody emesis and hematuria. He denied history of any trauma or lower urinary tract symptoms. He had suffered COVID-19 pneumonia 20 days back for which he required hospitalisation for 6 days. He had diffuse ground glass opacities on HRCT thorax with severity score of 10/25 and required oxygen support for mild hypoxia [arterial pO2/FIO2 (p/f ratio) –240]. C Reactive protein, ferritin and lactate dehydrogenase were mildly elevated with normal D- Dimer. He was categorised as mild-moderate COVID-19 disease and received inj Remdesivir with dexamethasone for 5 days. He was discharged on day 7 with tapering dose of oral prednisolone. The interval from discharge to current presentation of 14 days was uneventful.

On admission, he had tachycardia with blood pressure of 90/60 mm of Hg. On abdomen examination, there was diffuse abdominal tenderness without rebound or guarding. Laboratory assessment revealed creatinine of 1.4 mg/dl (0.66-1.2 mg/dl), with leucocyte count –14,080/mm$^3$ and platelet count –84,000/mm$^3$. He had elevated lipase levels of 360 U/L (23-300 U/L), c-reactive protein at 200 mg/dl (<10 mg/dl), lactic dehydrogenase level of 658 U/L (120-246 U/L), D- Dimer levels –7.37 (<0.53 mcg/ml) and prolonged prothrombin time of 20.2 seconds (control –11.3) and INR of 1.7. Urinalysis had plenty of red blood cells on microscopy. A computed tomogram of the abdomen and pelvis with intravenous contrast was done which revealed wedge shaped cortical hypodensities in both kidneys due to non-enhancement of contrast suggestive of renal infarcts [Figure 1]. Both renal arteries showed normal enhancement and were patent [Figure 2]. Cardiac enzymes and echocardiogram done for the evaluation of shock were normal. Intravenous meropenem was started after drawing blood and urine culture samples. In view of gross hematuria, thrombocytopenia and shock, anticoagulation was not started. Patient had worsening hypotension requiring high dose noradrenaline and vasopressin infusions. Over next 12 hours in ICU, he was put on positive pressure ventilation and initiated on continuous renal replacement therapy (CRRT) for anuria, hyperkalemia and severe metabolic acidosis. Patient succumbed 48 hours after admission due to refractory shock.

Discussion

Corona Virus disease 2019 (COVID–19) caused by SARS-CoV-2 virus, initially described in Wuhan, China has rapidly spread worldwide to proportion of global pandemic. Kidney involvement due to COVID-19 manifests as acute kidney injury, nephrotic or sub nephrotic range proteinuria and haematuria. Histopathological picture consists of podocytopathy, immune-mediated glomerulopathy, acute tubular injury and thrombotic microangiopathy, although the exact mechanism of injury remains unclear. Kidney injury due to bilateral renal infarction as in our case is a very rare manifestation of COVID-19 with very few reported cases in literature. Anticardiolipin antibody (IgA) and

Figure 1: Contrast enhanced computerized tomography (CECT) scan abdomen showing wedge shaped cortical hypodensities in both kidneys due decreased enhancement consistent with multiple bilateral renal infarctions

Figure 2: Reconstructed Contrast enhanced computerized tomography (CECT) scan abdomen showing patent bilateral renal arteries with renal infarcts
anti–b2-glycoprotein I (IgA and IgG) antibodies have been reported to be positive in patients with COVID-19 from Wuhan, China.\textsuperscript{[4]} This procoagulant state, called as “COVID-19 Coagulopathy” can lead to microvascular and macrovascular thrombi presenting as deep venous thrombi, pulmonary embolism and very rarely as stroke and acute coronary syndrome.\textsuperscript{[5]} Renal artery or venous thrombosis and bilateral renal infarction is rarely been reported. Anticoagulation with low molecular weight heparin (LMWH) has shown mortality benefit in patients with severe COVID-19 disease (LMWH - 40% versus controls - 64%).\textsuperscript{[6]} American Society of Haematology recommend prophylactic dose anticoagulation in critically ill and acutely ill hospitalised patients infected with SARS-CoV-2 who do not have established or suspected venous thromboembolism.

Our case had mild disease on presentation and the rare manifestation of bilateral renal infarction was seen on day 20 of the diagnosis of COVID-19. He did not have any COVID-19 coagulopathy during the previous admission and there is no guideline to add prophylactic anticoagulation in such cases. Further studies are required to evaluate the benefit of prophylactic anticoagulation in patients with milder disease and those who are non-hospitalised and suffered COVID-19 in past. The duration and choice of anticoagulation to be given too needs to be studied.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understand that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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