Renal artery thrombosis in COVID-19

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Case report

Renal artery thrombosis in COVID-19

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A B S T R A C T

COVID-19 has been described to have association with hypercoagulable state and thromboembolic events in major blood vessels, pulmonary artery, major limb vessels causing limb ischemia, associated with neurological symptoms and complications including stroke. We present a unique case of renal artery thrombus formation secondary to COVID-19. To the best of our knowledge this is the fourth manuscript describing renal artery thrombosis with evidence on imaging studies in medical literature.

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Case report

A 77-year old female presented to hospital with acute onset of generalized weakness and two episodes of watery, non-bloody diarrhea accompanied with poor appetite. She denied fever, chest pain, shortness of breath, abdominal pain, nausea, vomiting or urinary complaints. Her past medical history included hypothyroidism, stable coronary artery disease, hypertension, chronic obstructive pulmonary disease, and non-small cell lung carcinoma in remission. She had a surgical history of abdominal aortic aneurysmal repair and aortoiliac graft with bilateral renal artery stenting secondary to renal artery stenosis approximately two years before. She was hospitalized two weeks previous to presentation for an endovascular leak which was coiled embolized successfully by intervention radiology.

Her home medications included amiodipine, metoprolol, simvastatin, aspirin, tiotropium and levotyroxine. She was a former smoker with a 40 pack-year smoking history, quitting 16 years before, otherwise no other recreational drug use was reported. On admission, the vital signs included: blood pressure 90/50 mmHg, heart rate 90, temperature 97.9 °F, respiratory rate normal and oxygen saturation 100 % on room air. Initial laboratory workup revealed normal white blood cell count of 7000 /µL with mild lymphopenia, with hemoglobin 11.0 g/dL and platelet count of 305,000/µL. The chemistry panel was noted to be unremarkable without transaminitis. The inflammatory markers at the time of admission are summarized in Table 1.

The patient had a nasal swab for COVID-19 which resulted positive. Due to her acute generalized weakness and hypotension in the setting of recent aortic intervention, there was a suspicion for recurrent endovascular leak. Therefore, computed tomographic (CT) angiography with dissection protocol was obtained. The CT scan study ruled out any retroperitoneal bleeding or significant aortic structural defect but revealed intraluminal filling defects compatible with thrombus within renal artery stents bilaterally.

After hemodynamically optimizing the patient she was evaluated by the interventional radiology and vascular surgery teams. A renal duplex scan was subsequently performed showing no significant stenosis in right renal artery and but occlusion of the left renal artery. The patient’s blood pressure and renal function remained intact throughout the hospital stay. It was determined from imaging studies that the anatomical location of the thrombus was not amenable to any intervention. Secondly, given the history of aortic aneurysmal repair, any other intervention was deemed inappropriate as it may likely do more harm than benefit as the patient remained asymptomatic.

The hypercoagulable studies were performed to screen for prothrombotic diseases which were negative. It was concluded that the patient’s renal artery thrombosis was caused by the hypercoagulable state induced by COVID-19. The patient continues to follow up outpatient with her primary care physician and nephrology.

Discussion

Since the emergence of global pandemic of the novel coronavirus (COVID-19) in China in December 2019, it has been described as a causative agent for spectrum of clinical pathologies

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changes in renal function is not clearly determined and more studies are underway to describe the pathophysiology [5]. With this brief literature review, kidney dysfunction seen in COVID-19 patient can partly be described with undetected renal artery thrombosis and systemic microangiopathies with renal microthrombi [6–8]. Our patient described above presented with an incidental finding of thrombus formation in renal artery stenting. Patient has been asymptomatic with no changes in the urine production. The decision for intervention with revascularization mainly depends upon the size of the vessel involved, time of onset and size of infarction. The success of such intervention is unpredictable, and the patient may become dependent on renal replacement therapy if there is not enough renal reserve left as described by Philpponnet [6]. The timeline of renal artery thrombosis and renal infarcts may not necessarily align, and signs and symptoms may present after significant interval with possible no evidence of thromboembolism [9,10]. Although, not enough data is available, but antiplatelets and full anticoagulation should be considered in patients with diagnosed renal infarcts. As our patient had a significant risk of endovascular bleed due to recent abdominal aortic aneurysmal repair, it was decided to hold off with anticoagulation.

**Conclusion**

This article and literature review summarize the cases described with renal artery thromboembolism and infarcts secondary to thromboembolic state induced by COVID-19 infection. This limited data suggests that such macro and microthromboembolic activities may have played role in causing renal dysfunction seen with COVID-19 infections. Further studies are required to determine role of revascularization or long-term anticoagulation for treatment modalities.

**Authorship contributions**

Conception and design of study: S.Acharya, S.Anwar

Acquisition of data: F.siddiqui, S.shabih

Analysis and/or interpretation of data: U.Manchandani, S. Dalezman

Drafting the manuscript: S.Acharya, S.Anwar

Revising the manuscript critically for important intellectual content: F.siddiqui, S.shabih, U.Manchandani, S.Dalezman

Approval of the version of the manuscript to be published (the names of all authors must be listed): S.Acharya, S.Anwar, U. Manchandani, S.shabih, F.siddiqui, S.Dalezman

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**Table 1**

| MARKERS                      | RESULTS          |
|------------------------------|------------------|
| C-reactive Protein 5.55 mg/dL |                  |
| Ferritin 589 ng/mL           |                  |
| Procalcitonin 0.13 ng/mL     |                  |
| Lactate dehydrogenase 583    |                  |
| D-dimers 4620 ng/mL          |                  |
| Prothrombin time/INR 11.6/1.01|                  |
| Activate partial thromboplastin time 29 |