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Case Report

Bilateral renal infarction with COVID-19 pneumonia: a case report

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Abstract

Acute renal infarction is a rare and often underdiagnosed condition with estimated incidence of 0.5–1.5%. Coronavirus disease 2019 (COVID-19) has been shown to cause a hypercoagulable state in patients leading to arterial and venous thromboembolism. Renal infarction as a consequence of COVID-associated coagulopathy has been reported, sometimes resulting in acute kidney injury. Most of the patients so far reported had other existing comorbidities and risk factors that compounded the risk of precipitating an infarction. Here, we present a 37-year-old, the youngest patient reported so far, with no pre-existing comorbidities or risk factors, who developed bilateral renal infarction with COVID-19 pneumonia. The patient was treated with anticoagulation for renal infarction and discharged on apixaban. Anticoagulation is an important part of current treatment strategies for COVID-19 pneumonia and should extend beyond the acute phase of the disease to prevent long-term sequelae, especially in young patients.

INTRODUCTION

A new type of coronavirus, the SARS CoV-2, was identified in January 2020, which caused coronavirus disease 2019 (COVID-19) [1]. Though primarily shown to cause interstitial pneumonia worsening to acute respiratory distress syndrome, COVID-19 has also been reported to be precipitate a hypercoagulable state leading to arterial and venous thromboembolism [2]. Although pulmonary thromboembolism has been most commonly encountered, there have been cases where patients had cerebral, myocardial and abdominal visceral infarction. Very few cases with renal infarction have been reported to date, with most of them having multiple comorbidities and risk factors. Here, we present the youngest patient so far reported, with no comorbidities, who developed COVID-19 pneumonia with bilateral renal infarction.

CASE REPORT

A 37-year-old male presented to the emergency department with complaints of bilateral flank and suprapubic pain since 2 days. The pain was sudden in onset, continuous, 5/10 in severity and aggravated with coughing and deep breathing. His past medical history was significant for hospitalization for urinary tract infection with development of ureteral strictures requiring stents 5 years ago. There was no family history of cancer or blood disorders.

At presentation, his vitals were normal with blood pressure of 100/75 mm Hg. On examination, he was non-toxic, alert, oriented and in pain. Physical examination was significant for bilateral costovertebral angle tenderness. Imaging showed bilateral pneumonia on X-ray and computed tomography (CT) of chest. Contrast enhanced CT of abdomen showed brisk excretion of contrast, no hydronephrosis and bilateral wedge-shaped non-enhancing areas in the renal parenchyma consistent with infarcts (Fig. 1a and b).

Labs were significant for positive COVID-19 infection on reverse transcription-polymerase chain reaction of nasal swab and negative for IgG antibodies. Serum creatinine, blood urea nitrogen (BUN) and urinalysis were normal. Hypercoagulability workup including platelet count, prothrombin time (PT), partial thromboplastin time (PTT), international normalized ratio (INR), Factor V levels and mutation analysis, antineutrophil cytoplasmic antibodies (ANCA), complement, anti-double-stranded DNA, cardiolipin antibody, beta-2-microglobulin and serum homocysteine were all normal (Table 1). Renal arterial and venous duplex showed preserved renal perfusion and no significant stenosis in the visualized segments of renal arteries.

Patient was admitted for COVID-19 pneumonia and bilateral renal infarction. He was started on dexamethasone (6 mg) daily, Lovenox (70 mg) twice daily

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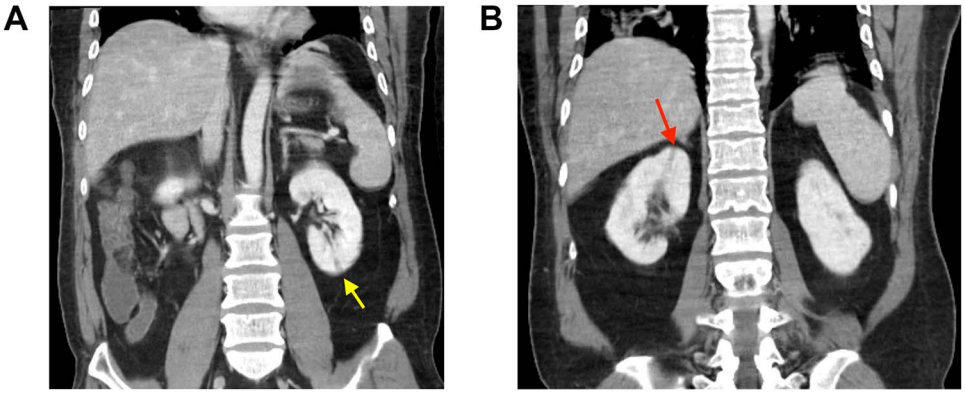

Figure 1. (**a**) Linear wedge-shaped infarct in left kidney (yellow arrow) on contrast enhanced CT. (**b**) Linear wedge-shaped infarct in right kidney (red arrow) on contrast enhanced CT.

Table 1. Laboratory values of the patient during hospitalization

		At admission	Day 3	Day 4	At discharge
	Weight in kg.	68			
	BMI (kg/m^2)	25			
Metabolic profile	Creatinine (mg/dl)	0.9	0.7	0.5	0.5
	BUN (mg/dl)	13	8	7	12
	AST (IU/L)	384	192	67	86
	ALT (IU/L)	414	309	188	221
	ALP (IU/L)	90	103	86	90
	Bilirubin (mg/dl)	0.5	0.5	0.4	0.5
	Albumin (g/dl)	3.7	3.4	3.2	3.2
	Cholesterol (mg/dl)	69			
	LDL (mg/dl)	25			
	LDH (IU/L)	366			
	HbA1c (%)	5.3			
	CRP (mg/dl)	9.482 (0–0.9)			
	Ferritin (ng/ml)	1990 (3.1–110.9)			
Coagulation profile	D-dimer (ng/ml)	226			
	PT (sec)	13.3			
	PTT (sec)	34.6			
	INR	1.2			
	Platelet count (per μl)	150 000	171 000	194 000	235 000
	Factor V	No mutation			
	Fibrinogen (mg/dl)	505 (217–521)			
Serology	ANCA	Negative			
	C3 (mg/dl)	108 (81–157)			
	C4 (mg/dl)	34 (13–39)			
	dsDNA (IU/ml)	<12			
	Cardiolipin antibodies	Negative			
	Homocysteine (μmol/l)	9.2 (<15)			
	Beta-2-microglobulin (mg/l)	2.1 (0.8–2.2)			
Urinalysis	Specific Gravity	1.040			
	pH	6			
	Protein	Trace			
	Hemoglobin	Negative			
	Sugar	Negative			
	Nitrite	Negative			
	Leucocyte Esterase	Negative			

LDL: low density lipoprotein; AST: aspartate transaminase; ALT: alanine transaminase; ALP: alkaline phosphatase; dsDNA: double-stranded DNA; CRP: C-reactive protein; LDH: lactate dehydrogenase. Normal range values in parentheses.

subcutaneously, IV fluids and supplemental oxygen at 5 liters/min through nasal canula. Because of his deranged liver enzymes, the patient was not considered a candidate for remdesivir. He received 1 unit of convalescent plasma. Pain gradually subsided and patient recovered saturating at 98% on room air at the time of discharge. He was discharged on apixaban (5 mg) daily for renal infarction.

Table 2. Summary of cases reported so far with renal infarction and COVID-19 pneumonia

No.	Study	Age	Gender	Kidney involved	Thrombus	AKI	Remarks
1	Mukerjee *et al.*	71	Male	Left	Left renal artery and ascending aorta	No	No comorbidities
2	Post *et al.*	62	Male	Allograft	N/A	Yes	HTN, HSP, post-transplant on immunosuppression
		58	Male	Bilateral	N/A	Yes	OSA
3	Anazco *et al.*	41	Female	Bilateral	Left renal artery	Yes	Obesity, untreated DM
4	Ammous *et al.*	62	Male	Left	Left renal artery	N/A	HTN, BA. Presented 14 days after COVID and while on LMWH prophylaxis; cardiolipin IgM Ab positive
5	Xu *et al.*	46	Male	Transplanted kidney	No	Yes	Kidney pancreatic transplant on Immunosuppression, HTN, Type 1 DM and dyslipidemia; rehospitalization after initial discharge
6	El Shamy *et al.*	60s	Female	Bilateral	Renal and celiac arteries	Yes	Afib on apixaban, HTN and HFpEF
7	Varner *et al.*	46	Male	Right	Right renal artery	N/A	No comorbidities
8	Kundal *et al.*	39	Female	Right	Aortic thrombus	No	OCP use, PFO, uncontrolled HTN, lupus anticoagulant positive. COVID antibodies present, PCR negative
9	Mantica *et al.*	67	Female	Right	N/A	No	Lobectomy for lung adenocarcinoma on chemotherapy
10	Lushina *et al.*	84	Male	Left	Aortic arch	N/A	HTN, Afib with RVR at presentation
11	Ramanathan *et al.*	54	Male	Bilateral	N/A	No	Obese, post COVID discharge
12	Tascon *et al.*	56	Male	Left	Left renal artery	No	DM, dyslipidemia and diverticulosis
13	Imoto *et al.*	64	Male	Bilateral	N/A	N/A	Gastric and duodenal ulcer; MCA, splenic infarctions
14	Besutti *et al.*	54	Male	Right	Renal artery	N/A	BA, Ulcerative colitis, Smoker
		53	Male	Left	N/A	N/A	HTN, MVR on aspirin

HTN: hypertension; HSP: Henoch-Schonlein glomerulonephritis; OSA: obstructive sleep apnea; DM: diabetes mellitus; BA: bronchial asthma; Afib: atrial fibrillation; HFpEF: heart failure with preserved ejection fraction; OCP: oral contraceptive pill; PFO: patent foramen ovale; RVR: rapid ventricular response; MCA: middle cerebral artery; MVR: mitral valve replacement; N/A: not available.

DISCUSSION

The incidence of renal infarction is 0.5–1.5% [3]. The most common causes of renal infarction are trauma, right atrial embolism from cardiac thrombus, dissection and iatrogenic complications of endovascular procedures [3]. Hypercoagulable states constitute a less common cause of renal infarctions reported. Infection with SARS CoV-2 has been shown to be associated with thromboembolic phenomena due to hypercoagulable state of the blood [2]. Various theories have been proposed regarding the pathophysiology of thrombi formation in the lungs of acutely ill COVID-19 patients including dysregulation of hemostasis, inflammation induced cytokine storm driven activation of endothelium and platelets, hypoxic vasoconstriction and direct viral effects [4]. The role of antiphospholipid antibodies has also been elucidated as a causative factor for COVID-19 associated coagulopathy [5].

COVID-19 has been shown to be frequently causing venous thromboembolism with arterial thrombus formation constituting a minor yet dangerous complication [6]. Hypercoagulability with elevated D-dimer, prolonged PT, APTT, thrombocytopenia and presence of fibrin degradation products have been shown to portend a poor prognosis in COVID-19 patients [7]. Although pulmonary thromboembolism and deep venous thrombosis are more commonly encountered, cerebral, myocardial and infarctions of the abdominal viscera have also been reported [6].

Renal infarction has also been reported with and without the presence of arterial thrombi in the renal vasculature. Thrombotic microangiopathy has also been described as a cause for acute kidney injury (AKI), after studying the post-mortem findings in COVID-19 patients [8]. Very few cases have been reported where patients developed renal infarction with COVID-19 pneumonia. Most of the patients had underlying comorbidities or factors, which added to the risk of developing infarction along with the hypercoagulability due to COVID-19. Bilateral renal infarction has been seen in less than five cases (Table 2). Our patient is the youngest that has so far been reported, with renal infarction and COVID-19 pneumonia. He did not have any comorbidities or risk factors that could have contributed to him developing bilateral renal infarction. Cardioembolic origin was ruled out with normal findings on 2D echocardiogram. Serology ruled out hypercoagulable states like Factor V mutation, protein C and S deficiency and autoimmune causes. There was no family history of bleeding or coagulation disorders, and his coagulation profile was

also found to be normal. In the light of all these findings, COVID-19 coagulopathy as the cause of his renal infarction is a strong possibility.

Although there have been recommendations on the prophylactic early use of anticoagulation with low molecular weight heparin (LMWH) in COVID-19 patients, especially in an intensive care unit setting, there is no consensus on the duration of anticoagulation treatment needed after resolution of the acute phase [9]. Few case reports have suggested the persistence of hypercoagulable state in COVID-19 patients even after the resolution of acute phase (Table 2). This requires constant monitoring and prolonged anticoagulation especially in young patients such as ours, who have no contraindication for prolonged anticoagulation. Further studies are required to delineate the duration of anticoagulation needed in COVID-19 patients to prevent thromboembolic phenomena.

Hypercoagulability due to COVID-19 is a challenging complication that needs to be addressed not only with early institution of anticoagulation therapy but also to be controlled even after resolution of acute phase. Extended duration of anticoagulation will help in preventing thrombi formation as a long-term sequela of the disease.

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CONFLICT OF INTEREST STATEMENT

None declared.

ETHICAL APPROVAL

No approval is required.

CONSENT

Written informed consent obtained from the patient for publication.

GUARANTOR

Kundan Jana.

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