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Extensive renal cortical necrosis in a postpartum patient after COVID-19 infection

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ABSTRACT

It has been more than two years since the COVID-19 pandemic affects the world. COVID-19 affects other organs in addition to the respiratory system. Renal cortical necrosis is a severe kidney injury due to decreased renal arterial perfusion induced by vascular spasm, microvascular injury and endovascular thrombosis. Acute kidney injury is a common finding in COVID-19; however, renal cortical necrosis is rare. To the best of our knowledge, only one case of renal cortical necrosis has been reported in the literature.

Case Report

Implication for health policy/practice/research/medical education:
COVID-19 infection is accompanied by several complications in addition to the respiratory disorders. Sufficient knowledge about all probable complications is needed for correct and on-time management of patients.

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Introduction

It is more than two years that the world is affected by COVID-19 pandemic which has caused huge mortality and morbidity worldwide. Although its main manifestations are fever and respiratory distress; however, now it is clear that this virus can target almost all of the organs during the infection. Several complications have been reported for COVID-19 infection, some were very mild such as skin rashes but some were life threatening. Renal cortical necrosis is a fatal kind of kidney injury with serious outcomes which may lead to end-stage renal disease or death(1). Renal cortical necrosis accounts for 2% and 6-7% of all cases of acute renal failure in developed nations and developing countries, respectively (2). It is mostly bilateral and unilateral cortical necrosis is rare (3). Renal cortical necrosis is the result of decreased renal arterial perfusion due to vascular spasm, microvascular injury and endovascular thrombosis (1). Histologically total ischemic necrosis of all of the renal cortical elements including glomeruli, blood vessels and tubules of the affected area is typical feature of Renal cortical necrosis (1). According to histologic findings two types of renal cortical necrosis have been described, diffuse and patchy cortical necrosis (1).

Among hospitalized patients with coronavirus infection, acute kidney injury (AKI) is a common finding (4); however, cortical necrosis is very rare and to the best of our knowledge, only one case of renal cortical necrosis has been reported in the literature (5). Here, we report the second case of renal cortical necrosis in a patient with COVID-19 infection.

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Case Presentation

The patient was a previous healthy 37-years-old woman. She had an uncomplicated term vaginal delivery 12 days before her admission to our hospital. Three days after natural vaginal delivery, the patient returned to emergency with dyspnea and cyanosis. On initial physical examination she was found to be conscious and oriented with normal vital signs. Body temperature was 37.1, respiratory rate was 30 with O_2 saturation of 93%. According to positive polymerase chain reaction (PCR) test for COVID-19 and computerized tomography (CT) scan findings she was treated for COVID-19 infection. During admission, the patient's clinical condition worsened and laboratory findings showed uremia, elevated serum creatinine level, hyperphosphatemia and hypocalcemia (laboratory findings are mentioned in Table 1).

Considering the laboratory data advanced renal failure and rhabdomyolysis was suspected and she underwent hemodialysis through jugular vein. Ultrasonography of kidneys showed increased renal cortical echo pattern and more prominent corticomedullary differentiation with a simple cortical cyst measuring 21 mm in greatest diameter in the middle pole of the left kidney. Color Doppler ultrasonography of the kidneys showed normal blood flow of renal arteries. Due to patient's progressive course renal biopsy was conducted for further evaluation of the kidney.

Renal biopsy showed extensive coagulative (ischemic) necrosis in about 60% of renal tissue mostly in cortical area involving both tubules and glomeruli. There was interstitial mixed inflammatory cells infiltration (about 60%) (mostly lymphocytes and rare neutrophils), hemosiderin deposition and mild edema in interstitium (Figures 1-3).

The patient was stabilized in hospital with corticosteroid therapy, supportive care and hemodialysis. After rigorous treatment efforts, the patient survived but the renal damage remained persistent. She was discharged from the hospital with serum creatinine level of 7.4 mg/dL with scheduled hemodialysis three times-per-week.

Discussion

We report the case of previously a healthy 37-year-old female with history of normal vaginal delivery and COVID-19 infection with CT scan findings of pulmonary involvement and laboratory findings of uremia and hyperphosphatemia who underwent

Table 1. Laboratory finding of the patient

	Laboratory test	Value	Unit	Reference value
Hematology	Hemoglobin	10.8	g/dl	11-16
	Total leukocyte count	15.72	×1000/µL	4-10
	Platelet count	138	×1000/µL	140-440
	Uncorrected reticulocyte count	0.7	%	0.5-1.5
Biochemistry	Blood urea	204	mg/dL	0.6-1.4
	Uric acid	10.58	mg/dL	3.5-7.2
	Serum Creatinine	8	mg/dL	0.6-1.4
	Serum sodium	134	mEq/L	136-145
	Serum potassium	5.4	mEq/L	3.5-5.5
	Serum phosphorus	10.77	mg/dL	2.8-4.5
	Serum calcium	8	mg/dL	8.5-10.5
	Serum magnesium	2.3	mg/dL	1.8-2.6
	Serum total bilirubin	2.21	mg/dL	0.1-1.2
	Serum direct bilirubin	0.13	mg/dL	Less than 0.3
	Serum albumin	2.64	gr/dl	3.4-5.4
	Aspartate aminotransferase (AST)	15	U/L	Less than 37
	Alanine aminotransferase (ALT)	9	U/L	Less than 41
	Alkaline phosphatase (ALP)	193	U/L	80-306
	Serum lactate dehydrogenase (LDH)	4055	U/L	140-280
Urine	Urine protein	Trace		Negative
	Urine blood	Negative		Negative
	Red blood cell	3-4	Cell/hpf	Less than 3/hpf
	Urine 24 h protein Urine 24 h volume Urine 24 h creatinine	399 700 175	mg/day mL/day mg/day	Less than 150 800-1800 800-2000
Others	HBsAg	Negative		Negative
	ANA (antinuclear antibody)	0.2	Index	Less than 1.5
	CH50	38	U/mL	42-95

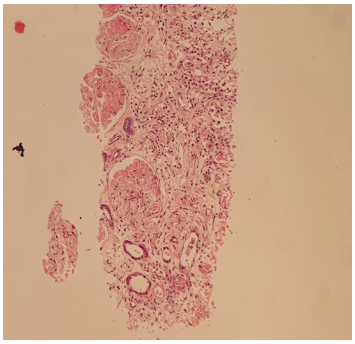

Figure 1. Renal biopsy with 3 totally necrotic glomeruli and chronic inflammatory cells infiltration of interstitium.

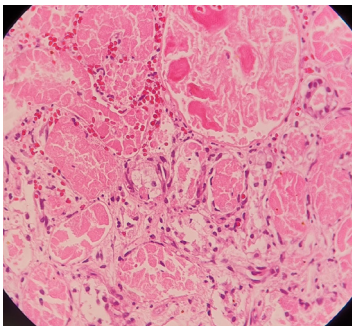

Figure 2. Extensive necrosis of glomeruli and tubules with interstitial chronic inflammatory cells infiltration.

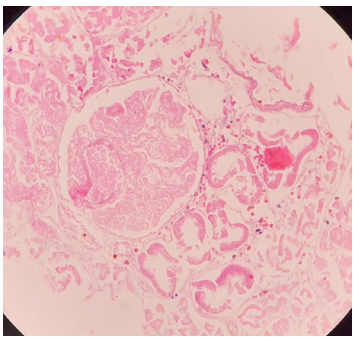

Figure 3. Extensive necrosis of all elements in kidney nephrons.

hemodialysis with initial suspicion of rhabdomyolysis. Diagnosis of renal cortical necrosis was made after biopsy and histopathologic evaluation. Cortical necrosis is a rare cause of AKI. Renal cortical necrosis is mostly a result of decreased renal arterial perfusion (3); however, our patient had a normal arterial blood flow of both kidneys on color Doppler ultrasonography. Renal cortical necrosis of this patient can be caused due to microvascular injury of the kidney due to covid19 infection.

The most common reported causes of intensive care unit admission in COVID-19 patients were either hypoxemic respiratory failure needing mechanical ventilation or hypotension and data on AKI are little (6).

Several clinical and autopsy reports of COVID-19 patients from China and the United States revealed increased clotting and disseminated intravascular coagulation resulting in microangiopathy of other organ systems such as splenic infarction (6).

The incidence of AKI in COVID-19 patients is reported from 0.5 to 80 percent. The three most common histopathologic findings of kidney biopsies in COVID-19 patients were acute tubular injury, collapsing glomerulopathy and thrombotic microangiopathy and these data about pathology of AKI in COVID-19 patients are primarily derived from case reports and case series (7,8).

Conclusion

The rarity of renal cortical necrosis, its important adverse outcomes and its unknown effects on vital systems prompts us to report this case. It is important to emphasis that early recognition and accurate management of COVID-19 infection is necessary to reduce its adverse complications.

Authors' contribution

Conceptualization: Ata Abbasi, Khadijeh Makhdoomi.
Methodology: Ata Abbasi.
Validation: Ata Abbasi, Leila Mahmoudzadeh.
Investigation: Shadi Babaei, Behzad Banaie.
Resources: Khadijeh Makhdoomi.
Data curation: Ata Abbasi, Khadijeh Makhdoomi.
Visualization: Ata Abbasi, Khadijeh Makhdoomi.
Supervision: Ata Abbasi, Khadijeh Makhdoomi.
Writing–original draft: Sepideh Rahimi, Parham Porteghali.
Writing–review and editing: Ata Abbasi, Leila Mahmoudzadeh.

Conflicts of interest

The authors declare that they have no competing interests.

Ethical issues

This case report was conducted in accordance with the World Medical Association Declaration of Helsinki. Written informed consent was obtained from the patient for publication of this report. Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

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