CASE REPORT: Coronavirus with acute kidney injury

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Abstract

Known case COVID-19 is with the appearance of acute kidney injury with no previous history. Pathology studies mainly showed histological findings are consistent with thrombotic microangiopathy. With a high prevalence of comorbidities such as acute kidney injury, patients could suffer from severe forms of COVID-19.

Introduction

Coronavirus disease (COVID-19) is a newly discovered contagious disease caused by severe acute respiratory syndrome (SARS)–coronavirus (CoV)-2 virus, primarily manifesting as an acute respiratory illness with interstitial and alveolar pneumonia, but it can affect multiple organs such as the kidney, heart, digestive tract, blood, and nervous system. The rapidly spreading outbreak, which first emerged in Wuhan, Hubei Province, China, in December 2019, has since been declared a global pandemic(1).The common clinical presentations of COVID-19 are fever (98%), cough (76%), and myalgia and fatigue (18% each), with accompanying leucopenia (25%), and lymphopenia (63%). Symptoms of upper respiratory infection with rhinorrhea and productive cough are uncommon, except in children. About 16% to 20% of cases have been classified as severe or critical(2). Early reports suggested a lower incidence (3%–9%) of AKI in those with COVID-19 infection(3, 4). Recent reports, however, have shown a higher frequency of renal abnormalities. A study of 59 patients with COVID-19 found that 34% of patients developed massive albuminuria on the first day of admission, and 63% developed proteinuria during their stay in hospital(5). Blood urea nitrogen was elevated in 27% overall and in two-thirds of patients who died. Computed tomography scan of the kidneys showed reduced density, suggestive of inflammation, and edema. AKI was an independent risk factor for patients' in-hospital mortality(6).

Case presentation

A 37-year-old woman patient, without a previous history of AKI, was admitted to Razi Hospital in Ahvaz for 10 days in April 2020 with two positive COVID PCR and Cr[1]= 0.8. She was treated with naproxen and hydroxychloroquine. After the symptoms of fever and dyspnea disappeared and the COVID PCR test was negative, she was discharged.

On April 23, 2020, the patient presented with symptoms of weakness, lethargy, and lower extremity edema. The COVID PCR test was negative and two creatinine tests were performed (1.7 and 2.1). The patient's echocardiography showed EF = 50-55% and mild PE (Fig. A).

She was sent to Golestan Hospital with high creatinine and acute kidney injury. The result of the patient's echocardiography was the same. From April 27 to 29, she received two doses of prednisolone (1g). Kidney biopsy and secondary tests were performed for the patient. The results of the second test were normal and the results are shown in Table 1.

Four days later, the patient was discharged from the hospital with a prescription, including Nephrovit, Folic acid, erythropoietin, Prednisolone 75 mg and Allopurinol.

The next day, the patient returned to the hospital with symptoms of dyspnea and a productive cough. She had symptoms of fever (37.8), 80% o2 sat[2], abdominal distension and ascites, three plus-limb edema (Anasarca edema), and Fine Crackles (rales) to the apex of both lungs. TAP ascites fluid was performed for the patient (Table 2). The results of the patient's body fluid test (ascites) showed high protein and high SAAG[3].

[1] creatinine
[2] O2 saturation
[3] serum ascites albumin gradient

According to counseling for the treatment of lung problems caused by a coronavirus, the patient was transferred to the ICU isolation ward. She was prescribed meropenem modified dose, vancomycin modified dose, furosemide, serum TNG (nitroglycerin), and prednisolone (50 mg).

According to the nephrologist's advice (suspected PCP), prednisolone and co-trimoxazole injection were started for the patient. Chest CT showed pleural congestion in the patient(Fig.B).

The next morning, IVIG was prescribed to the patient, but before receiving IVIG, the patient had decrease o2sat, leading to respiratory dystrophy. Intubation was performed for her, but the patient died. Two days later, the COVID PCR test was positive.

Results of kidney biopsy showed histological findings are consistent with thrombotic microangiopathy (Fig.C).

In terms of glomeruli, a maximum number of 27 glomeruli are seen in serial sections examined. The glomeruli show mild enlargement with a simplification of tuft architecture due to segmental to global mesangial expansion with a fluffy abd hyaline appearance (mesangiolysis). Mesangial cells are not proliferated. Glomerular capillaries are mostly devoid of RBCs and show a narrowing of lumen due to endothelial swelling. Rare fragmented RBCs or fibrin thrombi are seen in a few capillaries and hilar arterioles. Capillary walls show irregularity and corrugation of contours.

In terms of Tubules, degenerative vacuolization of renal tubular epithelial cells are seen in some of the proximal tubule profiles.

Discussion

Tens of thousands of humans were infected with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) within a short period of time, and the infection spread quickly across China and throughout the world. Acute kidney injury (AKI) is one of the important complications of the 2019 novel coronavirus disease (COVID-19), occurring in 0.5–7% of cases and in 2.9–23% of ICU patients(7, 8). Wong et al(9) showed AKI was uncommon in COVID-19. SARS-CoV-2 infection does not result in AKI, or aggravate CKD in the COVID-19 patients. In our case report, the patient had no history of acute kidney injury, but COVID-19 infection resulted in AKI.

The exact mechanism of kidney involvement is unclear. Postulated mechanisms include sepsis leading to cytokine storm syndrome or direct cellular injury due to the virus. Angiotensin converting enzyme (ACE) and dipeptidyl peptidase-4, both expressed on renal tubular cells, were identified as binding partners for SARS-CoV and MERS-CoV, respectively(10, 11). Viral RNA has been identified in kidney tissue and urine in both infections(12). Recently, Zhong's lab in Guangzhou successfully isolated SARS-CoV-2 from the urine sample of an infected patient, suggesting the kidney as the target of this novel coronavirus(13). Also, Similar to SARS-CoV infection, the spike (S) protein of SARS-CoV-2 binds to angiotensin-converting enzyme 2 (ACE2), a host cell receptor, and the S protein is activated and cleaved by cellular transmembrane serine proteases (TMPRSSs), allowing the virus to release fusion peptides for membrane fusion(14, 15). Therefore, the coexpression of ACE2 and TMPRSSs is a key determinant for the entry of SARS-CoV-2 into host cells and improves host conditions for coronavirus.

pan et al(15) clearly identified podocytes and proximal straight tubule cells as kidney host cells. Podocytes and proximal straight tubule cells play critical roles in urine filtration, reabsorption, and

excretion. Notably, podocytes are particularly vulnerable to viral and bacterial attacks, and podocyte injury easily induces heavy proteinuria(16). So, the cytopathic effects of SARS-CoV-2 on podocytes and proximal straight tubule cells may cause AKI in patients with COVID-19, especially in patients with SARS-CoV-2 infection in blood samples. Therefore, we need to pay more attention to the early monitoring of renal function and cautiously handle the urine of COVID-19 patients with AKI to prevent accidental infection.

Conclusion

We have presented the reported case of COVID-19 with AKI. Overall, with a high prevalence of comorbidities such as acute kidney injury, patients could suffer from severe forms of COVID-19. In patients with COVID-19 besides the risk of respiratory failure, the danger of acute kidney injury should be considered as well. Also, COVID-19 can show acute kidney injury symptoms that patients without a history.

Consent

Written informed consent was obtained from the patient for publication of this Case report and any accompanying images.

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Table 1 .IMMUNOASSAYS AUTOIMINNE DISEASE

TEST	RESULT	UNIT	NORMAL RANGE
Anti GBM Ab	Negative	Titer	Up to 1.10
Anti SSA(Ro) Ab	Negative(0.2)	Ru/ml	Negative<20 Positive>=20
Anti SSB(La) Ab	Negative(0.8)	Ru/ml	Negative<20 Positive>=20
Anti dsDNA Ab	Negative(1.1)	IU/ml	Negative<30 Grey zone: 30-35 Positive>35
ANCA(P)	Negative	Index	ELISA: Negative<1.0 positive>=1.0 IF: Negative<1.10 Positive>1.10
ANCA(C)	Negative	Index	ELISA: Negative<1.0 positive>=1.0 IF: Negative<1.10 Positive>1.10
Anti CCP Ab(CPA)	Negative(0.8)	U/ml	Negative<30 Positive>=30
Anti phospholipid Ab (IgG)	Negative	U/ml	Negative<10 Positive>=10
Anti phospholipid Ab(IgM)	Negative	U/ml	Negative<10 Positive>=10
Anti Cardiolipin Ab (IgG)	Negative	U/ml	Negative<10 Positive>=10
Anti Cardiolipin Ab (IgM)	Negative	U/ml	Negative<10 Positive>=10
COAGULATION			
Lupus anticoagulate	37.4	sec	31-45
DRVV screan	1.3	sec	Normal: absence<1.2 Abnormal: presence>1.2
Special biochemistry			
C3	1.24	g/l	0.9-1.8
C4	0.256	g/l	0.09-0.39
CH50	60.0	%	Low<50 Normal 50-150 High >150
Serology			
RF	Negative	IU/ml	Quantitiative (IU/ml) Up to 20 Qualitative: normal: negative

Table 2. Body fluid	Test		
Liquid test	Result	Unit	Normal range
Appearance	Yellow, semi clear		clear
Total cell count	68		
RBC	33		
Leukocytes	35		
Neutrophils	15	%	
Lumphocytes	20	%	
Glucose	129	Mg/dl	50-80
Protein	3.5	Mg/dl	15-45
Albumin	2.5		
LDH	139		
Other	*		
* The sample was xanthochromia after centrifugation.			

Fig A. Echocardiography

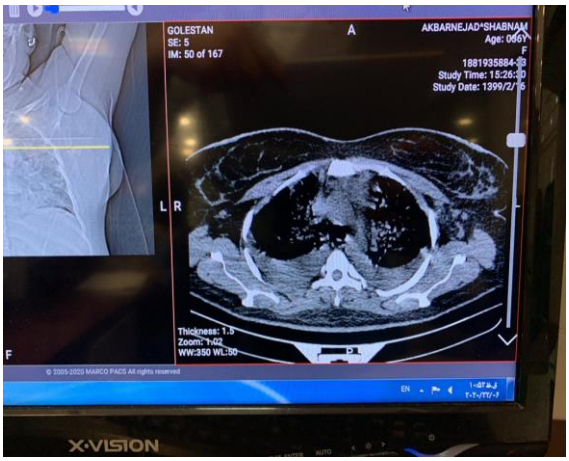
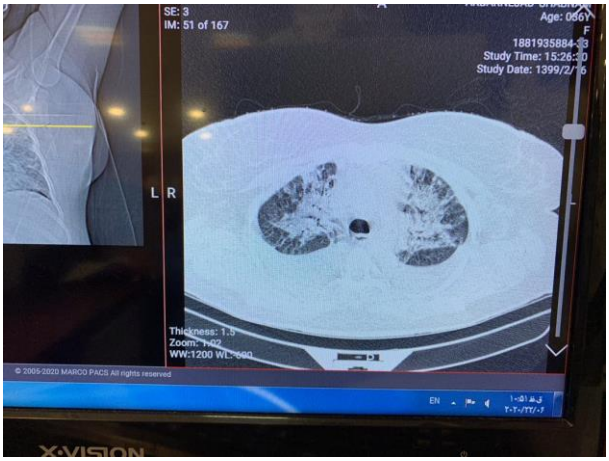


Fig B. Chest CT



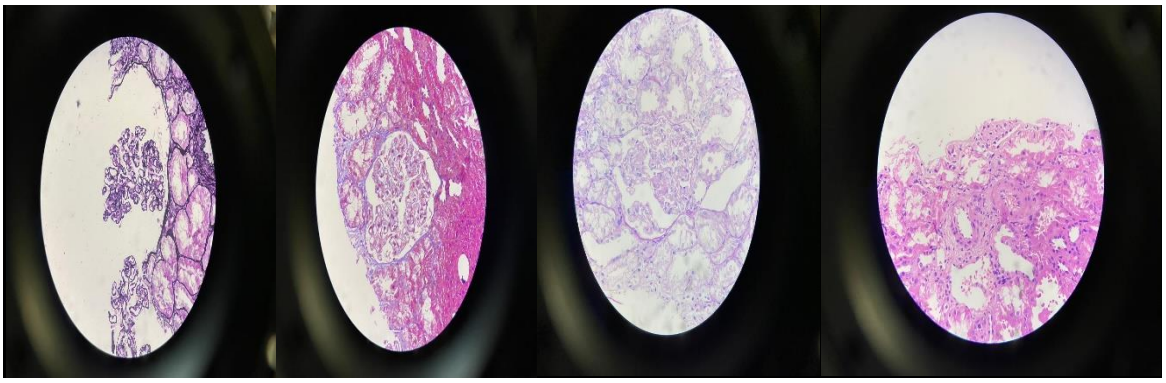


Fig C. Results of kidney biopsy