Full recovery of a patient with COVID-19-induced acute kidney injury

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ABSTRACT

Background: In this report, we discuss the diagnosis and management of a case of COVID-19-induced acute kidney injury (AKI).

Case Presentation: A 58-year-old male with PCR-based COVID-19 diagnosis (at a specialized hospital, Minia, Egypt) was admitted and received supportive medications along with corticosteroids and hydroxychloroquine. After 2 days, the patient developed tachypnoea and desaturation. Therefore, he was transferred to the intensive care unit with a continuous positive airway pressure. On the third day, he developed oliguria with spiking kidney function tests, metabolic acidosis, and eventually anuria on the 6th day. AKI diagnosis was established, and the patient received daily dialysis sessions for 10 days until discharge together with tocilizumab and methylprednisolone. The patient was discharged after normalization and stabilization of his clinical parameters and a second negative PCR swab with continuous follow-up.

Conclusion: Early monitoring of kidney function tests during the infection might help in preventing further kidney damage.

Keywords: Coronavirus, infection; COVID-19, kidney, renal failure.

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Background

Since the beginning of the COVID-19 pandemic, there were no specific global treatment guidelines, which forced many countries to develop their own treatment protocols for COVID-19. However, the management of COVID-19 is complicated due to the variety of clinical presentations and unknown facts about viral pathogenesis. In the early pandemic, respiratory involvement was the major presentation noticed among these patients. However, recent investigations have recorded other manifestations as acute kidney injury (AKI), and many other complications [1]. AKI has been linked to sepsis, which commonly causes multi-organ failure [2]. The reported incidence of AKI among COVID-19 patients is variable with increasing in-hospital high morbidity and mortality rates [1]. The most common clinical features include proteinuria and hematuria occurring in 44% and 27% of AKI patients [3]. However, diagnosis and management of COVID-19 induced AKI are challenging as indicated by the relatively high mortality rates [1]. In this report, we discuss the diagnosis and management of a case of COVID-19 induced AKI presenting the clinical and laboratory features throughout the disease.

Case Presentation

On May 16, 2020, a 58-year-old male presented to the Minia Fever Hospital, Egypt, with fever, sore throat, bony ache, and dry cough. Apart from his well-controlled hypertension, he had previous deep venous thrombosis. A computed tomography (CT) scan of the chest, complete blood count, as well as liver and kidney function tests, were performed on the same day of admission. His CT chest revealed mild-to-moderate ground glass opacities scattered posteriorly and peripherally. Accordingly, the patient was isolated, and a PCR swab was taken, which turned out to be positive. Accordingly, an urgent transfer to the Mallawi Specialized (Quarantine) Hospital, Minia, Egypt, was made where his O_2 saturation started to decline. Thus, he had a venturi mask 60 with 15 l O_2 to restore oxygen saturation (Figure 1). Main laboratory findings are detailed in Table 1.

Following the Egyptian Ministry of Health's guidelines for confirmed cases (back then), the patient was given a daily combination of Tamiflu 75 mg/12 hours, azithromycin 500 mg/24 hours for 5 days, hydrocortisone sodium succinate (Solu-Cortef ampoule)/6 hours, Zinctron 50

mg/24 hours, vitamin C 1 g/24 hours, Enoxaparin sodium (Clexane) 60 mg/12 hours, proton pump inhibitors/24 hours, and hydroxychloroquine 400 mg/12 hours at the first day, and then 200 mg/12 hours daily for 6 days. Two days after admission, he developed tachypnea and desaturation. Therefore, he was transferred to the intensive care unit (ICU) by applying a continuous positive airway pressure (CPAP) (pressure support ventilation 14, positive end-expiratory pressure 8, the fraction of inspired O_2 60%) to compensate O_2 and control the movement of respiratory muscles. Additionally, a central venous line was established.

On May 18th, he developed oliguria (200 ml/24 hours), subsequently, he had an ultrasound which revealed mild hepatomegaly, and normal kidneys in the site, size, and echogenicity. Furosemide (Lasix) infusion was given a trial

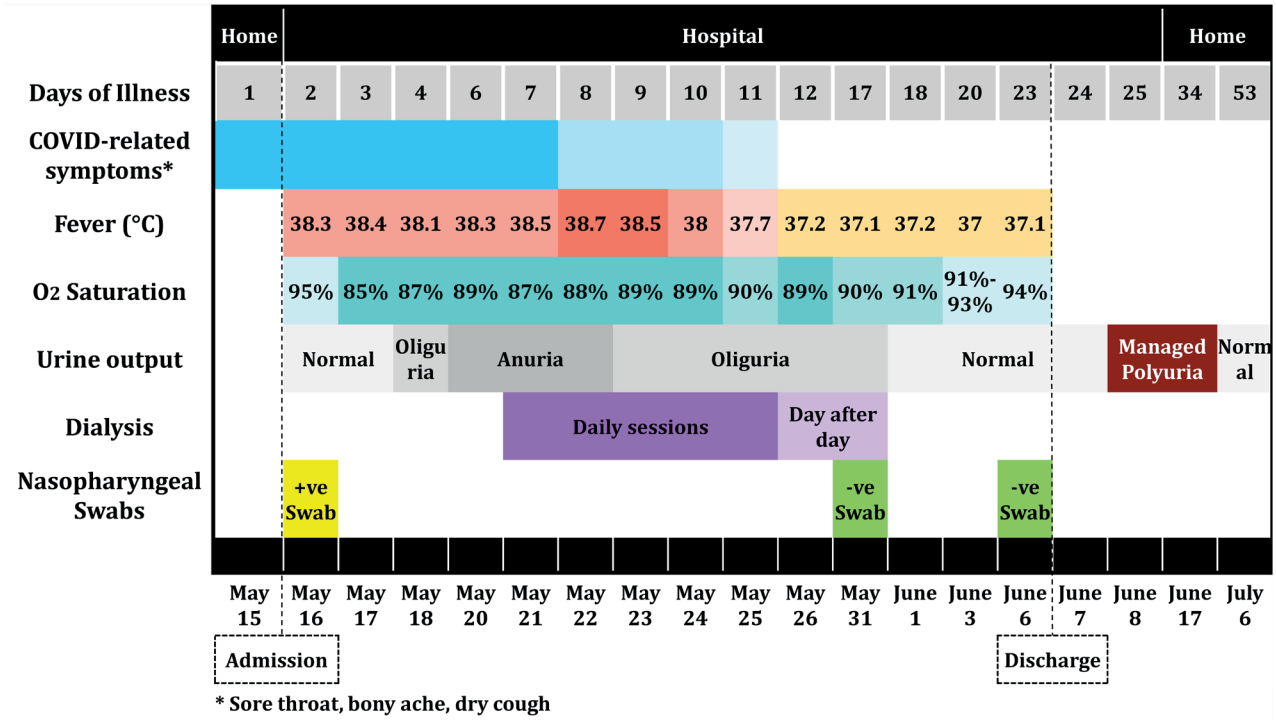

Figure 1. *The clinical course of the patient.*

Table 1. Laboratory results of the patient.

	Reference	16/5	17/5	18/5	19/5	20/5	21/5	24/5	25/5	26/5	27/5	28/5	1/6	7/6	17/6
Creatinine	0.8-1.43mg/dl	0.97	***1.8***	***4***	***5.5***	***6***	***9***	***7***	***8.1***	***7.4***	***6.7***	***6***	***4.9***	***2.7***	1.04
Urea	17-43mg/dl	22	***50***	***103***	***165***	***185***	***225***	***131***	***195***	***150***	***202***	***152***	***189***	***125***	***49***
Na	136-146 mmol/L	141	140	142	139	140	140	134	131	132	128	129			143
K	3.5-5.1 mmol/L	4.3	4.5	5	4.8	***5.8***	***5.5***	5.1	4.8	4.9	***5.2***	5.1			4
Ca	1.15-1.3 mmol/dl	-	-	-	-	-	1.21	1.21	1.1	0.89	**1.5**	1.14			
ALT	0-50U/L	***88***	***88***	-	-	-	-	***214***	***540***	***385***	***133***	***226***	***102***	34	38
AST	0-50U/L	***106***	***106***	-	-	-	-	***103***	***125***	***165***	***87***	***104***	25	11	18
ALB	4.5-5.5 g/dL	***4.1***	***4.1***	-	-	-	-	4.5	***3.9***	***4.1***	***3.7***	***4***	3.4	5.2	
Ferritin	20-250ng/mL	***3721***	-	-	-	-	-	-	-		-	-	-	-	-
d.Dimer	<0.5µg FEU/ml	***400***	-				-	-	-	***3.8***	-	-	-	-	0.5
CRP	<6mg/L	***117***	-	-	-	-	-	-	-	-ve	-	-	-	-ve	
TLC	4-11 x 10^3/uL	5.1	5.1	-	-	-	-	***24.5***	***21.1***	***18.15***	***20.2***	***23.42***	9.6	8.3	8.8
Neutrophils	20-80%	68.9	68.9	-	-	-	-	80		85	78	-	-	-	62
lymphocytes	20-45%	***18.7***	***18.7***	-	-	-	-	***8.7***	***9.7***	***13.2***	***14.7***	***13.3***	-	***12.6***	26
platelets	150-450 x 10^3/uL	176	176	-	-	-	-	357	341	289	270	228	***115***	***465***	277
Hb	13-17 g/dL	14.3	14.3	-	-	-	-						13.3		13.3
Glucose	60-170mg/dl	162	157	152	162	170	185	250	155	148	160	143			93

*Abbreviations; Hb = hemoglobin, ALT = alanine aminotransferase, AST = aspartate aminotransferase, ALB, albumin blood, CRP = C-reactive protein, TLC = total leukocyte count. *Abnormal values are in bold.*

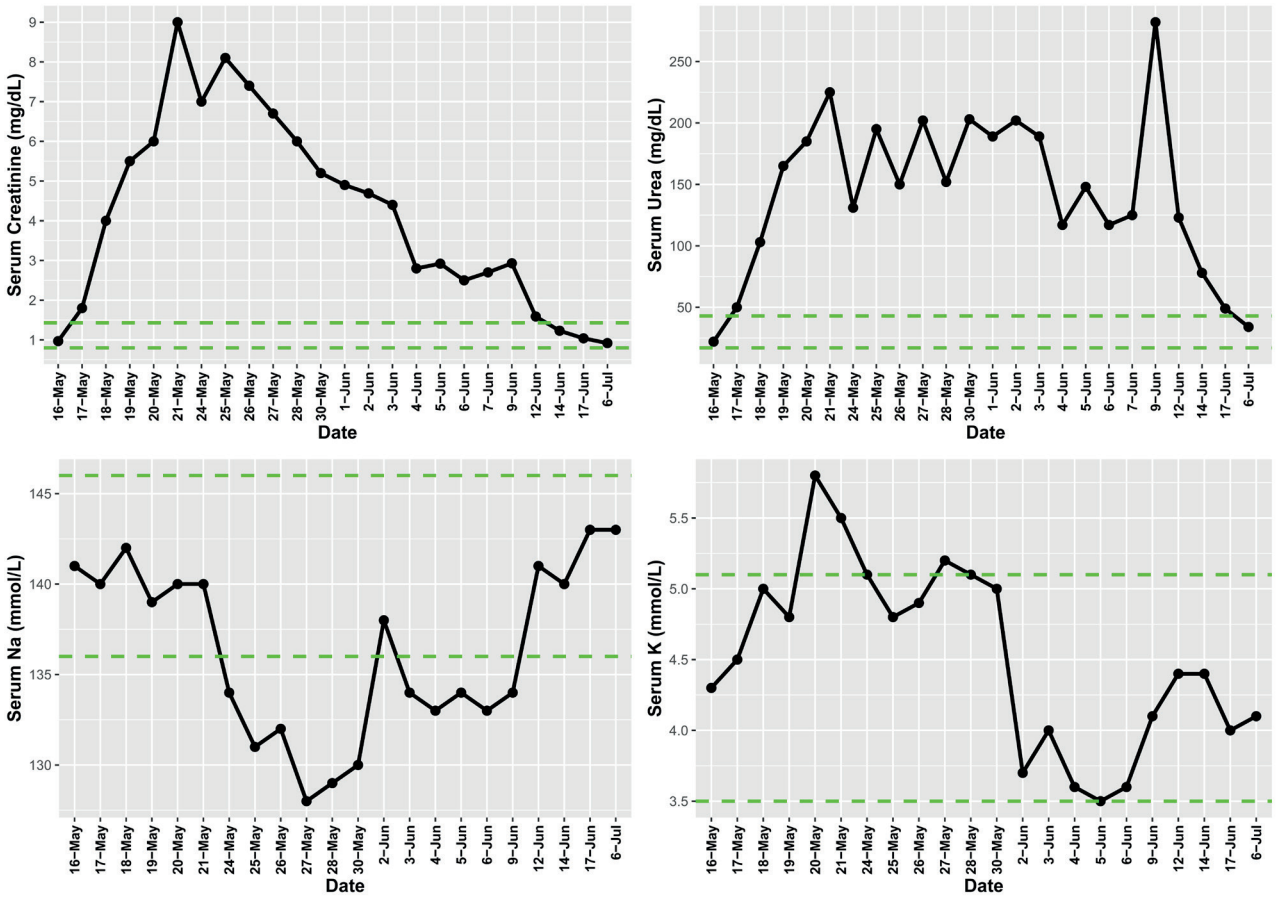

Figure 2. *The laboratory findings of the patient across the infection course.*

but with no increase in the urine output (UOP), which was associated with spiking kidney function tests (serum creatinine, urea, sodium, and potassium), metabolic acidosis, and eventually anuria on May 21st (Figure 2). An urgent nephrology consultation was made which established AKI diagnosis and the patient was ordered an immediate dialysis session (flux filter for 1.5 hours, blood pressure: 90/50, pump 230, bicarbonate: 4, sodium: 140, heparin free ultrafiltration of 1 l). At the same time, one vial of tocilizumab (400 mg) was administered along with steroid therapy in the form of initial pulse Methylprednisolone (Solupred) (500 mg/24 hours) for 3 days, followed by a maintenance dose of Methylprednisolone (Solupred) 40 mg/24 hours. Dialysis sessions were continued daily till May 25th when it was changed to be intermittent day-after-day sessions.

The first negative PCR came on May 31st, which was associated with improved UOP (400 ml/12 hours) and subsequent cessation of dialysis by June 1st. By that time, the gradual weaning from CPAP to a venturi mask was initiated, to reach complete weaning and discharge from ICU by June 3rd, with O_2 saturation of 91% in room air (RA). The second negative swab came on June 6th. The patient was discharged the next day after stabilization of his clinical parameters (normal pulse and blood pressure, O_2 saturation: 94% RA, and UOP: 1.5 l/day) and relative normalization of his lab studies (Figures 1 and 2). Home treatment included Methylprednisolone (Solupred) 40 mg/24 hours, Alfacalcidol 0.5 microgram (One-Alpha)/24 hours, Calcium Citrate-and Vitamin D3 combination 400 mg/24 (calcimate), Rivarospire 20 mg/24 hours, Aspirin 100 mg/24 hours, and Rosuvastatin Calcium 20 mg (Crestor). The case was followed up regularly by a nephrologist, who reported transient manageable polyuria (volume status control) and normalization of all kidney functions till the last recorded follow-up on July 6th (Figure 2).

Discussion

AKI is mainly attributed to acute tubular injury when multi-organ failure and shock occur. Su *et al.* [4] in their post-mortem case series of AKI showed that diffuse proximal tubule injury was the main characteristic. The pathogenesis of AKI development throughout COVID-19 infection is multi-factorial: first: histopathological reports have indicated that SARS-CoV-2 directly infects renal cells due to the presence of ACE2 receptors [5], second: microvascular dysfunction due to endothelial damage by lymphocytic endothelialitis and the presence of viral inclusion particles in glomerular capillary endothelial cells [6], third: the cytokine storm that may play a significant role in AKI pathology as a response to the viral infection [7].

AKI due to COVID-19 infection is a severe complication and should be treated promptly. AKI alone has a

mortality rate of 40%, which describes how urgently this disease should be managed [8]. In China, the incidence of COVID-19-induced AKI ranged between 0.5% and 29% [3,9] while in New York, a higher range was recorded (37%) [10]. Consequently, early care for kidneys at admission is essential due to the potential high incidence rate in addition to the risk of developing pre-renal AKI when avoiding hypervolemia which may worsen the patient's respiratory condition. Moreover, in our case, AKI developed on the third day after admission which lies within the range of previously published reports in China which reported a median of 7-14 days [3,9] and the USA which reported that a third of the COVID-19 patients developed AKI after one hour only from admission [10]. Besides, as mentioned before that proteinuria and hematuria are the most common AKI symptoms, neither of these were noticed in our patient.

The relatively high incidence of AKI impels conducting early diagnostic measurements as urine analysis, and protein-to-creatinine ratio should be conducted for early detection and better prognosis with the initiation of renal replacement therapy (RRT). Cummings et al. [11] reported that 31% of their patients received RRT. The ELIAN trial showed that early management of AKI with RRT had statistical significance than using it late in terms of decreasing the time spent on RRT, rapid kidney recovery. It enhanced renal function tests, in addition to less time spent on mechanical ventilation [12]. In critically ill individuals with AKI, early RRT may produce favorable outcomes through preventing life-threatening disorders, such as reducing hypervolemia, removal of toxins/electrolytes, correcting sepsis-associated acidosis, and prevention of complications associated with AKI [13], and therefore has economic advantages by decreasing the time spent in the ICU as with our case. Despite the importance of RRT, certain factors should be considered, including the maintenance of the extracorporeal circuits needed for RRT and the use of measures to prevent spreading the infection.

Conclusion

As our understanding continues to evolve about how COVID-19 induces AKI, the best therapeutic modality and high-risk populations; early monitoring of kidney function tests during the infection might help in preventing further kidney damage.

What is new?

COVID-19 may produce acute kidney injury. The authors report the first case of full recovery of a patient diagnosed with COVID-19 acute kidney damage.

Funding

None.

Conflict of interests

All authors have no conflict of interest.

Consent for publication

Informed consent was obtained from the patient to publish this case.

Ethical approval

All the methods were performed in accordance with the relevant approved guidelines, regulations, and declaration of Helsinki. Written informed consent was obtained from the patient to have her details and accompanying images published. Formal Ethical approval is not required at our institution to publish a case report.

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Summary of the case

1	**Patient (gender, age)**	Male, 58 years
2	**Final Diagnosis**	COVID-19 induced acute kidney injury
3	**Symptoms**	Tachypnoea and desaturation, oliguria with spiking kidney function tests, metabolic acidosis, and eventually anuria
4	**Medications**	The patient received daily dialysis sessions for 10 days until discharge together with tocilizumab and methylprednisolone.
5	**Clinical Procedure**	The patient received daily dialysis sessions for 10 days until discharge together with tocilizumab and methylprednisolone
6	**Specialty**	Infectious diseases