Journal of Global Public Health (JGPH)
Volume 2 Number 2, July- December 2020

Acute Renal Failure Triggered after Moderate to Severe Coronavirus Infection

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How to cite this article:

Banu Manickam Rajalu, Veena Kumari HB, Shashidhara HN et al. / Acute Renal Failure Triggered after Moderate to Severe Coronavirus Infection. Journal of Global Public Health. 2020;2(2):69–72.

Abstract

Background: SARS-CoV-2 infects multiple critical organs such as the kidney, besides the lungs. Acute kidney injury caused after coronavirus disease requires immediate renal replacement therapy. In this case report, we present a new case of developing severe acute renal failure after testing positive for COVID-19.

Case Description: This case was tested positive for COVID-19 twice. After the second testing, his renal function test increased drastically (serum creatinine, 9.68 mg/dl and urea 155mg/dl) along with deranged serum ferritin,823.6ng/ml; CRP, 4.61mg/dl; leucocytes, 12 × 1000 cells/cubic mm; D-Dimer, 269ng/ml and was diagnosed with severe acute renal failure after corroboration with radiological investigations. He was aggressively treated with renal replacement therapy (5 cycles of hemodialysis) for renal injury along with other conservative management for COVID-19 disease. After 14 days of hospitalisation, he became clinically stable with normal renal function and was tested negative for COVID-19. He resumed all his normal activities immediately after hospitalisation, and resumed duty after 3 weeks of post-discharge.

Clinical Relevance: Normal blood renal function test before the COVID-19 infection, sudden increase of blood values during the infection period, and reaching back to the normal range after treating the infection was highly suggestive of COVID-19 triggered severe acute renal failure. Regular monitoring of renal parameters along with other investigations is essential for managing the complications raised from COVID-19 infection. Health professionals need to be aware of kidney disease presentation during COVID-19 infection.

Keywords: AKI; RRT; Haemodialysis, Blood urea; Plasma creatinine, Before-during-after COVID-19.

Introduction

Coronavirus disease 2019 (COVID-19) is a contagious respiratory and vascular disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), that might cause systematic inflammation with the cytokine release and affecting multiple critical organs besides the lungs.[1] Though the initial studies indicated that the rate of acute kidney injury (AKI) was not common,[2,3] developing evidence demonstrated the prevalence of AKI among COVID-19 patients. Kidney involvement in patients with coronavirus disease 2019 (COVID-19) can range from the presence of proteinuria and haematuria to acute kidney injury (AKI), requiring renal replacement therapy (RRT). COVID-19-associated AKI (COVID-19 AKI) is associated with high mortality and serves as an independent risk factor for all-cause in-hospital death in patients with COVID-19.[4]

Case Report

A 56 years old man with, known case of type II Diabetes Mellitus, was isolated in our adopted COVID care center[5] on 14th July 2020 after testing positive for COVID-19, but asymptomatic. He was treated conservatively with Tab B. Compex, Vitamin C, Vitamin D, and Zinc capsules, and was later tested negative after 3 weeks. In October 2020, he was presented to us with the symptoms of fever, body ache, loose stools, loss of smell and taste with severe tiredness. The COVID-19 test turned positive for the second time on 13th October 2020, within 3 months from the first COVID-19 positive test. As he worked as a hospital ambulance driver, the secondary source of infection was assumed to

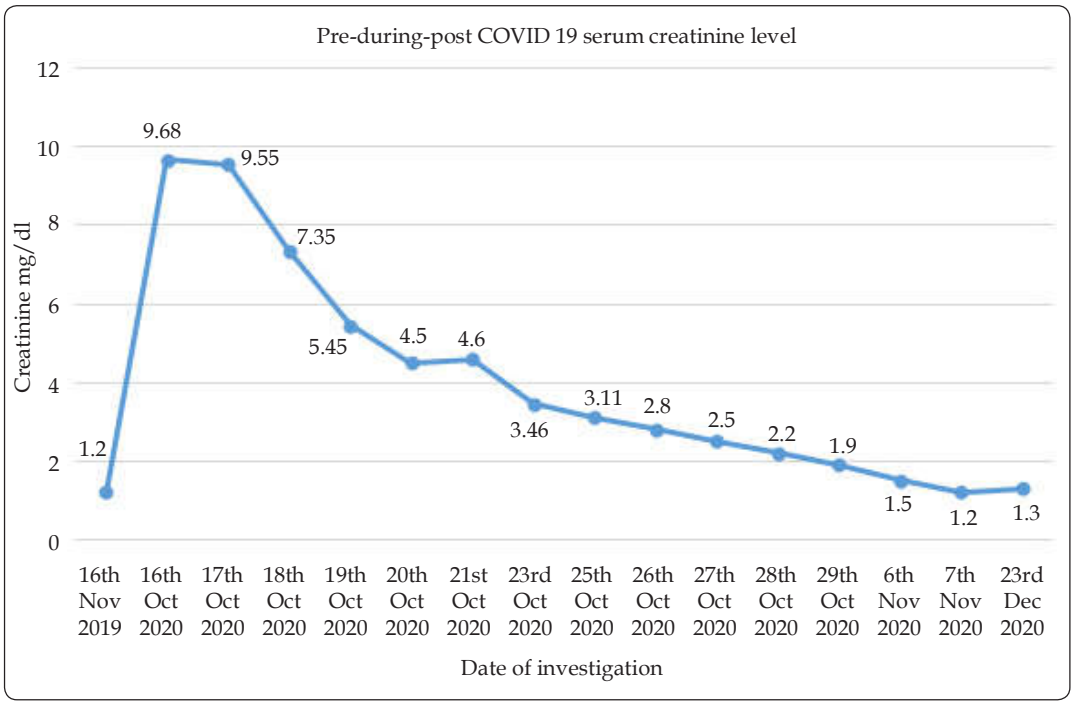

Fig. 1: Serum creatinine level before-during-after COVID 19.

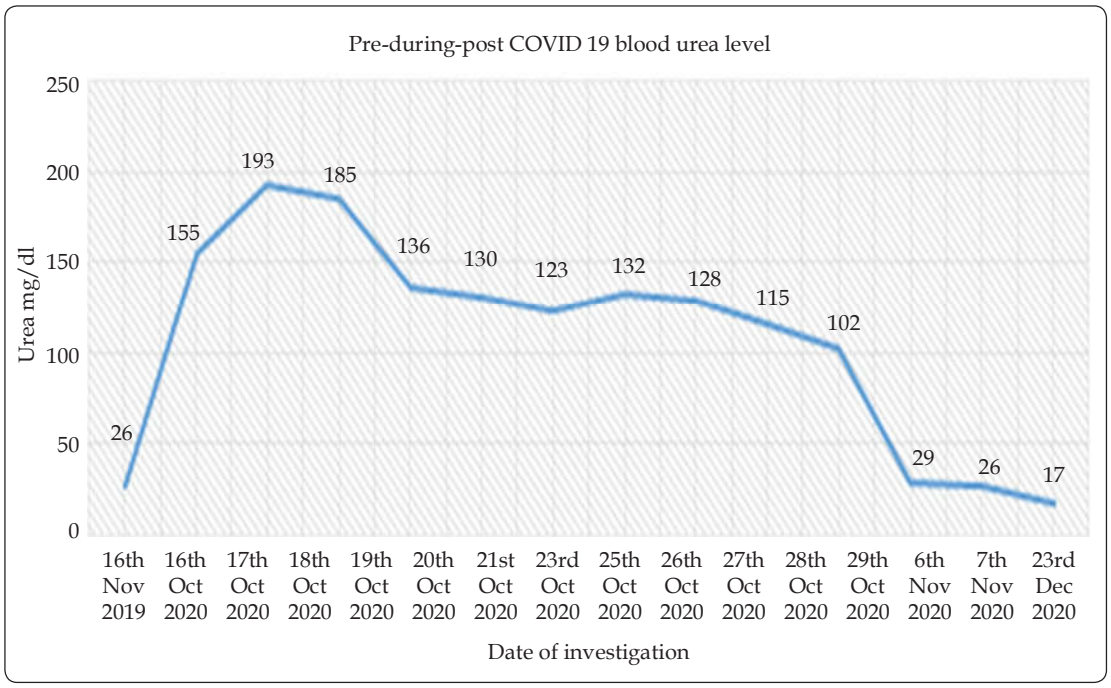

Fig. 2: Blood urea level before-during-after COVID 19.

be patients or caregivers infected with COVID-19, though could not be predicted accurately. He was isolated in our adopted COVID care center following the COVID-19 retest. After a couple of days, he developed breathing difficulty (Spo2 - 89%), had severe fatigue with a dull appearance. He was immediately shifted to a COVID designated hospital on 15th October 2020 in Bengaluru. No previous history of kidney disease, pulmonary disease, or hypertension was reported. No evidence of diabetic nephropathy was noted from his previous routine investigations (16th November 2019 report: blood urea 26.0 mg/ml, creatinine 1.2 mg/dl, total WBC count 9.8 × 1000/ul, and liver function test and serum electrolytes within normal limits). Also, he was not on medications such as angiotensin enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARB), or any other medications, except for the regular dose of Injection Human Mixtard twice a day.

On admission to the hospital, he was connected to oxygen by mask, and basic laboratory investigations, chest X-ray, and CT abdomen were done. Chest X-ray showed scattered air space opacity in both lungs. The CT chest showed multifocal confluent peripheral ground-glass opacities with interlobular septal thickening, having a crazy-paving appearance, and evolving consolidations in all the lobes of the lungs bilaterally - depicting the features of COVID-19 pneumonia, the extent of disease ranging from 50 to 75%. The kidney showed a moderate-sized exophytic renal cortical cyst in the mid pole of the left kidney measuring 4.5 × 4.7 cm with few small renal cortical cysts in the right kidney. The renal function test were high; blood urea -155 mg/dl, creatinine 9.68 mg/dl by Jaffe rate-IDMS standardization test, protein creatinine ratio 1.39 mg/mg (normal: <0.2 mg/mg), with deranged inflammatory markers – leucocytes: 12 × 1000 cells/cubic mm. The serum Ferritin was 823.6 ng/ml (normal 23.9 – 336 ng/ml) on chemiluminescence test, C - reactive protein (CRP) - 4.61 mg/dl (normal: <0.8mg/dl) by rate turbidometry test, and D-Dimer – 261ng/ml (normal: <250ng/ml) by automated latex enhanced immunoassay test. Urine appeared slightly cloudy with a trace of glucose and the presence of proteins. The other chemical examination on a urine test such as bilirubin, ketone bodies, and microscopic examination were in the normal range.

Nephrology opinion ruled out acute kidney failure. He underwent five cycles of hemodialysis through Right Internal Jugular Vein (RIJV) central line access. The other conservative treatment such as empirical ceftriaxone 2gm od × 3 days, 0.9% sodium chloride fluid transfusion for hyponatremia, Inj. Remdesivir 100mg × 2 days, Vitamin C 500 mg bd × 5 days, Tab. Zinc acetate 50 mg bd × 2 days, Inj pantoprazole od × 3 days, Tab. Dolo 650mg SOS and Inj. Human Mixtard 30/70 14 units in the morning and 10 units at night was administered.

Following hemodialysis and supportive management, the creatinine (Figure 1) and urea (Figure 2) reached normal values and bicarbonate ranged from 12.4 to 18.9 mmol/L during hospitalisation period. Serum potassium and chloride were within normal range except for serum sodium ranging from 129 to 134 mmol/L which was corrected with 0.9% sodium chloride solution intravenously.

Investigations after 14 days of hospital admission: C-reactive protein reduced from 4.61 mg/dl to 0.9 mg/dl. However, serum ferritin (1191.5 ng/ml), LDH (292 IU/L), leucocytes (17×1000 cells/cubic mm), fasting sugar (236 mg/dl) and postprandial (296 mg/dl) with HbA1C (11%), and D-Dimer (370 ng/ml) continued to be high. SARS COV-2 IgG serum antibodies were reactive - 31.57 S/CO (>/=1.0 S/CO is considered reactive by CMIA testing). The chest X-ray showed an interval increase in the air space opacities in both lungs. He was clinically stable with normal vital parameters and oxygen saturation except for severe tiredness. He was tested negative for COVID 19 on the 14th day of admission and subsequently was discharged from the hospital on 29th October 2020 on prescription with Tab. Predmet 8mg BD ×5 days, Tab. Pantoprazole 40 mg OD × 14 days, Tab. Vitamin C 500mg OD × 10 days, Tab Besozinc bd × month, Tab. Ecospirin 75mg od, and Inj. Human Mixtard 30/70 12 units in the morning and 10 units at night. He resumed his duty after 3 weeks of his discharge from the hospital.

Repeat blood investigations on 23rd December 2020: Serum creatinine 1.3 mg/dl (Figure 1), blood urea 17 mg/dl (Figure 2), liver function test and serum electrolytes within normal limits, Total WBC count 10.5 × 1000/ul, CRP 0.1mg/dl, and D-Dimer 208 ng/ml with the continued increase in serum Ferritin level 670 ng/ml and HbA1C 9.3%.

He is currently clinically stable, doing well, resumed duty, and on medical supervision for his diabetic control.

Discussion

Patent developed ARF after 3 months from his

first COVID-19 positive test or within 3 days from the second test that correlated with the late viremic response phase of the SARS infection.[6] His plasma urea and creatinine level were very high, suggestive of renal damage which warranted immediate RRT. However, impaired renal function with raised plasma creatinine is not commonly found during the clinical presentation at an earlier stage.[7] Unfortunately, we did not test his renal function test during the first test of COVID-19. COVID-19 has been hypothesized to trigger renal failure as he was re-infected and the condition was severe to cause acute kidney injury. Individuals who develop secondary infections are at increased risk of secondary sepsis-associated AKI.[8] Haemodialysis, in his case, helped to reduce the urea and creatinine level to a normal range within 14 days of hospitalisation that is in line with another study that reported a fall of creatinine level over 10 days.[7] Haemoperfusion sorbents were assumed to remove virus particles and cytokines in patients with high endotoxin levels.[4] In developed countries, hemodialysis is the mainstay of RRT[9] to optimize renal functions. The mechanism of AKI in patients with COVID-19 has not been fully studied which seems to have multifactorial pathophysiology. SARS-CoV-2 might display viral tropism and directly affect the kidney.[4] Though pulmonary damage and respiratory failure were commonly reported complications of COVID-19[10], the involvement of other organs such as the kidney is also evident from this case that needs immediate attention, as it carries the risk for mortality. Health professionals need to be aware of kidney disease presentation during COVID-19 infection. The relation with COVID-19 induced acute renal failure in a patient with comorbid diabetes mellitus merits further study which might have a direct pathogenic mechanism.

The patient's renal function parameters were normal before his COVID 19 status, the plasma creatinine and urea level suddenly peaked during the SARS-CoV-2 period and gradually returned to normal level at the time of discharge which is highly suggestive of COVID 19- triggered renal injury. Early investigations and diagnosis of COVID 19-related complications such as kidney diseases, and appropriate and meticulous interventions with adequate renal replacement therapy would help patients to recover, thereby preventing increase in mortality rate.

Acknowledgment

We acknowledge the health professionals of the COVID-19 designated hospital, Bengaluru for the management of the patient.

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