Acute Kidney Injury in a Nigerian Patient with COVID-19 and a Shrunken Kidney

Akpowaye A[1] and Okoye O[2]

[1]Department of Internal Medicine, Federal Medical Centre, Asaba, Delta State, Nigeria
[2]Department of Internal Medicine, Delta State University, Abraka, Delta State, Nigeria

ABSTRACT

A case report of a 42 year old Nigerian male who presented with complaints of high grade intermittent fever, dry unproductive cough and difficulty in breathing of 3 days duration; with associated history of protracted vomiting and generalized body weakness. There was no history of recent travels and he was not aware of exposure to COVID-19 virus. A reverse transcriptase polymerase chain reaction (RT-PCR) for SARS-COV2 was positive and he was treated for COVID-19 pneumonia. During the course of the illness, he had AKI with azotaemia, hyponatremia, hyperkalemia, acidosis, and anuria and had several sessions of haemodialysis among several medications. Renal ultrasound scan showed a shrunken right kidney and normal sized left kidney.On admission he developed deep vein thrombosis and was commenced on treatment. Renal function returned to near normal and he is being followed up in clinic.

The case report highlights the multifactorial risk factors of AKI in the setting of COVID-19 and the challenges of care in a low-resource setting.

Keywords: *Case Report, AKI, COVID-19, Shrunken Kidneys,*

INTRODUCTION

The incidence of AKI in COVID-19 patients ranges from 0.5% to 36.6%; and is associated with greater severity of illness and increased mortality [1]. We present the clinical findings of a middle-aged African man with COVID-19 associated AKI and discovered to have a shrunken kidney on evaluation. The aim of this case is to highlight multifactorial risk factors of AKI in the setting of COVID-19 and the challenges of care in a low-resource setting.

CASE PRESENTATION

A 42 year old African man presented at the emergency room with complains of high grade intermittent fever, dry unproductive cough, dyspnea and generalized body weakness of 3 days duration with associated vomiting and diarrhea. The notable past medical history was a gun-shot injury to the left thigh that required vascular surgery eight years before presentation; as well as deep vein thrombosis involving same limb two years after the surgery. There was no history of recent travel or exposure to a suspected COVID-19 patient. He takes alcohol occasionally, does not use tobacco or any other substance. He took over the counter antimalarial medicine, acetylsalicylic acid, and black tea-based herbal mixture prior to presentation.

Physical examination revealed a middle aged man, febrile with a temperature of 39.7^0C, pulse rate of 98 beats per minute, blood pressure of 138/90 mm Hg and normal heart sounds. The respiratory rate was 26 per minute and both lungs clear to auscultation with patient breathing ambient air. Abdominal and nervous system examination were normal.

Corresponding author: **Dr A Akpowaye**, Department of Internal Medicine, Federal Medical Centre Asaba, Delta State, Nigeria. *Email:* adrakpowaye@gmail.com, *Telephone*: +23407035120302

The working diagnosis was sepsis, focus being atypical pneumonia (COVID-19); other consideration was malaria with treatment failure. He received oral chloroquine, amoxicillin-clavulanic acid, azithromycin, paracetamol, zinc sulphate, vitamin C, vitamin B complex, vasoprin, and oxygen therapy via a rebreathing mask and rehydration for the first 48hours with normal saline.

Hospital Course (COVID-19 Treatment Ward)

He was on admission for 15 days and fever subsided after the 3rd day but his blood pressure increased to a maximum of 170/110 mmHg for which he received amlodipine and lisinopril. His respiratory rate increased to 42 cycles/min but oxygen saturation was 98% on 5 – 6 L of oxygen via face mask.

He developed bilateral leg swelling on the seventh day on admission, and vomiting had been intractable for six days despite having parenteral metoclopramide and ondansetron. Requested serum electrolytes urea and creatinine revealed hyponatremia (124 mmol/l), hyperkalemia (6.9mmol/l), and acidosis (bicarbonate 9mmol/l) with azotaemia (Table 1). Hyperkalemia was corrected with 50% dextrose infusion and insulin after cardiac stabilization with 10% calcium gluconate and he had the first session of haemodialysis. Urine output declined over the next three days despite fluid resuscitation and frusemide. He had further haemodialysis on the 10th and 14th day due to persistent azotaemia, electrolyte and acid base inbalance. He subsequently recovered and was discharged to the outpatient clinic.

Follow Up (Outpatient Clinic)

At his first clinic visit (three days post-discharge), he was clinically stable and excited to be well. Serum creatinine was creatinine 1.7mg/dl and other electrolytes were normal. Renal ultrasound showed the right kidney was shrunken with size 6.7 x 3.0cm while the left kidney was 14.7 x 7.7cm. Both kidneys had increased cortical echogenicity with loss of corticomedullary differentiation.

During his next clinic visit a week later, he complained of bilateral leg swelling, which was more on the left leg with associated calf pain, while still on aspirin. A doppler ultrasound of the left lower limb showed dilatation and loss of compressibility of the common femoral, entire femoral, popliteal veins, and the paired deep calf veins; the femoral and popliteal veins showed diffuse echogenic intraluminal thrombi. Prothrombin time was 48 seconds and INR was 1.9 and he recieved oral dabigatran and low dose loop diuretics. Bilateral leg swelling and pain subsided

Table 1: Laboratory results on the 7th day on admission.

Laboratory test	Patient test
Sodium, mmol/l	124
Potassium, mmol/l	6.9
Chloride, mmol/l	99
CO_2, mmol/l	9
Creatinine, mg/dl	9.93
Urea, mg/dl	279.6
Hemoglobin, g/dl	10.7
Wbc count x 10^9 /µl	5.7/µl
Platelet count/mm	249000
Calcium, mg/dl	5.1
Phosphate, mg/dl	6.8
AST U/L	19
ALT U/L	18
ALP U/L	62
Total bilirubin, mg/dl	0.7
Direct bilirubin, mg/dl	0.2
GGT U/L	40
Total protein, g/dl	6.9
Albumin, g/dl	3.0

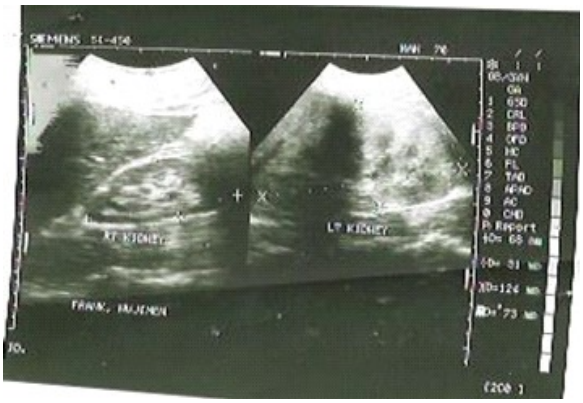

Fig. 1: Renal scan **Fig. 2a:** Doppler scan

over two weeks, and serum electrolytes returned to near normal values. (He had no baseline records of kidney function tests).

DISCUSSION

The pathophysiology of COVID-19 AKI is multifactorial with several sub-phenotypes [2]. This case report shows a black middle aged man treated for AKI following sepsis from COVID-19 infection, with associated protracted vomiting and diarrhea, exposure to over the counter herbal medication, use of diuretics and ACEI and background renal impairment with a shrunken kidney.

Suspected intrinsic AKI was reported in 82% of patients with AKI in a single-centre retrospective study [3]. The SARS-COV virus may have direct cytopathic effects on Renal epithelial cells and this is supported by the detection of PCR fragment in the blood and urine of patients; Renal epithelial cells reportedly express the ACE2 receptor by as much as 100 fold [4', however, pathological studies did not support the deposition of immune complex in the kidneys and subsequent stimulation of specific immunological effect or mechanisms such as T lymphocyte or antibody that may damage the kidney [5].

"Cytokine storm" with a massive elaboration of virus-induced cytokines or mediators may have an indirect effect on renal tissue such as hypoxia, shock and rhabdomyolysis. A collapsing glomerulopathy referred to as COVID associated nephropathy COVAN similar to that found in HIV patients occurs in COVID patient [6,7]. This glomerulopathy has been linked to the chemokine surge in interferon, furthermore, it presents with AKI and nephrotic range proteinuria, associated with capillary turf collapse, podocytes and parietal epithelia cells hypertrophy or hyperplasia, microcystic tubular dilation, and tubular injury [6,7]. Proposed risk factors include black race and homozygosity for the APOL1 gene [6,7].

This patient had background renal impairment with a shrunken kidney. A probable cause of the unilateral shrunken kidney is renal hypoplasia/ dysplasia (histology was not done); usually associated with compensatory enlargement and detection could be inutero or accidentally during a routine sonography [8]. The absence of compensatory enlargement, increase in dietary protein and salt intake increases the risk of renal injury. The presence of protracted vomiting with volume contraction as well as well as use of diuretic and ACEI are other risk factors that may be responsible for the development of AKI. Pre-existing CKD is a strong independent determinant of COVID-19 AKI with as much as three-fold increase in risk, black race, male sex, hypertension, use of ACEI and disuretics are other risk factors identified for the development of COVID-19 AKI [9].

A study in US reported the incidence of renal replacement therapy amongst AKI patient with COVID-19 as 14.3%; [10] it was more common among patients with severe pneumonia and was associatesd with higher mortality [11].

Limitations

A significant limitation in the management of this case was the absence of adequate laboratory support at the COVID-19 treatment centre which delayed investigations such as serum electrolytes, blood gases, ultrasound scan, urine microscopy, urine electrolytes, and chest X-ray. Histologic diagnosis was not possible since the patient had a unilaterally shrunken kidney and was taking anticoagulants. Excretory function of kidneys was not assessed to avoid exposure to contrast.

CONCLUSION

We present a case of an African male with COVID-19, a history of DVT and a unilateral shrunken kidney; who developed a sudden decline in renal function and required several sessions of intermittent haemodialysis but recovered. This case highlights that COVID-19 associated AKI may be multifactorial; and although patients present acutely, it is always essential to search for underlying risk factors or evidence of kidney disease and avoid nephrotoxins. There may be a racial variation in the presentation and course of AKI in patients with COVID-19. Finally, basic laboratory and radiological support are crucial in Infectious disease isolation/treatment centres to optimize patient outcomes.

REFERENCES

1. Zou X, Chen K, Zou J, *et al*. Single-cell RNA-seq Data Analysis on the Receptor ACE2 Expression Reveals the Potential Risk of Different Human Organs Vulnerable to

2019-nCoV Infection. Front Med. 2020;14(2):185-192.
2. Legrand M, Bell S, Forni L, *et al*. Pathophysiology of COVID-19 Associated Acute Kidney Injury. Nature Reviews Nephrology. 2021;17: 751-764
3. Pei G, Zhang Z, Peng J, *et al*. Renal Involvement and Early Prognosis in Patients with COVID-19 Pneumonia. J Am Soc Nephrol. 2020;31(6):1157-1165.
4. Batlle D, Soler MJ, Sparks MA, *et al.* Acute Kidney Injury in COVID-19: Emerging Evidence of a Distinct Pathophysiology. J Am Soc Nephrol. 2020:1-4.
5. Su H, Yang M, Wan C, *et al*. Renal Histopathological Analysis of 26 Postmortem Findings of Patients with COVID-19 in China. Kidney Int. 2020;98(1):219-227.
6. Wu H, Larsen CP, Hernandez-Arroyo CF, et al. AKI and Collapsing Glomerulopathy Associated with COVID-19 and APOL1 High-risk Genotype. J Am Soc Nephrol. 2020;31(8):1688-1695.
7. Velez JCQ, Caza T, Larsen CP. COVAN is the New HIVAN: The Re-emergence of Collapsing Glomerulopathy with COVID-19. Nat Rev Nephrol. 2020;16(10):565-567.
8. Jewell PD, Bramham K, Galloway J, *et al.* COVID-19 Related Acute Kidney Injury; Incidence, Risk Factors and Outcomes in a Large UK Cohort. BMC Nephrology. 2021(22);359.
9. Hiraoka M, Tskahara H, Ohshima Y, Kasuga K, Ishihera Y, Mayumi M. Renal Aplasia is the Predominant Cause of Congenital Solitary Kidneys. Kidney int. 2002;(May)61(5):1840-1844
10. Hirsch JS, Ng JH, Ross DW, *et al.* Acute kidney injury in patients hospitalized with COVID-19. Kidney Int. 2020;(May):209-218.
11. Pei G, Zhang Z, Peng J, *et al.* Renal Involvement and Early Prognosis in Patients with COVID-19 Pneumonia. J Am Soc Nephrol. 2020;31(6):1157-1165.