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It's complicated: A case report on a COVID-19-positive HIV patient presenting with rhabdomyolysis and acute kidney injury

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Abstract

The SARS-Cov-2/COVID-19 pandemic in early 2020 has had a devastating impact on health systems around the world. While viral pneumonia remains the most common complication, reports are surfacing of cases with neurological, cardiac, and renal involvement. Even less is known about the implications in special high-risk populations. In this report, we discuss a unique case of an HIV-positive patient in New York City who presented with a 2-week history of worsening fatigue, cough, dyspnea, and myalgias and was found to have COVID-19 pneumonia and acute kidney injury. He was managed for severe uremic metabolic acidosis and electrolyte abnormalities with emergent hemodialysis and supportive therapy with subsequent improvement. Direct involvement of SARS-CoV-2 and pneumonia-induced rhabdomyolysis were identified as the precipitating factors of his acute kidney injury. The pathophysiologic mechanisms of acute kidney injury, SARS-CoV-2 renal tropism, and the impact of highly active antiretroviral therapy on COVID-19 pneumonia are discussed. We highlight the importance of clinician awareness of this potentially fatal complication of COVID-19 pneumonia, particularly in the HIV-positive population as early recognition and management can have favorable outcomes.

Keywords

Acute kidney injury, rhabdomyolysis, HIV, SARS-CoV-2, COVID-19

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Introduction

In December 2019, SARS-CoV-2 a coronavirus caused an outbreak of viral pneumonia in Wuhan, China. The pandemic illness termed COVID-19 has now infected over 2 million people and has caused over 140,000 deaths worldwide.[1] Recent studies have demonstrated that approximately 20%–40% of COVID-19 patients admitted to the intensive care unit (ICU) develop acute kidney injury (AKI).[2,3] COVID-19-induced AKI presents with a more severe clinical course, particularly in the ICU setting, and is a negative prognostic factor with respect to survival. The mechanisms of kidney injury in COVID-19 infection have been hypothesized to be due to: (1) hypoperfusion-related injury of the renal tubule, (2) viral tropism for the kidney due to highly expressed angiotensin-converting enzyme (ACE)-2 receptors on podocytes and epithelial cells that are essential for viral uptake, and (3) cytokine-induced nephropathy.[4] Reports are limited regarding patients on antiviral therapy who present with COVID-19 and subsequently develop kidney injury. Furthermore, a common complication of infection in HIV-positive patients is rhabdomyolysis which tends to precede AKI. HIV, overlapping infections, and antiviral medications used to treat HIV are known to be nephrotoxic. Considering the potential of nephrotoxicity of SARS-CoV2, we must attempt to distinguish the causes

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and manifestations of kidney injury as well as the possibility of combined effects. Early recognition of COVID-19 and AKI in vulnerable populations is crucial to intervening early and lowering mortality.

Case report

A man in his 40s with a history of HIV on highly active antiretroviral therapy (HAART) and hypertension with a body mass index (BMI) of 34.9 presented to the Emergency Department (ED) after being seen at an urgent care clinic with hypotension and dyspnea. He visited his Primary Care Provider 2 weeks prior for fever, chills, and cough productive of grayish sputum; however, his symptoms persisted despite completing a course of azithromycin. During the interview, he denied recent travel or COVID-19 exposure. He endorsed fatigue, leg cramping, and poor appetite. CD4+ count and viral load were unknown to the patient. His vitals were unstable in ED with tachypnea (58 breaths/min) and hypotension (70/50 mmHg), heart rate of 91 beats/min, temperature of 37.1°C, and an oxygen saturation of 92% while on a non-rebreather mask at a flow rate of 15 L. On examination, he was in respiratory distress with decreased breath sounds in bilateral lung fields. The patients' baseline creatinine was unknown at the time of presentation. The remainder of the examination was noncontributory; patient had good skin turgor and appropriate capillary refill. Vital signs for the duration of the visit are provided in Table 1.

Fluid resuscitation with IV normal saline was initiated. On re-examination, patient was hemodynamically stable with persistent tachypnea (28 breaths/min). He had multiple electrolyte abnormalities which are presented in Table 2. Inflammatory markers, troponin, and coagulopathy studies were not performed.

Urinalysis was significant for a white blood cell (WBC) > 100/high-power field (HPF), red blood cell (RBC) 3/HPF, and pH 5.0. Bedside electrocardiogram (EKG) showed tachycardia (105 beats/min) and a prolonged QTc (518 ms) (Figure 1). A rapid nasopharyngeal swab collected was positive for SARS-CoV-2, prompting contact and droplet precautions. Patient was tested for Hepatitis B and C which were both negative; however, the patient was not tested for concomitant influenza. Chest X-ray showed patchy infiltrates laterally in the right mid-lung and the right base with lesser peripheral patchy infiltrates laterally from left mid to lower lung.

In the ED, IV magnesium and IV terbutaline were administered. Patient was admitted to the ICU, for management of acute renal failure with electrolyte imbalance, rhabdomyolysis, anion gap metabolic acidosis secondary to uremia, and COVID-19 pneumonia. Patient denied prior history of renal disease. In the ICU, 3 amps of IV sodium bicarbonate was administered followed by IV bicarbonate drip. A straight catheter drained 25 mL of tea-colored urine prompting the insertion of a Foley catheter. All nephrotoxic agents including HAART were held. Prolongation of the QTc on EKG contraindicated the use of hydroxychloroquine and azithromycin.

On hospital day 1, despite continued management, he showed no significant improvement with an elevated uric acid of 13.8 mg/dL. His renal function remained severely compromised with persistent severe metabolic acidosis; creatine kinase (CK) remained elevated at 2185 U/L with continued electrolyte abnormalities (hypokalemia, hypocalcemia, hyperphosphatemia, hypermagnesemia). Chest X-ray showed persistent patchy peripheral infiltrates in the right lung with lesser patchy peripheral infiltrates just above the left lung base (Figure 2). Given his intractable acidosis, a hemodialysis catheter was placed for emergent dialysis. Lopinavir and ritonavir were held due to nephrotoxicity. Viral HIV load was low at 47 HIV-1 RNA copies/mL. Patient was closely monitored in supplementary ICU area until discharged on hospital day 7.

During the hospital stay, following initial fluid resuscitation with Lactated Ringers at a rate of 100 mL/h for 10 h, normal saline was administered in three 1000 mL boluses initially and then at a rate of 150 mL/h for 6 h. For the remainder of the hospital stay, 6000 mL of Lactated Ringer's was administered at a rate of 150 mL/h for 40 h and 500 mL of normal saline on the final day of admission at a rate of 100 mL/h. Patient was dialyzed via hemodialysis and electrolyte imbalances were corrected resulting in normalization of the patient's vital signs except for persistent tachycardia (113 beats/min). Patients' blood pressure was 123/70 post-dialysis. Electrolyte imbalances, glomerular filtration rate (GFR), uric acid level, and CK level improved; hypokalemia and patient's anion gap metabolic acidosis resolved. Improvement in renal function prompted discontinuation of hemodialysis. Patient's Foley catheter was removed for a voiding trial, and he was in no acute distress on 4-L nasal cannula saturating at 96%. A renal biopsy was unable to be obtained from the patient.

Discussion

COVID-19 has challenged health care systems globally with a myriad of presentations. With an estimated 14-day incubation period, most individuals show symptoms 4–5 days post-exposure. Symptomatic COVID-19 patients generally present with fever, cough, malaise, and shortness of breath, usually managed at home with supportive care. Other commonly reported symptoms are headache, sore throat, rhinorrhea, and diarrhea.[5] Well-recognized risk factors for severe presentations include age, comorbidities such as lung disease, coronary artery disease, hypertension, diabetes, chronic kidney disease, and cancer.[6]

In hospitalized patients, lymphocytopenia, elevation of liver enzymes, and CK are associated with ICU admission and higher mortality.[7] This patient had an elevated white blood cell count and his CK was significantly elevated at

Table 1. Vital signs.

	Day 1 16:19	Day 1 17:46	Day 1 21:40	Day 1 04:26	Day 1 08:01	Day 2 21:52	Day 2 6:00	Day 2 8:00	Day 3 00:46	Day 4 21:44	Day 5 20:44	Day 5 7:58	Day 6 16:53	Day 7 21:55	Day 7 15:46
HR	91	88	101	119	115	120	122	118	123	121	120	113	117	117	93
RR	58	28	38	28	19	22	28	28	18	19	20	20	22	24	16
BP	70/50	100/72	84/50	89/67	136/71	114/74	109/66	104/61	133/90	121/75	115/68	108/88	140/90	113/72	125/63
O_2 Sat		100	94	94	95	93	93	93	96	98	98	96	96	95	95
T (°C)		37.1	36.4	38.6	37.3	37.2	38.3	37.9	36.5	35.9	35.9	36.6	36.4	36.7	35.4
BUN	151	157	119	126	112	111	109	98	92	75	69	62	48	33	30
Cr	17.4	17.5	9.4	7.8	6.5	4.3	3.8	3.3	3.2	2.8	2.6	2.4	2	1.6	1.6
Cr K		2185	3127		1785	1126			998			1679			

HR: heart rate; RR: respiratory rate; BP: blood pressure; O_2 Sat: oxygen saturation; T: temperature; BUN: blood urea nitrogen; Cr: creatinine; Cr K: creatine kinase.

2185 U/L on presentation. His chest X-ray revealed patchy infiltrates with peripheral, lower zone distribution and bilateral involvement, which is commonly seen in COVID-19 pneumonia.[8]

Table 2. Initial lab findings.

Labs	
WBC	15.6 µL
Neutrophils	88.5%
Lymphocytes	4.9%
Serum osmolality	317 mOsm/L
Na	126 mmol/L
K	3.2 mmol/L
Cl	88 mmol/L
Ca	7.7 mmol/L
Albumin	3.5 g/dL
HCO_3^-	9 mmol/L
Blood urea nitrogen	151 mg/dL
Creatinine	17.4 mg/dL
Anion gap	29
Lactic acid	1.0
AST	18
ALT	50
Uric acid	13.8 mg/dL
Glucose	155 mg/dL
pH	7.12
pCO_2	25 mmHg
pO_2	28 mmHg
Urine specific gravity	1.024
Creatine kinase	2185 U/L

WBC: white blood cell; AST: aspartate transaminase; ALT: alanine transaminase.

The patient presented with significant elevations in CK; his imaging revealed severe COVID-19 pneumonia, he was HIV positive, and he had a history of hypertension and was obese. Despite many risk factors and findings that would lend to poor prognosis, the patient was hospitalized for 7 days and required only a single round of hemodialysis. This lends the question of why his clinical course was brief compared with the average of 10 days for his age group and risk group.[9] Recently, hemodialysis has been proposed as a prospective treatment for SARS-CoV-2 due to the affinity of the virus for renal parenchyma. Our patient did have a remarkable outcome following a sole round of hemodialysis via Shiley catheter. Future large-scale studies will be needed to provide insight to the efficacy of hemodialysis as a treatment for COVID-19.

In a recent histopathologic examination of COVID-19 patients,[9] SARS-CoV-2-associated pulmonary lesions manifest as inflammation of the parenchyma and interstitium, with hyperplasia of the alveolar epithelium and formation of hyaline membranes. Damage was evidenced in areas outside of the lungs with lesions apparent in the cells of the immune, cardiac, hepatic, and renal systems.[8] This tissue tropism is due to high expression of ACE-2 receptors on the alveoli. SARS-CoV-2 potentially gains entry into cells through binding of a spike protein on its envelope to ACE-2 receptors; uptake is via a clathrin-dependent endocytosis mechanism.[10] Reports show that there are numerous ACE-2 receptors in podocytes and glomerular mesangial cells of the kidney.[11] Given these findings, renal involvement of COVID-19 is not an unexpected phenomenon. In a retrospective study of patients from Wuhan

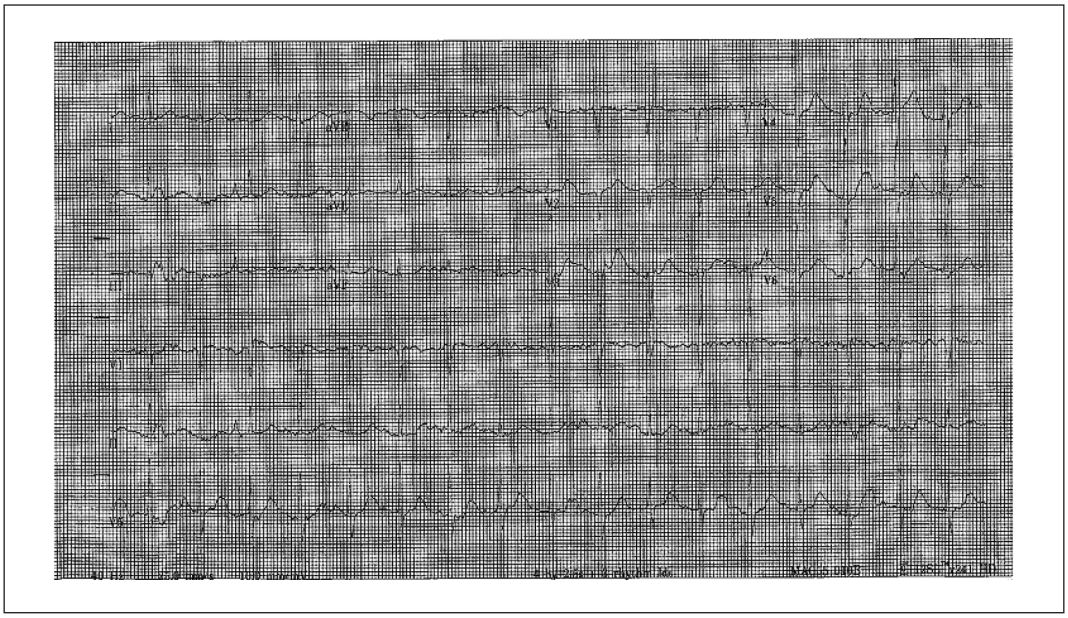

Figure 1. Initial EKG.

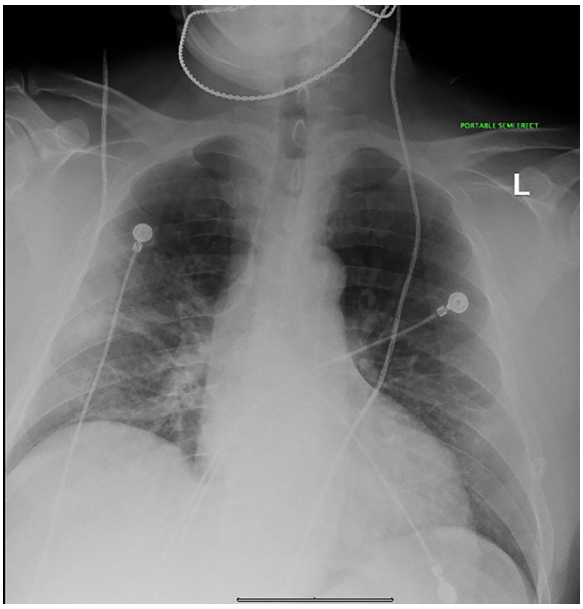

Figure 2. Initial chest X-ray. Posterior anterior chest X-ray demonstrating patchy peripheral infiltrates in the right lung with lesser patchy peripheral infiltrates just above the left lung base.

from 17 January 2020 to 3 March 2020 with lab-confirmed COVID-19, 27% of patients exhibited acute renal failure.[12] Post-mortem analysis showed severe acute tubular necrosis (ATN) and lymphocyte infiltration in the kidneys with SARS-CoV-2 NP antigen identified via immunohistochemistry and accumulated viral particles on electron microscopy.[11] Reports have also indicated severe hyponatremia and hypokalemia in patients infected with SARS-CoV-2, possibly due to an inflammatory response pathologically causing electrolyte impairment secondary to non-osmotic release of vasopressin.[13] A transtubular potassium gradient for this patient was scored as 8 indicating hypokalemia as a result of renal potassium wasting supporting the idea that SARS-CoV-2 renal damage was responsible for this patient's electrolyte abnormalities. These findings lend support to the hypothesis that SARS-CoV-2 may directly induce renal damage.

AKI is an abrupt decrease in the GFR resulting in electrolyte disturbances, retained metabolic waste, and is a well-recognized complication of HIV.[14] The symptoms of AKI are generally nonspecific apart from oliguria, making blood urea nitrogen (BUN) and creatinine essential for diagnosis.[14] Diagnostic criteria for AKI are defined as either a 50% increase in serum creatinine from the patient's baseline or a reduction in creatinine clearance by 50%.[14] A strong correlation exists between the onset of AKI symptoms and the patient requiring intubation with mechanical ventilation.[15] The rate of AKI in patients receiving mechanical ventilation was noted to be as high as 89.7% in some studies compared to that of patients not receiving mechanical ventilation which was 21.7%.[15] Despite previous studies from Italy and China which determined the rate of AKI to be from 0.5% to 29%, with many studies demonstrating percentages on the lower end of the spectrum, the United States had an astronomical number of AKI cases.[15] Since China and Italy had experienced surges in cases of Sars-CoV-2 prior to the United States, it is perplexing as to why the COVID-19 patients in the United States were more likely to suffer from renal injury.[15] Various risk factors have been studied such as hypertension, diabetes mellitus type II, age as well as obesity; however, mechanical ventilation and the use of vasopressors demonstrate the strongest correlations in terms of risk of developing AKI.[15]

Our patient was maintained on nasal cannula for the duration of his hospitalization and his hypotension was treated with fluid resuscitation instead of vasopressors. Although our patients' baseline creatinine was unknown, his elevations in CK, electrolyte derangements, BUN, and acute clinical presentation were suggestive of AKI. AKI in HIV patients has a wide range of causes including prerenal azotemia, rhabdomyolysis, microangiopathy, glomerulonephritis, Hepatitis B or C, ATN, acute interstitial nephritis, crystal-induced nephropathy, nephrotoxicity from HAART, and HIV-associated nephropathy.[13,14,16] ATN is an important cause to investigate in this patient particularly due to its association with AKI, COVID-19, and HIV. In our patient, his urinalysis only contained 3 RBC/hpf and no casts which made ATN, glomerulonephritis, microangiopathy, and crystal-induced nephropathy less likely. Thrombotic microangiopathy (TMA) has been noted in patients with COVID-19-induced AKI.[17] Underlying complement disorders and gemcitabine use have been demonstrated as the potential causes of TMA in such patients.[17] New data have shown that Sars-CoV-2 can activate the alternate and lectin pathways of the complement system resulting in an inflammatory response.[17] The inflammation leads to the development of coagulopathy and subsequently TMA in some cases.[17] It is a point of contention as to whether infection with COVID-19 is a secondary insult to patients with underlying predispositions to the development of TMA such as complement disorders or prior use of medications that have been associated with the development of TMA.[17] While AKI in this patient can be attributed to his initial hypotension and hemodynamic instability resulting in decreased renal artery perfusion, it is crucial to consider that SARS-CoV-2 infection of the kidneys with subsequent replication in the renal tissue may have played a substantial role in the patients' abrupt decline in renal function.[3]

Rhabdomyolysis is a common cause of AKI among HIV-positive patients with an incidence reported to be of 943 per 100,000 person-years.[18] Infection is most commonly identified as the precipitating etiology, particularly pneumonia which constitutes 38% of infectious causes.[16] In the HIV population, rhabdomyolysis is associated with increased mortality compared to the rest of the population.[18] Rhabdomyolysis can be evoked by various alternative

mechanisms including trauma, crush injuries, use of statins, cocaine, amphetamines, heroin, Phencyclidine, musculoskeletal diseases, hyperthermia, hypothermia, burns, muscular ischemia, hypophosphatemia, seizures, prolonged operations, and severe dehydration.[19] The patient stated he had not experienced any trauma, seizures, temperature extremes, prolonged operations, burns, or crush injuries. The patient was screened for illicit drug use upon presentation which was negative and phosphate levels were elevated on presentation. Severe dehydration was the only potential cause apart from infection which was significant in this patient as a cause for rhabdomyolysis. Studies have demonstrated that COVID-19 has been known to cause hypotension in greater than 50% of cases.[20] Whether directly through involvement of SARS-CoV-2 interaction with ACE-2 receptor or indirectly from causing hypotension from an undetermined mechanism, this patient's decline in renal function can be attributed to his acute infection with COVID-19.

In a patient with HIV on HAART, it is important to consider the potential impact of therapy on both renal function and the patients' ability to counter infections. Although HAART increases CD4+ counts and subsequently decreases the risk of certain infections, it is associated with an increased incidence of AKI from 2.9% to 6.0% in HIV patients.[16] Despite the concern of AKI from HAART, 52% of all renal injury in HIV patients are primarily caused by infection.[21] Delaying administration of HAART in HIV patients with AKI prevents further renal injury from the medications' nephrotoxic nature.[22]

Prior to discontinuation upon hospitalization, our patient's HAART regimen consisted of lopinavir and ritonavir. The patient endorsed compliance with his HAART regimen prior to hospitalization. Lopinavir acts via inhibition of viral 3 chromotrypsin-like (3CL) proteases which has been hypothesized as the mechanism for efficacy against SARS-CoV-2 since 3CL proteases play a fundamental role in the processing of viral RNA which interferes with the viral replication process.[23–25] Although the lopinavir/ritonavir combination drug has been investigated as a potential treatment regimen for COVID-19, there has been conflicting evidence in terms of efficacy.[21–26] Some studies have reported a reduction of symptoms and SARS-CoV-2 viral load when initiated early in the treatment course.[2] Despite the recommendation by early studies that lopinavir/ritonavir should be used in vulnerable populations with COVID-19 pneumonia, more evidence is needed to enforce this practice.[24–26] Our patient's exceptional clinical course despite having HIV lends to the idea that his HAART regimen as well as his azithromycin use prior to presentation may have decreased the total amount of SARS-CoV-2 viral replication in both the renal parenchyma and pulmonary tissue resulting in a rapid recovery and subsequent hospital discharge.

The potential of COVID-19 to antagonize renal function is crucial to note given most cases of both rhabdomyolysis and AKI in HIV patients are precipitated by infections. In the case presented, our patient had significantly elevated CK of 2185 U/L, with drastic elevations in creatinine and significant uremia. It is likely that the AKI in our patient was multifactorial involving direct tissue damage from SARS-CoV-2, COVID-19-induced hypotension, and indirect injury through another mechanism compounded by rhabdomyolysis.

Conclusion

In this report, we present a case of AKI in an HIV-positive patient with COVID-19 with a successful outcome. Clinicians should be cautious of this complication in HIV patients; those presenting with symptoms consistent with SARS-CoV-2 infection may be at an increased risk of AKI given they are immunocompromised. In our case, the damage to the kidney was likely a combination of pneumonia-induced rhabdomyolysis and direct effects of SARS-CoV-2 on renal parenchyma. The use of HAART therapy and azithromycin prior to admission may have been a contributing factor in the remarkably quick recovery of our patient.

Declaration of conflicting interests

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Ethical approval

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