Case Report

A case of coronavirus disease 2019 in acquired immunodeficiency syndrome patient: a case report and review of the literature

Abdallah Qasim^{1,*}, Mohamed Mansour², Omar Kousa¹, Dana Awad¹, Bader Abuhazeem¹, Paul Millner¹, Manasa Velagapudi³

¹Department of Internal Medicine, Creighton University, NE, United States;

²Department of Internal Medicine, Sheikh Shakhbout Medical City, Abu Dhabi, United Arab Emirates;

³ Division of Infectious Diseases, CHI Health Creighton University Medical Center. NE, United States.

SUMMARY Coronavirus disease 2019 (COVID-19) is a respiratory illness caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus that was identified in December 2019. The impact of COVID-19 virus on Acquired Immunodeficiency syndrome (AIDS) patients has been reported with variable outcome. We reported a patient that was immunosuppressed by AIDS disease and chemotherapy for cancer who contracted SARS-CoV-2 infection and had a mild disease. We did literature review for the cases published that had human immunodeficiency virus (HIV) infection and COVID-19 disease and analyzed the characteristics and outcomes of the reported cases. Our review yielded three case reports and four case series for patients with HIV infection and COVID-19 disease usually had other comorbidities. The findings from the case reports and case series indicate that the risk of death or severe disease from COVID-19 in HIV positive patients was lower than observed in the general population, which may indicate a possible protective effect of uncontrolled HIV in preventing the complications associated with the massive inflammatory response.

Keywords COVID-19, coronavirus, HIV, AIDS, chemotherapy, SARS-CoV-2, pneumonia

1. Introduction

Beginning in late December 2019, numerous cases were emerging from Wuhan, China, of a new type of severe pneumonia of unknown etiology. The etiologic pathogen has since been identified as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). This virus has since spread rapidly to many countries throughout the world (1). This is the seventh coronavirus identified so far and differs from the other coronaviruses that cause the common cold and mild pneumonia (229E, OC43, NL63, and HKU1) (2). In the United States, 1.2 million people are living with human immunodeficiency virus (HIV). Of note, in March 2020, the Centers for Disease Control and Prevention (CDC) identified people living with HIV (PLWH), cancer patients and those receiving chemotherapy as high risk for severe illness from the new coronavirus disease known as coronavirus disease 2019 (COVID-19) compared to the general population (3).

Here, we report a case of COVID-19 infection in an

immunocompromised HIV patient on chemotherapy that resulted in a mild disease with full recovery.

2. Case Report

A 37-year-old man with a history of AIDS and Kaposi's sarcoma presented to the infusion clinic to receive his second dose of doxorubicin. On arrival, he complained of high-grade fever for two days associated with sore throat, mild cough, occasional headaches, chills, and night sweats. A review of systems was negative for shortness of breath, chest pain, diarrhea, skin changes, or loss of smell or taste. He denied any sick contacts.

He was diagnosed with AIDS two years ago, was nonadherent with antiretroviral therapy (ART). He had recent hospitalization for severe *pneumocystis* pneumonia from which he recovered. He was diagnosed with Kaposi sarcoma two months ago and was started on doxorubicin. Since the diagnosis of Kaposi's sarcoma, he was adherent with his ART. He also history of treated chronic hepatitis C, syphilis, anxiety, and depression.

His medications include bictegravir-emtrictabinetenofovir alafenamide, atovaquone, prochlorperazine, ondansetron, and tramadol as needed. He is allergic to trimethoprim/sulfamethoxazole and intolerant to Dapsone. He is a never smoker, denies alcohol use, had remote history of methamphetamine and marijuana use, but has been sober for 3 years. Family history was unremarkable. He used to work as a bus driver in the past, currently unemployed. He denied any recent travel outside Nebraska state or recent exposure to COVID-19 or sick patients.

On examination, his temperature was 38.2°C, heart rate of 118 beats per minute, blood pressure was 136/72 mmHg, respiratory rate of 20 breath per minute, and his oxygen saturation was 99% on room air. The patient had normal respiratory effort, lungs were clear to auscultation. There was a healed incision in right groin, pruritic rash in bilateral inguinal areas. He had shallow perianal ulcers with minimal bleeding. The rest of his examination was normal.

Laboratory testing at the time of presentation was notable for leukocytosis, and mildly elevated

procalcitonin (Table 1 and Table 2). A recent HIV viral load of 517 copies/milliliter with a cluster of differentiation 4 (CD4) cell count of 67 and both respiratory pathogen screen and group-A streptococcus screening have been negative, and the rest of laboratory workup are listed in Table 1. The chest radiograph at the time for admission showed no signs of cardio/pulmonary disease (Figure 1), and computed tomography for abdomen and pelvis showed no focus of



Figure 1. Chest X-ray on admission. Postero-anterior chest x-ray showing normal lung fields with no reported abnormality.

Component	Reference range in adults	On admission	On discharge	
White blood cells (k/ul)	4.0-12.0	15.1	10.8	
Red blood cells (m/ul)	4.30-5.90	4.14	3.68	
Hemoglobin (gm/dl)	13.5-17.5	12.9	11.7	
Platelets (k/ul)	140 000-440 000	88	125	
Absolute neutrophil count (k/ul)	1.5-8.0	10.3		
Absolute lymphocytes count (k/ul)	1.0-4.5	1.1		
Creatinine (mg/dl)	0.60-1.30	0.84	0.62	
Sodium (mmol/L)	135-145	135	140	
Potassium (mmol/L)	3.7-5.1	3.5	3.9	
Albumin (gm/dl)	3.5-5.0	3.0	2.9	
Aspartate aminotransferase (u/l)	10-40	27	24	
HIV viral load (Copy/mL)	Negative	517		
T-cell count differential	-			
CD4/T4 cells (%)	40.0-60.0%	4.2		
CD4 T cell abs. (cells/ul)	436-2,168	67		
CD8/T8 cells (%)	15.0-43.0	47.3		
CD8 T cell abs. (cells/ul)	164-1,456	757		
CD19 cells (%)	5.0-22.0	19.0		
CD19 abs. (cells/ul)	56-745	290		
CD56 cells (%)	3.0-21.0	14.1		
Absolute CD56 (cells/ul)	33-711	215		
CD4/CD8 ratio	0.9-3.4	0.1		
Infectious tests				
Blood culture	Negative	No growth at 5 days		
Respiratory pathogen panel	Negative	No respiratory pathogens detected by multiplex PCR		

Table 1. Laboratory investigation on admission and on discharge

Table 2. Inflammatory markers related to COVID-19

Component	Reference range in adults	Hospital day 1	Hospital day 2	Hospital day 4
Ferritin (ng/mL)	22-388	452	430	416
C-reactive protein (mg/L)	≤ 9.00	41.90	35.80	25.50
D-dimer quantitative (mg/L)	< 0.25	0.79		0.90
Fibrinogen (mg/dl)	200-400	437		
Procalcitonin (ng/mL)	≤ 0.05	0.55		
COVID-19 qualitative	Not detected	detected		

infection. Blood cultures were drawn, and COVID-19 testing was sent (Table 1 and Table 2).

Given his recent methicillin-resistant Staphylococcus aureus (MRSA) abscess infection, a current cutaneous candidiasis infection, and overall septic picture, he was started on empiric therapy with vancomycin, cefepime, metronidazole, fluconazole, and topical Miconazole. On day 3 of admission, COVID-19 testing was positive, rest of the work up was negative so the antibiotics were stopped. During his entire hospital stay, he did not require any supplemental oxygen therapy, and all his symptoms of fever, sore throat, headache, and mild cough completely resolved. Anticoagulation was deferred due to his thrombocytopenia and recent episode of rectal bleeding. He was discharged home on day 4 of hospitalization in stable condition with instructions for self-quarantine for 14-days. The patient remained asymptomatic and healthy at post discharge follow up visit.

3. Discussion

Multiple risk factors have been linked to worse outcomes in COVID-19 infection including, age (> 60 years), hypertension, diabetes, cardiovascular disease, lung disease, and chronic kidney disease (4). Immunosuppressed patients are at a higher risk of being infected with COVID-19. Multiple studies and case reports showed the role of massive immune response and excessive release of inflammatory cytokines which the CD4 T-cells play a significant role - in the damage that occurs in the lung tissues (5-7). However, the question of whether being immunosuppressed is a risk factor for more severe disease or not is still under investigation.

We conducted a systematic review of the literature for studies published to date in PubMed, Scopus, Web of Science, and Cochrane Central databases. The following search terms were used: "acute respiratory syndrome coronavirus 2 (SARS-CoV-2)", "COVID-19" and "Human Immunodeficiency Virus". Our search was limited to individuals 18 years and older. Our search revealed a total of 3 case reports and 4 case series.

Zhu *et al.* (2020) was among the first to report a case of SARS-CoV-2 and HIV co-infection in a patient from Wuhan. The patient was diagnosed with COVID-19 pneumonia and was found to be HIV positive during hospitalization (8). Despite his hospital course complicated by severe pneumonia requiring treatment with steroids, he recovered completely from the illness. Our findings were similar to Louisa *et al.* (2020), who reported a SARS-CoV-2 infection from a patient with previously diagnosed HIV infection, on antiretroviral therapy. The patient developed a mild illness and recovered completely without any specific therapy for COVID-19 (9). Also, Wu *et al.* (2020) reported a patient with HIV on antiretroviral therapy (tenofovir disoproxil fumarate, lamivudine, and efavirenz), stage-4 diffuse large B-cell lymphoma and previously treated pulmonary tuberculosis, who was diagnosed with COVID-19 after presenting to the hospital with fever and symptoms of viral respiratory tract infection that progressed to pneumonia then he recovered (10).

Furthermore, four prior case series were found, the first case series by Blanco et al. (2020), describes five HIV positive patients who were generally less than 50 years old. Two patients were virologically suppressed with protease inhibitor (darunavir-boosted cobicistat) based antiretroviral therapy, while the other two were suppressed with integrase inhibitor (dolutegravir) based antiretroviral therapy. Nevertheless, the fifth patient had elevated viral load, low CD4 count, and was antiretroviral therapy naïve. Mortality was low amongst these patients, with four cured of COVID-19 and one remaining in ICU at the time of publication of the study (11). The second case series by Haerter et al. (2020) was that 33 people living with HIV patients were included. All patients were on antiretroviral therapy at the time of diagnosis of COVID-19, 60% of patients included had comorbidities, including hypertension, COPD, diabetes mellitus, cardiovascular disease, and renal impairment. 76% of patients had mild disease, 6% had severe disease, while the remaining were critical cases (12).

The third case series by Gervasoni *et al.* (2020) reported 28 HIV patients with COVID-19. Out of these 28 patients, 13 required hospitalization, and 6 had severe disease. The majority (96%) of patients in this cohort recovered with good outcomes, while the rest (4%) died (13). The fourth case series by Aydin *et al.* (2020) reported four cases of patients diagnosed with COVID-19 pneumonia who had co-existent previously diagnosed HIV infection. Most of the patients were maintained on antiretroviral therapy, except for one noncompliant patient. All patients without comorbidities (three patients out of the four) recovered; and the fourth patient who died had co-comorbidities; diabetes mellitus, essential hypertension, and chronic obstructive pulmonary disease (14).

Our patient was immunosuppressed, as evidenced by his low CD4, high viral load, and being on chemotherapy. However, he did not develop any complications such as pneumonia, acute kidney injury, stroke, or coagulopathy. We hypothesize that the fact his immunocompromised state with low CD4 count resulted in a lessened immune response and fewer disease complications, in addition to the possible potential protective effect of antiretroviral therapy (bictegravir, emtricitabine & tenofovir alafenamide). Our assumption was supported by the prior studies which showed similar outcomes of patients with high viral load and low CD4 T-cell count, and in organ transplant patients who are on immunosuppressive therapy that contracted COVID-19 and had mild symptoms (*15*). The possible protective effect of antiretroviral therapy was based on the current ongoing trials that are being done to evaluate the role of tenofovir and emtricitabine in protection against COVID-19 infection (16). However, our review of the literature failed to support it.

4. Conclusion

Findings from the above case series indicate that the risk of death, severe disease, or admission to ICU from COVID-19 in HIV positive patients was lower than observed in the general population. This might suggest a possible protective effect of poorly controlled HIV in avoiding the cytokine storm induced COVID-19 complications despite being more susceptible to infection. However, as current knowledge about COVID-19 is still evolving, more studies are needed to validate this observation.

References

- Cho HJ, Koo JW, Roh SK, Kim YK, Suh JS, Moon JH, Sohn SK, Baek DW. COVID-19 transmission and blood transfusion: a case report. J Infect Public Health. 2020; doi: 10.1016/j.jiph.2020.05.001.
- Costa IBSDS, Bittar CS, Rizk SI, *et al.* The heart and COVID-19: what cardiologists Need to Know. Arq Bras Cardiol. 2020; 114:805-816.
- Shiau S, Krause KD, Valera P, Swaminathan S, Halkitis PN. The burden of COVID-19 in people living with HIV: a syndemic perspective. AIDS Behav. 2020; 24:2244-2249.
- Guan WJ, Ni ZY, Hu Y, *et al.* Clinical characteristics of coronavirus disease 2019 in China. N Engl J Med. 2020; 382:1708-1720.
- Small BA, Dressel SA, Lawrence CW, Drake DR 3rd, Stoler MH, Enelow RI, Braciale TJ. CD8(+) T cellmediated injury *in vivo* progresses in the absence of effector T cells. J Exp Med. 2001; 194:1835-1846.
- Fang M, Siciliano NA, Hersperger AR, Roscoe F, Hu A, Ma X, Shamsedeen AR, Eisenlohr LC, Sigal LJ. Perforin-dependent CD4+ T-cell cytotoxicity contributes to control a murine poxvirus infection. Proc Natl Acad Sci U S A. 2012; 109:9983-9988.
- Xu Z, Shi L, Wang Y, *et al.* Pathological findings of COVID-19 associated with acute respiratory distress syndrome. Lancet Respir Med. 2020; 8:420-422.

- Zhu F, Cao Y, Xu S, Zhou M. Co-infection of SARS-CoV-2 and HIV in a patient in Wuhan city, China. J Med Virol. 2020; 92:529-530.
- Sun LJ, Wong SXL, Gollamudi S. A case of HIV and SARS-CoV-2 co-infection in Singapore. J Acquir Immune Defic Syndr. 2020; 84: e23-e24.
- Wu Q, Chen T, Zhang H. Recovery from the coronavirus disease-2019 (COVID-19) in two patients with coexisted (HIV) infection. J Med Virol. 2020; doi: 10.1002/ jmv.26006.
- Blanco JL, Ambrosioni J, Garcia F, Martínez E, Soriano A, Mallolas J, Miro JM; COVID-19 in HIV Investigators. COVID-19 in patients with HIV: clinical case series. Lancet HIV. 2020; 7:e314-e316.
- 12. Härter G, Spinner CD, Roider J, Bickel M, Krznaric I, Grunwald S, Schabaz F, Gillor D, Postel N, Mueller MC, Müller M, Römer K, Schewe K, Hoffmann C. COVID-19 in people living with human immunodeficiency virus: a case series of 33 patients. Infection. 2020; 1-6. doi: 10.1007/s15010-020-01438-z.
- Gervasoni C, Meraviglia P, Riva A, Giacomelli A, Oreni L, Minisci D, Atzori C, Ridolfo A, Cattaneo D. Clinical features and outcomes of HIV patients with coronavirus disease 2019. Clin Infect Dis. 2020; ciaa579. doi: 10.1093/cid/ciaa579.
- Altuntas Aydin O, Kumbasar Karaosmanoglu H, Kart Yasar K. HIV/SARS-CoV-2 coinfected patients in Istanbul, Turkey. J Med Virol. 2020; doi:10.1002/ jmv.25955.
- Li F, Cai J, Dong N. First cases of COVID-19 in heart transplantation from China. J Heart Lung Transplant. 2020; 39:496-497.
- ClinicalTrials.gov. Randomized Clinical Trial for the Prevention of SARS-CoV-2 Infection (COVID-19) in Healthcare Personnel (EPICOS). Identifier: NCT04334928. https://clinicaltrials.gov/ct2/show/ NCT04334928 (accessed May 27, 2020).

Received July 20; 2020; Revised August 5, 2020; Accepted August 13, 2020.

*Address correspondence to:

Abdallah Qasim, Department of Internal Medicine, Creighton University, Education Building, 7710 Mercy Road, Suite 301, Omaha, Nebraska 68124-2354, United States. E-mail: Abdullah_qasem2005@hotmail.com

Released online in J-STAGE as advance publication September 4, 2020.