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Visceral Leishmaniasis in An Ethiopian Patient with COVID-19: A Case Report

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Case Report

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Abbreviations: COVID-19: Coronavirus Disease 19; VL: Visceral Leishmaniasis; HIV/AIDS: Human Immunodeficiency Virus/ Acquired Immunodeficiency Syndrome; SARS CoV-2: Severe Acute Respiratory Syndrome Coronavirus 2; RTPCR: Reverse Transcription Polymerase Chain

ABSTRACT

Coronavirus disease-19 has dominated global health conversations. It can seem as if the hugely significant burden of infectious diseases, including visceral leishmaniasis, is no longer a public health issue. We discussed, in this case, the clinical and public health implications of COVID-19 and *Leishmania donovani* co-infection. The patient was an 18 years old male from Northwest Ethiopia diagnosed with COVID-19 and concomitant culture-confirmed visceral leishmaniasis that ended in a fatal outcome. It is of crucial importance that clinicians have a high index of suspicion for a combination of COVID-19 and visceral leishmaniasis in endemic areas.

Reaction; NNN: Novy-McNeal-Nicolle medium

INTRODUCTION

Coronavirus disease-19 is an ongoing global pandemic claiming millions of human lives. Medical conditions associated with severity and death from COVID-19 includes diabetes, cardiovascular diseases, hypertension, pre-existing pulmonary disease, and cancer [1]. Little is known of co-morbidities from tropical infectious diseases.

Ethiopia is one of the countries with the highest Visceral Leishmaniasis (VL) burden and the highest incidence of VL and HIV co-infection [2]. The disease is widely distributed throughout the country's lowlands, especially in the north-western, south-western, and south-eastern regions. Herewith, we report a case of COVID-19 with underlying VL in a patient from northwest Ethiopia. This is yet another co-infection of VL with another infectious disease, that clinicians may find challenging to diagnose and treat.

CASE SUMMARY

An 18-year-old male patient from Northwest Ethiopia presented with 2 weeks history of recurrent high-grade fever with chills and rigors associated with extreme fatigue, loss of appetite, and significant weight loss. He also complained of painful lower abdominal cramps and frequent loose stool. He showed no improvement with empiric anti-malarial and ceftriaxone treatment. He had no history of cough, hemoptysis, or tuberculosis. He never smoked cigarettes. His past medical history was unremarkable.

His vital signs were: blood pressure 102/53 mmHg, pulse rate 110 beats per minute, respiratory rate 34, body temperature 38.4°C, and 90% oxygen saturation on room air. Conjunctivae were pale. He had shotty axillary lymphadenopathy and tipped splenomegaly.

Initial laboratory tests showed pancytopenia and hyperbilirubinemia. Serology for HIV, hepatitis B and C were negative. No hemoparasite was demonstrated on blood film. Peripheral blood smear showed normocytic normochromic anemia with no blasts. RT-PCR for SARS CoV-2 became positive (Table 1).

While on care for severe COVID-19 (intranasal oxygen 3-4 liter per minute, dexamethasone, prophylactic heparin) and work up for the possible causes of cytopenia, he developed septic shock (fever with a body temperature of 38.8 degree Celsius, change in mentation, tachycardia, hypotension, and hyperlactatemia). He was resuscitated, started cefepime and vancomycin, transfused with blood components, intravenous hydrocortisone 200 mg/day, and vasopressor initiated together with other supportive care. Over 2 days, the shock state was corrected. The pressor was de-escalated. He later required blood products transfusion and prophylactic antimicrobials due to severe neutropenia. Plumpy'nut was also given for subjective global assessment C (severe) malnutrition. Over the subsequent days, he deteriorated with progressive liver dysfunction and bleeding.

As some of the clinical presentations were likened to VL, a request for a leishmanial serologic test was made. The rK39 rapid diagnostic test gave a positive result. Examination of the Giemsa stained bone marrow smear demonstrated amastigotes with very low parasite density (grade 1:1-10 amastigotes/1000 fields of oil immersion).

There was a challenge about the VL therapy options at the time as the patient was already deteriorating with severe liver dysfunction. Sodium Stibo Gluconate (SSG) was started - the only anti-leishmanial available at the time. On point-of-care ultrasound examination, he was edematous with serosal effusions. He later became hypotensive. He was resuscitated, transfused with blood components, and antibiotics revised to meropenem for empiric coverage of hospital-acquired infection. SSG was withheld after 2 doses. He, unfortunately, passed away from hemorrhagic

bleeding and septic complications after 10 days of hospitalization. Culture of the bone marrow specimen in Novy-McNeal-Nicolle (NNN) medium grew promastigotes after 2 weeks of inoculation days after the patient died.

Table 1. Haematological and biochemical profile during hospitalization.

Test	Day 1	Day 2	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10
WBC (cells/ml)	2,700	2,100	1,900	870	1,500	1,070	2,020	2,500
Hgb (g/dl)	6.5	9.5	8.7	7.4	8	7.6	8.3	7.1
Platelet (cells/µl)	75,000	36,000	22,000	14,000	49,000	14,000	12,000	26,000
BUN (mg/dl)	13	16	11	-	-	-	15	15
sCr (mg/dl)	0.3	0.3	0.3				0.7	0.7
ALT (IU/L)	21	29	-	29	-	-	41	35
AST (IU/L)	113	214		166			307	299
ALP (IU/L)	247	273		368			372	234
Bilirubin [T/D], (mg/dl)	2.6/1.8	-		-			7.7/6.9	10.0/8.0
PT (sec)	16	-	-	-	64	20	-	-
INR	1.1				7	1.5		
aPTT (sec)	25				101	30		
Na (mEq/l)	130	132	-	133	-	139	129	127
K (mEq/l)	3.6	3.5		2.4		3.9	3.3	3.4
RBS (mg/dl)	109	71	64	58	54	47	120	110
Other tests	TSH=2.5 ml U/L; Lipase 81 U/L; Amylase=25 U/L; Uric Acid=1.9 mg/dl; LDH=527 U/L; ESR=60 mm/hr; Urinalysis unremarkable.							

Note: WBC: White Blood Cells; Hgb: Haemoglobin; BUN: Blood Urea Nitrogen; SCR: Serum Creatinine; ALT: Alanine Transaminase; AST: Aspartate Aminotransferase; ALP: Alkaline Phosphatase; PT: Prothrombin Time; INR: International Normalized Ratio; aPTT: activated Partial Thromboplastin Time; Na: Sodium; K: Potassium; RBS: Random Blood Sugar; TSH: Thyroid-Stimulating Hormone; LDH: Lactate Dehydrogenase; ESR: Erythrocyte Sedimentation Rate

DISCUSSION

There are no specific clinical features that can reliably distinguish COVID-19 from other infections. The commonly reported clinical symptoms of COVID-19 infection include fever, cough, chills and rigors, fatigue, diarrhea, and abdominal pain. Lymphopenia and elevated liver enzymes are also common laboratory features [3]. Most of these COVID-19 manifestations could also be a presenting feature of VL. Such diagnostic confusion could contribute to delayed diagnosis and treatment.

The literature on VL and HIV coinfection showing reciprocal impacts on pathogenesis, diagnosis, and treatment is abundant. VL in HIV-positive patients is an AIDS-defining condition, and co-infection is commonly found in endemic areas [4].

Even though this patient had some clinical features consistent with VL, parasitemia was low. The patient likely had an asymptomatic *L. donovani* infection before SARS-CoV-2 exposure. One wonders if SARS-CoV-2 has the potential to reactivate latent *L. donovani* infections - reminiscent of what HIV does to asymptomatic infections of *L. donovani*. The corticosteroids used for covid-19 treatment could also contribute to immunosuppression resulting in VL reactivation.

The fact that this young patient with COVID-19 with no other co-morbidity, presented with VL and ended in a fatal outcome raises a query about reciprocal impacts of the co-infection. In VL, plasma levels of cytokines and chemokines are high and contribute to cytokine storms [5, 6]. Given this, it is conceivable that an underlying VL can exacerbate symptoms of COVID-19. Albeit anecdotal, it could suggest that VL could be added to the list of co-morbid conditions that predispose SARS-CoV-2 infected individuals to severe and fatal COVID-19.

CONCLUSION

In summary, this case illustrated that VL-COVID-19 co-infection could be challenging to diagnose and treat. In endemic regions, VL should be suspected in patients with COVID-19 infection. Like *leishmania* and HIV co-infection, SARS-CoV-2 and *leishmania* co-infection may prove to be a deadly gridlock. The incursion of SARS CoV-2 into VL endemic areas where HIV co-infection is also common can bring a profound immunopathological paradigm of triple co-infections. Epidemiologically speaking, the situation can turn dire in the face of the ongoing leishmaniasis control program.

COMPETING INTERESTS

The authors declare no conflict of interest.

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