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Management of a Patient with Multi-Drug Resistant HIV and COVID-19 Infections with Associated Left Hand Ischemia from Probable Micro-Embolic Disease

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Introduction

Coronavirus infection caused by the SARS-COV-2 virus (COVID-19), a current worldwide pandemic, manifests mainly with pulmonary complications, causing acute respiratory distress syndrome (ARDS) in its most lethal form, but can also manifest with rare complications such as coagulopathy. To our knowledge, there are no published case reports of a patient with HIV and COVID-19 infections experiencing coagulopathy. Hence, we present a case of multidrug resistant HIV in a patient with COVID-19 and subsequent micro-embolic disease of the left upper extremity.

Keywords: SARS-COV-2; COVID-19; Multi-drug resistant HIV; Micro-embolic disease; Ischemia

Case Presentation

A 39-year-old female patient diagnosed with HIV/AIDS (acquired immune deficiency syndrome) 14 years prior, with multiple antiretroviral drug resistance due to nonadherence with the following resistance-associated antiretroviral mutations K103N, K65R, M184V, L10I, I71T, 140C, 148R, currently prescribed darunavir ethanolate 800 mg daily, lamivudine-zidovudine 150-300 mg daily, ritonavir 100 mg daily. The antiretroviral treatment regimen was chosen to improve adherence with two active agents and the patient has been doing well with undetectable viral load ever since switched to this regimen 3 years ago. Despite continued patient education and discussions of newer antiretroviral therapies available, the patient had been unwilling to change to another possible option.

The patient presented to the emergency department with complaints of cough, myalgia, subjective fever, and chest pain of 5 days duration. Her vital signs were significant for temperature 39.9C, respiratory rate 24 breaths per minute, and pulse 129 beats per minute, blood pressure 133/78 mmHg, and oxygen saturation 94% on room air height of 165 cm, weight 151 lb. The lab work-up was significant for ferritin 777ng/mL, C-reactive protein 8.0 mg/dL, LDH 544 U/L, Creatinine 0.88 mg/dL and positive PCR SARS-COV-2 on a nasopharyngeal sample. The patient was diagnosed with COVID-19 and was placed on 2 L nasal cannula, started on dexamethasone 6 mg IV daily and empiric piperacillin-tazobactam 3.375 mg IV every 8 hours. After 2 days of being hospitalized, blood cultures were negative, antibiotics were discontinued, and symptoms improved, the patient was discharged home on room air with home medications.

Seven days following hospital discharge, the patient experienced painful discoloration of the thenar eminence of the left thumb and left index finger with associated coolness. She reported placing the hand in a dependent position worsened the discoloration and subsequently reported to the emergency department. A physical examination revealed a bluish discoloration of the left arm index finger with tenderness to palpation (Figure 1). An arterial duplex was ordered, showing no evidence of large vessel flow limiting disease, and radial and ulnar arteries with monophasic, hyperemic waveforms.

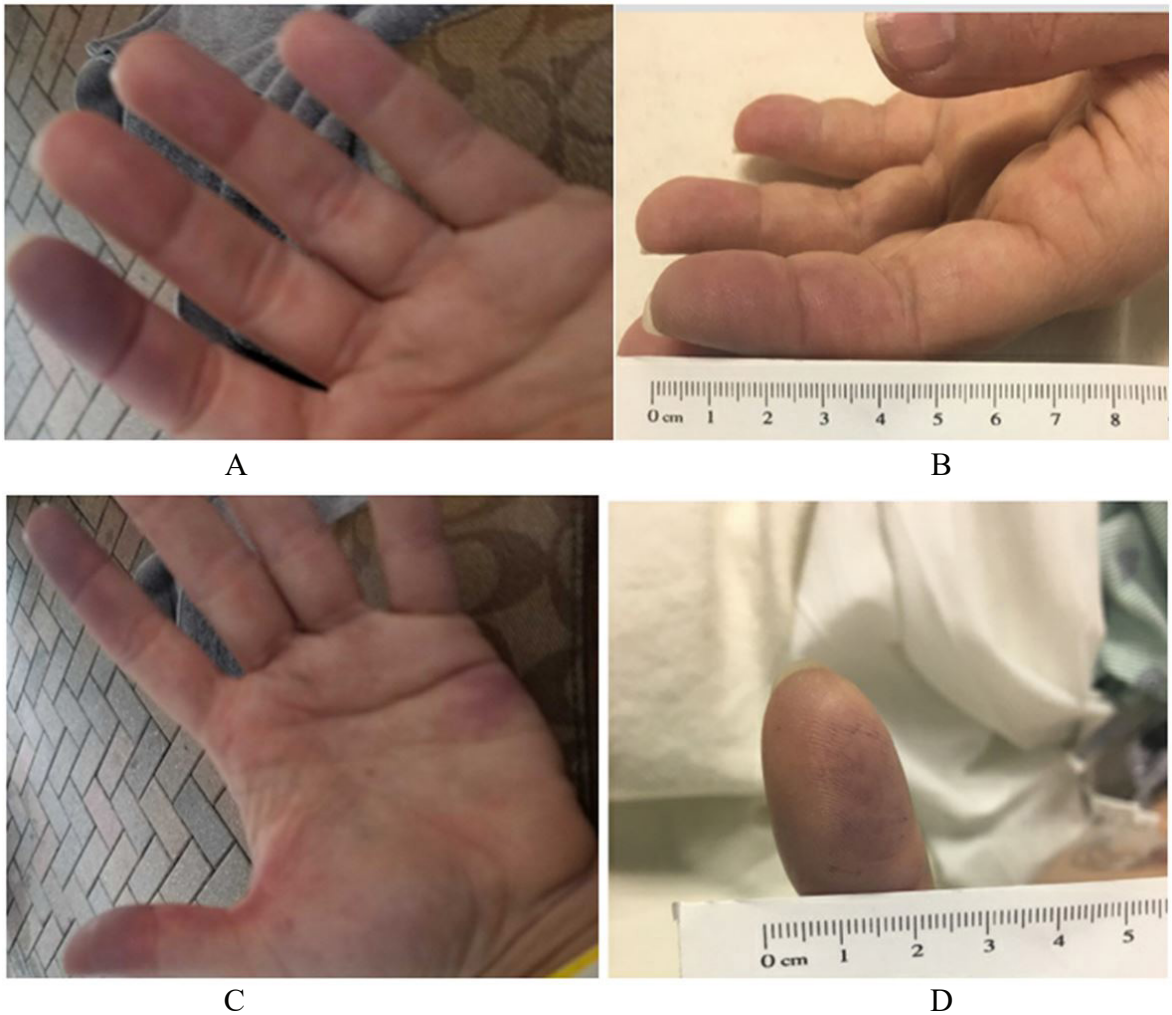

Figure 1: Left hand of the patient showing bluish discoloration of her thumb and index finger on the day of presentation to Emergency Department

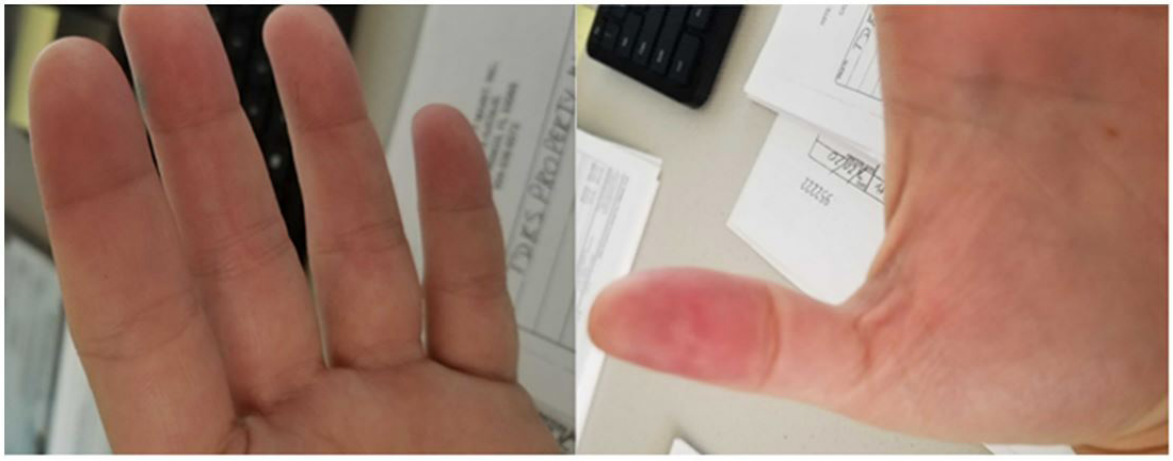

Figure 2: Left hand showing improvement in skin discoloration of the same patient, 13 days later

She was placed on heparin drip for the suspicion of an embolic source, and a vascular surgeon was consulted. A CT angiogram of the left upper extremity was ordered with no definite left upper extremity arterial filling defect. She was diagnosed with a possible micro embolic disease related to COVID-19 infection. The patient was discharged on apixaban 2.5 mg orally every 12 hours due to the high suspicion of embolic source. On follow up office visit 13 days later, the patient reported much improvement in skin discoloration and no pain (Figure 2). However, at this time apixaban was discontinued due to a drug-drug interaction with darunavir, and anticoagulation was continued with dabigatran 150 mg orally every 12 hours, for a total duration of 3 months.

Discussion

Viral, bacterial, or fungal infections initiate a complex inflammatory responses as part of innate immunity, which results in activation of coagulation pathways. The patients with SARS-CoV-2 infection have elevated levels of IL-6, C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and increased fibrinogen level at the time of presentation which indicates significant inflammation [1]. COVID-19 infection could be associated with coagulopathy. COVID-19 coagulopathy is similar to other systemic coagulopathies found with severe infection such as Disseminated Intravascular Coagulation (DIC). A number of patients with COVID-19 infection have developed arterial and venous thromboembolic complications [2].

Patients with COVID-19 coagulopathy have an increased D-dimer and fibrinogen concentration, prolonged prothrombin time and mild thrombocytopenia. In a study of the first 99 patients hospitalized in Wuhan, China with COVID-19, researchers found that 36% of the patient had an elevated D-dimer , 6% with elevated activated partial thromboplastin time (aPTT), 5% with elevated prothrombin (PT), and increased biomarkers of inflammation including interleukin-6 (IL-6), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) [3]. In a series of 1099 patients with COVID-19 from China, elevated D-dimer (>0.5 mg/L) was found in 260 (46%) of 560 patients [4]. Another study found that patients with severe COVID-19 infection, who were admitted to the intensive care unit (ICU) had significantly higher D-dimer concentrations (2.4 mg/L, IQR 0.6–14.4) than patients who received no ICU care (0.5 mg/L, 0.3–0.8) [5]. These coagulation changes which have been found on the patients with COVID-19 infection suggest that there is a hypercoagulable state associated with the infection, which might increase the risk of thromboembolic complications.

An endotheliopathy with vascular endothelial dysfunction contributes to the pathophysiology of circulatory changes and prothrombotic state of the COVID-19 infection [6]. SARS-CoV-2 virus adheres to ACE2 receptor on endothelial cells [7]. The replication of virus leads to inflammatory cell infiltration, endothelial cell inflammation and microvascular prothrombotic effects.7 In the recent studies which includes postmortem evaluation of the COVID-19 patients, researchers have found viral inclusion inside endothelial cells of blood vessels, along with mononuclear, polymorphonuclear cell infiltration and endothelial apoptosis [8]. These microvascular endothelial injury with clot formation found in the postmortem evaluation of COVID-19 patients, supports the clinical picture of micro embolic disease in our patient.

We believe our patient had SARS CoV-2 induced endotheliopathy which led to her clinical presentation. Her emboli imaging workup Doppler study was negative and CT arteriogram showed no evidence of vessel flow limiting disease. However, she showed significant improvement of her ischemic symptoms on intravenous heparin drip. After a proximal thrombus was ruled out, we plan to treat her with oral anticoagulation for 3 months.

In patients with HIV on antiretroviral therapy, drug-drug interactions between antiretroviral drugs and direct oral anticoagulants (DOACs) have limited the use of this new class of anticoagulant. Our patient had multidrug resistant HIV infection with multiple resistances associated viral, and required the use of an HIV protease inhibitor in her antiretroviral regimen. Navigating DOACs drug interactions in her was challenging. She was initially started on anticoagulation with apixaban 2.5 mg two times a day for 3 months. However, due to the interaction between the darunavir component of her ART regimen and apixaban, (darunavir can increase apixaban levels and potentially cause bleeding), apixaban had to be discontinued [9]. This patient could have been considered for a 50% dose reduction of apixaban, but she was already on the lowest dose. The patient was switched to dabigatran, which has no interactions with her antiretroviral regimen and no concerns for possible thromboembolic and bleeding complications. The mechanism of the apixaban interaction is via P450 inhibition and that dabigatran is metabolized by conjugation, avoiding the P450 interaction.

Conclusion

This case report highlights the importance having a suspicion of micro embolic disease in patients with COVID-19 infection especially in HIV patients in which treatment options can be challenging due to medications interactions.

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