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Acroyschemia in a Male Patient with Covid 19: A Case Report

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INTRODUCTION

Coronavirus disease-19 (6COVID-19) which is an enveloped RNA beta coronavirus. First emerged in December 2019 in China and rapidly spread worldwide. Although various studies have reported that COVID-19 is associated with a hypercoagulable state and thrombotic complications in critically ill patients. The current SARS-CoV-2 pandemic is causing high morbidity and mortality, its main affectation is at the pulmonary level, however, the virus also affects the cardiovascular system.[1] The vasculature is affected directly by the virus and indirectly by a storm of inflammatory cytokines, causing an excessive inflammatory process, platelet activation, endothelial dysfunction and stasis.[2] Biochemically it manifests itself with altered coagulation parameters, increased levels of D-dimer and Ferritin, which are the most common laboratory abnormalities related to mortality.[3]

In our investigation, acroischemic lesions were found in critically ill patients with severe limb ischemia.[4,5,6,7,8.]

OBJECTIVE

Demonstrate the importance of the endothelium in COVID-19, the risk of the appearance of vascular complications in the short and long term, promoting research on endothelial dysfunction, for future approaches and treatments.

CLINICAL CASE

33-year-old male, with no chronic degenerative history. The clinical condition began on 07/08/20, with an event of seizures, deterioration of alertness, dyspnea and desaturation of 67%, so he came to our institution. He required supplemental oxygen support with a mask showing improvement in saturation and clinical status, a PCR test for SARS cov2 was performed, with a positive result. However, 48 hours after admission, cyanosis and ischemia of fingers and toes were detected. (Figure 1 and 2)

Laboratories: leukocytes 19.96 x 10 ^ 3, platelets 223 x 10 ^ 3, Hb 13.56g / dl, D-dimer 554ng/ml, Ferritin 1376ng / ml. Glucose 102mg / dl, creatinine 0.69mg / dl.

With adequate clinical evolution at the pulmonary level, but with persistent necrosis of both extremities, being assessed by angiology, performing an angiotomography, which did not identify focal or diffuse stenoses, or filling defects.

Metacarpophalangeal disarticulation was performed. (Three fingers in the right hand, and four fingers of the left hand) Metatarsophalangeal disarticulation was performed of one toe of the left foot and two toes of the right foot. He received antibiotic therapy with cefotaxime 1gr IV every 8hrs, thromboprophylaxis with Lower Molecular Weight Heparin, being discharged with clinical improvement 7 days after surgery without new zones of ischemic areas.

DISCUSION

The current body of literature has been focused on the severe to critical cases of COVID-19 and their association with hypercoagulability and risk of thrombotic events.[9]

These vascular changes could be explained by the utilization of ACE2 receptors by the virus. The ACE2 receptor is also widely expressed on pneumocytes as well as endothelial cells that traverse the multiple organs. The binding between the virus and the ACE2 receptors leads to systemic inflammation as well as endotheliitis that subsequently systemic impaired microcirculatory function in different vascular beds and their clinical sequelae in patients with COVID-19.[10]

Tang et al. demonstrated that elevation of fibrin degeneration products, including D-dimer, and changes affecting the fibrinolytic system can be present from the initial phases of COVID-19, predisposing to pro-thrombotic states and worse prognosis. Anatomopathological studies have identified fibrin deposits in the alveoli and interstitial space in the lungs, with additional evidence of microcirculation thrombosis.[11]

Klok et al presented a 31% incidence of thrombotic complications in (ICU) patients with COVID-19, demonstrating that the infection may predispose patients to

both venous and arterial thrombosis.[12] Zhang et al reported seven cases of patients in the ICU with COVID-19 who subsequently developed marked cyanosis and gangrene of the digits.[13]

Clinical variables poorly predicted which patients had imaging manifestations of coagulopathy. Thus, in patients with COVID-19, pulmonary CTA, extremity Doppler US, contrast-enhanced abdominal CT, and contrast-enhanced brain MRI and MRA may all be appropriate if systemic coagulopathy is suspected.[14]

The American Society of Hematology recommends that all hospitalized patients with COVID-19 should receive pharmacologic thromboprophylaxis with LMWH or fondaparinux, unless bleeding risk exists, and full therapeutic-intensity anticoagulation in the appropriate clinical scenario. Anticoagulant therapy mainly with LMWH appears to be associated with better prognosis in patients with severe COVID-19 meeting sepsis-induced coagulopathy criteria or with markedly elevated D-dimer levels.[15] Acute Limb Ischemia can occur during in-hospital evolution of COVID-19 infection or they can be admitted to the hospital for this vascular condition with mild or no respiratory symptoms. Some patients are able to achieve revascularization with observation or medical and/or surgical intervention, but other patients succumb to either amputation or death.[16]

CONCLUSION

It is important to always take into account that the SARS-CoV2 virus has a lung condition, however it unleashes an important inflammatory process, which comes to present thrombotic complications in various systems, so surgeons must be alert to the signs of arterial thrombotic complications in these patients.

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Figure 1.

Figure 2.