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SARS-Cov2andlimb Ischemia

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1. Abstract

Patients with severe acute respiratory syndrome coronavirus 2 (SARS-Cov2) infection mainly present severe pneumonia associatedwithcomplicationsrelatedtocytokinestormsyndrome.So,it wasassociatedwiththromboticincidentslikeacutelimbischemia and pulmonary embolism.

Wereport3casesofCOVID-19infectioncomplicatedbyarterial thrombosis in the form of acute limb ischemia.

2. Introduction

Attheendof2019,anovelcoronavirusdisease2019(COVID-19) caused by the severe respiratory syndrome coronavirus 2 (SARS-Cov2) was declared a worldwide epidemic.

Patients with COVID-19 usually present with respiratory symptoms like cough, fever, and shortness of breath. We found also a very high incidence of thromboembolic complications in patients hospitalized with COVID-19 pneumonia.

We report 3 cases of COVID-19 infection complicated by acute limb ischemia.

3. CasePresentation1

A-71-year-oldmanwithamedicalhistoryofdiabetesmellitusand dyslipidemia presented to the emergency department with shortness of breath. One week previously, he had started to develop fever, asthenia, and anorexia.COVID 19 rapid test was positive. Clinical examination showed a Glasgow scale of 15/15, blood pressure120/60mmHg,heartrate74/min,oxygensaturation86% onroomair,andtemperature37,1C°.Electrocardiographyshowed the left anterior fascicular block. On initial laboratory evaluation, wefound:lymphopenia(820cells/mm^3),hyperglycemia(18 mmol/l),hepaticcytolysis(1,5xN),andhigh

C-Reactive-Protein (254 ng/ml). According to SFR, CT Scan showed SARS-CoV2 pneumonia with parenchymal involvement estimatedat25%-50%.Thepatienthadreceivedoxygen(10liter/min),dexamethasone12mgaday,andlowmolecularweighthep- arin (LMWH) 0,4 mg a day. He had also received cefotaxime 3 g fortheday.Afteroneweek,hisbreathingwasimprovedbuthehad acutepaininhisleftlimb.Thedistalthirdofthelimbwascoldand mottled.Hehadasensory-motordeficitofhislimb.Theleftpedal pulsewasabsent.Hewastreatedwithunfractionatedheparindrip (UFH)210mgfordayandvasodilatordrug.Butthelimbischemia was extended. The patient had a mid-leg amputation. Two days after, he had a cardiac arrest and died.

4. CasePresentation2

A-73-year-old man with a medical history of diabetes mellitus and hypertension presented to the emergency department with complaints of shortness of breath.One week previously he had started to have fever and cough.Vital signs on presentation were: Glasgow scale 15/15, heart rate 92/min, blood pressure 140/70 mmHg, oxygen saturation 84 % on room air, and 95% on supplemental oxygen (12 liter/min). The COVID 19 rapid test was positive. The electrocardiography was normal. On initial laboratory evaluation, we found: hyperleukocytosis at 15900/mm^3, lymphopeniaat660/mm^3,anemiaat11.1g/dl,elevatedC-reactiveprotein at 91 mg/l, and hepatic cytolysis (3x reference). The patient had receivedLMWH0.7mgtwiceaday,dexamethasone12mgaday, cefotaxime 3 g a day, and furosemide 20 mg twice a day.After7 days, he mentioned lower limb pain. On examination, his right footwascoldtothetouchandcyanosisinappearance.Hislegwas

mottled and cold. His right popliteal artery and pedal pulse were not palpable. Computed tomography angiography showed complete occlusion of the right superficial femoral artery extended to the popliteal artery (figure 1).

Hewasstartedonatherapeuticheparininfusion.Heunderwentan emergency right lower artery embolectomy Fogarty catheter. But hisfootwascyanosisandhispedalpulsewasnotpalpable.So,the patient had a mid-leg amputation.Two days after, the amputation stump was necrotic.

He had a mid-thigh amputation then transferred to intensive care for respiratory distress and he died a few days later.

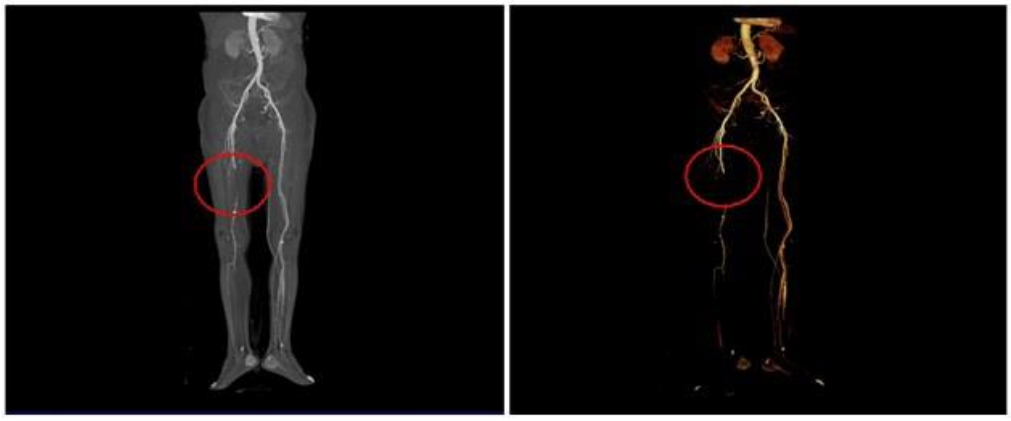

Figure1:CTangiographyofaortaandiliofemoralarteries:Completeocclusion oftherightsuperficialfemoralarteryextendedtothepoplitealartery

5. CasePresentation3

An A-58-year-old man without medical history presented to the emergency department complaining of fever, asthenia, and shortness of breath.

Vital signs on presentation were:Glasgow scale, 15/15; temperature, 37 C°; heart rate, 99 beats/min; blood pressure, 122/64 mmHg; oxygen saturation, 80% on room air and 96% on supplementaloxygen(16liter/minofoxygen).Hisnasopharyngealswap was positive for COVID-19 infection. The electrocardiography wasnormal.Oninitiallaboratoryevaluation,wefound:hyperleukocytosis,10700/mm^3;lymphopenia,510/mm^3;elevatedD-dimer 635 ng/mL and elevated C-reactive protein, 195 ng/mL.

Thepatienthasbeentreatedwithdexamethasone12mgforaday, LMWH 0,4mg twice for a day, cefotaxime 3 g for a day. He had receivedinsulinfornewlydiagnoseddiabetes.Twodaysafter,he had painful lower extremities. On examination, his bilateral extremitieswere mottledand cold. His femoral, posterior tibial,and pedal pulses were not palpable. Computed tomography angiography of the aorta and iliofemoral arteries showed complete occlusion of the abdominal aorta with extension to both common iliac arteries, complete occlusion of the right popliteal artery, the left anteriortibialartery(figure2).Itshowedasplenicinfarction.The patient had received UFH. But he had a distress respiratory and a cardiac arrest and died.

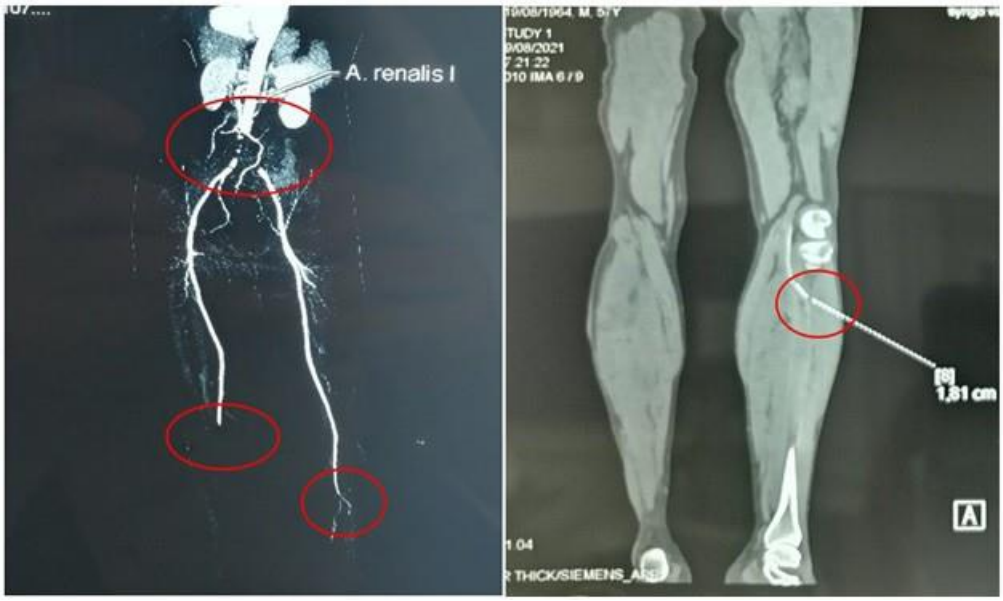

Figure2:CTangiographyoftheaortaandiliofemoralarteries showed:
- Complete occlusion of the abdominal aorta extension to both common iliac arteries
- Completeocclusionoftherightpoplitealartery,theleftanteriortibialartery

6. Discussion

COVID-19 significantly impacted healthcare worldwide. It can present with a variety of clinical manifestations including fever, cough, myalgia, dyspnea, headache, diarrhea, and vomiting [1]. Although respiratory symptoms predominate, COVID-19 is a high risk for arterial thromboembolism.Patients with severe COVID-19infectioncandevelopintravascularcoagulopathywith endothelial dysfunction [2].

Multipleretrospectiveanalyseshavesimilarincidentsfrom12 to 31% of thrombotic complications in patients infected with COVID-19[2].Aminority ofthese eventsare arterial(4%) [1, 3].

Themechanismofthromboemboliccomplicationsismultifactorial:adirectviralinfectionoftheendothelialcellleadingtodiffuse endothelialinflammationincreasedpro-coagulantfactorssuch as fibrinogen, factor VIII, and high inflammatory state associatedwiththecytokinestormleadingtocoagulationandfibrinolysis activation [4]. So, endothelitis and hypercoagulability, together withprolongedimmobilizationofcriticallyillCOVID-19patients complete the

Virchow'striadexplainsthemechanismofarterialthrombosis[3].

Elevated D-dimer was associated with high mortality [5].The study of Tang et al showed that abnormal coagulation results,

characterized by markedly elevated D-dimer and fibrin degradation products

arecommoninpatientswhohavediedofthisviralpneumonia[6].

In China, a study of 1099 covid-19 cases showed that 46,4% of patientshadahighlevelofD-dimer(≥500μg/l),60%ofthemhad severe pneumonia [7].

Parminder K et al reported a similar case of a 43-year-old patient whohadasevereformofCOVID-19infection.Hehadanelevated D-dimer and fibrinogen. He was found to have a thrombus of the proximalrightsuperficialfemoralarteryandtwodaysafteradmission, he had a cardiac arrest and died [8].

Inareview,CheruiyotIetalfoundthatmostpatientswitharterial thrombosis were elderly and had pre-existing co-morbidities [3], confirming the current evidence that pre-existing chronic disease increases the incidence and severity of COVID-19 [3, 9]. Pre-existing cardiac diseases have been known to prejudice vascular inflammation which could be exacerbated by the SARS-CoV-2 infectionandtheenhancedprevalenceofarterialthrombosisinthese particularly predisposed individuals [3].An observation has been reportedinthepediatricpopulationwherethepresenceofcongenitalcardiovasculardiseasepredisposedaffectedchildrentosevere COVID-19 infection and thrombotic complications [10].

We have two elderly patients with preexisting chronic illness in ourcases,butthethirdcasehad58years,withoutmedicalhistory.

The American Society of Hematology (ASH) recommends that allhospitalizedpatientswithCOVID-19infectionshouldreceive anticoagulantthromboprophylaxis,intheabsenceofacontraindication [11].

Therecommendationsregardingtherapeuticanticoagulationinpatientswithlimbischemiaandsars-CoV-2infectionarenotwell established yet [12, 13].

BellostaRetal,inastudyon20patientswhounderwentrevascularization, showed that the usage of systemic heparin was associated with increased survival [5]. In this study, all the patients receivedLMWHafterrevascularization,andnoneofthemrequired reintervention, which leads to the possibility that heparinization prevents recurrent thrombosis and improves the rate of survival and limb salvage [12].

However, in our case, a patient, treated with therapeutic LMWH after Fogarty artery embolectomy catheter, had a mid-leg amputation for a necrotic lower.This may be explained by the patient's comorbidities and the delay of the revascularization.

7. Conclusion

COVID-19 can be associated with an increased risk of limb ischemia.Ourcasesandreviewoftheliteraturerevealthathealthcare providers should be aware of life-threatening thromboembolic events.So,weshoulddiscusstherapeuticanticoagulationandlaboratory values such as D-dimer may help with the decision.

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