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Case Report

Concomitant renal and splenic infarctions in a COVID-19-patient with a catastrophic thrombotic syndrome ☆

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ABSTRACT

Since the outbreak of the COVID-19 pandemic, thrombotic events have been accurately described in patients infected by the SARS-CoV-2, especially venous thromboembolism. However, the mystery of arterial thrombosis is still unclear. Here, we report the case of a 59-year-old man with diabetes mellitus, admitted for COVID-19-pneumonia complicated by pulmonary embolism, a thrombus in the aortic isthmus, the descending thoracic aorta associated with splenic and left renal infarctions, and an acute right limb ischemia. The etiological assessment of this catastrophic thrombotic syndrome showed no evidence for preexisting inherited or acquired thrombophilia. Our case emphasizes the hypercoagulability state in COVID-19-patient leading to both arterial and venous thromboembolisms and the need to establish adequate strategies for the diagnosis and management of thrombo-embolisms to prevent these potentially fatal complications.

Introduction

Since the outspread of the novel coronavirus disease 2019 (COVID-19), there is increasing evidence of thrombotic events, especially in critical ill patients [1]. Moreover, this hypercoagulable state seems to induce both venous and arterial thrombosis [2]. Nonetheless, little is known about arterial thrombosis related to COVID-19 disease. In fact, some authors reported widespread vascular thrombosis with microangiopathy in the lungs from patients with Covid-19 during autopsy [3,4]. To date just a few cases of thrombotic arterial complications related to the COVID-19 infection have been reported. Herein, we report the case of a 59-year-old man who presented with a COVID-19

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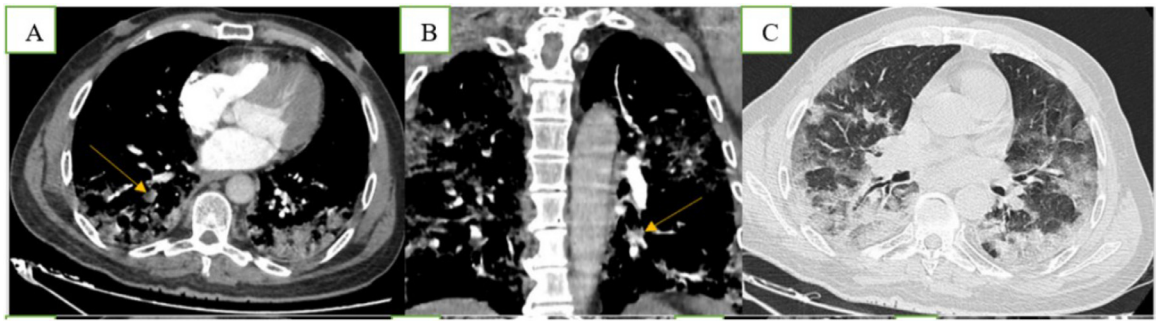

Fig. 1 – computed tomography pulmonary angiography (CTPA): (A) axial and (B) coronal reformatting (mediastinal windows) with a filling defect in the right segmental branch artery (yellow arrows), (C) axial reformatting (parenchymal window) showing typical patterns related to COVID-19-infection at the lung parenchyma including peripheral distribution of ground-glass opacities associated with consolidation.

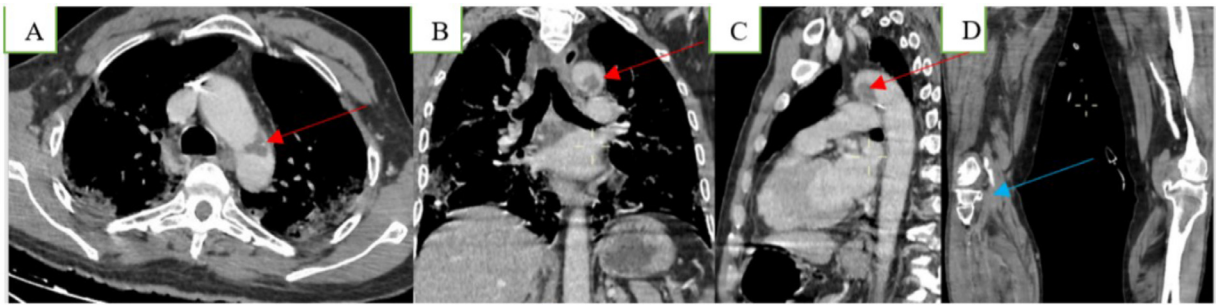

Fig. 2 – Chest, abdominal, pelvic computed tomography angiography (CTA): (A) axial (B) coronal and (C), sagittal reformatting (mediastinal windows) showing an acute thrombus in the aortic isthmus and the descending thoracic aorta. (Red arrows). (D) lower limbs CTA show.

infection, complicated by a catastrophic thrombotic syndrome including pulmonary embolism, aortic thrombosis and acute limb ischemia, associated with splenic and left renal infarctions.

Case presentation

A 59-year-old man with a past medical history of diabetes mellitus was admitted to the emergency department for sudden dyspnea after two weeks of cough, subjective fever, and asthenia. He was febrile at 39°C and hemodynamically stable on physical examination, but saturating 75% on room air. Biological findings revealed elevated white blood cells (WBC) at 15,000 elements/mm^3 (normal range (NR): [4000-10,000]), with lymphopenia at 450 elements/mm^3 [NR: 1500-4000], decreased level of platelets counts at 120,000 elements/mm3 [NR: 150,000-450,000]; a high D-Dimer level at 33,620 ng/mL [NR: 0-500]; a high level of fibrinogen at 4,5 g/L [NR: [1,5-3], elevated CRP at 189.88 mg/L [NR: 0-6]; a high level of ferritin at 4150 ng/mL, [NR: 30-300], and LDH at 1221 unit/L [NR: 140-245]. The SARS-Cov-2 reverse transcriptase-polymerase chain reaction test (RT-PCR) was positive. The patient received Azithromycin, Ceftriaxone, hydroxychloroquine and prophylactic anticoagulation therapy with low-molecular-weight-heparin (LMWH).

The day after, the patient reported pain in the right lower limb. The physical examination revealed coolness, numbness and no peripheral pulse of the right popliteal, anterior tibial, posterior tibial, and dorsalis pedis artery were palpable. We emergently performed a venous and arterial doppler ultrasound that showed no vascular flow at the right popliteal artery level. An urgent blood test showed an elevated creatinine kinase (CK) level at 20,500 U/L (NR: 0-195 UI /l), and renal function was normal with creatinine level at 9.59 mg/L (NR: 6-12 mg/l).

An urgent chest, abdominal, and pelvic computed tomography angiography (CTA) revealed a filling defect in the left segmental pulmonary artery and typical patterns related to COVID-19 infection at the lung parenchyma, including peripheral distribution of ground-glass opacities associated with crazy paving as well as consolidation (Fig. 1, A, B, C), a thrombus in the aorta isthmus and the descending thoracic aorta (Fig. 2, A, B, C) associated with renal and splenic infarctions (Fig. 3). The lower limbs CTA showed an abrupt arrest in the right femoral artery. The patient underwent an emergent femoropopliteal Fogarty embolectomy by the vascular surgery department with a good final blood flow.

After embolectomy, the patient underwent anticoagulation treatment with low molecular weight heparin (LMWH) 60 mg twice a day. Two days after the embolectomy, the patient presented a poor clinical response. We discussed his case in a

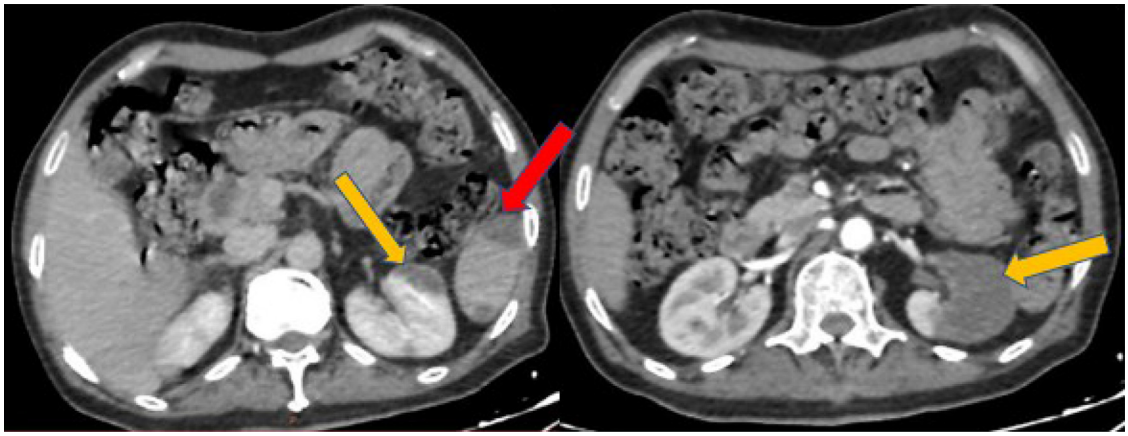

Fig. 3 – abdominal CTA in axial reformatting showing left renal infarction (yellow arrows) and splenic infarction. (Red arrow).

multidisciplinary session then the patient underwent infracondylar amputation of the right lower limb. His condition improved and discharged 15 days later with anticoagulation treatment.

The etiological assessment of this catastrophic thrombotic syndrome showed no evidence for pre-existing inherited or acquired thrombophilia. (Protein S, protein C, and antithrombin III were normal, mutation for factor II and V Leiden and antiphospholipid antibodies were absent.

Discussion

Catastrophic thrombotic syndromes are distinguished by the rapid onset of multiple thromboembolic occlusions in various vascular beds [5]. Thromboembolic complications are commonly described in patients with COVID-19 disease [1]. However, the blood clots' pathogenesis related to COVID-19 is still unclear. The pathophysiological mechanism proposed includes the three elements of Virchow's triad. Endothelial cell dysfunction, blood flow stasis due to immobilization, and coagulation abnormalities such as elevated fibrinogen, factor VIII have been reported in patients with COVID-19 [3,6,7]. This state of hypercoagulability, seems to induce both venous and arterial thrombosis [2]. Nevertheless, little is known about the incidence of arterial thrombosis. A recent study has reported an incidence of 1.8% in 2021 confirmed-COVID-19-patients [8].

Berre et al. [9] have reported the first case of acute arterial thrombosis related to COVID-19 and was an association between aortic thrombosis and pulmonary embolism. Woehl B et al. have reported the first series of 4 cases of aortic thrombosis in COVID-19 patients. As 50% of these patients died, they suggested that the outcomes in patients infected by the SARS-cov2 affected by arterial thrombosis seem to be severe [10]. Then, a few reports of acute arterial thrombosis were described in the literature, which occurred in the mesenteric, limb and cerebral arteries [11]. Indeed, COVID-19-patients are at high risk for developing acute arterial thromboembolic complications, especially in large vessel distributions [12]. Kashi et al. [13] have reported a case series of seven severe arterial thrombotic events COVID19-patients despite the use of antiplatelet or anticoagulant therapy. Moreover, there has been a single report of arterial thrombosis in an ambulatory patient in recovery [14] and patients with a mild COVID-19 [15].

Our patient, who presented a catastrophic thrombotic syndrome (thrombotic storm) as reported in a previous single report, [16] required amputation after Fogarty embolectomy. Madani et al. reported a case of a 40-year-old with COVID-19 who required amputation of the right lower extremity due to acute limb ischemia [17]. The mortality reported in patients with arterial thrombosis is higher in COVID-19-patient than those without COVID-19, as well as the rate of amputation [18].

Regarding previous reports, including a concomitant renal and splenic infarction in a patient with COVID-19, [19] we hypothesized in our patient that the severe COVID-19-pneumonia induced a prothrombotic state, resulting in the descending aortic thrombosis. This thrombus likely embolized, leading to both renal and splenic infarction.

For arterial thromboprophylaxis, a few studies have demonstrated the importance of the use of antiplatelet therapy either alone or in combination with an anticoagulant in COVID-19-patients [20]. However, some authors propose to use routinely antiplatelet therapy with either low-dose aspirin (75-150 mg/d), or $P2Y_{12}$ inhibitors such as clopidogrel and ticagrelor, especially in those who are deemed with a high risk of acute arterial thrombosis [21]. In contrast, a recent large study reported no effect of these antiplatelet therapies [22].

Conclusion

This case emphasizes the hypercoagulability state in COVID-19 patients leading to arterial thromboembolism. However, further studies are needed to confirm this novel and unusual form of thrombotic events associated with COVID-19. In the light of this case and current data, we suggest that physicians should adopt a high index of suspicion for associated thromboembolic events of to diagnose and manage this disease's novel manifestations.

Authors' contribution

F. Laouan Brem: conception, literature review, analysis, data collection, writing - review & editing. **T. Abu Al Tayef**: conception, software, writing - review & editing. **H. Rasras**: conception, software, writing - review & editing. **O. EL MAHI**: conception, methodology, supervision. **N. El Ouafi**: conception, methodology, supervision. **Z. Bazid**: conception, methodology, supervision.

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Patient consent

Written informed consent was obtained from the patient for anonymized data to be published in this article.

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