



MULTIPLE THROMBI COMPLICATING PATIENT WITH COVID-19 PNEUMONIA.

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ABSTRACT Patients with coronavirus disease (COVID-19) have an increased incidence of thromboembolic events compared to the general population. Pulmonary embolism (PE), deep vein thrombosis, ischemic stroke, and myocardial infarction are complications described with increasing frequency in these patients. Renal artery thrombosis in COVID-19 infection is extremely rare. We describe a 73-year-old man with a positive IgG COVID-19 test, who presented with mucocutaneous jaundice and fatigability lasting 20 days, without other symptoms. Laboratory tests showed elevated D-dimer levels and renal markers, with negative phospholipid antibodies. A CT angiography revealed the presence of two left ventricular thrombi, renal artery thrombi, pulmonary embolism, and "crazy paving" patterns. Echocardiography demonstrated a dilated and severely hypokinetic right ventricle with biventricular thrombi. This necessitated the initiation of therapeutic anticoagulation with heparin, leading to a favorable outcome. This case highlights that thromboembolic complications in patients infected with COVID-19 may include renal artery thrombosis, which has important implications for prevention and therapy.

KEYWORDS : phospholipids antibodies, renal artery thrombosis, pulmonary embolism, biventricular thrombi, covid-19

INTRODUCTION

Patients with COVID-19 are at a heightened risk of thromboembolic events compared to the general population. Various studies have consistently shown that severe cases of COVID-19, especially those requiring intensive care, are associated with increased rates of deep vein thrombosis (DVT), pulmonary embolism (PE), and other thrombotic complications. A meta-analysis has indicated that venous thromboembolism (VTE) affects between 10% and 30% of COVID-19 patients, with pulmonary embolism being the most commonly encountered complication [1, 2]. In this case report, we present a patient who developed right distal pulmonary embolism, along with occlusion of the right renal artery and thrombi in both ventricles. This case underscores the potential for multiple thromboembolic events to occur simultaneously in COVID-19 patients and highlights the importance of early detection and timely intervention to manage thrombotic complications.

Case Presentation

A 73-year-old male with a history of gout, managed with colchicine for the past three years, presented with a productive cough, mucocutaneous jaundice, and fatigue lasting for 20 days. He denied experiencing fever, dyspnea, or chest pain. On examination, he exhibited mucocutaneous jaundice and had a body mass index (BMI) of 26 kg/m². He was hemodynamically stable, with a respiratory rate of 16 breaths per minute and was eupneic. His oxygen saturation was 98% on room air, and there were no signs of critical limb ischemia or right heart failure. The electrocardiogram showed sinus rhythm without ischemic changes. A nasopharyngeal swab tested negative for SARS-CoV-2 using Reverse-Transcription Polymerase Chain Reaction (RT-PCR). However, the patient tested positive for COVID-19 with a rapid Immunoglobulin G (IgG) test.

The laboratory work-up revealed leukocytosis with a white blood cell count of 13270/mm³, lymphopenia with a lymphocyte count of 900/mm³, and thrombocytopenia with a platelet count of 118000/mm³.

Prothrombin time was prolonged at 24.5 seconds, and there was evidence of hepatic cytolysis. The patient also presented with acute renal failure, evidenced by a serum creatinine level of 270 µmol/L, and elevated D-dimer at 21 mg/L. Hyperlactatemia was noted with a lactate level of 621 U/L, and serum ferritin was significantly elevated at 2000 ng/mL. Troponin T levels were markedly elevated at 1680

pg/mL. Testing for lupus anticoagulant, anti-beta-2-glycoprotein antibodies, and anticardiolipin antibodies was negative. (table1)

Table 1: Laboratory findings for a 73 year old Moroccan male patient affected by COVID-19.

Variables	Result	Reference range
White-cell count (per mm3)	13270	400-11000
Total neutrophils(per mm3)	11380	2500-7000
Total lymphocytes(per mm3)	950	1000-4800
Total monocytes(per mm3)	890	200-600
Platelet count (per mm3)	118000	150000-450000
Hemoglobin (g/dl)	12.6	12.5-16
Alanine aminotransferase (U/liter)	1271	29-33
Aspartate aminotransferase (U/liter)	544	8-33
Lactate dehydrogenase (U/liter)	621	135-225
Creatinine (µmol/liter)	270	60-120
EGFR (ml/min/1.73 m2)	20.5	Sup 90
High-sensitivity cardiac troponin I (pg/ml)	1680	inf 14
Prothrombin time (sec)	24.5	11-13.5
Fibrinogen (g/liter)	2.56	2-4
D-dimer (mg/liter)	21.1	0-5
Serum ferritin (ng/ml)	2000	30-400
Procalcitonin (ng/ml)	0.503	Inf 0.5
High-sensitivity C-reactive protein (mg/liter)	53.34	Inf 5
Lupus anticoagulant, anti-beta-2-glycoprotein and anticardiolipin antibodies	negative	-

Computed Tomographic (CT) angiography imaging of the chest and abdomen revealed the presence of 2 left ventricular thrombus measuring 37 mm thick were found and the second small thrombus measuring 8 mm, on the other side, a right ventricular thrombus measuring 12 mm were found Figure 1a, Pulmonary Embolism in the right lower lobe as shown in Figure 1b, with bilateral and peripheral ground-glass opacities predominantly on the right, associated with anterior consolidations of the right inferior pulmonary lobe with Crazy Paving as indicated in Figure 1c, and Occlusion of the Right Renal Artery as pointed out in Figure 1d (Figure1).

(a) Typical Covid-19's of biventricular thrombosis, (b) Pulmonary Embolism in the right lower lobe, (c) diffuse reticulation with crazy paving, (d) and occlusion of the right renal artery

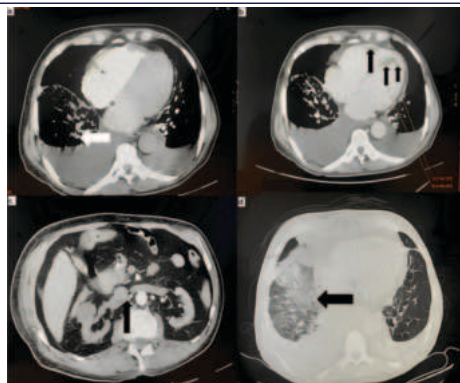


Figure 1: CT angiography scans Imaging for a 73 year old Moroccan male patient affected by COVID-19.

The 2D transthoracic echocardiography, performed in modified views to improve the visualization of left ventricular (LV) thrombi in the four-chamber views, revealed several findings (Figure 2).

A thrombus attached to the distal interventricular septum and left ventricle apex, extending through the left ventricle cavity, and dangling above the mitral valve (a), another thrombus attached to the infero-lateral segment with a small region of echolucency (b, c). There was an enlargement of the right ventricle (RV) and atrium (c) with a round, mobile thrombus seen in a modified apical view of the right ventricle apex (d). a dilated and severely hypokinetic right ventricle

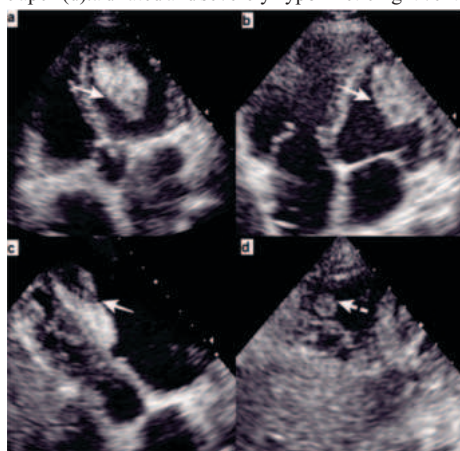


Figure 2: 2D-trans-thoracic echocardiography in parasternal and short-axis views demonstrated two echo-dense masses attached to the infero-septal and antero-lateral segments of the left ventricle and grossly dilated left ventricle (LV).

The coronarography imaging was not done in order not to aggravate the deterioration of the renal function. Given the presence of multiple venous and arterial thrombi, therapeutic anticoagulation with intravenous heparin was initiated. At day 7 after admission in critical care the patient was later transferred to the cardiology department.

DISCUSSION

This case highlights the thromboembolic complications observed in COVID-19 patients, including biventricular thrombi and associated cardiac dysfunction. The exact mechanisms underlying cardiac injury and myocarditis in COVID-19 remain unclear, but several theories suggest direct viral damage via the ACE2 receptor, myocarditis, systemic inflammatory response (cytokine storm), destabilized coronary plaques, and exacerbated hypoxia.

Marwa Soltani et al. reported biventricular thrombi associated with myocardial infarction and severe systolic dysfunction in a COVID-19 patient [2].

Mahine Kashi et al. reported seven cases of severe arterial thrombotic events in COVID-19 patients, all with a history of cardiovascular disease [3]. The risk of pulmonary embolism is further compounded by obesity, advanced age, and immobilization during hospitalization. In this case, the patient presented with elevated CRP and D-dimer levels,

along with advanced age, indicating a COVID-19-induced hypercoagulable state, which likely contributed to thrombosis in the pulmonary vasculature. Elevated D-dimer levels are common in COVID-19 patients, even in the absence of thrombophlebitis or acute pulmonary embolism [4]. In our case, occlusion of the right renal artery led to acute kidney injury. While no renal artery thrombi were detected, a post-mortem histopathologic study of 26 COVID-19 patients found segmental fibrin thrombi in the glomerular capillaries of three cases [5]. This supports the possibility of microthrombus formation in our patient. Elevated renal markers in our patient were likely due to the renal artery occlusion.

While some cases of COVID-19-associated thrombosis are linked to prothrombotic conditions like antiphospholipid syndrome [6]. Our patient tested negative for antiphospholipid antibodies. This suggests that the thrombotic complications may result from the hypercoagulability induced by SARS-CoV-2 infection.

Given the high incidence of thrombotic events in hospitalized COVID-19 patients often elderly, immobile, and with coagulopathy, it is essential to monitor for venous thromboembolism (VTE) and optimize antithrombotic therapies. COVID-19 promotes thrombotic disorders through mechanisms such as excessive inflammation, platelet activation, endothelial dysfunction, and stasis. Furthermore, the concurrent use of antithrombotic therapy in COVID-19 patients may require adjustments in treatment, dosing, and monitoring [7].

This case underscores the importance of early detection and management of thromboembolic complications in COVID-19, particularly in patients with underlying comorbidities, to improve outcomes during the pandemic.

CONCLUSIONS

This case report highlights a patient with COVID-19 who developed concurrent renal artery thrombosis, pulmonary embolism, and biventricular thrombi, as demonstrated by CT angiography. These thromboembolic complications underscore the critical need to recognize the hypercoagulable state induced by SARS-CoV-2 infection, which carries important implications for both therapeutic intervention and prevention. Additional research is essential to further elucidate the underlying pathophysiology and establish optimal management strategies for these life-threatening thrombotic events in COVID-19 patients.

Additional Information

Disclosures

Human Subjects: Consent was obtained or waived by all participants in this study. **Conflicts of Interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/Services Info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial Relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other Relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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