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Thrombosis Update 3 (2021) 100042

Contents lists available at ScienceDirect

Thrombosis Update

journal homepage: www.journals.elsevier.com/thrombosis-update

# Concomitant acute pulmonary embolism, intracardiac thrombus and renal artery thrombosis in COVID-19 patient

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ARTICLE INFO

*Keywords:*
Pulmonary embolism
Venous and arterial thromboembolic complications
Intracardiac
Thrombus
Renal artery thrombosis
Covid-19

## 1. Introduction

Currently the entire word is facing an unprecedented situation; the corona virus disease 2019 pandemic (COVID-19). In addition to the well known pulmonary complications, recently cardiovascular damages were therefore noticed in patients with COVID-19 [1–3]. It may be attributable to the hypercoagulable state associated with the disease [4–9]. The exact physiopathology remains unknown. Radiologists helped by thoracic computed tomography and CT angiography of the chest play a key role in the diagnosis of severe cases and complications of COVID-19 pneumonia [10]. We present herein a case of concomitant pulmonary embolism, intracardiac thrombus and thrombosis of the right renal artery in COVID-19 patient reflecting the hypercoagulable state associated with the disease.

## 2. Case report

A 73 years old man who had no medical history, except for gout for which he followed a diet without any drug intake, was admitted in the emergency department for acute severe dyspnea at rest (stage IV of NYHA), he was unable to carry on any physical activity without discomfort, dyspnea and dry cough, symptoms starting whiting 5 hours.

The physical examination found a conscient patient (15 on the Glasgow score), weighing 71kg, febrile at 39.2°, extreme asthenia with respiratory struggle signs and perioral cyanosis, tachypnea at 22 cycles per minute with a peripheral saturation of oxygen at 82% while breathing in ambient air, his blood pression was at 133 mmHg/82 mmHg with no tachycardia. The pulmonary auscultation shows crackles on lung bases, the patient had also lower limbs white pitting edema and the Homans sign was negative.

Blood test showed: lymphopenia at 250/mm$^3$, (normal >1500/mm$^3$), elevation in LDH (Lactate Dehydrogenase) at 899U/l (normal between 135 and 225 U/l) and an elevation of the inflammatory markers like serum D-dimer level of 11200ng/ml for a normal <500ng/ml, C- reactive protein 115mg/l (normal <5mg/l), ferritin at 1050ng/ml (normal:30–400ng/ml) and procalcitonin elevation at 0.885ng/ml (normal:<0.5ng/ml).

Further, other laboratory abnormalities were found such as a renal failure with a glomerular filtration rate (GFR) of 23ml/min/1.73m$^2$ estimated with MDRDs formula, a prothrombin time of 10 sec, platelets count of 120 × 10$^3$/μL and a troponin elevation at 2.45ng/ml (normal<0.014ng/ml).

An old annual medical check-up shows no history of kidney failure (7 months prior GFR was 75 ml/min/1.73m$^2$, estimated with MDRDs formula).

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https://doi.org/10.1016/j.tru.2021.100042
Received 7 June 2020; Received in revised form 27 January 2021; Accepted 1 March 2021

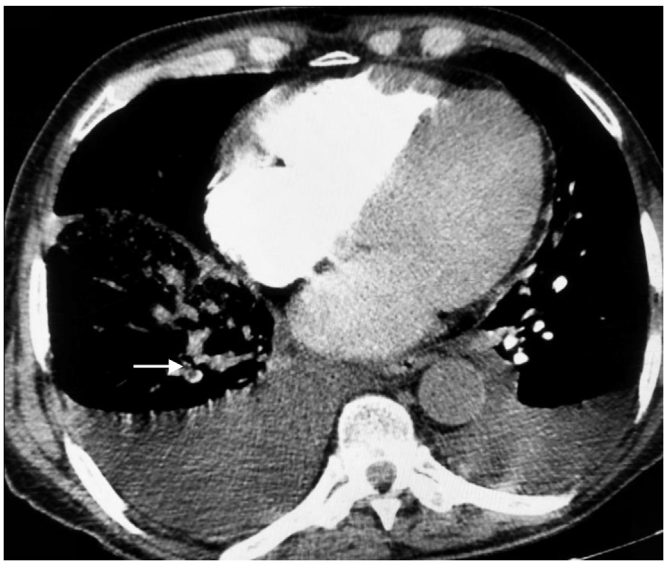

**Fig. 1.** Pulmonary CT angiography shows pulmonary embolism in a right lower lobe segmental branch (arrow).

A thoraco-abdominal computed tomography angiography performed one hour after admission showed a right distal pulmonary embolism (Fig. 1), left ventricular thrombus (Fig. 2) and a thrombosis of the right renal artery with right renal hypoperfusion (Fig. 3).

In addition lung lesions reminiscent of COVID-19 pneumonia and bilateral pleural effusion were also found (Fig. 4). Subsequently the COVID-19 infection was confirmed with real-time reverse transcription polymerase chain reaction (RT-PCR) test. A transthoracic echocardiography has later been performed and showed a dilated cardiomyopathy with severe biventricular dysfunction and a left ventricular ejection fraction of 15% in addition a left ventricular thrombus located in the apex.(Fig. 5). The 12-lead electrocardiogram showed normal sinus rhythm with no sign of myocardial infarction.

Oxygen therapy, anticoagulant and heart failure treatment had been initiated. For the management of heart failure, it was initially based on dobutamine and furosemide then, we introduced progressive doses of bisoprolol in association with the antiviral therapy based on the Moroccan Ministry of Health recommended protocol (hydroxyl-chloroquine with azythromycin); The anti-thrombotic therapy was based on Sodic Enoxaparin 100U/kg (subcutaneous) per day and acenocoumarol 2mg per day; the Enoxaparin was stopped after we got an INR of 2.7. The INR ratio was stable (between 2 and 3) during all the hospital duration.

Later 2 real-time reverse transcription polymerase chain reaction (RT-PCR) tests were performed at Day 10 and Day 11 to test the healing. They were both negatives. After 55 days of hospitalizations, the control shows that LVEG rose to 28% and no echocardiographic signs of a thrombus was found (Fig. 6) and after 3 months from his infection, the echocardiographic control showed an improvement of the LVEF which is now about 40%. For AKI during the hospitalization the GFR improved in 4 weeks, it raised from 23 to 65 ml/min/1.73 $m^2$ with no sign of renal artery thrombus.

## 3. Discussion

Recently some studies related venous and arterial thromboembolic complications in COVID- 19 patients that may reflect the potential hypercoagulability state associated with this infection [11–13,23]. There is growing evidence that COVID-19 may be associated with exaggerated inflammatory response with an abnormal activation of the coagulation system and signs of small vessel vasculitis and microvascular thrombosis. However few cases described multi-organ thrombosis and their management [8]. Our observation reflects pertinently those findings. Several investigational agents are being tested in the management of COVID-19, especially for patients who develop severe disease [20,21,27,34]. As far as its known there is no major interaction between investigational COVID-19 therapies and parenteral anticoagulants [24].

Despite the very limited information on kidney involvement in COVID-19, Acute Kidney Injury (AKI) appears to involve a complex process driven by virus-mediated injury, cytokine storm, AngiotensinII

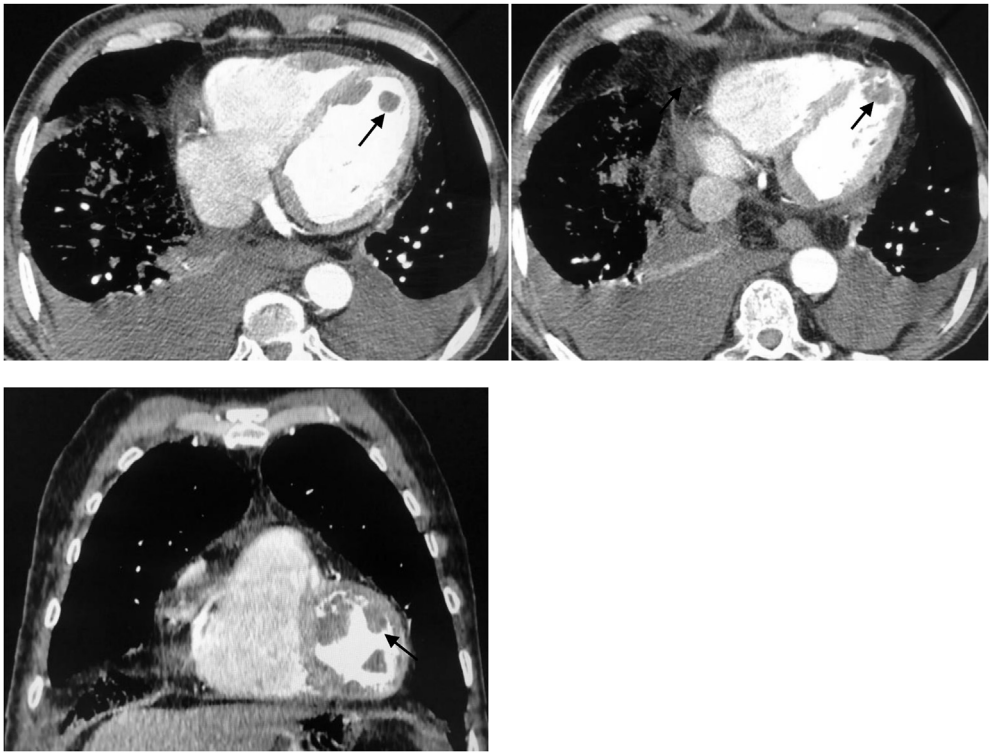

**Fig. 2.** Pulmonary computed tomography angiography images in the axial and coronal planes showing thrombus in left ventricle (arrow).

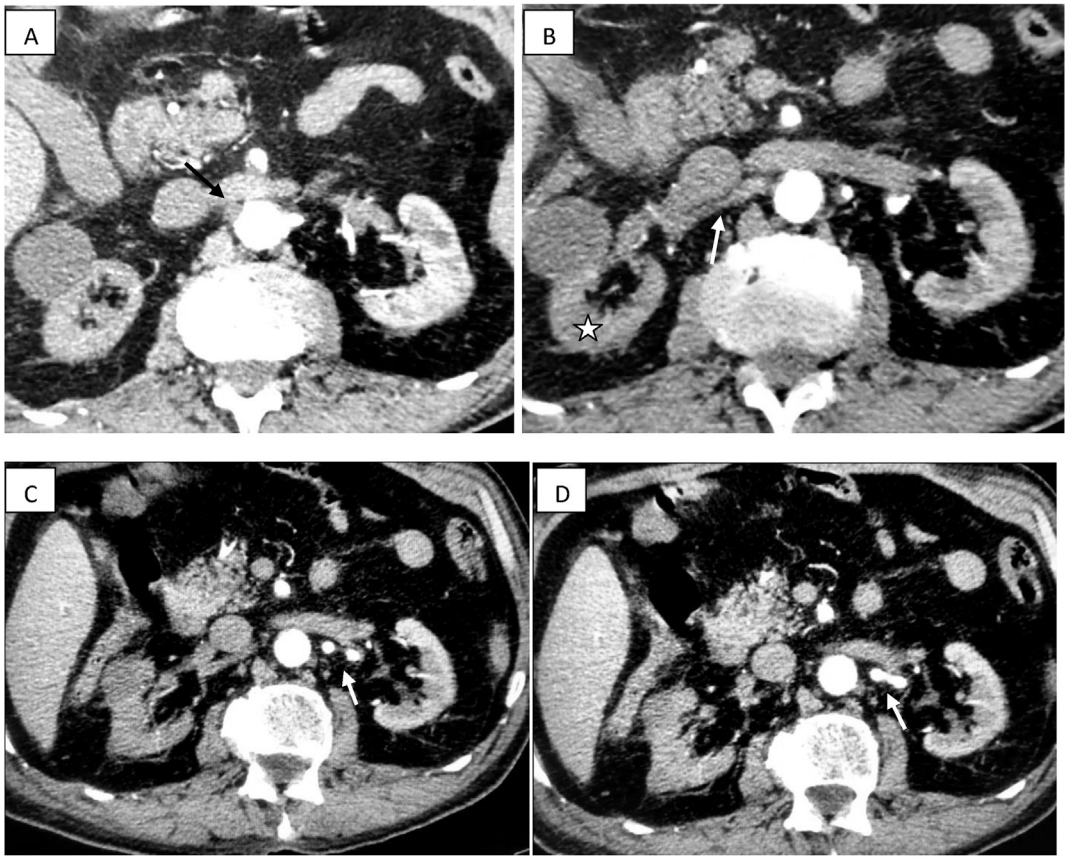


**Fig. 3.** Abdominal CT angiography images in the axial plane show: A. a filling defect in the emergence of right renal artery (arrow), B. a non opacified right renal artery (arrow) and non enhanced right kidney (star), C. and D. Left renal artery well opacified (arrow).

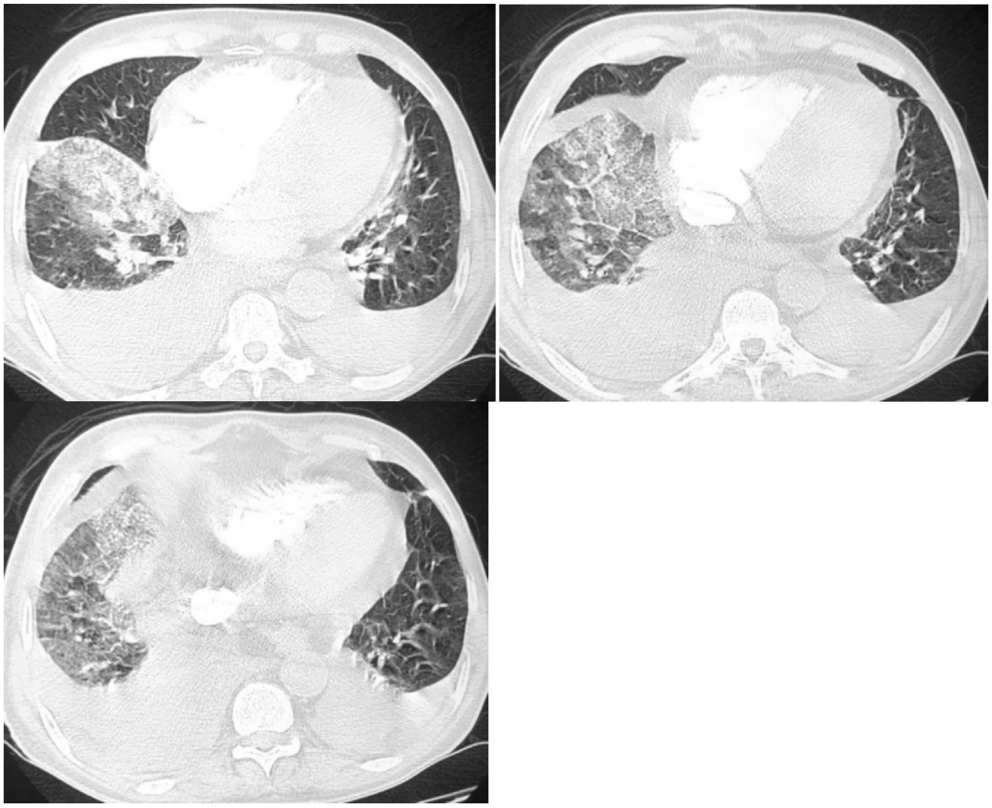

**Fig. 4.** Axial CT scan (lung window) of the chest shows lung damage and involvement due to the coronavirus such as right basal ground-glass opacities associated with crazy paving appearance, in addition to bilateral pleural effusions.

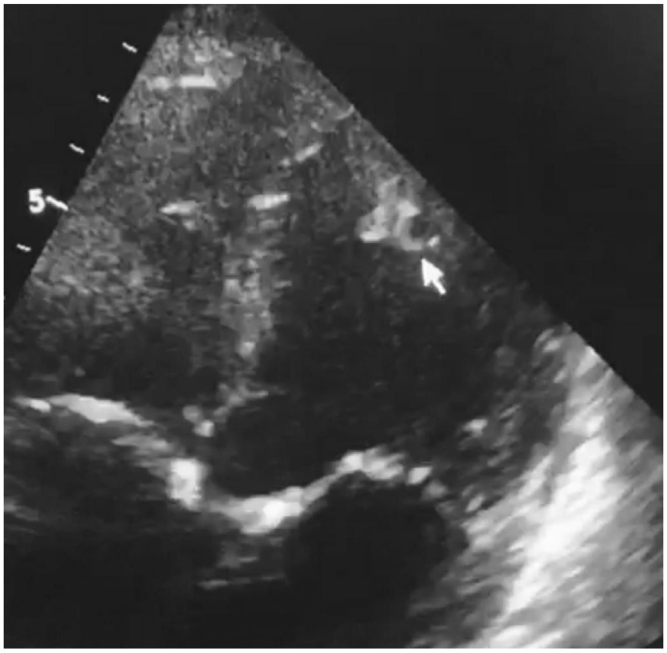

**Fig. 5.** Transthoracic echocardiography shows a voluminous left ventricular thrombus (arrow).

pathway activation, deregulation of complement, hyper-coagulation, and micro-angiopathy interacting with common and known risk factors for AKI in COVID-19 [28]. In our cases many factors were associated with the severity of COVID-19 infection: renal artery thrombus and low cardiac output.

Multiples studies have highlighted the major role of chest computed tomography (CT) scan for identifying the typical pulmonary lesions in COVID-19 cases. Among the published chest CT findings related to the COVID-19 infection, the typical images found are: ground-glass opacities, crazy paving appearance (ground-glass opacities and inter-/intralobular septal thickening), air space consolidation, bronchovascular thickening in the lesion and traction bronchiectasis. Usually ground-glass or consolidative opacities are bilateral, peripheral and basal in distribution [14].

In contrast atypical CT findings are: mediastinal lymphadenopathy, pleural effusion, tree-in- budd, pneumothorax and cavitation [14]. In our case we found most of the typical lesions but the distribution was both central and peripheral. The bilateral pleural effusion noticed may be bind to the cardiac failure. The CT pulmonary angiography is the gold standard investigation for the diagnosis of pulmonary embolism [15]. However it poses some disadvantages including the contrast allergy and the complication of contrast induced nephropathy [16]. The non contrast Magnetic Resonance pulmonary Angiography (MR-PA) could be an alternative tool in diagnosis of pulmonary embolism without radiation exposure or the risk of contrast media complications [17]. For our patient we use a CT angiography for its easy availability in an emergency context.

Multiple cases of acute pulmonary embolism in COVID-19 patients had been reported [1,3,18,19] as well as concomitant acute aortic thrombosis and pulmonary embolism [11]. To our knowledge, none of acute renal artery thrombosis was published. In fact many studies conclude at a strong association between COVID-19 infection and high rate of thromboembolic complications [1,2]. This had led to an increasing use of chest computed tomography angiography on a population already at risk of renal dysfunction [25].

The exact physiopathology of the thromboembolic predisposition in COVID-19 patients is still unknown. Therefore some authors such as Corrado Lodigiani et al. [23] attributed those complications to the activation of the coagulation and the endothelial dysfunction owed to the severe inflammatory response, thus explaining the coagulation and cardiac biomarkers elevation. In fact, those biological abnormalities were found in our case. Furthermore, other studies such as the one realized by Dr Behnood Bikdeli et al. suggest that the hemostatic changes could be

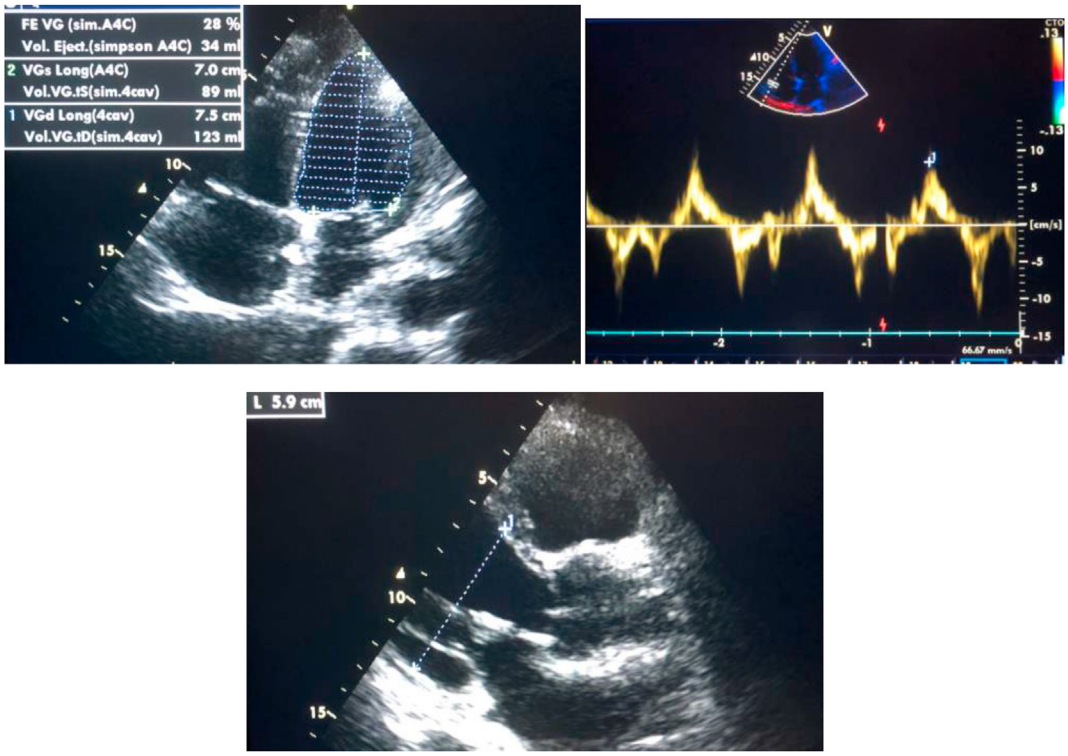


**Fig. 6.** Transthoracic echocardiography showing a biventricular dilatation and the disappearance of the left ventricular thrombus.

related to the exacerbate cytokin production caused by systemic inflammatory response syndrome (SIRS) or to the liver dysfunction [3]. Some others talk about anti-phopholipid syndrome recorded in patients with COVID-19 [22,30,31]. However those biological modifications are also found in other category of patients in intensive care department and are not specific to COVID-19. There is a global consensus that all patients may receive prophylaxis [26,29], many international societies suggest to realize a CT angiography at admission, in order to propose efficient anticoagulant therapy despite thromboprophylaxis in case of concomitant pulmonary embolism [32,33].

## 4. Conclusion

Coronavirus disease (COVID-19) is a serious disease caused by the severe acute respiratory syndrome coronavirus 2 (SARS COV2), clinically and biologically miming a systemic vascularitis with a high rate of thromboembolic complications. We related herein a case illustrating those complications and combining pulmonary embolism, cardiac thrombosis and renal artery thrombosis in a COVID-19 patient. Thereby raising the issue of a systematic use of CT angiography instead of a CT scan of the chest, despite the risk of renal failure. And also the matter of the use of a thromboprophylatic treatment.

## Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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