

Pulmonary Embolism and Internal Carotid Thrombosis in COVID 19 Infections

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Abstract

Infection with the SARS-Cov-2 virus induces an unusual pre-thrombotic state, of multifactorial origin, responsible for a high incidence of venous thromboembolic events but also of arterial thromboses which have a negative impact on the prognosis of patients. Knowledge of the mechanisms of thrombosis formation has improved significantly since the onset of the pandemic pending appropriate protocols for preventive and curative anticoagulant therapy during COVID 19, emerging from randomized controlled clinical trials. We report the case of a young patient hospitalized with a moderate form of COVID 19 who presented with pulmonary embolism and acute ischemic stroke related to internal carotid thrombosis.

Keywords: COVID 19, pulmonary embolism, ischemic stroke, internal carotid thrombosis.

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INTRODUCTION

The SARS-Cov-2 virus is a single-stranded RNA virus of the coronaviridae family responsible for the covid 19 pandemic. Respiratory signs are in the foreground with a potential development in acute respiratory distress syndrome, in addition to a trait systemic and hypercoagulable confirmed of this disease causing venous and arterial thrombosis especially in severely affected patients.

OBSERVATION

This is a 48-year-old patient who has a chronic smoking as a cardiovascular risk factor. No particular history. He reported, a week before his admission, frontal headaches with cough and slight difficulty in breathing in a context of fever and asthenia. On physical examination, the patient presented an altered general condition without dyspnea at rest with a temperature of 39 ° and Oxygen saturation at 94% in

ambient air. The PCR test for the SARS Cov 2 virus came back positive and the chest computed tomography showed bilateral and diffuse ground-glass opacities classified as CORADS 5 (Fig. 1), supplemented by a thoracic CT angiography which revealed a marginal thrombosis of the branches of lobar divisions of right pulmonary artery (Fig. 2). In addition to treatment for covid 19, the patient received a curative dose of anticoagulation with low molecular weight heparin (LMWH) (Enoxaparin 60 mg subcutaneous every 12 hours). On the 3rd day of his hospitalization, he presented a right sensitivomotor deficit related to an acute left sylvian ischemic stroke confirmed by cerebral CT (Fig. 3). As part of the aetiological assessment, supra-aortic trunk ultrasound revealed a thrombosis of the proximal part of the left internal carotid artery floating in the bulb responsible for a complete occlusion (Fig. 4). Aspect confirmed by CT angiography of supra-aortic trunk (Fig. 5).

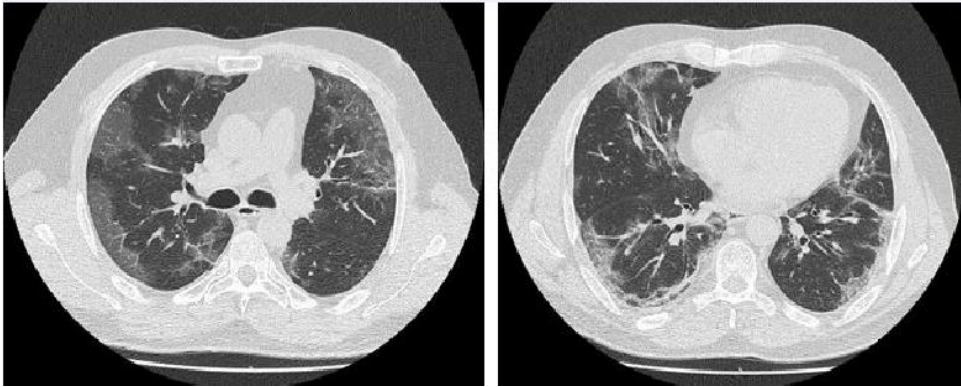


Fig-1: Chest CT: bilateral and diffuse ground-glass opacities classified as CORADS 5



Fig-2: Thoracic CT angiography: marginal thrombosis of the branches of lobar divisions of right pulmonary artery

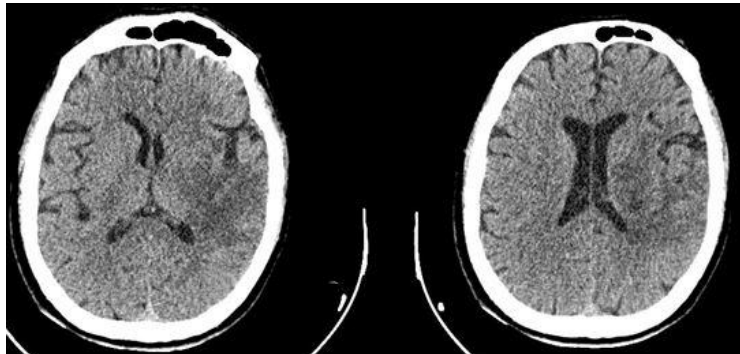


Fig-3: cerebral CT: acute left sylvian ischemic stroke

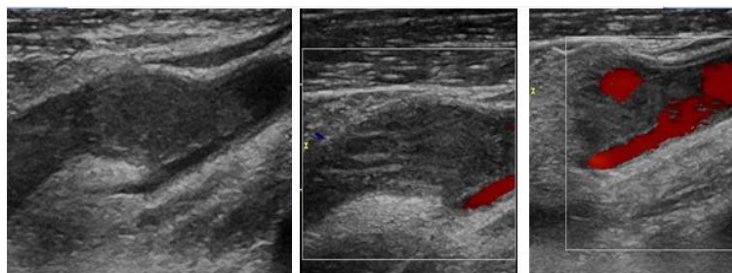


Fig-4: Echodoppler of the supra-aortic trunks (2D and color mode) objectifying a thrombosis of the proximal part of the left internal carotid artery floating at the bulb



Fig-5: CT angiography of supra-aortic trunks confirming the complete occlusion of the left internal carotid artery

DISCUSSION

Since the start of the covid 19 pandemic, there has been a strong thrombotic trend among patients with the SARS-Cov-2 virus. These are not only venous thrombosis, but also arterial thrombosis, hemodialysis filters and extracorporeal membrane oxygenation equipment [1].

The increased proportion of venous thromboembolic events (VTE) is explained on the one hand by the presence of risk factors making up the Virchow triad (stasis related to bed rest and obesity, parietal damage by endothelial inflammation and hypercoagulability due to sepsis), and on the other hand, endothelial activation and thrombogenic inflammation (increase in Von Willebrand factor and factor VIII) due to the virus itself [2].

Indeed, SARS-Cov-2 possesses the ability to penetrate inside host cells via the interaction between its transmembrane glucoprotein and its functional angiotensin converting enzyme 2 (ACE 2) receptor expressed in endothelial cells. This results in an intense local inflammation responsible for endotheliitis, leading to the formation of extensive immunothrombosis in the pulmonary vascular bed [3].

Sepsis secondary to covid 19 causes a massive and sustained release of proinflammatory cytokines, also called “cytokine storm”, responsible for endothelial dysfunction that causes an imbalance between procoagulant and fibrinolytic factors in favor of thrombosis [4].

The SARS-Cov-2 virus can also be the cause of ischemic stroke. On the one hand, it can cause direct damage to the central nervous system, by hematogenous propagation or retrograde invasion via ACE2 receptor also expressed by nerve cells [5, 6]. On the other hand, inflammation affects atherosclerotic plaque vulnerability and promotes a thrombogenic environment [7]. The fact that all patients presented with involvement at the carotid bifurcation may be due to its pre-existing propensity for atherosclerosis and

turbulent flow; the latter can damage the endothelium and lead to platelet aggregation.

Among the recently advanced mechanisms we find the notion of cerebral vasculitis. In fact, anti-phospholipid antibodies have been shown to be positive in patients severely infected with SARS-Cov-2 and presenting with multiple ischemic stroke [8, 9].

Thromboprophylaxis with LMWH, unfractionated heparins or fondaparinux should be routine in all patients hospitalized for covid 19, as several studies have found a decrease in mortality in patients who received anticoagulation during hospitalization for covid19 in comparison with those who did not receive it [10, 11]. Some authors report the occurrence of VTE in patients hospitalized for covid 19 despite preventive anticoagulation. Thus, it seems that VTE associated with covid 19 have specific risk factors such as male sex, the time between the onset of symptoms and hospitalization, the absence of preventive anticoagulation and a marked inflammatory syndrome [12, 13].

CONCLUSION

Infection with the SARS cov2 virus or covid 19 diseases is an aggressive endothelial disease that provides a unifying pathophysiological picture and also a framework for a rational treatment strategy, at a time when we have a modest evidence base to guide our therapeutic attempts and face this pandemic.

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