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A case of Covid-19 revealed by extensive portomesenteric thrombosis

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Abstract

Covid-19 is an inflammatory disease primarily affecting the respiratory system, but can affect multiple organs causing multiple organ failure. Thrombosis is a major prognostic complication during SARS-Co-2 infection. We report the case of a young patient consulting for an acute febrile abdomen, whose abdominal CT scan showed extensive porto-mesenteric thrombosis. A systematic Covid-19 PCR test coming back positive. The assessment objectified a positive JAK2 mutation. This reflects the interest of broad screening for SARS-Cov-2 infection, by PCR test and initial imaging aimed at looking for thromboembolic disease, and of a comprehensive etiological approach.

Keywords: SARS-Cov-2 infection, Thromboembolic disease, PCR Covid-19, systematic screening, Extensive thrombosis, mortality, JAK2.

Introduction

SARS-Cov-2 infection is an emerging virus that appeared in December 2019 in Wuhan, China [1].The reference diagnostic method remains laboratory research for viral RNA by PCR-test using nasopharyngeal swabs, with good specificity but less sensitivity at 60-70% [2].

Although the respiratory picture of Covid-19 is in the foreground, extra-pulmonary manifestations have been reported, especially in the cardiovascular system. Indeed, an abnormally high incidence of venous thromboembolic events has been observed [3].

We report the observation of a young patient consulting for intense abdominal pain and whose etiological investigation revealing porto-mesenteric thrombosis on

SARS-Cov-2 infection and essential thrombocythemia.

Patient-Observation

It is about 39 years old Mrs. CH, married and mother of two children, having as antecedent a medicalized delivery by vaginal route at term of a stillbirth, with continuations of simple childbirth.

On questioning, the patient had no idea of taking medication, in particular oral contraceptives; no known autoimmune disease or neoplasia.

She had consulted in the emergency room for diffuse abdominal pain, intense, evolving for 5 days associated with a cessation of materials and vomiting in a context of fever not quantified. The

clinical examination found an abdominal defense, with a finger cot clean to the digital rectal examination.

The initial biological assessment showed an inflammatory syndrome (hyperleukocytosis at 28000/mm^3 predominantly PNN at 19610/mm^3, thrombocytosis at 614000/mm^3, microcytosis at 73 with ferritinemia at 367 and CRP at 177 mg / l), cholestasis at twice normal without cytolysis, correct renal function and hypokalaemia.

An abdomino-pelvic CT revealed intestino-mesenteric ischemia of venous origin with thrombosis of the portal vein, its branches, the superior mesenteric vein, the spleno-mesaraic trunk and the splenic vein with suffering from the small intestine and peritoneal effusion; without surgical indication.

In this epidemic context and as part of the systematic screening adopted by our service given the fragility of our patients, a PCR test on a nasopharyngeal swab was carried out with a positive result. A chest CT without injection of contrast product showing an aspect of a viral pneumopathy of the COVID-19 type with minimal impairment classified as CoRADS 6.

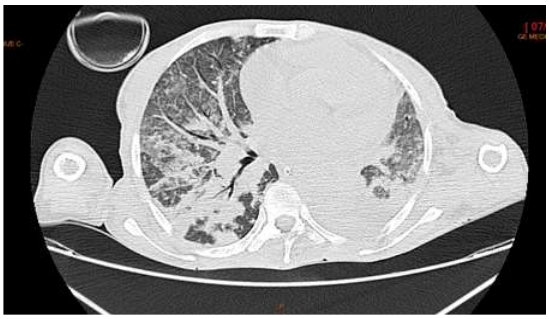

Figure 1: CT image showing areas of ground glass "Crazy paving", foci of pulmonary parenchymal condensations. CoRADS-6.

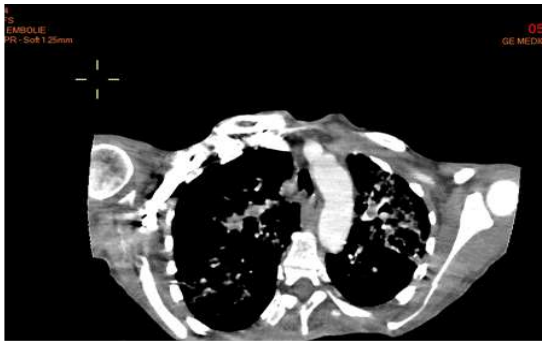

Figure 2: CT image in favor of right upper lobar segmental bronchial pulmonary embolism.

Before young age and extent of thrombosis; an exhaustive etiological assessment was necessary: the immunological assessment (AAN, ANCA and APL), infectious (viral serologies, Quantiferon), paraneoplastic and thrombophilia were negative. Celiac disease and HLA B51 antibodies also negative. An electrophoresis of normal serum proteins, 24-hour proteinuria negative and a bone marrow biopsy revealing amedullary hyperplastic mainly involving the megakaryocytic lineage**.**

The patient received an anticoagulant treatment at a curative dose and the Covid-19 treatment protocol composed of Hydroxychloroquine (200mg 1cp ^{x} 2 / d for 10d), Azythromycin (500mg 1cp on the 1st day then ½ cp per day for 6d), vitamin C (1g 3^{x} / day for 7 days) and Zink (500mg 1cp / day for 7 days).

The evolution was initially favorable marked by apyrexia, decrease in pain, disappearance of vomiting, normalization of his blood count and negativation of CRP.

Thirty-six days later, a patient admitted to the intensive care unit in a state of hypovolemic shock following uncontrollable vomiting. Biologically, the patient had hyperleukocytosis at 22320 / mm^3 still predominantly PNN (18680 / mm^3), thrombocytosis at 597000 / mm^3 with fibrinogen at 4.6g / l, very positive D-dimers, renal failure of functional appearance, and a CRP of 183 mg / l. The patient is put on filling and probabilistic antibiotic therapy with slight improvement.

A TAP angiogram is performed, revealing a right superior lobar segmental bronchial pulmonary embolism, an infectious alveolo-interstitial lung disease (Figure 1,2), with a portal cavernoma and the absence of visualization of the portal trunk, the superior mesenteric vein and the spleno-mesaraic trunk. .

The patient died two days later from acute respiratory distress syndrome. The JAK2 mutation, recovered later, came back positive. The diagnosis of essential thrombocythemia is retained.

Discussion

Coronavirus disease 2019 (COVID-19) is a viral infection caused by “Severe Acute Respiratory Syndrome coronavirus 2” (SARS-CoV2). SARS-CoV2 is a single-stranded RNA virus that enters the body's cells via the angiotensin converting enzyme receptor. This receptor is widely expressed in particular in pulmonary alveoli and vascular endothelium [4]. Along with respiratory infection, the pro-thrombotic potential of the virus has been the subject of case descriptions or case series [5]. In a Chinese study, carried out on 48 patients hospitalized for a severe form of COVID-19, whose systematic screening by venous doppler ultrasound of the lower limbs, made it possible to objectify an incidence of occurrence of deep vein thrombosis in 85.4% of patients [3].

According to the studies published to date, the coagulopathy associated with COVID-19 appears to be different from that conventionally observed during sepsis and results in a higher prevalence of prothrombotic events [1]. Many phenomena are mentioned and probably entangled in coagulopathy linked to COVID-19, including an excess of inflammation linked to the massive release of cytokines, activation of platelets, vascular stasis and endothelial dysfunction. Pre-existing comorbidities also form the bed for thrombotic events [6], but the prevalence of these classic risk factors for venous thromboembolic disease (VTE), such asthat active neoplastic disease, venous insufficiency, a history of thrombophilia, a history of pulmonary embolism or deep vein thrombosis, is rare [7,8]. On the contrary, age, male sex, obesity, the time between the onset of symptoms and hospitalization, hyoxemia, the extent of the inflammatory syndrome, the presence of catheters for the most severe forms, as well as the absence of preventive anticoagulant treatment during the disease constitute specific factors of the VTE associated with COVID-19 [3,8].

The exact pathophysiological mechanisms leading to the pro-thrombotic phenotype during SARS-Cov-2 infection remain unknown and difficult to clarify with certainty [1]. If we take again the Virchow triad describing the mechanisms of venous thrombosis, three factors can be brought into play: hypercoagulability, endothelial aggression and venous stasis. Infection with SARS-CoV-2 cause’s lung damage, mainly described as diffuse alveolar damage. Hypoxemia occurs in severe damage. In response to hypoxemia, there is an induction of the “hypoxia inducible transcription factors” signaling pathway which helps to activate coagulation, suppress fibrinolysis and inhibit natural circulating anticoagulants. At the same time, the infection causes recruitment of mononuclear cells at the level of the alveolar-capillary barrier. These, following the induction of the NF-κB pathway, will secrete pro-inflammatory cytokines (TNF-α, IL-1 and IL-6, 2 and 7, G-CSF, IP10, MCP1, MIP1A) which will promote the release of the plasminogen activator inhibitor (PAI-1) and the inhibition of natural anticoagulants. Also, they promote the activation of coagulation by the generation of tissue factor. This activation is supported by platelet activation. Activation of the endothelium, secondary to specific viral damage and/or complement activation, promotes coagulation and interaction with circulating platelets. Eventually, fibrin, red blood cells and platelets aggregate, producing a fibrino-cruoric thrombus [1,9].

Biologically, an increase in fibrinogen and D-dimers makes it possible to identify patients at high risk of thromboembolic complications. Thus, the GIHP retained the thresholds for fibrinogen> 8 g / L and D-dimers> 3 g / L to define this high risk. In view of these elements, the GIHP recommends monitoring the following hemostasis parameters at least every 48 hours: platelet count, prothrombin time (PT or PT),

TCA, fibrinogen, and D-dimers [4,10]. The presence of the lupus anticoagulant in some patients is also frequent [11].

The association of venous and arterial thrombotic complications with SARS-Cov-2 infection is now well established [12], with an increased risk of mortality [13]. In view of the high frequency, the severity of the thrombotic complications and the clinical polymorphism of the SARS-Cov-2 infection, it would be advisable to have a complete reference imaging at the admission allowing to look immediately for thrombosis ; to improve management and prognosis [1,2]. Anticoagulation is now one of the essential prophylactic and curative therapeutic means for SARS-Cov-2 infection (Figure 3).

In our observation, we note that the patient was initially respiratory stable (no hypoxemia), had an inflammatory syndrome with positive D-dimers at 1.46 and Fibrinogen levels reaching up to 6g / dl. All of these make her at risk for VTE. In addition to the lack of improvement despite appropriate treatment and curative anticoagulation, we are encouraged to adopt a broad and well-established etiological strategy, particularly in young subjects without comorbidities. The etiological assessment in our patient had demonstrated an essential thrombocythemia (unfortunately after death), for which cytoreductive therapy is necessary, given the active thrombotic event [14].

Conclusion

This observation shows that Covid-19 is no longer a virosis with respiratory tropism alone. However, it can present as an inflammatory disease, cause multivisceral damage and cause extensive thrombosis; hence the interest of screening by nasopharyngeal PCR in front of any atypical picture. Besides, thromboembolic events during SARS-Cov-2 infection do not necessarily occur in patients with a severe form of the infection, but seem associated with a poor prognosis. The search for a predisposing ground is necessary in particular in the face of extensive or repeated thrombosis.

Conflicts of interest: The authors declare that they have no link of interest

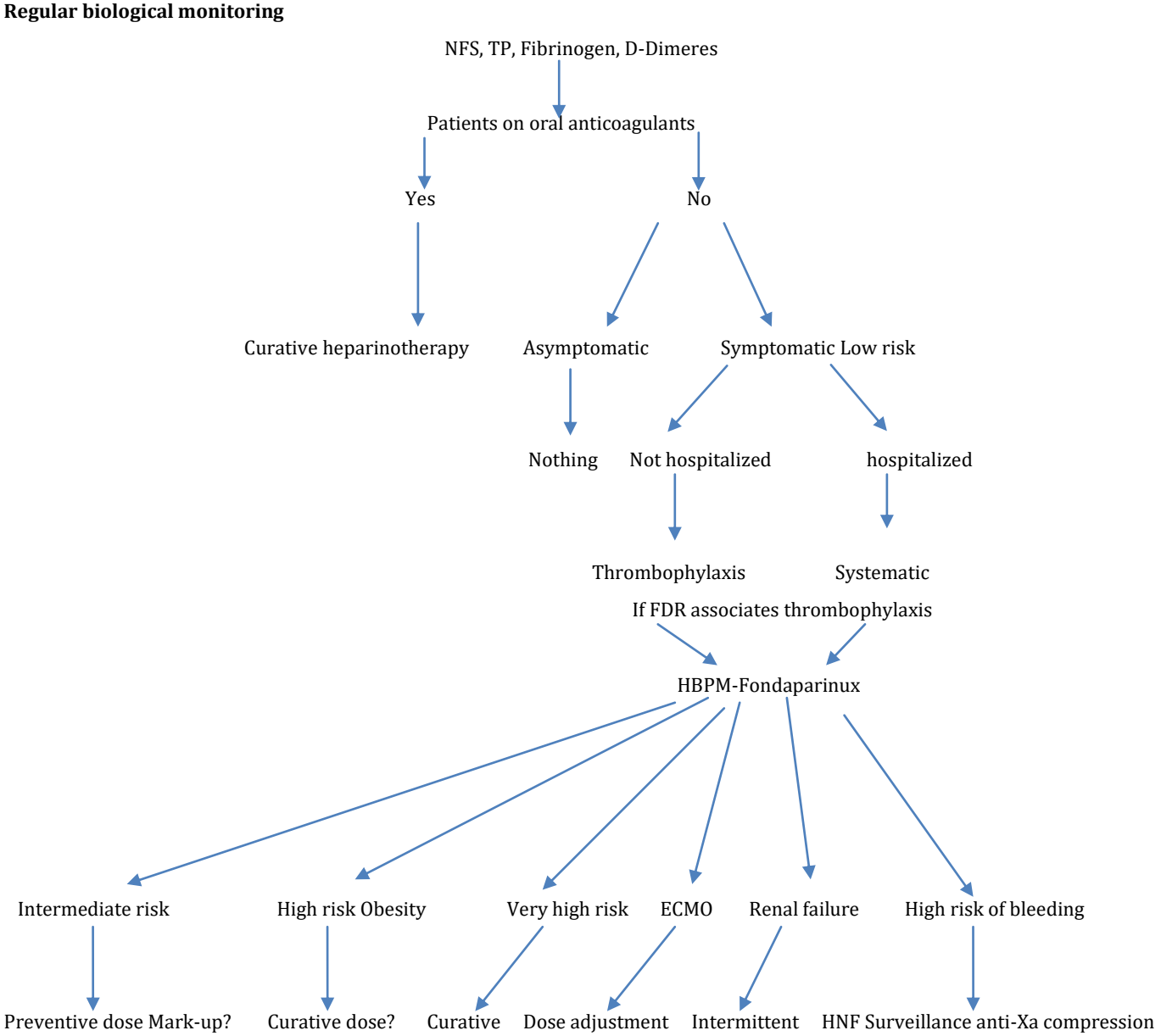

Figure 3: Thromboprophylaxis algorithm during SARS-CoV-2 infection [1]

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