Aortic thrombosis associated with COVID 19 pneumonia

Thrombose de l'aorte associée à une pneumopathie COVID 19

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RÉSUMÉ

La maladie à coronavirus 19 (COVID 19) prédispose aux évènements thrombotiques veineux et artériels, en particulier chez les patients sévères. Il a été rapporté des cas de patients à faible risque dont l'évolution a été compliquée par une thrombose artérielle. Nous rapportons le cas d'une femme de 53 ans qui a présenté une détresse respiratoire aigüe due au COVID 19 avec thrombose de l'aorte descendante. L'évolution a été favorable sous traitement anticoagulant.

Mots clés : COVID19, thrombose de l'aorte, traitement anticoagulant

Abstract

Coronavirus disease 19 (COVID-19) predispose to both venous and arterial thrombotic events, especially in severe patients. There are reports of lowe risk patients whose courses are complicated by arterial thrombosis. We report the case of 53 year-old woman who presented with severe acute respiratory syndrome due to COVID-19 with descending aortic thrombosis. The evolution was favorable under anticoagulant treatment.

Keys words : COVID 19, aortic thrombosis, anticoagulant therapy.

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INTRODUCTION

Coronavirus disease 19 (COVID-19) could predispose to both venous and arterial thromboembolism, in an exaggerated immune response to the virus, especially in severe patients (1). We report the case of a low risk patient who presented with a severe acute respiratory syndrome due to COVID-19 with descending aortic thrombosis. The evolution was favourable under anticoagulant treatment.

CASE REPORT

A 53 year-old woman presented with 2 weeks of cough and shortness of breath.The patient had a history of diabetes and arterial hypertension. A nasopharyngeal swab specimen tested positive for SARS-CoV-2 by reverse transcription-PCR. On presentation she was afebrile, blood pressure was 138/67, tachycardic with a pulse of 100/min, tachypeic at 30 breaths /min and hypoxic at 87% breathing room air. Body Mass Index (BMI) was 29. Supplemental oxygen via high concentration oxygen mask was initiated at 14l/minute with improvement of saturation to 95%.

Initial laboratory investigations revealed : white blood 10500 cell/ml, lymphocytes 1500 cell /ml, hemoglobin 10.2 g/dl, platelet 360.000 /ml, D-dimer 1186 ng/ml, troponin I 2.41 pg/ml, creatinine 42 mg/dl, Lactate dehydrogenase LDH (U/L) : 400, Fibrinogen : 3.34g/l and international normalized ratio 1.08. Electrocardiogram showed a sinus rhythm.

Computed tomography (CT) thorax angiography showed a severe COVID 19 related bilateral ground-glass pattern lungs (50-75%) without signs of pulmonary embolism. Moreover, CT angiography revealed a non-obstructive descending aortic thrombus formation (14mm-22mm), with no evidence of atherosclerosis or vessel wall abnormality at the side of the aortic thrombus. Treatment with low molecular weight heparin at curative dose was initiated.

An etiological investigation has been started. Cholesterol level was 4.95mmol/l and triglyceride was 3.2 mmol/l. A transthoracic echocardiogram did not reveal any source of potential embolism. The abdomino-pelvic and cerebral CT was normal. Immunological assessement was normal. Tumors markers were negative. Thrombophilia testing including cardiolipin, beta-2glycoprotein-1 antibodies and lupus anticoagulant, Factor V Leiden gene mutation, prothrombin gene G202010A mutation; protein C, protein S, and antithrombin deficiencies; increased homocysteine; and methylenetetrahydrofolate reductase (MTHFR) was normal.

The patient's oxygenation improved after 12 days, and she no longer required supplemental oxygen. Follow-up CT aorta 3 weeks later showed resolution of the thrombus (Figure 2). The patient was discharged home with prescription for acenocoumarol (target INR 2.0–3.0) for a scheduled duration of at least 3 months.

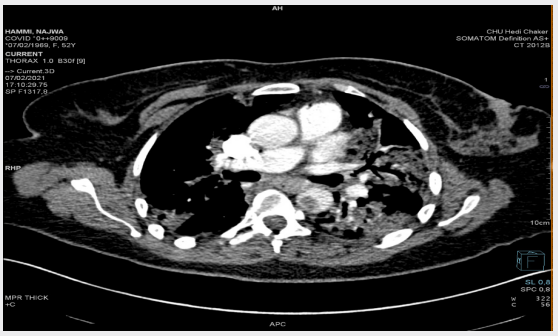

Figure 1 : CT thorax angiography shows bilateral ground-glass pattern lungs and a descending aortic thrombus formation

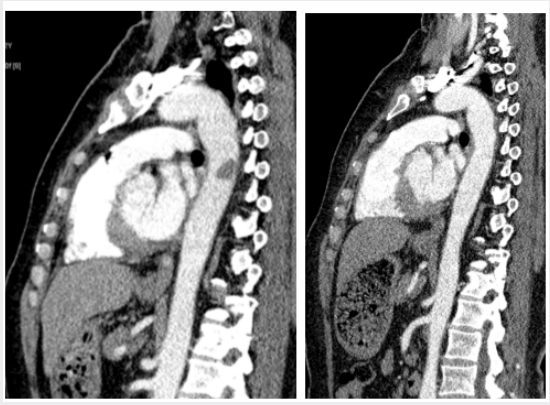

Figure 2 : CT thorax angiography (before and after anticoagulant treatment**) :** Resolution of thrombus.

DISCUSSION

An aortic thrombus is an uncommon condition even in common hypercoagulability states as sepsis, polycythemia, disseminated intravascular coagulation, autoimmune diseases, pregnancy, and cancer (2). Normally, acute arterial thrombosis arises in the setting of arterial atherosclerosis or cardiac diseases. With the novel coronavirus, patients can suffer from thrombosis in the absence of traditional risk factors for acute thromboembolism (3).

We have described the case of a 53- year-old patient with a severe form of COVID19, who presented acute aortic thrombus. There are reports of low-risk patients whose courses are complicated by arterial thrombosis (4). Cardiovascular risk factors of our patient were diabetes and hypertension. In our case, there was no evidence of atherosclerosis or vessel wall abnormality at the side of the aortic thrombus.

Microvascular thrombosis is characteristic of severe COVID-19 and it has been proposed that a virus-induced prothrombotic state culminates with hyper-inflammatory effectors and platelets to form immunothrombotic clots (5,6). Reports of microangiopathic complications in severe COVID-19—including disseminated intravascular coagulation (DIC), venous thromboembolism, and pulmonary embolism—support a role for immunothrombosis in viral pathogenesis (7).

Our patient was relatively young compared to other case reports described in the literature (8). The symptoms were not specific, and the aortic thrombosis was secondarily diagnosed in CT pulmonary embolism screening. Abnormal coagulation and inflammation parameters provide evidence for endothelium dysregulation in severe COVID-19 patients. Prolonged clot formation indicated by an elevated prothrombin time, as well as increased D-dimers, fibrin degradation products, and ferritin levels are all correlated with hypercoagulation (9). Deleterious inflammation is indicated clinically by an elevation in C-reactive protein and interleukin-6, both of which are associated with increased disease severity (9). In ou case, there was no evidence of inflammation parameters except elevation of D-dimers.

In several cases, the aortic thrombosis was complicated by limb ischemia requiring a surgical treatment (8). In our case, treatment with heparin was effective for disappearance of the aortic thrombus and for preventing new thrombi formation.

CONCLUSION

In summary, in this case, a 53-year-old woman presented with severe acute respiratory syndrome due to COVID-19 with descending aortic thrombosis. The evolution was favourable under anticoagulant treatment.

Thromboembolic complications should be suspected and screened in patients with severe COVID 19 forms.

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