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# Sars-Cov-2 Virus Coinfection and Mycobacterium Tuberculosis-A Case Report

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***Abstract***

*The SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2) infection, called COVID-19 (coronavirus disease 2019) started in December 2019 in the Wuhan, region of China. It is the cause of life threatening pneumonia and has spread globally to the point of becoming a pandemic. Alveolocapillary barrier alteration secondary to cytokine storm and immunosuppression, induced by SARS-CoV-2 can reactivate dormant bacterial infections. COVID-19 and tuberculosis coinfection is under-studied. We report here the case of a patient with respiratory failure linked to Covid-19 and who was diagnosed with pulmonary tuberculosis.*

***Keywords:*** *COVID-19, SARS-COV-2, Tuberculosis, Mycobacterium Tuberculosis, Intensive care.*

## Introduction

In December 2019, the SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2) infection, called COVID-19 (coronavirus disease 2019), started in the Wuhan, region of China. It has spread globally with 4,444,670 people declared infected and 302,493 deaths. (May 2020).

The World Health Organization (WHO) announced COVID-19 as an international public health emergency on January 30, 2020 and as a pandemic on March 11, 2020 [2].

Unlike COVID-19, tuberculosis is a long standing infection that about 10 million people contracted tuberculosis in 2018, with 1.2 million deaths [6].

A new study published on bioRxiv proposed in May 2020 that the long-term shock of the virus could include the activation of dormant bacterial infections like tuberculosis [3]. The characteristics of COVID-19 and tuberculosis coinfection are unclear and under studied.

## Medical Observation

60-year-old male patient with a history of: BPCO, poorly controlled hypertension, type 2 diabetes under insulin, bedridden for 6 months following an ischemic stroke and operated on for a fracture of both arms following a stroke. Declared Covid19 after a positive PCR carried out following symptoms of dyspnea, cough and deterioration of general condition.
On admission;

- Neurologically: drowsy (Glasgow Coma Score at 13/15), symmetrical and reactive pupils, right hemiplegia, central facial palsy, aphasia and dysarthria.
- On the respiratory level: Eupneic, 92% oxygen saturation in the open air, PAO2/FIO2=211 and bilateral crackling rales predominantly at the bases on pleuropulmonary auscultation.
- On the hemodynamic level: normocardium 100 bpm/min, normotension at 14/7.
- Apyretic.
- Dehydration fold.

A brain CT was performed showing a subacute left parietal ischemic stroke (sylvian territory). (Figure 1).

**ICONOGRAPHY:**

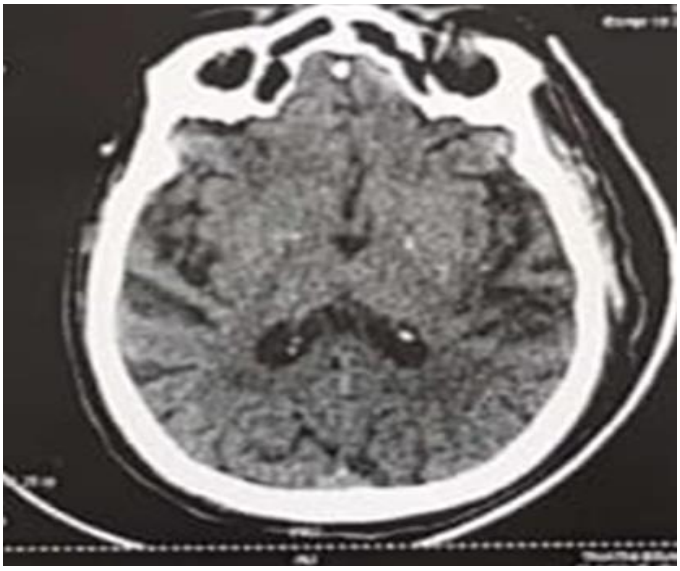

**Figure 1:** Cerebral tomography.

A chest CT was done revealing extensive to severe COVID-19 viral lung disease, an excavated lesion in the left Fowler whose infectious or tumor origin cannot be identified, as well as multiple mediastinal lymphadenopathy. (Figure 2).

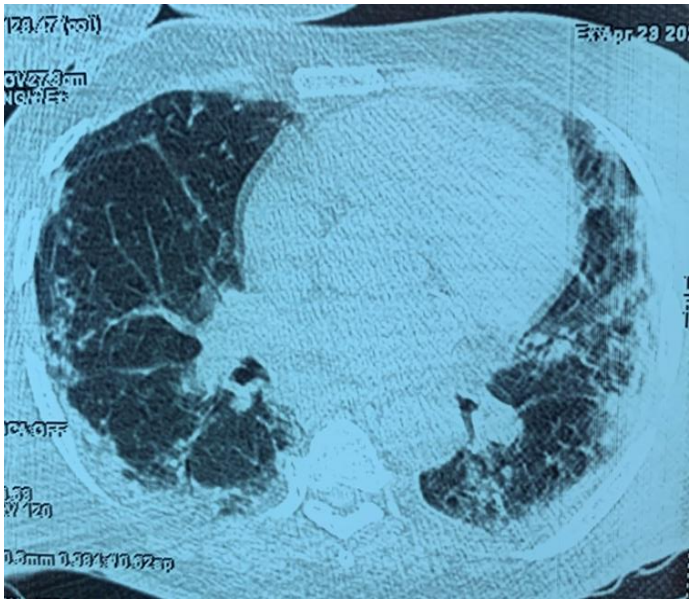

**Figure 2:** Thoracic tomography

Seen the excavated lesion revealed on thoracic CT, sputum BKs were performed showing the presence of BAAR (+). Biological assessment: Hb at 11.2 g/dl, GB at 9460/µL, PNN at 8080/µL, lymphocytes at 660/µL, fibrinogen at 7.47 g/l, D-dimers at 1490 µg/l, urea at 0.34 g/l, creatinine 5.1 mg/L, ASAT at 29 IU/L, ALAT at 14 IU/L, albumin at 19 g/L, ferritin at 600 ng/ml, CRP at 138.3 mg/L, NT- Pro BNP at 749 pg/ml and troponin at 13.30 ng/L.

A transthoracic ultrasound was done revealing a dilated left ventricle site of overall hypocontractility with FE at 25-30%, elevated PRVG, a slightly dilated left atrium, retained RV function, IM grade I, IT grade I, RV retained, uncompliant IVC at 19mm, dry pericardium, no thrombus visualized.

A mask-based oxygenation at high concentration was initiated with a treatment based on:

- Azithromycin, Hyroxychloroquine, 3rd generation cephalosporin and quinolone.
- Corticosteroids.
- Effective anticoagulation at curative dose.
- Vitamin supplementation
- Treatment of heart failure: an ACE inhibitor, a beta blocker, a statin, an antiplatelet agent and a diuretic.
- Antibacillary treatment: 2RHZE / 4 RH.

The evolution was marked by marked clinical as well as biological improvement.

## Discussion

The pathogenic mechanism of the immune response against the Sars-Cov-2 virus in the pulmonary alveoli is as follows [1]:

- The virus infects alveolar epithelial cells; mainly type 2 pneumocytes in lung tissue, thanks to its anchoring on a receptor.
- The destruction of lung cells where the coronaviruses have proliferated by replication of their RNA, and the increase in their cell permeability, results in the release of viruses.
- There is an awakening of the innate immune system whereby the host's defense agents (macrophages, lymphocytes, monocytes, and granulocytes) not only start capturing the virus but also produce a discharge of chemokines and cytokines. An appropriate immune response is also activated by cells presenting antigens or viral particles, mainly dendritic cells. T and B lymphocytes are thus able to ensure the production of IgM and IgG antibodies and the secretion of inflammatory cytokines, directly or indirectly, through mediators or signals.

Compared to humoral responses, there is more research on cellular immunity to coronaviruses. The latest report shows that the number of T CD4+ and CD8+ cells in the peripheral blood of patients infected with SARS-CoV-2 is significantly reduced [2]. The alteration of the alveolocapillary barrier secondary to cytokine storm and immunosuppression, induced by SARS-CoV-2 can reactivate dormant bacterial infections, such as tuberculosis [3].

In fact, the risk of tuberculosis is 2 to 5 times higher in case of early infection with HIV-1 and more than 20 times higher in case of advanced disease with HIV-1. HIV-1 infects CD4 + T cells and macrophages. Mycobacterium tuberculosis primarily infects macrophages, which require CD4 + T cells to increase the intracellular clearance of microbial pathogens. They therefore believe that the depletion of CD4 + T cells associated with HIV-1 infection plays a major role in increasing the risk of tuberculosis in people infected with HIV-1 [10].

The Russian (1889) and Spanish (1918-1920) influenza pandemic led to an increase in the number of cases of lung tuberculosis. The highest death rate was in the patient subgroup, who had the flu with tuberculosis [7,8,11].

The H1N1 influenza pandemic (2009) also showed the same trend, an increase in the number of cases of pulmonary tuberculosis with the presence of multidrug-resistant strains of mycobacterium tuberculosis [9].

As with our patient, tuberculosis developed after the patient contracted SARS-Cov-2, most likely as a result of reactivation of a previous infection or a new infection with mycobacterium tuberculosis, while he was temporarily immunocompromised.

Tuberculosis in patients with SARS-Cov2 has been reported on rare occasions. In a Chinese study, three pulmonary tuberculosis patients with COVID-19 were followed prospectively from hospital admission to discharge.

In this study, elderly patients with tuberculosis easily progressed to the severe type of COVID-19 and had a long healing process. These case reports remind us of the possibility of coinfection with tuberculosis in patients with COVID-19 whose recovery is incomplete, as well as the importance of prudent use of steroids in the management of their case [12].

The results of a Spanish study describing a group of patients who died from tuberculosis (active disease or sequelae) and COVID-19 in two cohorts, show that mortality is likely to occur in elderly patients with comorbidities, tuberculosis may not be a major determinant of mortality, and in settings where advanced forms of tuberculosis frequently occur and are caused by drug-resistant strains of mycobacterium tuberculosis, high rates can be expected mortality rates among young people [5].

Current directions for studying dormant tuberculosis show that mycobacterium tuberculosis is largely found in stem cells called CD271+ BM-mesenchymal (CD271+ BM-MSCs). These cells can live in the bone marrow and the lung. Using a strain-1 murine hepatitis virus (MHV-1), which is a murine coronavirus capable of representing the clinical characteristics of SARS-CoV-2 in humans MHV-1 induces acute respiratory infection in 2-4 days, which leads to acute lung inflammation with high levels of chemokines and inflammatory cytokines like TNFalpha. Furthermore, MHV-1 can activate the activation of innate defense mechanisms, resulting in increased proliferation of mycobacterium tuberculosis [4].

## Conclusion

As the world comes together to fight the COVID-19 pandemic, dormant tuberculosis is already affecting a quarter of the world's population according to the World Health Organization.

COVID-19 and tuberculosis coinfection is under-studied to be able to draw definitive conclusions. More studies to measure this association are needed in order to derive recommendations that could help prevent another tuberculosis pandemic.

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