Tuberculosis And Covid-19 Association: About A Case And Literature Review

H.Arfaoui, A.Boumehdi, H.Elkihel, R.Cherkaoui, S.Elhanafy, H.Jabri, W.Elkhattabi, H.Afif

Respiratory Disease Department Hospital 20 August 1953, UHC IBN Rochd, Faculty of medicine of Casablanca

ABSTRACT

Tuberculosis and COVID-19 are two serious diseases that primarily affect the lungs. Co-infection TB and SARS-CoV-2 pose a serious risk of COVID-19 with increased mortality.

We report the observation of a 28-year-old patient, admitted for febrile miliary assessment, whose PCR revealed co-infection tuberculosis, COVID-19 and enterovirus.

Keywords: Co infection, Tuberculosis, COVID19, Miliary

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I. Introduction

Coronavirus disease 2019 (COVID-19) was first identified in Wuhan, China in December 2019. As of April 2023, more than 761 million people were infected with SARS-CoV-2, and more than 6 million people had died from the disease (2). It is estimated that in October 2022, more than 10 million people worldwide contracted TB and more than 1.4 million people died from it (3).

Morocco is a TB endemic country where the Covid-19 pandemic has taken hold. The two pathologies mainly affect the lungs, with a variable mode of installation, acute for tuberculosis and subacute for Covid 19. The risk of ignoring TB during this Covid-19 pandemic is high (4). Work on COVID-19 TB co-infection is rare. It is in this context that we report a case of COVID-19-tuberculosis co-infection observed at the 2O August Hospital in Casablanca.

II. Observation

A 28-year-old patient, married and mother of a daughter, originally from Casablanca, who worked as a caregiver in a hospital setting, receiving suspected COVID-19 patients. She went to Pulmonology for respiratory distress.

Three months before, she had a chronic cough, and exercise dyspnoea, evolving in a context of prolonged fever, night sweats, asthenia and weight loss of 10 kg.

Five days before her admission, the symptomatology worsened, by the appearance of rhinorrhoea and the increase of his dyspnea, becoming at rest.

Physical examination on arrival, revealed a patient in respiratory distress, 85% desaturated at ambient area, cyanotic, polypneic at 25 cycles per minute, with signs of respiratory struggle, febrile at 38.5°C, tachycardic at 112 beats per minute, and normotensive at 10/6 cmHg.

Pleuropulmonary examination showed a decrease in vocal vibrations, with a mattness of the lower 1/3 of both hemithoraxes and crepitating rales in the basithoracic area. The rest of the examination was unremarkable. The X-ray and chest CTscann, show a miliary aspect (Figures 1 and 2).

The diagnosis retained is that of a febrile miliary (with bronchiolar distribution) hypoxemic. Two diagnoses were raised: tuberculosis and pneumocystis.

The patient was put on oxygen therapy, corticosteroids(1mg/kg/day for 7 days) and antibacillary treatment under 2RHZE/7RH regimen.

The assessment showed, a lymphopenia at 530/mm3, an acceleration of the sedimentation rate at 90 mm, at the 1st hour and an elevation of the CRP at 35 mg/l, the search for BK in sputum was negative, while the PCR Mycobacterium Tuberculosis performed on induced sputum was positive.

Nasopharyngeal samples for COVID-19 PCR were positive and Multiplex PCR came back positive for Enterovirus, negative for Pneumocystis Jiroveci. The patient was put on COVID-19 treatment according to the Moroccan national protocol with continuation of antibacillary treatment.

The rest of the assessment in the tuberculosis miliary showed, at the osteeomedullary biopsy, an epithelio-giganto-gigantocellular granuloma without caseous necrosis, there were no Bouchet's nodules at the fundus. Flexible bronchoscopy not done in front of the distress panel.

The evolution was marked by significant clinical improvement after 7 days of antibacilar and anti-COVID-19 treatment.

The particularity of our observation is co-infection with tuberculosis miliary, COVID19 and enterovirus infection.



Figure 1 : Chest x-ray on admission: miliary appearance



Figure 2: Chest CT scan on admission: miliary appearance

III. Discussion

COVID-19 and tuberculosis mainly affect the lungs. Co-infection TB and SARS-CoV-2 pose a serious risk of COVID-19 with increased mortality. Both diseases are recognised by common clinical signs, leading to misdiagnosis (1).

Morocco is a TB endemic country with an estimated 87 cases/100,000 inhabitants. In 2022, a total of 29,327 cases were reported and treated under the National Tuberculosis Program (5).

Risk factors predisposing to these two diseases are essentially: age over 70 years, male sex, immunosuppression, heart disease, diabetes, chronic respiratory failure, chronic renal failure on dialysis, neoplasia, chemotherapy, organ transplantation, and liver cirrhosis(6).

SARS-CoV-2 is propagated by respiratory droplets and manual contamination (7.8). Tuberculosis is transmitted by airborne inhalation of Koch's bacillus. In co-infection tuberculosis and COVID-19, the latter, allows the progression of latent or active infection of Mycobacterium tuberculosis, modifies the clinical presentation and exacerbates pulmonary tuberculosis, while cytokines play an essential role in the pathophysiology of COVID-19 and tuberculosis (plasma concentrations related to severity of the disease).

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SARS-CoV-2 infection could initiate aggressive inflammation by increasing the secretion of cytokines, such as interleukin-1 β (IL-1 β), interferon-c (IFN-c), α tumor necrosis factor (TNF- α), interleukin-2 (IL-2), interleukin 4 (IL-4) and interleukin-10 (IL-10), their high plasma levels, leads to a "cytokine storm" (9).

Clinically, TB and COVID-19 have similar signs (fever, cough and dyspnea) with different characteristics.

In COVID-19, cough and fever have a rapid onset and incubation period of 1 day to 2 weeks, although clinical manifestations of tuberculosis develop over a longer period. Coughing in TB patients is generally productive and occasionally haematopoietic, while coughing is usually dry during COVID-19. For dyspnea, it develops early in COVID-19 and late in tuberculosis (10).

In our patient the symptomatology was chronic (3 months), with aggravation of dyspnea. This could be due to TB-induced lesions or overadded SARS CoV-2 infection.

Biologically, the diagnosis of COVID-19 is based on RT-qPCR of nasopharyngeal swab and that of tuberculosis on sputum bacilloscopy for BAARs, confirmed by genexpert MTB/IFR and culture (11). Other biological disturbances in our case are elevated CRP, accelerated sedimentation rate and lymphopenia. These last two abnormalities are rarely described in COVID-19, whereas raised CRP is frequent (12, 13). All three disorders can be seen in tuberculosis as the stigma of chronic inflammation.

Radiologically, the miliary aspect is not reported in the context of COVID-19, related in our patient to tuberculosis.

COVID-19 lung lesions are due to viral inclusions, lymphocyte-predominant interstitial infiltrations, pulmonary edema lesions, and thrombosis most often related to a thrombotic microangiopathy(6).

Pulmonary lesions of tuberculosis are due to the formation of granulomas with caseous necrosis, which soften before being expelled into the bronchi, explaining the formation of the pulmonary cavities and fibrous lesions (14).

Thus, in the event of tuberculosis-COVID-19 co-infection, these lesions mutually worsen. Literature suggests that latent or active TB is a risk factor for SARS CoV-2 infection (higher rate of occurrence of COVID-19 in tuberculosis patients than bacterial or viral infection) (15).

In Morocco, the treatment of COVID-19 is based on hydroxychloroquine and azithromycin, and tuberculosis on anti-tbacillary drugs. No side effects observed by combining the two medications. However, both therapies should be managed with caution, given the risk of drug interaction between rifampicin, azithromycin and hydroxychloroquine (4).

IV. CONCLUSION

Co-infection TB and COVID-19 pose an increased risk of severe form and mortality. Clinical signs are common and imaging is disease specific. Hence the interest of vaccination and barrier measures.

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