

COVID-19, HIV and pulmonary tuberculosis. A triple threat to consider

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The World Health Organization declared the outbreak of novel SARS-CoV-2 as a pandemic in mid-March, 2020 [1]. The majority of coronavirus cases have a benign course, however, some strains have showed higher morbidity and mortality [2]. SARS-CoV-2 currently has an overall mortality rate of 14.6%. Most deaths in COVID-19 are reported in older patients with co-morbidities (23.7%), with systemic hypertension being the most frequent and only 0.2% related with medical history of immunosuppression [3]. Approximately 38 million people live with human immunodeficiency virus infection (HIV) and based on the available information, the rate of co-infection with COVID-19 is 0.92% [4,5]. On the other hand, roughly 10 million people suffer from tuberculosis (TB) worldwide, [6] nonetheless, there are few cases of co-infection with COVID-19 [7].

It is well documented that certain microorganisms might affect patients with HIV, in particular, when absolute CD4+ cells are less than 200 [8]. Current information suggest that tuberculosis could be a more important risk factor for COVID-19 than the comorbidities commonly reported in epidemiological studies such as diabetes mellitus and hypertension [9]. The immune status that prones people with HIV to tuberculosis might also have influence to coronavirus infection [10]. Here we present the case of a male in his 3rd decade of life with previous history of HIV infection in whom co-infection with COVID-19 and Tuberculosis was diagnosed.

Case summary

A 29-year-old male whose relevant medical history included HIV infection for 8 years in treatment with darunavir/cobicistat 800/150 mg plus tenofovir/emtricitabina 245/200 mg once a day. In 2018, his CD4+ T cell count was 242 cells/ μ L, and the viral load was less than 20 copies/ml, however, the patient abandoned the treatment for 2 years and restarted 2 months prior to hospital admission.

He complained in the previous 2 weeks to hospital admission with fever, cough, myalgias, nausea and vomiting. A nasopharyngeal swab test confirmed COVID-19 infection and was referred to our facility. Baseline blood test analysis revealed mild anemia (Hemoglobin 10.8 gr/dL), lymphopenia 0.45 x10³/mL, elevated inflammation-related biomarkers (LDH 467 U/L, Ferritin 2778 ng/ml, procalcitonin 13.180 ng/mL) mild elevation in D-dimer (600 mg/dL), creatinine 0.80 mg/dl and albumin 2.80 g/dL among relevant serum findings.

Hospital evolution was insidious, in particular, with persistent fever despite multiple antibiotic schemes including cephalosporins, amikacin, macrolide, carbapenems. A chest CT-scan (Figure 1) showed ground-



Figure 1. Chest CT scan showing ground-glass opacities with nodular opacities and air bronchogram in the right upper lobe.

glass opacities and nodular opacities in the right upper lobe; also, a cavitation in the right lower lobe. A sputum culture was negative for bacterial, fungus or virus co-infection but a gene-Xpert test confirmed TB and a 4-drug treatment (isoniazid, rifampicin, pyrazinamide and ethambutol) was initiated. After 48hrs the patient continued with fever, however, overall symptoms diminished in intensity and maximum temperature registered was 38 °C. He was hemodynamically stable with oxygen level of 92% on room air and was referred to a community hospital to continue the treatment under strict surveillance. Currently, the patient is alive, without fever, in his home and continuous anti-tuberculosis treatment.

Discussion

COVID-19 disease has spread rapidly worldwide since the first reports. Similarly, people infected with HIV are at risk for severe

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coronavirus disease compared to the general population [7]. The disease is usually characterized by initial signs and symptoms similar to those of related viral infections (e.g., influenza, SARS, Middle East Respiratory Syndrome [MERS]) and TB, although complications and prognosis differ.

Worldwide, the incidence of HIV and pulmonary TB is increasing. The relevance of HIV co-infection and TB morbidity/mortality is highlighted to the fact that in 2016 more than 20% of all TB-related deaths were associated with HIV infection [8]. To date, the experience with COVID-19 infection in TB patients is limited, it is anticipated that people with TB and COVID-19 may have worse results, especially if TB treatment is delayed.

So far, SARS-CoV2 interacts with the host tissues through the angiotensin-converted enzyme 2 (ACE2) receptor facilitating the binding and entry in host cells [8]. Unlike SARS-CoV-2, tuberculosis requires cellular immunity, and in case of coinfection with HIV, this causes a progressive depletion and dysfunction of CD4 lymphocytes leading to immune suppression with negative implications in patients who might have asymptomatic latent TB infection rendering an increase risk to develop active TB. Latent TB infection is more likely to become a tuberculosis disease among individuals with HIV with a 5-10% lifetime risk. A weakened immune system with a chronic pre-existing lung infection could increase the risk of developing a SARS-CoV-2 infection, especially in patients with poor control of pre-existing diseases [6,8].

However, due to the recent discovery of COVID-19, there is currently no data on whether patients with HIV co-infection and TB will have greater complications. such as a greater number of days of hospitalization, a greater requirement for invasive mechanical ventilation or greater mortality.

Conclusion

In patients with COVID-19 who have medical history of HIV and showed persistent fever despite prescribed treatment, physicians might search for differential diagnosis including tuberculosis, in particular, in highly prevalent endemic areas.

Conflicts of interest

The authors have no conflicts of interest to disclose.

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