Medicine & Clinical Science

Case Report

Respiratory Worsening at the End of a Hospital Stay for Severe Covid19 Pneumonia Revealing Pulmonary Tuberculosis

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Abstract

The coexistence of a viral and tuberculous respiratory infection increases mortality and the transition to a severe form in both directions. We report an observation for a 55-year-old woman with untreated diffuse interstitial pnuemopathy, investigated by immunological and infectious tests, and found negative for pulmonary tuberculosis. Bronchial biopsies revealed non-specific inflammation, responsible for chronic dyspnea. Having presented ten days previously with myalgias, fatigue, dry cough and fever, with worsening of basic dyspnoea, without digestive or neurological manifestations, she was referred to the emergency department, where physical examination showed SpO2 at 80% on room air, reduced to 92% under 15 L/mn delivered via a high-concentration mask, polypnoea at 40 cycles per minute with bilateral diffuse crackling rales on auscultation, scattering just the anterior quadrants. The initial evolution was favorable, with clinical improvement in oxygen saturation to 94% on two liters per minute of oxygen delivered by nasal cannula, with respiratory rate at 23 cycles per minute with modest effort tolerance. On day 18 of hospitalization, she presented with a febrile peak of 38.3°C, polypnoea at 33 cycles per minute, increased oxygen requirements with a flow rate of 6l/m by face mask to reach an oxygen saturation of 92%, tachycardia at 130 beats per minute, and was therefore readmitted to intensive care with high-flow nasal oxygen therapy and antibiotic therapy with imipenem and amikacin. Multiplex PCR showing no germ, and a PCR test for pulmonary tuberculosis on a sputum sample which was positive, prompting the introduction of antibacillary treatment based on a fixed quadric-association of rifampicin, isoniaside, pyrazinaamide and ethombutol. Non-specific antibiotic therapy was discontinued after improvement of the inflammatory syndrome, and antibacillary treatment is still ongoing.

Conclusion: *Through this observation we discuss the difficulties in terms of diagnosis, management and outcome of a coinfection by SARS-COV 19 and mycobacterium tuberculosis.*

Introduction

Covid 19 pneumonia due to the SARS-COV 19 virus is a global health emergency, with the potential for significant spread, healthcare system exhaustion, mortality and morbidity. The interaction with other specific respiratory infections such as pulmonary tuberculosis remains poorly understood. The latter is endemic in our country, with a high frequency of latent tuberculosis, hence the interest of our observation.

Historically and according to the literature, the coexistence of a viral and tuberculous respiratory infection increases mortality and the transition to a severe form in both directions [1].

Case Report

This is a 55-year-old woman with untreated diffuse interstitial pnuemopathy, investigated by immunological and infectious tests, and found negative for pulmonary tuberculosis. Bronchial biopsies revealed non-specific inflammation, responsible for chronic dyspnea. Having presented ten days previously with myalgias, fatigue, dry cough and fever, with worsening of basic dyspnoea, without digestive or neurological manifestations, she was referred to the emergency department, where physical examination showed SpO2 at 80% on room air, reduced to 92% under 15 L/mn delivered via a high-concentration mask, polypnoea at 40 cycles per minute with bilateral diffuse crackling rales on auscultation, scattering just the anterior quadrants. The patient was tachycardic at 135 beats per minute with a correct blood pressure of 130/75 mmHg, with a tricuspid murmur on auscultation, while conscious with a capillary blood glucose of 2.5 g/L and a temperature of 38.3°C under the effect of an antipirant. A thoracic CT scan revealed diffuse ground-glass lesions covering more than 75% of the lung fields, associated with condensation images. PCR for SARS-COV 19 was positive, with a laboratory work-up showing an inflammatory syndrome with CRP 220 mg/l, white blood cells 5200/mm3, correct renal function with urea at 0.16 g/l and creatinine at 8 mg/l, correct transaminases with

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ASAT at 22 U, ALAT at 15 , a transthoracic echocardiography showed chronic pulmonary heart disease with pericardial effusion. The patient was hospitalized in intensive care, where she was treated with oxygen therapy, using an anti-Covid 19 protocol adopted by the national health authorities, including hydroxychloroquine at a dose of 600 mg/d, combined with azythromycin at a dose of 250 mg per day, plus vitamin C and zinc for seven days. In addition, methylprednisolone-based corticosteroid therapy at a dose of 40 mg/d was given, along with curative heparin therapy with enoxaparin and probabilistic antibiotic therapy with third-generation cephalosporins and a fluoroquinolone and spironolactone at a dose of 50mg daily.

The evolution was favorable, with clinical improvement in oxygen saturation to 94% on two liters per minute of oxygen delivered by nasal cannula, with respiratory rate at 23 cycles per minute with modest effort tolerance, and biologically with CRP at 24 mg/l. The patient was discharged from the intensive care unit after a few days, and hospitalized in the clinical sector for rehabilitation and oxygen weaning, the latter objective not being achieved, and it was decided to discharge her on home oxygen therapy.

On day 18 of hospitalization, she presented with a febrile peak of 38.3°C, polypnoea at 33 cycles per minute, increased oxygen requirements with a flow rate of 6l/m by face mask to reach an oxygen saturation of 92%, tachycardia at 130 beats per minute, and was therefore readmitted to intensive care with high-flow nasal oxygen therapy and antibiotic therapy with imipenem and amikacin, in the face of a clinical and biological worsening, with Procalcitonin at 78 ml/l and CRP at 170 mg/l, with a multiplex PCR showing no germ, and a PCR test for pulmonary tuberculosis on a sputum sample which was positive, prompting the introduction of antibacillary treatment based on a fixed quadric-association of rifampicin, isoniaside, pyrazinaamide and ethombutol. Non-specific antibiotic therapy was discontinued after improvement of the inflammatory syndrome, and antibacillary treatment is still ongoing.

The patient had a good clinical evolution at D 40 of her hospitalization, but still had problems with oxygen weaning and exercise tolerance.

Discussion

At the time of writing, we are experiencing the second wave of the SARS-COV 19 pandemic, which is responsible for respiratory disease of varying severity. At the same time, tuberculosis is endemic in our country, and a quarter of the world's population probably carries latent tuberculosis [2]. These two diseases have much in common, and represent a short- and long-term challenge for the healthcare system. The STOP Tuberculosis operation in Switzerland indicates that the Covid 19 panemia is having a profound effect on efforts to prevent, detect and manage tuberculosis cases [3]. The true incidence of the association of these two respiratory pathologies remains poorly known and probably under-studied, due to the diagnostic difficulties of low-income endemic countries and the state of emergency that the Covid 19 pandemic brings, exhausting the resources of the health system, as well as the similarities between the two conditions . The first described 49 cases of coinfection from 8 different European countries [4], while Stochino, et al. [5] and Motta, et al. [6] from Italy described two cohorts of 20 and 69 patients respectively from immigrants. A larger cohort was described by Say [7] from the Philipines and Davies [8] from South Africa, with 172 and 155 respectively. The interaction between these two pathologies remains debated, ranging from being a factor favoring each other, to influencing clinical results. Thus, latent tuberculosis induces a chronic local inflammatory state necessary for the maintenance of the granuloma [7,8], and through lung damage and local immunity promotes infection by airborne pathogens [2]. On the other hand, viral infection, including Covid 19, induces a cytokine storm and an inflammatory state with impaired cellular immunity that can reactivate latent tuberculosis [9]. The outcome of patients suffering from this coinfection remains controversial. In our case, pulmonary tuberculosis was diagnosed in the face of a secondary worsening of the condition, while in the literature, through past coronavirus pandemics, coinfection has had more dismal results, as has the case with influenza viruses. For the current Covid 19 pandemic, researchers believe that competition between the two conditions leads to poor results [10], and that the interaction is synergistic in terms of respiratory severity.

Conclusion

Through this observation we discuss the difficulties in terms of diagnosis, management and outcome of a coinfection by SARS-COV 19 and mycobacterium tuberculosis. Our patient presented two episodes of respiratory distress requiring intensive care, and posed a weaning problem given chronic interstitial damage aggravated by Covid 19 and pulmonary tuberculosis.

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