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Accelerated progression of pulmonary tuberculosis in a COVID-19 patient after corticosteroid treatment



KEYWORDS

SARS-CoV-2;
Steroid;
Immunosuppression

Dear Editor,

During the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pandemic, systemic investigations of bacterial or fungal co-infections remain limited to guide the antimicrobial therapy.¹ The frequent use of corticosteroids that are now used for the SARS-CoV-2-infected patients who present with desaturation may raise concerns about reactivation of latent infection including hepatitis B or tuberculosis, as described in non-COVID-19 scenarios. Here we present a patient with pulmonary tuberculosis that progressed after a 10-day course of corticosteroid therapy for COVID-19.

A 46-year-old mechanic in the hospital, previously healthy, presented with acute onset of fever up to 39.1 °C two days prior to this admission, associated with malaise, sore throat, chest tightness and productive cough. There was no dysosmia, dysgeusia or diarrhea. On arrival at our hospital, the oxygen saturation was 97% while he was breathing ambient air despite high fever and tachycardia (120 beats/min). A chest radiograph disclosed increasing infiltration of both lung fields (Fig. 1A and B). Blood examinations were remarkable for leukocytosis ($11.42 \times 10^3/\mu\text{L}$) and an elevated C-reactive protein (CRP) level (3.92 mg/dL). He tested negative for HIV. A real-time reverse transcription-polymerase chain reaction test of nasopharyngeal swab specimens yielded positive results for SARS-CoV-2.

Investigations for the etiology of community-acquired pneumonia were performed on admission. Blood cultures and acid-fast stain of sputum specimens collected on three consecutive days were negative. Ceftriaxone was administered because of a positive result for urine pneumococcal antigen. However, fever persisted and desaturation (oxygen saturation, 92%) ensued on the 5th hospital day. A follow-up chest radiograph showed increases of pulmonary infiltrates (Fig. 1C). Betamethasone 6 mg per day was administered for 10 days with improvement of oxygenation and he was discharged on the 18th hospital day. Mild dyspnea on exertion was reported after discharge and follow-up chest radiographs showed further increases of pulmonary infiltrates (Fig. 1D). A sputum culture on admission subsequently grew *Mycobacterium tuberculosis*. With 14 days of anti-tuberculous therapy, the respiratory symptoms resolved and pulmonary infiltrates decreased (Fig. 1E). The chest radiograph performed 6 weeks after initiation of anti-tuberculous therapy revealed significant resolution of pulmonary infiltrates (Fig. 1F).

Previous meta-analysis of mainly small case series by Lansbury et al. found that concurrent bacterial infections might be less common in the COVID-19 than in influenza pandemics; however, bacterial co-infection could still have a major adverse impact on the prognosis, especially among patients with severe COVID-19 who receive corticosteroids.² Concomitant SARS-CoV-2 infection of patients with invasive pneumococcal disease had a seven-fold increased risk of mortality compared with those who had only invasive pneumococcal infection.³ Whether short-course exposure to corticosteroids may have an adverse impact on the risk of secondary bacterial pneumonia in the setting of COVID-19 remains unclear, although long-term use of inhaled steroids in individuals with chronic lung disease might increase the risk of bacterial infection such as invasive pneumococcal infection.⁴ In animal model, corticosteroids may inhibit pneumococcal clearance by reducing alveolar

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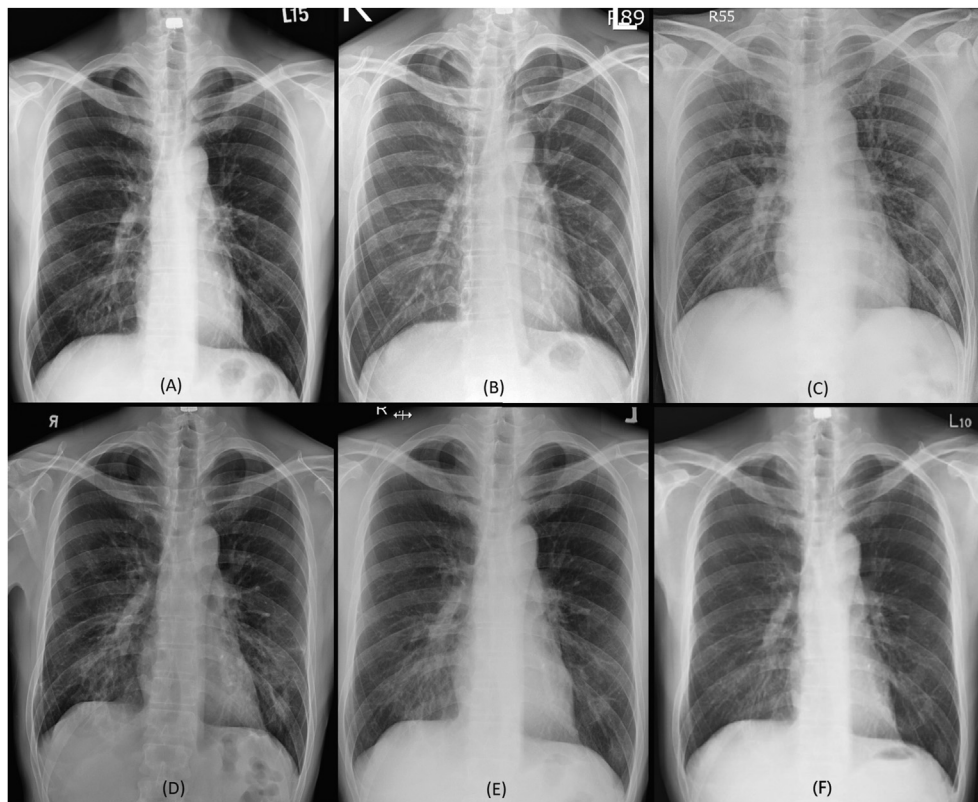


Figure 1. Chest radiographs two months prior to COVID-19 (A), and on the day of admission (B), initiation of corticosteroids (5th hospital day) (C), discharge (18th hospital day) (D), after a 2-week anti-tuberculous therapy (41 days of diagnosis of COVID-19) (E) and after a 6-week anti-tuberculous therapy (69 days of diagnosis of COVID-19) (F).

macrophage bactericidal function.⁵ Therefore, active surveillance of co-infection with the use of antigen-, culture- or polymerase-chain-reaction-based methods are warranted to facilitate timely initiation of appropriate antibacterial therapy and decrease subsequent mortality among the high-risk patients.⁶

Two retrospective studies recently described patients with tuberculosis diagnosed simultaneously or shortly after SARS-CoV-2 infection, highlighting the challenges of correct and timely diagnosis and treatment of tuberculosis in patients with COVID-19, particularly in those receiving corticosteroids.^{7,8} The recent study by Patel et al. showing increasing incidence of mucormycosis in India during COVID-19 pandemic also demonstrates the emerging threat associated with inappropriate use of corticosteroids.⁹

While short-course corticosteroid therapy is a cheap and effective treatment for patients with COVID-19 who present with oxygen desaturation, our case should alert the health care providers to the risk that reactivation of latent infection or progression of undetected infection may occur in such a setting.

Declaration of competing interest

None to declare.

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