COVID-19 pneumonia in a patient with sarcoidosis: A case report

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Abstract
Key prognostic elements to consider in sarcoidosis patients who contract COVID-19 pneumonia are pulmonary involvement, the underlying immune system dysfunction, immunosuppressive therapies’ use, and the increased risk for hypercoagulability.

KEYWORDS
chest ct, COVID-19 pneumonia, ground-glass, mediastinal lymphadenopathy, sarcoidosis

1 | INTRODUCTION

We report the case of a patient with sarcoidosis who contracted Sars-Cov-2 as limited data is available regarding this association and there is concern about its poor outcome. Key elements to consider are sarcoidosis pulmonary involvement, the underlying immune system dysfunction, the immunosuppressants’ use, and the increased hypercoagulability risk.

As limited data is available regarding COVID-19 pneumonia in patients with sarcoidosis, we share the case of a 66-year-old female patient with sarcoidosis who contracted Sars-Cov-2. Through this case report, we address clinical presentation, radiological features, and therapeutic approach uncertainties.

Although the presentation is similar to that of the general population, sarcoidosis patients might have a poorer prognosis due to the pulmonary involvement present in 90% of cases with a frequently diminished baseline lung function, the underlying immunologic dysregulation, the common use of immunosuppressive drugs, and the increased risk for hypercoagulability.1-3

2 | CASE PRESENTATION

A 66-year-old female patient with remarkable medical history for pulmonary sarcoidosis diagnosed in 2016 and a depressive disorder under amitriptyline 25 mg per day since 2018 had been lost to follow-up for 2 years and was addressed to our radiology department for an evaluation chest CT. Thoracic computed tomography scan with pulmonary angiography revealed bilateral hilar and mediastinal lymphadenopathy (Figure 1), multifocal, bilateral ground-glass opacities, traction bronchiolectasis and reticular opacities (Figures 2-3). Upon interrogation, the patient reported a 4 days course of fever, cough, myalgia, and diarrhea. She was admitted to the hospital. Body temperature was 38°C and arterial oxygen saturation in room air 98%. Lung auscultation was normal. Moroccan COVID-19’s first intention therapeutic protocol consists of administering a 7 days course of chloroquine (500 mg 2×/day) or hydroxychloroquine (200 mg 3×/day) in association with azithromycin 500 mg on the first day then 250 mg/day from the 2nd to the 7th day. It was not introduced for on one hand; the association with amitriptyline yields a risk of QT interval prolongation, on the other hand, sarcoidosis cardiac involvement can manifest with arrhythmias. The patient was closely monitored. Supplemental oxygen was
not required. Symptoms rapidly improved under supportive treatment and the patient was discharged on day 5 with a recommendation of 14 days' home isolation.

3 | DISCUSSION

The emerging COVID-19 pandemic continues its rapid spread challenging healthcare systems worldwide in a new and unpredictable manner. First reported in Wuhan, the probably zoonotic causative agent has been identified: A single-stranded RNA novel coronavirus (Severe acute respiratory syndrome Coronavirus 2 = Sars-CoV-2).4 Patients with sarcoidosis are at high risk of complications of COVID-19 because of their weakened immunity and underlying lung disease.1

Major initial symptoms of COVID-19 include fever, cough, sore throat, and dyspnea, and can mimic worsening of sarcoidosis.5 Myalgia, diarrhea, and vomiting are reported in some patients. An altered mental status in the elderly may be an atypical COVID-19 presentation.6 Critical forms manifest with respiratory insufficiency, shock, and multiorgan failure.7 Leukopenia and lymphopenia are frequent. Liver function anomalies may be detected in severe forms.8 Confirmatory diagnosis of COVID-19 infection relies on nucleotide detection. Chest CT is a complementary diagnostic modality with a considerable sensitivity reaching 97% in epidemic territories.4

Chest radiography’s sensitivity for COVID-19 pneumonia is limited for it fails to detect ground-glass opacities; the infection’s major manifestation. COVID-19’s pneumonia chest CT imaging findings include multifocal, bilateral ground-glass opacities with a predominant peripheral, posterior, and lower lobes distribution, vascular dilatation, and traction bronchiectasis. Thicken interlobular and intralobular septa within the ground-glass opacities define ‘Crazy paving’. Consolidation appears in the peak stage mainly in the elderly. Subpleural bands and architectural distortion manifest in later disease stages.9 Lung involvement’s extent on CT correlates with the severity of the disease and can be assessed visually.

Pulmonary sarcoidosis radiologic patterns are diverse. The most common feature is bilateral hilar lymphadenopathy followed by interstitial lung disease with perilymphatic micronodules, fibrotic changes, masslike or alveolar opacities, honeycomb-like cysts, tracheobronchial disease, mosaic attenuation, pleural involvement, and complications including aspergilloma.10 Of note; patchy ground-glass opacities are seen in 40% of patients with parenchymal pulmonary sarcoidosis. Bronchoalveolar structures are frequently seen within them generating air bronchograms at CT.

In the setting of COVID-19 pneumonia in a sarcoidosis patient; the Sars-Cov2 commonly reported pulmonary infiltrates are associated with baseline pulmonary sarcoidosis lesions.11-13

Our patient's imaging findings overlap with pulmonary sarcoidosis atypical features, organizing pneumonia and other viral pneumonia; nevertheless, the diagnosis was asserted based on the epidemiological context, the patient’s symptoms, the superimposed bilateral multifocal ground-glass opacities on the mild baseline sarcoid lesions on CT and
the positive SARS-CoV-2 RT-PCR from the nasopharyngeal swab.

The Sars-Cov2 mortality rate (3.8%) is lower than that of Sars-CoV (10%) and Mers-CoV (37.1%); however, infection cases are 10 times higher, principally as a result of transmission from moderately symptomatic or asymptomatic patients. COVID-19’s presentation is more grave in older patients with comorbidities and mortality reaches 6.3% in those with a chronic respiratory disease. Sarco. osis raises the risk of poor outcome and mortality from COVID-19 pneumonia for:

First, patients with sarcoidosis have a propensity for respiratory failure since 90% present pulmonary involvement with a commonly diminished pulmonary reserve.

Second, COVID-19 could cause acute sarcoidosis exacerbations especially in patients with pulmonary fibrosis.

Third, immunologic dysfunction’s role in sarcoidosis development is fundamental. Immunosuppressants—primarily glucocorticoids—are first-line treatment drugs. They are linked to an increased risk of serious infection. The prevalence of glucocorticoids associated comorbidities (hypertension, diabetes, and obesity) is higher in sarcoidosis patients. These comorbidities are recognized as COVID-19 related death independent risk factors. Thus; in sarcoidosis patients exposed to COVID-19; immunosuppressive regimen adjustment based on disease stability and risks of reactivation is believed to improve outcome.

And fourth, COVID-19 associated coagulopathy induces diverse thromboembolic complications, notably in critically ill patients; an effect conceivably potentialized by sarcoidosis—a chronic immune-mediated inflammatory disorder—also associated with an increased risk of venous thromboembolism.

Great efforts and active research are ongoing to develop a specific antiviral medication and vaccine against the novel coronavirus. Current therapies include isolation, supportive care, and administration of some antiviral therapies: Nucleoside analogs (Favipiravir, Remdesivir...), Chloroquine, and Protease inhibitors (Lopinavir and Ritonavir).

Further evidence-based recommendations are needed to guide the management of patients requiring long-term immunosuppressants such as those with sarcoidosis. In practice; physicians carefully adjust immunosuppressive drugs to treat the Sars-Cov-2 infection and prevent prolonged viral shedding whilst taking into account the underlying auto-immune disease stability and exacerbation risk.

Our patient was lost to follow-up and was not under treatment for sarcoidosis at the time of COVID-19 pneumonia diagnosis. Imaging showed both mild baseline sarcoid parenchymal abnormalities and COVID-19 pneumonia features which explains symptoms’ rapid resolution.

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CONFLICT OF INTEREST
None declared.

AUTHOR CONTRIBUTIONS
HR and KB: Contributed to conception, acquisition, analysis, and interpretation of data; drafted the manuscript. NMB and IN: Critically revised manuscript. All the authors have read and approved the final draft of the manuscript.

ETHICAL APPROVAL
Ethical approval is not required for de-identified single case reports based on institutional policies.

DATA AVAILABILITY STATEMENT
Data available on reasonable request from the authors.
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