

# Tension hydrothorax in a patient with SARS-CoV-2 pneumonitis and pleural *Mycobacterium tuberculosis*

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## SUMMARY

Unilateral pleural effusions are uncommonly reported in patients with SARS-CoV-2 pneumonitis. Herein, we report a case of a 42-year-old woman who presented to hospital with worsening dyspnoea on a background of a 2-week history of typical SARS-CoV-2 symptoms. On admission to the emergency department, the patient was severely hypoxic and hypotensive. A chest radiograph demonstrated a large left-sided pleural effusion with associated contralateral mediastinal shift (tension hydrothorax) and typical SARS-CoV-2 changes within the right lung. She was treated with thoracocentesis in which 2 L of serosanguinous, lymphocyte-rich fluid was drained from the left lung pleura. Following incubation, the pleural aspirate sample tested positive for *Mycobacterium tuberculosis*. This case demonstrates the need to exclude non-SARS-CoV-2-related causes of pleural effusions, particularly when patients present in an atypical manner, that is, with tension hydrothorax. Given the non-specific symptomatology of SARS-CoV-2 pneumonitis, this case illustrates the importance of excluding other causes of respiratory distress.

## BACKGROUND

The COVID-19 is a contagious disease caused by the novel SARS-CoV-2, first identified in December 2019 in Wuhan, China.<sup>1</sup> As of January fourth 2021, the COVID-19 pandemic has resulted in more than 83 million confirmed cases with more than 1.8 million deaths.<sup>2</sup> The typical symptoms of SARS-CoV-2 are non-specific and include dyspnoea, fever, cough, fatigue and sore throat.

The diagnosis of SARS-CoV-2 should not imply the absence of concomitant diseases.<sup>3</sup> It is particularly important for non-SARS-CoV-2 causes of respiratory symptoms to be identified and excluded in patients who present in an atypical fashion.

Herein, we describe the case of a woman who presented to the emergency department with severe dyspnoea secondary to a tension hydrothorax in the context of *Mycobacterium tuberculosis* infection and SARS-CoV-2 pneumonitis.

## CASE PRESENTATION

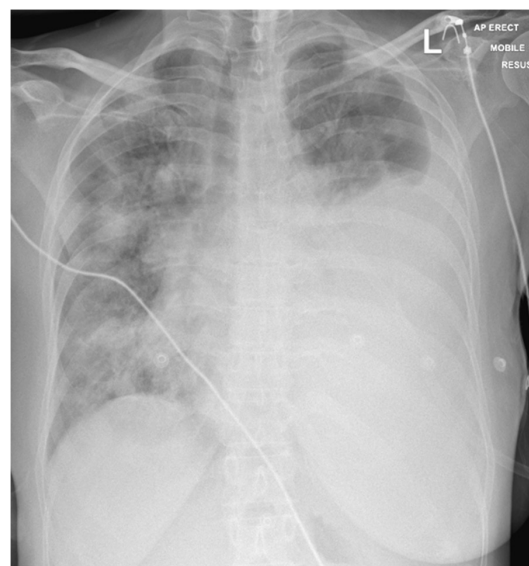
A 42-year-old, previously fit and well woman, presented to the emergency department with a 3-day history of severe shortness of breath on minimal exertion. This was on the background of a 2-week history of gradually increasing shortness of breath, fever, dry cough and fatigue. She denied weight loss, chest pain or nightsweats. There was no significant recent travel history and she had last visited Pakistan 2 years ago. There was no known

previous personal history of tuberculosis or exposure to asbestos. She had no medical history, was not on any regular medications and was a socially independent individual.

On arrival to the emergency department, she was severely hypoxic (SpO<sub>2</sub> 79%, room air), requiring 15 L/minute of oxygen to maintain target saturations of 90%–94%. She was tachycardic (heart rate of 110 beats/minute), mildly hypotensive (blood pressure of 91/63 mm Hg) and febrile (37.9°C). On examination, there was clinical evidence of right-sided tracheal deviation with reduced chest expansion on the left hand side. The left hemithorax was dull on percussion and, on auscultation, there was significantly reduced air entry at the left lung base.

## INVESTIGATIONS

Routine blood tests on admission were as follows: haemoglobin 117 g/L (normal range: 115–165 g/L); white cell count:  $7.88 \times 10^9/L$  (normal range:  $4.5\text{--}11 \times 10^9/L$ ); platelets:  $385 \times 10^9/L$  (normal range:  $150\text{--}450 \times 10^9/L$ ); neutrophils:  $5.64 \times 10^9/L$  (normal range:  $2.0\text{--}7.5 \times 10^9/L$ ); lymphocytes:  $1.9 \times 10^9/L$  (normal range:  $1.5\text{--}4.5 \times 10^9/L$ ); D-dimer: 8714 ng/mL (normal range: 220–460 ng/mL); and C reactive protein: 57 mg/L (normal range: <10 mg/L). There was no serological

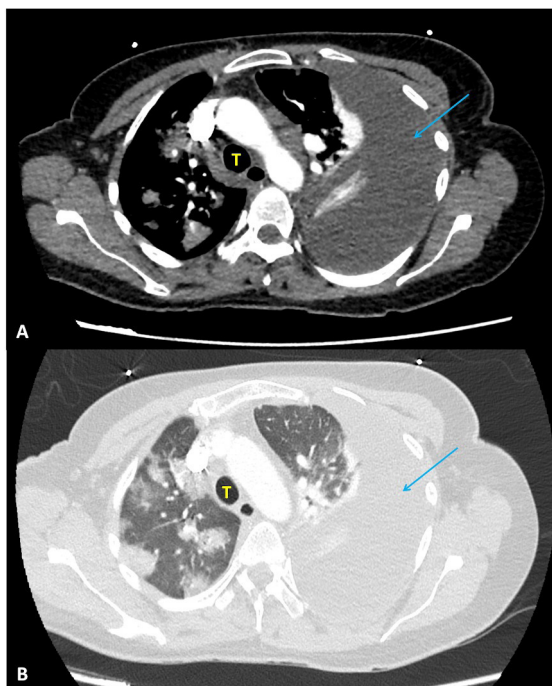


**Figure 1** Mobile anteroposterior chest radiograph demonstrating a large left-sided pleural effusion extending into the left upper zone causing contralateral tracheal deviation and mediastinal shift. Within the right lung, there is diffuse patchy consolidation consistent with SARS-CoV-2 pneumonitis.



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**Figure 2** CT pulmonary angiogram in axial soft tissue windowing (A) and lung windowing (B). This demonstrates a large low-density left-sided pleural effusion (blue arrow), causing almost complete collapse of the left lung and minimal left apical aeration. This results in mediastinal and tracheal deviation to the right (T). Within the right lung and the aerated apical segment of the left lung, there is patchy bilateral ground-glass attenuation and consolidation, consistent with SARS-CoV-2 pneumonitis. No evidence of pulmonary emboli or enlarged intrathoracic lymph nodes.

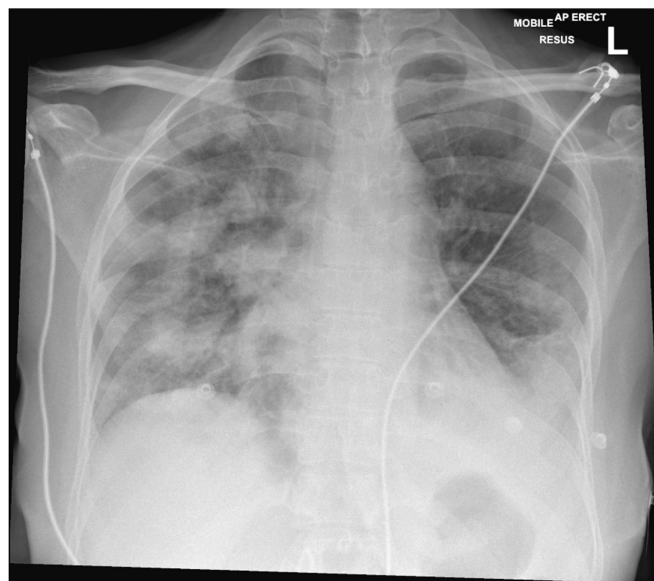
evidence of hepatitis B, hepatitis C or HIV. Laboratory diagnosis of SARS-CoV-2 infection was confirmed by nasopharyngeal swab PCR.

A chest radiograph demonstrated a large left-sided pleural effusion extending into the left upper zone (figure 1). This caused a right-sided tracheal and mediastinal shift. These findings were on a background of diffuse, patchy airspace shadowing within the right lung and partially visualised left lung apex, consistent with features of SARS-CoV-2 pneumonitis. An urgent CT pulmonary angiogram was requested with a view to evaluate the nature and size of the pleural effusion prior to intervention and to exclude a pulmonary embolism (figure 2). The study was negative for pulmonary emboli and supported the chest radiograph findings by demonstrating a large, left-sided pleural effusion causing right-sided mediastinal shift, that is, tension hydrothorax. There was also evidence of bilateral peripheral ground-glass attenuation and patchy consolidation, in keeping with SARS-CoV-2 pneumonitis.

Pleural fluid analysis confirmed an exudative lymphocytic-rich effusion (total protein: 59 g/L; lactate dehydrogenase: 307 U/L). Microscopy revealed a moderate white blood cell count with no growth of organisms. No malignant cells were identified by cytology. Following 16 days of incubation, the pleural aspirate tested positive for *M. tuberculosis*.

#### DIFFERENTIAL DIAGNOSIS

Low oxygen saturations (SpO<sub>2</sub> 79%, room air) in the presence of haemodynamic compromise (blood pressure of 91/63 mm Hg) and an elevated D-dimer (8714 ng/mL (normal range: 220–460



**Figure 3** AP erect chest radiograph following left-sided thoracocentesis. This demonstrates almost complete resolution of the left-sided pleural effusion, with minimal residual fluid within the left costophrenic recess. There is bilateral patchy consolidation, more pronounced within the right hemithorax, consistent with SARS-CoV-2 pneumonitis.

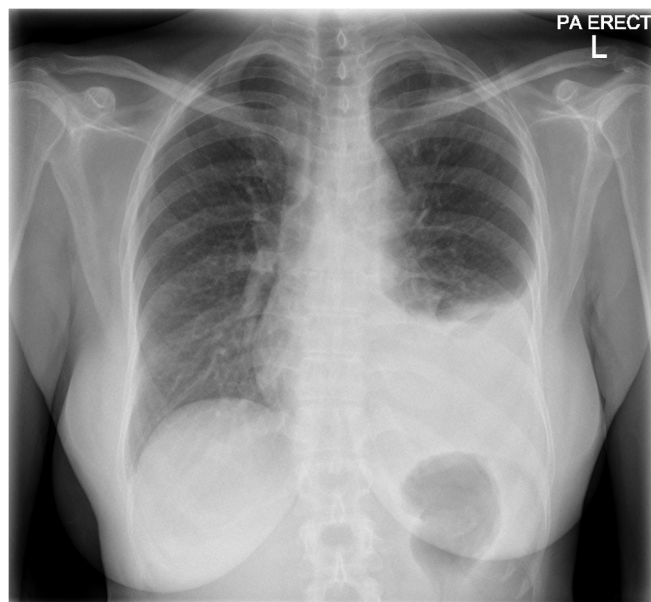
ng/mL)) could signal a large pulmonary embolism. In the absence of a CT pulmonary angiogram, treatment dose low-molecular-weight heparin could potentially have been administered, but the clinical and preliminary X-ray findings were in keeping with a tension hydrothorax as the most probable cause of symptoms. Dual pathology is not unheard of; however, it may have been reasonable to commence treatment dose low-molecular-weight heparin while awaiting evidence of pulmonary embolism on a CT pulmonary angiogram.

#### TREATMENT

A left-sided intercostal drain was inserted within the emergency department to treat the tension hydrothorax, which drained approximately 2 L of serosanguinous fluid. The patient's oxygen saturation improved to 92% on 3 L of oxygen on removal of the drain. In the acute setting, treatment was delivered in a ward-based setting and the patient received supplementary oxygen, dexamethasone and remdesivir for treatment of SARS-CoV-2 infection.

#### OUTCOME AND FOLLOW-UP

A repeat chest radiograph (figure 3) the following day revealed near-complete resolution of the left pleural effusion following removal of the intercostal drain. The mediastinal shift had simultaneously resolved. The patient was discharged after 1 week of treatment in the hospital. A chest radiograph 2 months after initial presentation to the hospital demonstrated reaccumulation of fluid in the left costophrenic recess, but resolution of the SARS-CoV-2 parenchymal changes (figure 4). The patient did not require readmission to hospital. Within 2 weeks of the *M. tuberculosis*-positive pleural aspirate finding, the patient was initiated on 6 months of targeted treatment for tuberculosis (rifampicin 150 mg, isoniazid 75 mg, pyrazinamide 400 mg and ethambutol 275 mg, four times a day for 2 months followed by rifampicin 600 mg and isoniazid 300 mg once daily for 4 months)



**Figure 4** Chest radiograph 2 months following the initial acute presentation. There has been interval reaccumulation of fluid within the left costophrenic recess, in keeping with a moderate left-sided pleural effusion. The previously demonstrated bilateral patchy consolidation secondary to SARS-CoV-2 pneumonitis has resolved.

and remained under respiratory follow-up in the community for the duration of treatment.

## DISCUSSION

To our knowledge, we are the first group to describe a tension hydrothorax in the context of SARS-CoV-2 and *M. tuberculosis*.

A tension hydrothorax is defined as a massive pleural effusion resulting in haemodynamic compromise secondary to mediastinal compression.<sup>4</sup> An increasing volume of fluid in the pleural space may result in reduction in lung volume, contributing to tachypnoea and hypoxia. Left untreated, the increasing intrathoracic pressure may result in decreased venous return and compression of the right ventricle, manifesting as hypotension. Prompt thoracocentesis is the cornerstone of management for tension hydrothorax.

The recognised radiological pulmonary manifestations of SARS-CoV-2 include multifocal and bilateral ground-glass opacities, interlobular septal thickening, bronchiectasis, lymphadenopathy, cavitation, pulmonary emboli, pneumothoraces and pneumomediastinum.<sup>5 6</sup> Pleural effusions are uncommonly reported in patients with SARS-CoV-2.<sup>7</sup> Indeed, one meta-analytic review of 15 studies showed that approximately 3% of SARS-CoV-2 patients who underwent CT chest imaging had evidence of a pleural effusion.<sup>8</sup> One multicentre study (n=476) of patients based in China showed that the presence of a pleural effusion on chest CT at admission was strongly correlated with disease severity.<sup>9</sup>

Pleural effusions in the context of SARS-CoV-2 may be incidental, caused directly by SARS-CoV-2 or be secondary to comorbid conditions. Given the low prevalence of pleural effusions in patients affected by SARS-CoV-2, it is important to exclude other causes. In a general hospital setting, the most common causes of pleural transudates are left ventricular failure and liver cirrhosis, while malignancy, parapneumonic effusions and tuberculosis are common causes of pleural exudates.<sup>10</sup> Common causes of lymphocyte-rich effusions can include

tuberculosis, sarcoid, lymphoma and asbestosis.<sup>10</sup> In this case, incubation grew *M. tuberculosis*, which was the most likely cause of pleural effusion, and treatment was only commenced once this was confirmed. GeneXpert—a rapid diagnostic test for tuberculosis detection as well as rifampicin resistance—could have been used in this setting to ensure a quicker diagnosis of *M. tuberculosis*, but culture is considered the ‘gold standard’ approach and is not limited by the lack of specificity that affects GeneXpert.<sup>11 12</sup>

Patients infected with *M. tuberculosis* may more likely be infected with SARS-CoV-2 and have been shown to have more adverse outcomes once affected.<sup>13 14</sup> The cause for this association is unknown and may be directional: temporary immunosuppression induced by *M. tuberculosis* could potentially increase susceptibility to SARS-CoV-2, and SARS-CoV-2 may, in turn, increase susceptibility to *M. tuberculosis*.<sup>13</sup> In patients who are known to have *M. tuberculosis*, therapy for SARS-CoV-2 could potentially result in reactivation of latent *M. tuberculosis*, which is an important consideration in high endemic areas, such as India and Pakistan.<sup>15 16</sup>

This case highlights the importance of considering tension hydrothorax as a potential cause of haemodynamic compromise in patients with SARS-CoV-2. Other causes of rapid haemodynamic decline may include massive pulmonary embolism and tension pneumothorax, which we have described elsewhere,<sup>17</sup> and are important considerations when a patient presents with severe hypoxia and breathlessness.

This case also demonstrates the need to exclude non-SARS-CoV-2-related causes of pleural effusions, particularly when patients present in an atypical fashion that is, tension hydrothorax. Moreover, given the non-specific symptomatology of SARS-CoV-2 pneumonitis, this case illustrates the importance of identifying and ruling out other underlying causes of respiratory distress.

## Learning points

- ▶ Pleural effusions in the context of SARS-CoV-2 infection warrant systematic identification of other possible aetiologies, including *Mycobacterium tuberculosis*.
- ▶ SARS-CoV-2 pneumonitis may occur in addition to other infectious diseases, requiring contact tracing and isolation, such as *M. tuberculosis*.
- ▶ Clinicians should consider tension hydrothorax in haemodynamically compromised patients on a background of SARS-CoV-2.

**Contributors** MFB was a member of the medical team who clerked the patient, played a significant role in writing the manuscript, obtained patient consent and followed-up the patient. MS contributed to writing the manuscript and edited the manuscript. RK is a specialist registrar in radiology and was a member of the medical team who clerked the patient, provided expert opinion, supervised and contributed to writing the manuscript.

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