EUROPEAN CLINICAL RESPIRATORY JOURNAL
2022, VOL. 9, 2028423
https://doi.org/10.1080/20018525.2022.2028423

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CASE REPORT

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Post-COVID-19 pneumonia pneumatoceles: a case report

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ABSTRACT

Introduction: Pneumatocele formation in COVID-19 pneumonia is arguably a common occurrence.

Case Presentation: We present a case of pneumatoceles, developing as a sequel of COVID-19 infection. We argue that pneumatocele formation in COVID-19 pneumonia is a common occurrence. Importantly pneumothorax, which can lead to a raised morbidity and mortality in these patients, can be a complication of a pneumatocele rupture.

Conclusion: As pneumatocele in COVID-19 pneumonia patients can lead to life-threatening complications, we emphasize the need to formulate appropriate and standardized monitoring and management guidelines. Our literature review also discusses various plausible mechanisms leading to pneumatocele formation and points to management strategies that may prevent pneumatocele formation and its complications.

ARTICLE HISTORY
Received 22 December 2021
Accepted 9 January 2022

KEYWORDS
Pneumatocele; cyst; COVID-19; pneumothorax

Introduction

Pneumatocele formation in COVID-19 pneumonia is arguably a common occurrence. While Shi et al reported cystic changes in 10% of their subjects[1], Werberich et al. reported similar changes in up to 37.5% in their retrospective study of 78 consecutive patients with COVID-19 infection [2]. It has also been reported in pediatric COVID-19 pneumonia [3]. Pneumothorax has been associated with COVID-19 infection and has been reported to cause higher morbidity and mortality in COVID-19 patients [4–7]. Anthony et al reported that approximately 1% of all patients requiring hospitalization with COVID-19 develop pneumothorax [8]. Rupture of pneumatoceles is one of the mechanisms leading to pneumothorax in these patients. We present a case of a patient who developed pneumatoceles as a sequela of his COVID-19 pneumonia. We also discuss various plausible mechanisms leading to cyst formation in COVID-19 patients.

Case presentation

A 34-year-old gentleman, a lifelong non-smoker, and an IT consultant was transferred to our hospital from a quarantine facility with a worsening shortness of breath and oxygen desaturation. Six days before his hospital transfer, he was admitted to a quarantine facility with a positive COVID-19 real-time reverse transcription-polymerase chain reaction (rRT-PCR) test; and has had symptoms of cough, fever, and body aches for 3 days. His past medical history included hypothyroidism. On admission to the hospital, he was febrile at 38°C and had 25 breaths/minute respiratory rate. His oxygen saturations were 89% on air, which improved to over 94% with 4 L/min oxygen administration. His chest was clear on auscultation. His CXR showed bilateral infiltrates (Figure 1(a)). His blood tests revealed a CRP of 121.6 mg/L, Ferritin 1730 ug/L, D-dimer of <0.30 mg/L, WBC of 7.0 × 10^3/μL with a lymphocyte count of 1.2 × 10^3/μL and LDH of 507 U/L. He was diagnosed with severe COVID-19 pneumonia and was commenced on treatment in accordance with the local guidelines (ceftriaxone, azithromycin, dexamethasone, favipiravir, and prophylactic enoxaparin). His symptoms improved during his hospital stay, and he was discharged after 15 days of his hospital admission. On discharge, he was off oxygen and had no significant chest symptoms.

Approximately 4 weeks after his hospital discharge, he attended the emergency department with intermittent shortness of breath. He was apyrexial, clinically stable, and his oxygen saturations were over 94% on

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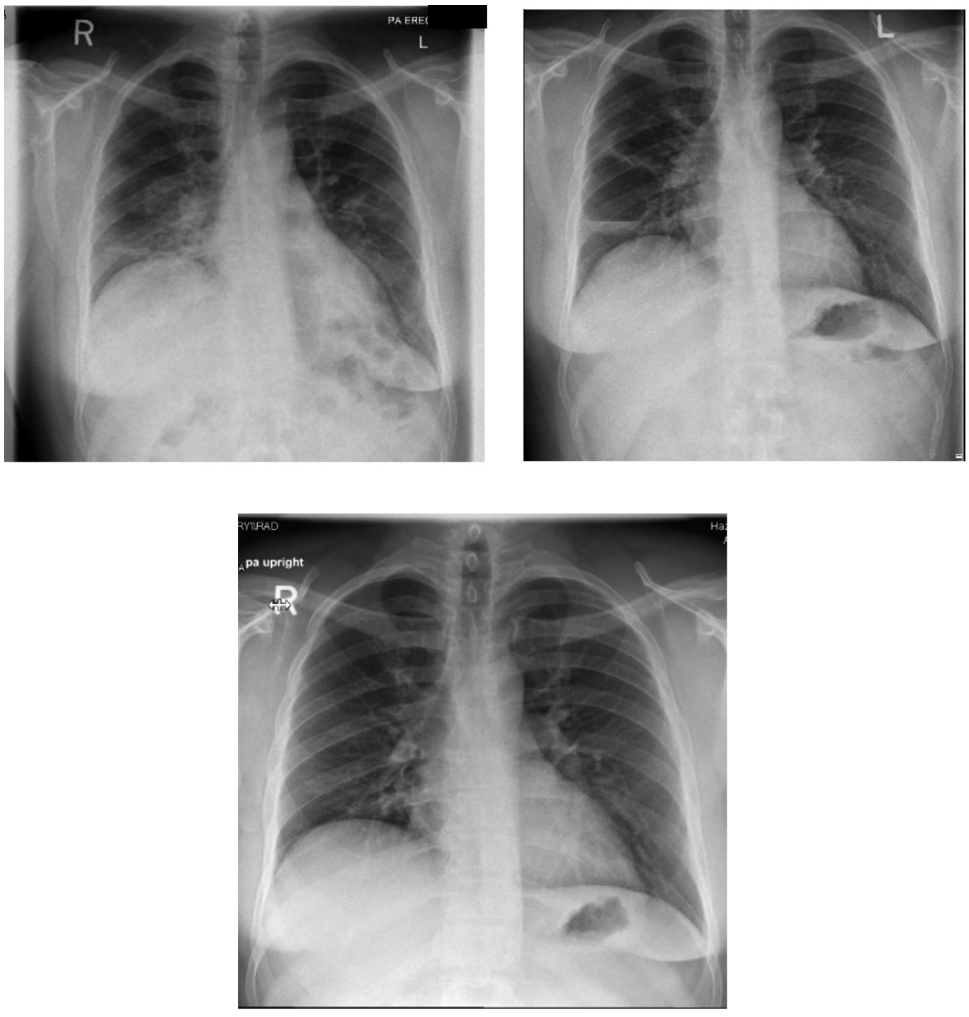

Figure 1. (a) Chest radiograph: multiple bilateral mid and lower zone airspace opacities. (b) Chest radiograph: pneumatocele with air fluid level in right lower lobe. (c) Follow up chest radiograph: resolution of pneumatocele in the follow up CXR.

air. He had a chest X-ray which indicated a cavitating lesion on the right side (Figure 1(b)). His subsequent CT chest indicated findings consistent with bilateral pneumatoceles. There were two pneumatoceles on the right side and one on the left side (Figure 2 indicating the largest pneumatocele on the right side). Other CT chest findings included bilateral ground-glass changes. The pneumatocele also had an air-fluid level. His blood tests revealed unremarkable inflammatory markers. His WBCs were also normal. He was arranged to have a bronchoscopy. His bronchial washings did not grow any organism, and they were also negative for acid-fast bacilli. As the pneumatocele had an air-fluid level, there was a possibility of infection. However, as he was apyrexial, clinically well, had unremarkable inflammatory markers, and bronchial washings did not grow any organism, he was not commenced on any antibiotics. He was sent home and was arranged to have a follow-up with a repeat CXR. A three-week CXR's follow-up revealed almost complete resolution of his pneumatocele (Figure 1(c)).

Discussion

Pneumatoceles are gas-filled intrapulmonary cystic spaces with thin walls. They are also referred to as pseudocyst in the literature since they lack an epithelial lining and have walls that are formed by the lung tissue. Most often, they occur as a sequel to acute pneumonia. Various mechanisms have been described causing pneumatocele formation. One of the processes consistently reported in the literature is the 'endobronchial check valve' mechanism [9,10]. This allows air trapping and development of cyst formation distal to the obstruction. Depending upon the duration and severity of blockade, cystic spaces may become

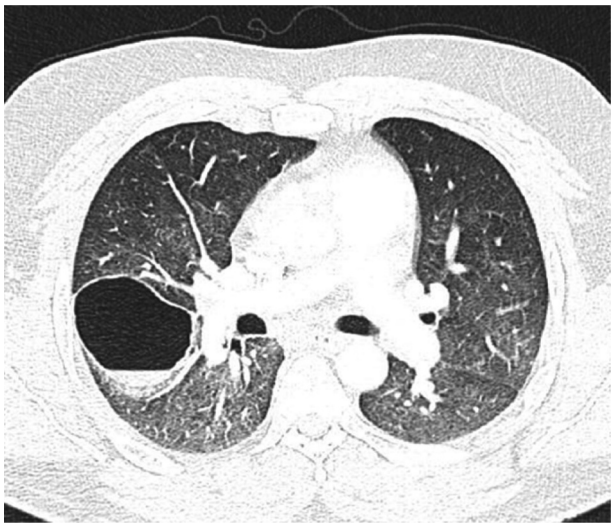

Figure 2. CT Chest: Right lower lobe pneumatocele with air fluid level. Other findings included bilateral ground glass changes.

impressively large. In COVID-19 patients, there has been mention of inflammatory exudates causing blockade of airways [11,12] and this was perhaps the cause of a large pneumatocele formation in our case. Liu et al [12] mentioned the role of N acetylcysteine in the treatment of COVID-19 cases. In addition to their role as antioxidants and anti-inflammatory agents, clearing the airways, keeping them patent, and helping with the gas exchange can potentially also help decrease the number of cyst formations and their subsequent complications. Other prominent mechanism described is diffuse alveolar damage, eventually leading to necrosis in the lung parenchyma. Necrosis is followed by focal air collections in the interstitial tissue, which can then expand and extend, causing cyst formation. As the air expands, its shearing effect on the lung parenchyma and small blood vessels can lead to bleeding and blood-stained fluid accumulation in the cavity, as most likely happened in our case. We reached this conclusion as pneumatocele in our case had the fluid; however, the inflammatory markers were unremarkable, and bronchial washings did not reveal any organism. Also, the cavity was resolved without any antibiotics. Non-infectious causes of pneumatocele include positive pressure ventilation, chest trauma, and chemical pneumonitis. Lung cavitation can also occur because of aseptic liquefaction of pulmonary infarctions [13]. A pulmonary embolus may lead to pulmonary infarction, and pulmonary emboli have been commonly diagnosed in COVID-19 cases [14], proposing another mechanism leading to pneumatocele formation in COVID-19 cases. In our case, however, there was no evidence of pulmonary embolism.

A pneumatocele rupture can cause complications, including pneumothorax or pneumomediastinum. Hence it is crucial to identify cystic changes on the CT chest. This would assist physicians in risk stratifying COVID-19 patients for these complications. As almost all cases of pneumatoceles resolve spontaneously, they are treated conservatively. Percutaneous catheter drainage or surgical intervention is considered in case of tension pneumatocele and cardiovascular compromise. Infected pneumatocele may also require consultation with thoracic surgeons. However, it has been debated to consider inserting percutaneous drain for management of pneumatocele in COVID-19 pneumonia cases with a high likelihood of clinical deterioration [15]. Pneumatocele rupture resulting in a pneumothorax in severe COVID-19 patients, especially those on positive pressure ventilation, can cause life-threatening clinical deterioration. Brahmbhatt et al reported treatment of a case of pneumatocele in COVID-19 patient on high flow nasal cannula with insertion of a pigtail catheter [16]. Although there seems to be a benefit of this approach in carefully selected patients, this can cause complications, including the development of bronchopleural fistula, and hence should be carefully advocated balancing risks and benefits. It also points to a need for standardized guidelines for managing pneumatoceles in COVID-19 cases, especially those requiring intubation and mechanical ventilation.

Conclusion

While most post-pneumonia pneumatoceles resolve spontaneously, we emphasize the importance of careful monitoring and follow-up of these patients, as their complications, notably pneumothorax, have been associated with increased morbidity and mortality. We also hypothesize a possible role of mucolytics and sputum clearance techniques in COVID-19 patients. In addition, by maintaining airways patent 'ball-valve obstruction' which may lead to pneumatocele formation and subsequent complications, can potentially be avoided. Finally, there is also a need for standardized guidelines regarding percutaneous drainage of pneumatocele in COVID-19 pneumonia patients who are at high risk of clinical deterioration.

Acknowledgments

None

Authors' contributions

WJ, MS, AS, SR, AJN: Data Collection, Literature Search, Manuscript Preparation. All authors read and approved the final manuscript.

Availability of Data and Material

All data generated or analyzed during this study are included in this published article.

Consent for publication

The consent for publication was obtained from the patient.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Funding

This study was not funded.

Ethics approval and consent to participate

The article describes a case report. Therefore, no additional permission from our Ethics Committee was required.

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