Acinetobacter cavitations in COVID-19 interstitial pneumonia: a case report and review of the literature

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Abstract

Acinetobacter baumannii is commonly known to cause infection in immunocompromised patients. During COVID-19 pandemic, outbreaks of multidrug-resistant organisms, including Acinetobacter, have been well documented in acute care hospitals, particularly among critically ill patients. In the case reported, a woman was admitted to our ICU because of a severe COVID-19 pneumonia. During her stay, she worsened due to Acinetobacter-related lung cavitations and only after proper antibiotic treatment she eventually recovered. To our knowledge, very few cases have been reported pointing to Acinetobacter as a causal agent for the acute development of lung cavities, especially in COVID-19 patients.

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Introduction

Acinetobacter baumannii is commonly asserted to cause infection in immunocompromised patients as a result of exposure to broad-spectrum antibiotics and disruption of anatomic barriers with the use of ventilators, central lines and urinary catheters commonly used in critical care settings.[1,2]

During COVID-19 pandemic, outbreaks of multidrug-resistant organisms, including Acinetobacter, have been well documented in acute care hospitals, particularly among critically ill patients.[3-5] The rate of *A. baumannii* infection during the pandemic was found to be higher when compared to pre-pandemic data.[2]

We report a singular case of COVID-19 pneumonia superinfected by *A. baumannii* which developed a severe lung cavitation pattern. In order to further understand the complexity of this case, we also searched PubMed for similar records, dividing the review in two sections: cases of lung cavitations in Acinetobacter pneumonia and cases of lung cavitations in COVID-19 pneumonia.

Case Report

In January 2021, a 60-year-old woman presented dyspnoea 10 days after a close contact with a COVID-19 positive relative. She had a medical history of diabetes type II and hypercholesterolemia and was on regular medication with metformin and statin. She denied tobacco use and recent travelling. In the Emergency Department she was diagnosed with COVID-19 related pneumonia due to a nasopharyngeal RT-PCR swab and presence of bilateral interstitial infiltrates on chest X ray. She was admitted to the general medicine ward where she was administered oxygen-therapy with High Flow Nasal Cannula (HFNC) and immunomodulatory therapy with tocilizumab, a monoclonal antibody against the interleukin-6 receptor. The progressive worsening of symptoms led to an escalation of oxygen-therapy from Continuous Positive Airway Pressure (CPAP) to Non-Invasive Ventilation (NIV) until, five days later, she was intubated and put on prolonged analgo-sedation and pharmacological paralysis. She was therefore admitted to our ICU. A full thoracic CT scan including high resolution computed tomography (HRCT) and computed tomography angiography (CTA) showed

multiple ground glass areas, while excluding pulmonary embolism. Pronation cycles were started due to respiratory impairment and need for increased ventilatory support, with some benefit. A temporary percutaneous tracheostomy was performed as it became evident that prolonged mechanical ventilation was required. A few days later she presented symptoms from low respiratory tract super-infection such as high fever, shivers, purulent secretions, slowly progressing to septic shock. Usual diagnostic work-up for sepsis included blood, urine and sputum cultures. An empiric antibiotic therapy with piperacillin tazobactam and gentamicin was started according to local protocols. Because a bronchoaspirate culture turned positive to *Staphylococcus intermedius* and *Serratia marcescens* (with a MIC to pip/tazo ≤4) but also to Multidrug-Resistant (MDR) *A. baumannii*, intravenous colistin was added. The MDR *A. baumannii* isolated showed non-susceptibility to ciprofloxacin, gentamycin and meropenem on the antibiotic susceptibility test. Another CT scan was performed which showed a new image of consolidation in the Right Middle Lobe (RML) with a 6 centimetres diameter air-filled cavity inside it. Even though she had developed Acute Respiratory Distress Syndrome (ARDS), it is worth noting that it had not caused severe lung stiffness. Compliance values and ventilatory pressures stayed contained through the whole disease, so that barotrauma was not taken into account further in this case. Conversely, the presence of a large pulmonary cavity in a clinical setting of septic shock and immunosuppression warranted a full work-up for usual cavitary diseases, including Galactomannan (GM) on both sputum and serum, culture for *Aspergillus* and other filamentous fungi, *Nocardia* spp. and Acid-Fast Bacilli smear and culture. In the meantime, prompt anti-*Aspergillus* therapy with voriconazole was started. The antibiotics were also changed into colistin and meropenem. Despite the antifungal therapy, no clinical improvement occurred, with fever and inflammation worsening with time. Laboratory tests showed leucocytosis with a peak of 29×10^9/L, 90% of which being neutrophils, and a sudden increase in C Reactive Protein (CRP) up to 309 mg/L. Procalcitonin (PCT) was found to be weakly positive with a value of 0.48 ug/L. A CT scan check displayed an enlargement of the cavity previously noted in the RML, together with the appearance of a contralateral cavity in the lingula (Figure 1). At that stage of the disease, both cavities had acquired the typical features of abscesses in the context of a large consolidation and seemed not to respond to the current therapy. GM together with cultures for fungi were also found to be negative on both serum and sputum, warranting the interruption of voriconazole. Six more bronchoaspirate samples analysed during this septic escalation (almost a time span of one month) revealed persistence of *A. baumannii*, that by that time had turned into an extensively drug-resistant pattern (XDR, only susceptible to colistin). On the contrary, *S. marcescens* was never isolated again. In accordance with the infectious disease consultant, *A. baumannii* was targeted with the recently approved antibiotic cefiderocol, thanks to a compassionate-use programme. Metronidazole and fosfomycin were added to broaden the spectrum of antimicrobial activity. Serial sputum and bronchoaspirate cultures were conducted to follow up on the infection and found a progressive decrease in the number of XDR *A. baumannii* colony-forming units. A confirmation bronchoalveolar lavage was also performed. The patient improved with the new treatment and gradually underwent a progressive recovery. After fourteen days of treatment, antibiotics were discontinued and weaning from mechanical ventilation was completed. The sputum eventually turned negative. The CT revealed a marked decrease of both cavities. After two months from the admission she was COVID-19 free and was discharged in stable conditions to a long-term facility, to continue pulmonary and physical rehabilitation. Eventually, the patient showed a good long-term clinical outcome with full recovery and no perceived functional limitation. The latest CT scan found residual lung cavities consistently smaller than previously found, meaning that the radiological picture was on its way to healing too (Figure 2).

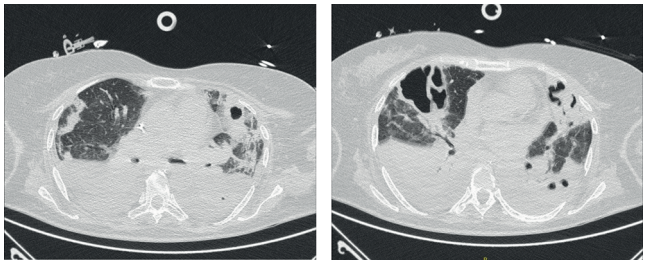

Figure 1. Lung CT scan showing bilateral dorsal consolidated tissue and pleural effusion, ground-glass opacities, an image of consolidation in the Right Middle Lobe (RML) with an abscessual cavity, together with a contralateral cavity in the lingula.

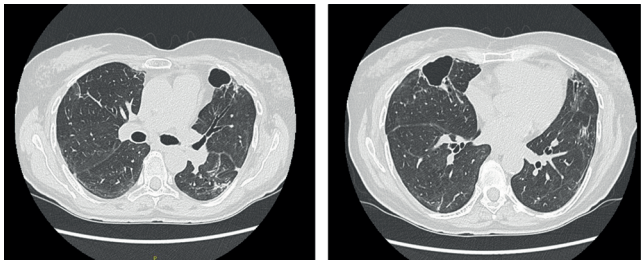

Figure 2. Small residual lung cavities in fully recovered lung tissue.

Discussion

Acinetobacter spp. are glucose-non-fermentative, non-motile, non-fastidious, catalase-positive, oxidative-negative, strictly aerobic Gram-negative coccobacilli.[6-8] While Acinetobacter species are ubiquitous and can be found in several ecological niches including environment, food and water, animals and human (as part of the human skin and enteric flora), *A. baumannii* is found almost exclusively in the hospital environment, particularly in intensive care units.[6,7,9] Among Acinetobacter species, *A. baumannii* is the most important member associated with hospital-acquired infections worldwide.[10] It was previously regarded as a low-grade pathogen, but it is now a major pathogen responsible for opportunistic infections of the skin, bloodstream, urinary tract, and other soft tissues.[6,11] The frequency of community-acquired *A. baumannii* infections has been increasing gradually,[10] still it accounts for up to 20% of all ICUs infections,[8] where ventilator-associated pneumonia and bloodstream infections are the most common, and mortality rates can reach 35%.[12]

A. baumannii has been classified as one of the six most serious MDR organisms, named by the World Health Organization with the acronym "ESKAPE", together with *Enterococcus faecium*, *Staphylococcus aureus*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, and *Enterobacter* species.[13,14] Several virulence factors have been identified by genomic and phenotypic analyses, including outer membrane porins, phospholipases, proteases,

lipopolysaccharides (LPS), capsular polysaccharides, protein secretion systems, and iron-chelating systems.[10,11] Resistance mechanisms include enzymatic degradation of drugs, target modifications, multidrug efflux pumps, and permeability defects.[10,11] Aside from microbiological mechanisms, it should be considered that the nature of the host (*i.e.* human patient) may also play a large part in the outcome of infections caused by *A. baumannii*, with patients in hospitals now being much more vulnerable because of their serious underlying conditions and increased use of highly selective antibiotics, indwelling lines and other invasive devices.[12] Specific host factors predisposing to *A. baumannii* infection were identified and they include major surgery, major trauma especially major burns and prematurity newborns.[15]

Ventilator-associated pneumonia

In hospitalized patients, isolating *A. baumannii* from the respiratory tracts does not help distinguish upper airway colonization from true pneumonia. However, true Ventilator-Associated Pneumonia (VAP) due to *A. baumannii* occurs and when bacteraemic it has a particularly poor prognosis.[6,15] VAP caused by Acinetobacter was previously considered to have a similar prognosis to pneumonia caused by other virulent pathogens and Acinetobacter itself was regarded to have a low-grade pathogenicity.[16] During the past decades the rapid development of resistance to the majority of antimicrobials has led MDR Acinetobacter to gain a higher mortality rate in critically ill patients. Carbapenem resistance was almost universal in the isolates collected from patients with VAP in Southern Europe (Italy, Greece, Spain) in a characterization study.[17] The emergence of XDR/PDR *A. baumannii* is paving the way for new molecules such as cefiderocol as well as for the revival of older antibiotics, like colistin.[18] Risk factors for VAP due to *A. baumannii* include previous neurosurgery, head trauma or large-volume aspiration as well as prolonged hospital stay and mechanical ventilation, prior episodes of sepsis, reintubation and prior antibiotic use.[16,19] Previous comorbidities and degree of associated organic injury seem to be more important factors in the prognosis, together with associated bacteremia which leads to a greater mortality rate.

Acinetobacter infection diagnosis is based on the growth of the pathogen from a patient specimen (*e.g.*, sputum, blood) in the setting of other clinical findings that suggest an infection at that site, such as fever, leucocytosis, increased respiratory secretions, need for additional respiratory support, or a new abnormality on chest imaging. Classically, the definition of VAP include the presence of clinical, radiological and microbiological findings. More recently, a faster VAP screening has been proposed, including the presence of new chest x-ray infiltrates plus one of the three clinical variables (fever ≥38°C, leucocytosis or leukopenia, and purulent secretions).[20] Unfortunately, COVID-19-related pneumonia usually presents with diffuse interstitial infiltrates so that, like ALI/ARDS, it is difficult to demonstrate a deterioration of radiological images. In such cases, at least one of fever ≥38°C, leucocytosis or leukopenia, and purulent secretions may warrant initial screening.[20]

Lung cavitations in Acinetobacter pneumonia

As in most cases of VAP, Acinetobacter pulmonary infection and pneumonia show no specific radiological patterns. An image of chest X-ray infiltrates is the common finding taken into account as VAP diagnostic tool. However, a review of the medical literature revealed a few reported cases regarding unusual radiological presentation of Acinetobacter pneumonia. Hunt *et al.* reported three cases of ICU ventilator-associated pneumonia with the formation of pneumatoceles due to Acinetobacter infection.[21] They were actually late-onset super-infections associated with or responsible for the pneumatoceles, which seemed to have been caused primarily by very high PEEP and barotrauma. In our case the cavity was firstly referred to as a pneumatoceles by the radiological report, but it soon developed an air fluid level and it was never associated with pneumothorax or pneumomediastinum as it happened in the three cases reported by Hunt. Moreover, our patient did not develop such an ARDS pattern to require very high pressure ventilation, thus barotrauma does not seem to explain her radiological picture. Hospital acquired lung abscesses associated to Acinetobacter were also reported in two different post-operative settings: Markelic *et al.* described the first case of MDR *A. baumannii*-related multiple lung abscesses after lung transplantation in a young woman affected by cystic fibrosis;[22] Cheng *et al.* reported the case of a tricuspid valve replacement in a young drug-abuser man, complicated by multiple lung abscesses and thoracic empyema.[23] Interestingly, in both cases Acinetobacter was not the only pathogen found, as it was isolated together with *P. aeruginosa* in the first case and *C. albicans* in the second one. In one case report MDR *A. baumannii* was responsible for a hospital-acquired necrotizing and cavitating pneumonia which also evolved into bronchopleural fistula and hydropneumothorax.[24] The man did not seem to have any structural disease or predisposing host condition, apart from diabetes and a brain tumor for the treatment of which he was admitted to a hospital in the first place.

Regarding community-acquired pneumonia, it's easier to find reports of severe cases since community-acquired *A. baumannii* pneumonia is more serious than nosocomial pneumonia and is known to be generally fulminant, with rates of mortality as high as 60%.[12] However, searching for atypical and cavitary presentation, only four cases were found: an 11-month-old girl diagnosed with septic pulmonary embolism after radiological evidence of multiple cavitary nodules and emboli;[25] a 16-year-old female with multiple lung abscesses;[26] a young adult with right lobe necrotizing pneumonia and lung abscess formation;[27] four large cavities and nodules due to an *A. pittii* strain in a smoking patient with systemic lupus.[28] All of them completely healed after appropriate and prolonged antibiotics. Not surprisingly, in all the community-acquired cases, Acinetobacter resulted susceptible to most antibiotics, in contrast with the prevalence of MDR *A. baumannii* found in nosocomial infections reports.

Lung cavitations in COVID-19 pneumonia

Until early COVID-19 pandemic, no lung cavitations were recognized in the range of radiological findings associated to COVID-19 pneumonia,[29] with the most common patterns being Ground Glass Opacification (GGO), sometimes with superimposed consolidative opacities.[30] In May 2020 Xu *et al.* described the first case of a COVID-19 positive patient presenting with a cavitary lesion.[31] No secondary infectious agents were found and it was therefore suggested that a link to the viral infection itself could not be excluded. One more unsolved case, where no causative agents or diseases were found, was later described by Afrazi *et al.*:[32] the patient presented with cavities combined with pneumothorax and recovered after thoracostomy. The two cited cases have in common an early presentation with cavitary lesions in COVID-19 patients, having no previous record of normal CT scan. On this basis, the temporal criteria and direct causative link is arguable. Moreover, all radiological reports and reviews agree that cavities, when present, are a late-stage finding in COVID-19 pneumonia, whether a cause is identified or not.[30,33] More cases, this time with delayed appearance of cavities and with no identified pathogen, were interpreted as having an infectious aetiology due to clinical signs and good response to antibiotics.[34-36]

Apart from the unsolved cases above cited, the majority of lung

cavitary lesions in COVID-19 patients were linked to a clear aetiology: thromboembolism, with cavity being the evolutionary stage of pulmonary infarction or microinfarcts;[37,38] infectious disease with one case of positivity to Aspergillus and *P. aeruginosa*,[39] one case as an evolution of nosocomial pneumonia due to *E. faecalis*,[40] one necrotizing pneumonia with *E. coli* isolation[41] and a case series of *Mycobacterium tuberculosis* (MTB) positivity in an endemic area.[42] In their review Mishra *et al.* identified a few more cases of co-infection of COVID-19 and pulmonary MTB presenting with typical cavitary lung lesions,[43] suggesting that in endemic areas and in migrants it is not an unusual finding. Zoumot *et al.* came to a similar conclusion after a retrospective review of 689 COVID-19 hospitalized patients, 12 of which had developed lung cavitations with features of abscesses during hospital stay and most of them had bacterial superinfections.[33] Remarkably, all of them had received treatment with tocilizumab, leading the authors to speculate on the multifactorial nature of such lesions with infections, immunosuppression, inflammation and microinfarcts as contributing factors.

Only two cases of *A. baumannii* related lung cavitations in a COVID-19 patient were found, both published in 2022. A Russian case of a 61-year-old male patient with confirmed COVID-19 infection who developed nosocomial pneumonia complicated by lung abscess associated with multi-drug-resistant isolates of *K. pneumoniae* and *A. baumannii* was described by Rachina *et al.*[44] The lung abscess was a late finding, concomitant with clinical worsening and positivity of sputum samples, that healed after antibiotic therapy and clinical recovery. The second report describes a high-risk long-COVID patient who developed cavitary lesions in both lungs colonized by Acinetobacter.[45] Neither case turned into XDR *A. baumanni* and neither required cefiderocol use.

Enumerating cases of the so-called bullae or emphysema recorded in COVID-19 patients is beyond the scope of this review, since those lesions are typically and predictably related to barotrauma or direct trauma. However, pneumatoceles (*i.e.* thin-walled, air-filled intraparenchymal cysts) must be paid close attention, since they might either be caused by localized bronchiolar infectious processes or be superinfected after development. In fact, findings of pneumatoceles and pulmonary cysts, alone or associated to pneumothorax, are reported as usually appearing some weeks after onset of symptoms due to COVID-19, in spontaneously breathing patients suffering from cough and shortness of breath.[46,47] Alternatively, some records refer to long-COVID patients after return to spontaneous breathing.[48,49] Most of them were treated with pigtail catheter placement and two required additional surgery.[48,49] None of them was temporally or clinically related to any septic manifestation, making room to mechanical hypotheses as the cause of tension pneumatoceles. In one case septic manifestations were also present and surgical resection of two pulmonary cysts led to recovery.[50] Even though most of cases have not identified a responsible pathogen, there is a good chance that some lung cavitations might be related to pathogens that have not been isolated due to technical difficulties or slow growth.

Conclusions

Until now, very few cases have been reported pointing to Acinetobacter as a causal agent for the acute development of lung cavities, especially in COVID-19 patients. Even if some hypothesis has been posed, its pathogenesis is so far unclear due to its rarity and so much is yet to be learned. Bringing to light such rare cases can help us ring the bell of possible super-infection every time new cavitary lesions are found in a patient rapidly developing septic clinical signs. Prompt antibiotic therapy according to antibiotic stewardship and specialist consultation is warranted. In the described cases, complications were present but eventually the right treatments proved successful, suggesting there is a good chance of full recovery.

References

1. Wong D, Nielsen TB, Bonomo RA, et al. Clinical and pathophysiological overview of Acinetobacter infections: A century of challenges. Clin Microbiol Rev 2017;30:409–47.
2. Boral J, Genç Z, Pınarlık F, et al. The association between *Acinetobacter baumannii* infections and the COVID-19 pandemic in an intensive care unit. Sci Rep 2022;12:1–7.
3. Eckardt P, Canavan K, Guran R, et al. Containment of a carbapenem-resistant *Acinetobacter baumannii* complex outbreak in a COVID-19 intensive care unit. Am J Infect Control 2022; 50:477–81.
4. Perez S, Innes GK, Walters MS, et al. Increase in Hospital-Acquired Carbapenem-Resistant *Acinetobacter baumannii* Infection and Colonization in an Acute Care Hospital During a Surge in COVID-19 Admissions — New Jersey, February–July 2020. Morbidity and Mortality Weekly Report/Centers for Disease Control and Prevention. 2020;69.
5. Thoma R, Seneghini M, Seiffert SN, et al. The challenge of preventing and containing outbreaks of multidrug-resistant organisms and *Candida auris* during the coronavirus disease 2019 pandemic: report of a carbapenem-resistant *Acinetobacter baumannii* outbreak and a systematic review of the literature. Antimicrob Resist Infect Control 2022;11:12.
6. Peleg AY, Seifert H, Paterson DL. *Acinetobacter baumannii*: Emergence of a successful pathogen. Clin Microbiol Rev 2008;21:538–82.
7. Carvalheira A, Silva J, Teixeira P. Acinetobacter spp. in food and drinking water – A review. Food Microbiol 2021;95.
8. Nasr P. Genetics, epidemiology, and clinical manifestations of multidrug-resistant *Acinetobacter baumannii*. J Hosp Infect 2020;104:4–11.
9. Fournier PE, Richet H. The epidemiology and control of *Acinetobacter baumannii* in health care facilities. Clin Infect Dis 2006;42:692–9.
10. Lin M-F. Antimicrobial resistance in *Acinetobacter baumannii*: From bench to bedside. World J Clin Cases 2014;2:787.
11. Lee CR, Lee JH, Park M, et al. Biology of *Acinetobacter baumannii*: Pathogenesis, antibiotic resistance mechanisms, and prospective treatment options. Front Cell Infect Microbiol 2017;7:55.
12. Antunes LCS, Visca P, Towner KJ. *Acinetobacter baumannii*: Evolution of a global pathogen. Pathog Dis 2014;71:292–301.
13. Rice LB. Federal funding for the study of antimicrobial resistance in nosocomial pathogens: No ESKAPE. J Infect Dis 2008;197:1079–81.
14. Boucher HW, Talbot GH, Bradley JS, et al. Bad bugs, no drugs: No ESKAPE! An update from the Infectious Diseases Society of America. Clin Infect Dis 2009;48:1–12.
15. Dijkshoorn L, Nemec A, Seifert H. An increasing threat in hospitals: Multidrug-resistant *Acinetobacter baumannii*. Nat Rev Microbiol 2007;5:939–51.
16. Garnacho-Montero J, Ortiz-Leyba C, Fernández-Hinojosa E, et al. *Acinetobacter baumannii* ventilator-associated pneumonia: Epidemiological and clinical findings. Intensive Care Med 2005;31:649–55.
17. Nowak J, Zander E, Stefanik D, et al. High incidence of pandrug-resistant *Acinetobacter baumannii* isolates collected from

patients with ventilator-associated pneumonia in Greece, Italy and Spain as part of the MagicBullet clinical trial. J Antimicrob Chemother 2017;72:3277–82.
18. Karakonstantis S, Kritsotakis EI, Gikas A. Pandrug-resistant gram-negative bacteria: A systematic review of current epidemiology, prognosis and treatment options. J Antimicrob Chemother 2020;75:271–82.
19. Inchai J, Pothirat C, Bumroongkit C, et al. Prognostic factors associated with mortality of drug-resistant *Acinetobacter baumannii* ventilator-associated pneumonia. J Intensive Care 2015; 3:9.
20. Bassetti M, Taramasso L, Giacobbe DR, Pelosi P. Management of ventilator-associated pneumonia: Epidemiology, diagnosis and antimicrobial therapy. Expert Rev Anti Infect Ther 2012; 10:585–96.
21. Hunt JP, Buechter KJ, Fakhry SM. Acinetobacter calcoaceticus pneumonia and the formation of pneumatoceles. J Trauma - Inj Infect Crit Care 2000;48:964–70.
22. Markelić I, Jakopović M, Klepetko W, et al. Lung abscess: an early complication of lung transplantation in a patient with cystic fibrosis. Int J Organ Transplant Med 2017;8:213–6.
23. Cheng YF, Hsieh YK, Wang BY, et al. Tricuspid valve infective endocarditis complicated with multiple lung abscesses and thoracic empyema as different pathogens: A case report. J Cardiothorac Surg 2019;14:41.
24. Widysanto A, Liem M, Puspita KD, Pradhana CML. Management of necrotizing pneumonia with bronchopleural fistula caused by multidrug-resistant *Acinetobacter baumannii*. Respirol Case Reports 2020;8:e00662.
25. Wade P, Ananthan A, David J, Ghildiyal R. A case of acinetobacter septic pulmonary embolism in an infant. Case Rep Infect Dis 2016;2016:5241571.
26. Kokkonouzis I, Christou I, Athanasopoulos I, et al. Multiple lung abscesses due to acinetobacter infection: a case report. Cases J 2009;2:9347.
27. Yang CH, Chen KJ, Wang CK. Community-acquired Acinetobacter pneumonia: A case report. J Infect 1997;35: 316–8.
28. Larcher R, Pantel A, Arnaud E, et al. First report of cavitary pneumonia due to community-acquired *Acinetobacter pittii*, study of virulence and overview of pathogenesis and treatment. BMC Infect Dis 2017;17:477.
29. Kaufman AE, Naidu S, Ramachandran S, et al. Review of radiographic findings in COVID-19. World J Radiol 2020;12: 142–55.
30. Salehi S, Abedi A, Balakrishnan S, Gholamrezanezhad A. Coronavirus disease 2019 (COVID-19): A systematic review of imaging findings in 919 patients. Am J Roentgenol 2020; 215:87–93.
31. Xu Z, Pan A, Zhou H. Rare CT feature in a COVID-19 patient: cavitation. Diagnostic Interv Radiol 2020;26:380–1.
32. Afrazi A, Garcia-Rodriguez S, Maloney JD, Morgan CT. Cavitary lung lesions and pneumothorax in a healthy patient with active Coronavirus-19 (COVID-19) viral pneumonia. Interact Cardiovasc Thorac Surg 2021;32:150–2.
33. Zoumot Z, Bonilla MF, Wahla AS, et al. Pulmonary cavitation: an under-recognized late complication of severe COVID-19 lung disease. BMC Pulm Med 2021;21:24.
34. Muheim M, Weber FJ, Muggensturm P, Seiler E. An unusual course of disease in two patients with COVID-19: pulmonary cavitation. BMJ Case Rep 2020;13:e237967.
35. Zamani N, Aloosh O. Lung abscess as a complication of COVID-19 infection, a case report. Clin Case Reports 2021;9: 1130–4.
36. Renaud-Picard B, Gallais F, Riou M, et al. Delayed pulmonary abscess following COVID-19 pneumonia: A case report. Respir Med Res 2020;78:100776.
37. Marchiori E, Nobre LF, Hochhegger B, Zanetti G. Pulmonary infarctions as the cause of bilateral cavitations in a patient with COVID-19. Diagnostic Interv Radiol 2020;27:690–1.
38. Selvaraj V, Dapaah-Afriyie K. Lung cavitation due to COVID-19 pneumonia. BMJ Case Rep 2020;13:e237245.
39. Ammar A, Drapé JL, Revel MP. Lung cavitation in COVID-19 pneumonia. Diagn Interv Imaging 2021;102:117–8.
40. Amaral LTW, Beraldo GL, Brito VM, et al. Lung cavitation in COVID-19: co-infection complication or rare evolution? Einstein (São Paulo) 2020;18:eAI5822.
41. Peeters K, Mesotten D, Willaert X, et al. Salvage lobectomy to treat necrotizing SARS-CoV-2 pneumonia complicated by a bronchopleural fistula. Ann Thorac Surg 2021;111:e241–3.
42. Yousaf Z, Khan AA, Chaudhary HA, et al. Cavitary pulmonary tuberculosis with COVID-19 coinfection. IDCases 2020;22: e00973.
43. Mishra A, George AA, Sahu KK, et al. Tuberculosis and COVID-19 co-infection: an updated review. Acta Biomed 2020;92:e2021025.
44. Rachina S, Kiyakbaev G, Antonova E, et al. A clinical case of nosocomial pneumonia as a complication of COVID-19: how to balance benefits and risks of immunosuppressive therapy? Antibiotics 2023;12.
45. Chowdhury T, Mainali A, Bellamkonda A, Gousy N. Acinetobacter: a rare cause of rapid development of cavitary lung lesion following COVID-19 infection. Cureus 2022;14.
46. Brahmbhatt N, Tamimi O, Ellison H, et al. Pneumatocele and cysts in a patient with severe acute respiratory syndrome coronavirus 2 infection. J Thorac Cardiovasc Surg Tech 2020;4: 353–5.
47. Sanivarapu RR, Farraj K, Sayedy N, Anjum F. Rapidly developing large pneumatocele and spontaneous pneumothorax in SARS-CoV-2 infection. Respir Med Case Reports 2020;31: 101303.
48. Capleton P, Ricketts W, Lau K, et al. Pneumothorax and pneumatocoele formation in a patient with COVID-19: a case report. SN Compr Clin Med 2021;1–4.
49. Hamad AM. Post COVID-19 large pneumatocele: clinical and pathological perspectives. Interact Cardiovasc Thorac Surg 2021;1–3.
50. Castiglioni M, Pelosi G, Meroni A, et al. Surgical resections of superinfected pneumatoceles in a COVID-19 patient. Ann Thorac Surg 2021;111:e23-5.