

Severe Acute Necrotizing Community-Acquired Pneumonia with Pyopneumothorax Due to *Pseudomonas aeruginosa* in a Healthy Young Girl

ABSTRACT

Aims: We report this case to acknowledge the presence of multidrug-resistant *Pseudomonas aeruginosa* in the community setting, which transpired in a young female, without any known comorbidity or exposure risk. **Background:** *P. aeruginosa* is an extremely unusual pathogen in community-acquired pneumonia (CAP), especially in previously healthy young adults. Immune status, underlying lung disease and possible exposures like hot tub use, should always be ruled out. There have been reports of bacterial etiology being next common after tuberculosis for pyopneumothorax, but ours is one of the first case as per our extensive research to report *P. aeruginosa* as a cause of CAP and pyopneumothorax in an absolutely healthy young adult. **Case Description:** A young female, developed pneumonia from the community setting which grew a nosocomial organism. Complications involved cavity formation and pyopneumothorax, which required intercostal drainage (ICD) and extensive intravenous antibiotics. Immediate and appropriate management is lifesaving. **Conclusion:** CAP due to heinous organisms like *P. aeruginosa* should be suspected in any patient who presents with a rapidly progressive course, even in the absence of risk factors and exposures. In cases of pleural effusion or pneumothorax or both, ICD drain should be inserted. Broad-spectrum antibiotic coverage, especially in hemodynamically unstable patients, should be initiated immediately, followed by antibiotic stewardship protocols. **Clinical Significance:** We would like to emphasize the importance of acquiring cultures from respiratory site, for early initiation/switching of appropriate antibiotics. Furthermore, *Pseudomonas* being an ominous organism yields a finer prognosis only when treated with appropriate antipseudomonal antibiotics.

Key words: Community-acquired pneumonia, Necrotizing pneumonia, *Pseudomonas aeruginosa*, pyopneumothorax

INTRODUCTION

Pseudomonas aeruginosa is a Gram-negative bacillus found widely in soil and water. It causes disease infrequently in normal hosts, but is a major cause of infection in patients with an immune compromised state. *P. aeruginosa* as an etiologic agent for community-acquired pneumonia (CAP) is quite rare and can cause necrotizing pneumonia. The evolution of this new pathogen ecology is menacing our treatment toward CAP. IDSA recommends abandoning the use of prior categorization of health care-associated pneumonia to guide selection of extended antibiotic coverage in adults with CAP.^[1] *P. aeruginosa* is one of the most common causes of pneumonia requiring intensive care unit (ICU) admission, with high morbidity and mortality. Therefore, it is important to have a high index of suspicion, as early empiric treatment tends to have a better prognosis.

CASE REPORT

An 18-year aged girl was absolutely healthy until 3 weeks back, when her symptoms started insidiously; which consisted of fever with chills, breathlessness on exertion, and bilateral chest pain. She received symptomatic treatment from a general physician,

Tasneem Bohra, Pralhad Prabhudesai

Lilavati Hospital and Research Centre, Bandra (West), Mumbai, Maharashtra, India

Corresponding Author:

Tasneem Bohra, Lilavati Hospital and Research Centre, Bandra (West), Mumbai, Maharashtra, India.
E-mail: tasneemshabbir1512@gmail.com

but without complete cure. One week later into the illness, she developed sudden respiratory distress and was brought to the casualty. Her SpO₂ on room air was 50%, respiratory rate – 50/min, heart rate – 150/min, and blood pressure of 100/60 mmHg. On auscultation, there were diminished breath sounds on the right side. An immediate chest X-ray [Figure 1a] showed right-sided hydropneumothorax. An intercostal drainage (ICD) insertion was done within no time and ICU admission was warranted. Her breathlessness settled and vitals stabilized, but fever persisted. On further taking a detailed history, the patient had no history of travel or exposure to any pets. There was no history of hospitalization in the past. Serial chest X-rays showed no expansion of the lung [Figure 1b]. Pleural fluid

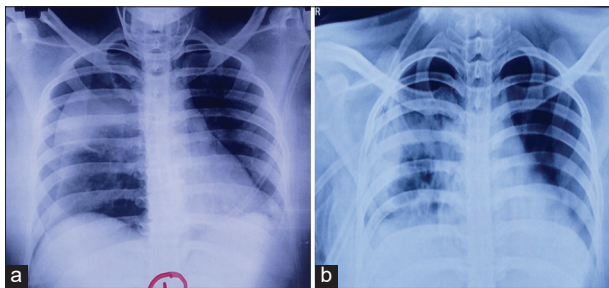


Figure 1: (a) Chest X-ray showing right side pneumothorax, (b) chest X-ray (day 4) showing right-sided intercostal drainage *in situ* without lung expansion

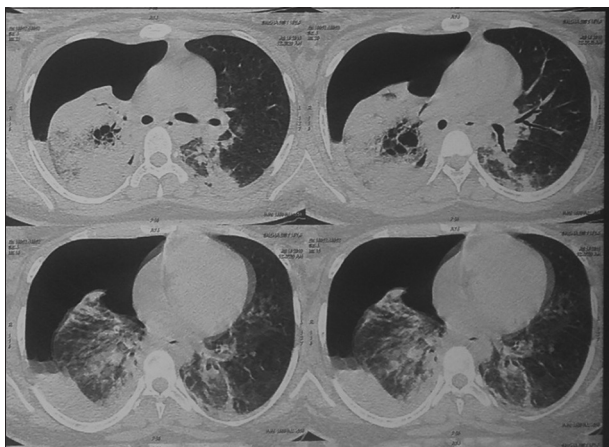


Figure 2: HRCT chest showing right side hydropneumothorax with cavity and collapse of underlying lung

was turbid, exudative (as per Light's criteria) with neutrophilic predominance and low adenosine deaminase (18.6). Since there was no lung expansion, HRCT chest [Figure 2] was performed; a right-sided hydropneumothorax with cavity and collapse of the underlying lung was noted. Serum investigations showed hemoglobin – 10.3, WBC – 10,700 of which 91% were neutrophils, and platelet count – 359,000. CRP was mildly raised (23.04) and PCT was negative. Liver function test, renal function test, and blood sugars were within normal limits. HIV, HBsAg, and HCV were non-reactive. Connective tissue markers (ANA, c-ANCA, and p-ANCA) were negative. Blood culture grew no organism. Pleural fluid and sputum for aerobic culture, GeneXpert, and AFB stain were negative. For ongoing infection, the patient was initially started on meropenem and metronidazole. Despite broad-spectrum antibiotics being administered for 1 week, her fever persisted. Hence, ciprofloxacin and linezolid were added. Since no change was observed in the fever trend, a bronchoscopy was performed. There was no structural abnormality in the tracheobronchial tree. Thick secretions were noted bilaterally, predominantly on the right side of bronchial tree. Bronchoalveolar lavage grew *P. aeruginosa* (>1 lakh colony-forming unit/ml), which was multidrug resistant and sensitive only to cefepime, levofloxacin,



Figure 3: Chest X-ray on follow-up after completion of treatment showed total resolution of hydropneumothorax

and colistin. Antibiotics were changed to intravenous cefepime-tazobactam and colistin for 2 weeks. As minimal drain was noted, ICD was removed and the patient improved gradually. She was discharged on 3 weeks of oral levofloxacin. Later, AFB culture reports of pleural fluid and sputum (>8 weeks) were also negative. On follow-up after 3 weeks, the patient showed significant clinical and radiological improvement [Figure 3].

DISCUSSION

Pneumonia is defined as inflammation and consolidation of lung tissue due to an infectious agent. Pneumonia that develops outside the hospital is considered CAP. Typical bacterial agents that cause CAP include *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*.^[2] Community-acquired *P. aeruginosa* cause mild and superficial infections such as otitis externa, varicose ulcers, and folliculitis.^[3] It is an infrequent pathogen to cause CAP. However, in patients with severe CAP, *P. aeruginosa* can be the etiology in 1.8%–8.3% of patients.^[4] During the past few decades, the etiology of CAP has been changing, wherein antibiotic-resistant bacteria that were thought to be important only in hospital settings are now becoming more prevalent in community settings.^[5] Hydropneumothorax or pyopneumothorax is a rare complication of community-acquired bacterial pneumonia. Studies have reported stating that bacterial etiology is second to *Mycobacterium tuberculosis* in causing hydropneumothorax, but we could not come across any study which reported the presence of an *P. aeruginosa* presenting with hydropneumothorax.^[6] *P. aeruginosa* more commonly causes nosocomial pneumonia. Most *P. aeruginosa* caused CAP is seen in patients with structural lung diseases, chronic obstructive pulmonary disease, or cystic fibrosis. Other risk factors for CAP due to *P. aeruginosa* include chronic heart failure, cerebrovascular disease, advanced age, smoking, and malnutrition. Several studies have found that

fatal *P. aeruginosa* pneumonia in previously healthy patients was associated with contaminated hot tubs.^[7] Our patient had none of the above risk factors or exposures, hence regarding it as an extremely rare case. Clinical presentation of CAP due to *P. aeruginosa* could be variable including cough, sputum production, pleuritic chest pain, fever, and/or hemoptysis.

High virulence of *P. aeruginosa* is due to factors which include bacterial surface components such as flagella/pili, secreted enzymes/toxins, and the ability to form biofilms. Mechanisms of antimicrobial resistance include multidrug efflux pumps, ss-lactamases, and downregulation of outer membrane porins. *P. aeruginosa* is a challenging bacterium to treat, due to its intrinsic resistance to the antibiotics used most frequently in the management of CAP. Drug-resistant *P. aeruginosa* is defined when the isolated pathogen is resistant to at least one antibiotic and MDR *P. aeruginosa* is defined by resistance to at least three of the evaluated antibiotics. IDSA recommends on obtaining pre-treatment Gram stain and culture of respiratory secretions and blood cultures, only in adults with CAP managed in the hospital setting, who are classified as severe CAP or are being empirically treated for methicillin-resistant *Staphylococcus aureus* or *P. aeruginosa*. Empiric treatment options for *P. aeruginosa* include piperacillin-tazobactam (4.5 g every 6 h), cefepime (2 g every 8 h), ceftazidime (2 g every 8 h), aztreonam (2 g every 8 h), meropenem (1 g every 8 h), or imipenem (500 mg every 6 h). If *P. aeruginosa*-CAP in adults is being empirically covered, it is recommended on continuing empiric coverage while obtaining culture data. If these pathogens are present, it is justified to continue treatment for these pathogens after the first few days of empiric treatment.^[1] In cases of recovery, complications include parenchymal scarring and recrudescence – requiring repeated antibiotic courses. In the cases reviewed with recovery, duration of therapy varied from 2 to 6 weeks.^[8]

CAP pseudomonas has a higher CURB-65 score and pneumonia severity index with mortality of approximately 18–61%.^[7] In fatal cases, the disease involves development of septic shock and organ failure. CAP due to *P. aeruginosa* has a rapid progression, requires more respiratory/vasopressor support, and has a poorer prognosis as compared to other pathogens.^[5]

CONCLUSION

P. aeruginosa is a rare etiology of CAP. Nonetheless, it should be suspected in any patient who presents with a rapidly progressive course, particularly in the presence of risk factors and exposures. In cases of pleural effusion or pneumothorax or both, ICD drain should be inserted. In recent years, circulating strains of *P. aeruginosa* have tended to have higher resistance patterns. Therefore, combination therapy, including

antipseudomonal agent, should be considered in the treatment, to prevent future resistance.

CLINICAL SIGNIFICANCE

Cultures from respiratory site and blood should be obtained before initiating antibiotics. Thereafter, antibiotic stewardship protocols should be adhered to. *Pseudomonas* being an ominous organism yields a finer prognosis only when treated with appropriate antipseudomonal antibiotics. Having a high index of suspicion with appropriate initial management reduces morbidity and mortality.

REFERENCES

1. Pletz MW, Blasi F, Chalmers JD, Cruz CS, Feldman C, Luna CM, et al. International perspective on the new 2019 American thoracic society/infectious diseases society of america community-acquired pneumonia guideline: A critical appraisal by a global expert panel. *Chest* 2020;158:1912-8.
2. von Baum H, Welte T, Marre R, Suttrop N, Ewig S. Community-acquired pneumonia through Enterobacteriaceae and *Pseudomonas aeruginosa*: Diagnosis, incidence and predictors. *Eur Respir J* 2010;35:598-605.
3. Huhulescu S, Simon M, Lubnow M, Kaase M, Wewalka G, Pietzka AT, et al. Fatal *Pseudomonas aeruginosa* pneumonia in a previously healthy woman was most likely associated with a contaminated hot tub. *Infection* 2011;39:265-9.
4. Cillóniz C, Gabarrús A, Ferrer M, de la Bellacasa JP, Rinaudo M, Mensa J, et al. Community-acquired pneumonia due to multidrug- and non-multidrug-resistant *Pseudomonas aeruginosa*. *Chest* 2016;150:415-25.
5. Restrepo MI, Babu BL, Reyes LF, Chalmers JD, Soni NJ, Sibila O, et al. Burden and risk factors for *Pseudomonas aeruginosa* community-acquired pneumonia: A multinational point prevalence study of hospitalised patients. *Eur Respir J* 2018;52:1701190.
6. Kasargod V, Awad NT. Clinical profile, etiology, and management of hydropneumothorax: An Indian experience. *Lung India* 2016;33:278.
7. Wang T, Hou Y, Wang R. A case report of community-acquired *Pseudomonas aeruginosa* pneumonia complicated with MODS in a previously healthy patient and related literature review. *BMC Infect Dis* 2019;19:130.
8. Maharaj S, Isache C, Seegobin K, Chang S, Nelson G. Necrotizing *Pseudomonas aeruginosa* community-acquired pneumonia: A case report and review of the literature. *Case Rep Infect Dis* 2017;2017:1717492.

How to cite this article: Bohra T, Prabhudesai P. Severe Acute Necrotizing Community-Acquired Pneumonia with Pyopneumothorax Due to *Pseudomonas aeruginosa* in a healthy young girl. *Bombay Hosp J* 2022;64(1):27-29.

Source of support: Nil, **Conflicts of interest:** None

This work is licensed under a Creative Commons Attribution 4.0 International License. The images or other third party material in this article are included in the article's Creative Commons license, unless indicated otherwise in the credit line; if the material is not included under the Creative Commons license, users will need to obtain permission from the license holder to reproduce the material. To view a copy of this license, visit <http://creativecommons.org/licenses/by/4.0/> © Bohra T, Prabhudesai P. 2022.