DOI: 10.7860/JCDR/2022/52806.16026

Case Report

Internal Medicine Section

Chronic Indolent Community-acquired Pneumonia due to *Pseudomonas* Infection in an Immunocompetent Patient- A Case Report

GAURAV KARNA[1], SHRIDHAR PATTAR[2], PRASAN KUMAR PANDA[3], RAMIT SHAH[4]

ABSTRACT

Pseudomonas is an uncommon cause of community-acquired pneumonia in immunocompetent patients. It is an opportunistic pathogen resulting in serious infection in patients who are hospitalised, mechanically ventilated, or immunocompromised. Here, authors reported a case of 47-year-old male, forest worker without any co-morbidities presented with a history of chronic cough, fever, and shortness of breath complicated with pseudohaemoptysis for 45 days. This patient was admitted and treated as a lower respiratory tract infection. Work-up for tuberculosis, invasive fungal balls was negative but sputum culture revealed *Pseudomonas aeruginosa* growth. This case report demonstrates a rare *Pseudomonas* infection which can also cause chronic indolent respiratory illness in immunocompetent.

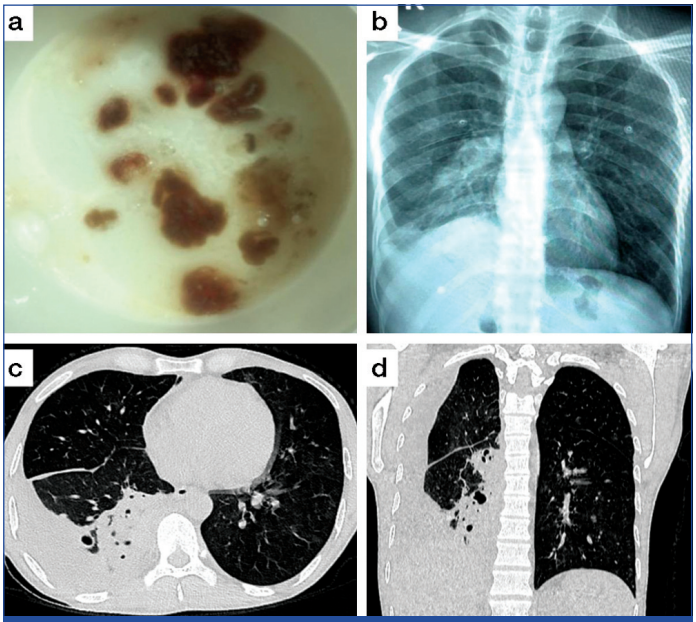
Keywords: Forest-dweller, Healthy, Lower respiratory tract infections, Pseudomonads, Pseudohaemoptysis

CASE REPORT

A 47-year-old male, forest patrol, presented with complaints of fever for 45 days and cough and shortness of breath for 30 days. Fever was undocumented, low-grade intermittent associated with chills and rigour, without sweating responsive to medication, without any diurnal variation. It was followed by cough, gradual onset, initially dry then became productive with mucopurulent sputum, later became dark brownish, non foul smelling which increased on lying down and during night time. It was associated with the pruritic type of right sided chest pain and breathlessness. Breathlessness was an insidious onset, gradually progressive, initially with marked exertion, which progressed later to cause dyspnoea even with regular household activities, without orthopnea and paroxysmal nocturnal dyspnoea. It was not associated with decreased urine output, palpitation, or body swelling. The patient had lost weight (not documented, in the form of loosening of clothes). He was taking over the counter medications like analgesic and antitussive for symptomatic relief. He did not have a history of any co-morbidities like hypertension, diabetes mellitus, tuberculosis, or any known chronic illness attributed to the heart, lungs, liver or kidney. He was a chronic smoker (smoking index-400) and chronic alcohol consumer (180 mL/week for 20 years). In mean time, he had shown to local pharmacist and taken unknown cocktail treatments but with no major improvements.

On examination, the patient was conscious, oriented, pale-looking with vitals including temperature of 99.7 F, pulse rate was 110/min, respiratory rate was 23/min, blood pressure was 100/60 mmHg, Oxygen Saturation (SpO_2) was 86% at room air and 97% at 3 L/min oxygen through nasal prongs. Systemic examination revealed decreased intensity of breath sound in right infra-scapular and infra-axillary area and dull note on percussion with coarse inspiratory crept over same areas. Other system examinations were unremarkable. Sputum was grossly examined that looked thick, brownish, and dark [Table/Fig-1a].

The patient was admitted on the same day of visit to hospital and was investigated for lower respiratory tract infection. Chest X-ray (day 1) showed right sided pleural effusion with patchy infiltration in the middle zone of the right lung [Table/Fig-1b]. High-Resolution Computed Tomography (HRCT) chest (day 1) demonstrated multiple patchy areas of ground-glass attenuation in bilateral lungs, predominantly in a peripheral location; areas of consolidation were seen in basal segments of lower lobe of right lung and posterior segment of right upper lobe; fibroatelectatic bands with mild traction bronchiectasis in bilateral lungs; right pleural effusion was noted with underlying basal atelectasis, maximum thickness was 10.5 mm [Table/Fig-1c,d]. Routine investigations [Table/Fig-2] revealed bicytopenia (anaemia and thrombocytopenia). Findings were suggestive of active infective aetiology. [COVID-19 Reporting and Data System (CORADS-4), CT Severity Score (CTSS-23/40)].



[Table/Fig-1]: Sputum and imaging findings; a) shows brownish sputum (pseudo-haemoptysis) collected in a pot during hospitalisation; b) shows chest X-ray PA view having right sided pleural effusion with patchy infiltration in the middle zone of right lung; (c&d) shows HRCT chest (axial and coronal sections) having areas of consolidation in the basal segment of the lower lobe of the right lung and fibroatelectatic bands with mild bronchiectasis (not shown).

Considering the epidemiology, chronic symptomatology, pathology affecting lung parenchyma, and image characteristics, following differentials were considered: tuberculosis, invasive fungal granuloma, or cavitary pneumonia. To narrow down the differential, Interferon-Gamma Release Assays and β-D glucan were done which turned

Investigations (with reference range)	Day 1	Day 7	Day 12
Haemoglobin (13.00-17.00 gm/dL)	11.77	9.7	9.87
Total leucocyte count (4.00-10.00 *1000/uL)	6.95	7.02	4.33
Differential count (N/L/M/E/B)	82/11/5.2	66/20/12	45/30/3.1
Platelets (150-450 1000/cumm)	40	207	223
Urea (3-43 mg/dL)	52.1	-	15
Creatinine (0.7-1.2 mg/dL)	0.7	-	0.45
Sodium (136-145 meq/L)	136	-	133
Potassium (3.5-5.3 meq/L)	4.62	-	3.60
Calcium (8.0-10.8 meq/L)	7.67	-	8.3
Total bilirubin (0.30-1.20 mg/dL)	2.06	-	0.99
Direct bilirubin (<0.20 mg/dL)	0.99	-	0.30
Aspartate transaminase (0-40 U/L)	58	-	49
Alanine transaminase (0-45 U/L)	54.5	-	70
Alkaline phosphatase (0-240 U/L)	163	-	155
Gamma-glutamyl Transferase (8-61 U/L)	60	-	35
Total protein (6.40-8.30 gm/dL)	4.8	-	5.1
Albumin (3.50-5.00 gm/dL)	2.51	-	2.5
Globulin (2.50-3.50 gm/dL)	2.29	-	2.6
C-Reactive protein (upto 6.0 mg/L)	80.2	-	-
Procalcitonin (<0.5 µg/L)	0.31	-	-
Troponin-I (kit)	Negative	-	-
Iron (70-180 µg/dL)	51	-	-
Ferritin (18-270 µg/L)	399	-	-
Transferrin saturation (20-50%)	19.7	-	-
Peripheral blood smear	Normocytic normochromic picture	-	-
Folic acid (>5.38 ng/mL)	16.9	-	-
Vitamin B12 (156-672 pmol/L)	650	-	-
Reticulocyte count (%)	6 (RPI-2.72)	-	-
Stool for occult blood	Negative	-	-

[Table/Fig-2]: Basic and advanced investigations during hospitalisation of the patient.

out negative. Sputum gram stain showed gram negative rods which was evident in sputum culture and sensitivity demonstrating growth of *Pseudomonas* aeruginosa [Table/Fig-3].

Special investigations	Results
HIV/HBsAg/HCV (day 1)	Non reactive
COVID-19 RT-PCR for nasopharyngeal sample (day 1 and day 5)	Negative
Blood and urine culture and sensitivity (day 1)	Sterile
Pleural fluid analysis (day 7)	
TLC	5400 cells/cumm
DLC-P/M	70/30 %
Sugar	104 mg/dL (with corresponding blood sugar 110)
Protein	3.6 mg/dL (with corresponding serum protein-4.9 mg/dL)
AFB and CBNAAT	Negative
Culture	Sterile
Sputum AFB and CBNAAT (day 8)	Negative
Sputum culture and sensitivity (day 8)	*Pseudomonas aeruginosa*-moderate growth, resistant to majority antibiotics except Amikacin (MIC 2), Trimethoprim/Sulfamethoxazole (MIC 20), and Minocycline (MIC 4)

[Table/Fig-3]: Special investigations during hospitalisation of the patient.
CBNAAT: Cartridge-based nucleic acid amplification test; AFB: Acid-fast bacillus; DLC: Differential Leukocyte Count; HBsAg: Hepatitis B surface antigen; HIV: Human immunodeficiency virus; HCV: Hepatitis C virus

On the first day of hospitalisation, patient was started empirically on intravenous ceftriaxone 1 gm twice daily along with intravenous azithromycin 500 mg once daily for 5 days for pneumonia. After sputum culture sensitivity reports on 8th day of hospitalisation, the patient was started on amikacin. Amikacin was given for 7 days parenterally by an intravenous route at a once daily dose of 15 mg/kg.

The patient's expectoration of brownish-coloured sputum disappeared gradually after the initiation of treatment based on culture sensitivity, and he was discharged with stable vitals after 12 days of hospitalisation. On follow-up after one month, the patient was clinically asymptomatic without fever, cough, or shortness of breath. His chest X-ray was clear with disappearance of the patchy heterogenous opacity.

DISCUSSION

This case report showed *Pseudomonas* can cause chronic respiratory illness and pseudohaemoptysis in healthy individual. *P. aeruginosa* is one of the common pathogens involved in acute or chronic respiratory tract infections in various clinical settings [1]. It is ubiquitous in the hospital environment and is suspected in individuals who are immunocompromised, who have an extended hospitalisation or mechanically ventilated, have malignancies or Human Immunodeficiency Virus (HIV) infection, have received prolonged antibiotic administration or multiple antibiotics, or who have indwelling catheters [2,3]. In chronic lung infection due to *Pseudomonas*, host responses are complex and dynamic ranging from vigorous activation of immune response to relative host tolerance favouring bacterial persistence [4]. *Pseudomonas* is an uncommon cause of community-acquired pneumonia in immunocompetent individuals [5]. In the study conducted in patients with the same bacteremia, 47.6% were immunocompetent and the rest were immunocompromised [6]. A point prevalence international study was done among 2564 immunocompetent patients of which *Streptococcus pneumoniae* (8.2%) was the most frequently identified pathogen, followed by *P. aeruginosa* (4.1%) and *Klebsiella pneumoniae* (3.4%) [7]. Even after being immunocompetent, here patient developed a chronic indolent infection of *P. aeruginosa*, which is not usually observed.

Data on the global burden and risk factors associated with *P. aeruginosa* are limited. Restrepo MI et al., conducted a study among 3193 diagnosed cases of CAP from 54 different countries, out of which 4.2% had *P. aeruginosa*. In contrast, the rate of *P. aeruginosa* CAP was 2% in patients without prior same infection or colonisation and chronic lung diseases similar to the present case [8]. Respiratory diseases have long been recognised in association with the variety of occupational exposure. To quantify the burden of respiratory disease among agricultural workers, Greskevitch M et al., examined various national health statistics data in 2007 and found that obstructive respiratory abnormalities were more prevalent in farm workers [9]. This study also showed that forestry workers had significantly elevated mortality for pulmonary tuberculosis, chronic airway obstruction, and pneumonia, organism unspecified. Considering this case had forest indwelling, it may be associated with indolent community-acquired pneumonia due to *P. aeruginosa*.

Chronic cough with fever and weight loss complicated by haemoptysis are moreover prevalent in tuberculosis and bronchiectasis [10]. But in this patient, the colour of the sputum was brownish and was initially thought to be blood-tinged so tests were carried out for tuberculosis. Rather the culture and sensitivity for the sputum revealed *Pseudomonas* infection. In addition, there is literature, which illustrates that deepening sputum colour from yellowish to brownish is associated with an increased yield of gram negative bacteria such as *Pseudomonas* or *Enterobacteriaceae*, illustrates pseudohaemoptysis [11].

In various studies conducted, combinations of beta-lactams and aminoglycosides have benefits over the monotherapy against *Pseudomonas* infection [12,13]. Here, this patient was started on

beta-lactams as empirical treatment and later, it was switched to amikacin for being sensitive. Among aminoglycosides, amikacin holds better bactericidal activity being concentration-dependent killing and usually due to lower resistance to *P. aeruginosa* [12]. Likewise, a retrospective cohort study was done to establish the relationship between initial inappropriate antimicrobial treatment and the clinical outcomes for *P. aeruginosa* infections which showed that hospital mortality was significantly higher for patients receiving inappropriate initial antimicrobial treatment than for those receiving appropriate therapy (31% versus 18%; p-value=0.02) [13]. Hence, the right dose and duration are important in the treatment of *P. aeruginosa* infection.

In the study conducted in 126 patients with *P. aeruginosa* infection, the mortality in immunocompetent patients was lower than immunocompromised patients {7 day mortality-8% vs 30% (p-value=0.01); 30 day mortality-23% vs 39% (p-value=0.053)} [6]. Initial antibiotic therapy (Heart rate: 0.21, p-value=0.01) and patient's immune status (Heart rate: 0.29, p-value=0.02) also had a significant impact on survival. A subgroup analysis showed that in immunocompromised, but not immunocompetent patients, initial appropriate antibiotic therapy was associated with lower mortality {30 day mortality 20.5% vs 66.7% (p-value <0.01 by log-rank test)} [6]. Even here, this patient showed remarkable improvements both clinically and radiologically after initiation of the definitive treatment based on culture sensitivity.

CONCLUSION(S)

Pseudomonas aeruginosa can cause chronic indolent pneumonia in immunocompetent. Forestry workers can be a risk factor for the same. All sputum history of haemoptysis is not true, haemoptysis deepening sputum colour from yellowish to brownish can be due to indolent pseudomonas lung infection. Monotherapy with amikacin can be used for primary treatment of chronic *Pseudomonas* infection.

REFERENCES

[1] Sadikot RT, Blackwell TS, Christman JW, Prince AS. Pathogen-host interactions in pseudomonas aeruginosa pneumonia. Am J Respir Crit Care Med. 2005;171(11):1209-23.
[2] Fujitani S, Moffett kathryn S, Yu VL. Pseudomonas aeruginosa. Antimicrobe: Infectious Disease & Antimicrobial. 2017. Available from: http://www.antimicrobe.org/new/b112.asp.
[3] Qureshi S. Pseudomonas aeruginosa Infections. Medscape. 2020. Available from: https://emedicine.medscape.com/article/226748-overview.
[4] Faure E, Kwong K, Nguyen D. Pseudomonas aeruginosa in chronic lung infections: How to adapt within the host? Frontiers in Immunology. 2018;9:2416.
[5] Gharabaghi MA, Abdollahi SMM, Safavi E, Abtahi SH. Community acquired Pseudomonas pneumonia in an immunocompetent host. BMJ Case Rep. 2012;26;2012:bcr0120125673. Available from: /pmc/articles/PMC3369319/.
[6] Migiyama Y, Yanagihara K, Kaku N, Harada Y, Yamada K, Nagaoka K, et al. Pseudomonas aeruginosa bacteremia among immunocompetent and immunocompromised patients: Relation to initial antibiotic therapy and survival. J Infect Dis. 2016;69(2):91-96.
[7] Carugati M, Aliberti S, Sotgiu G, Blasi F, Gori A, Menendez R, et al. Bacterial etiology of community-acquired pneumonia in immunocompetent hospitalised patients and appropriateness of empirical treatment recommendations: An international point-prevalence study. Eur J Clin Microbiol Infect Dis. 2020;39(8):1513-25.
[8] 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(2):1701190.
[9] Greskevitch M, Kullman G, Bang KM, Mazurek J. Respiratory disease in agricultural workers: Mortality and morbidity statistics. J Agromedicine. 2007;12(3):05-10.
[10] Walker HK, Hall WD, Hurst JW, editors. Clinical Methods: The History, Physical, and Laboratory Examinations 3rd edition. Boston: Butterworths; 1990;211-13. Available from: https://www.ncbi.nlm.nih.gov/books/NBK360/#A1225.
[11] Allegra L, Blasi F, Diano PL, Cosentini R, Tarsia P, Confalonieri M, et al. Sputum colour as a marker of acute bacterial exacerbations of chronic obstructive pulmonary disease. Respir Med. 2005;99(6):742-47.
[12] Bassetti M, Vena A, Croxatto A, Righi E, Guery B. How to manage Pseudomonas aeruginosa infections. Drugs in Context. 2018;7:212527. Available from: /pmc/articles/PMC5978525/.
[13] McDonald LC, Owings M, Jernigan DB. Clostridium difficile infection in patients discharged from US short-stay hospitals, 1996-2003. Emerg Infect Dis. 2006;12(3):409-15.

PARTICULARS OF CONTRIBUTORS:
1. Resident, Department of Medicine, All India Institute of Medical Sciences, Rishikesh, Uttarakhand, India.
2. Resident, Department of Medicine, All India Institute of Medical Sciences, Rishikesh, Uttarakhand, India.
3. Associate Professor, Department of Medicine, All India Institute of Medical Sciences, Rishikesh, Uttarakhand, India.
4. Resident, Department of Radiology, All India Institute of Medical Sciences, Rishikesh, Uttarakhand, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Prasan Kumar Panda,
Associate Professor, Department of Medicine, All India Institute of Medical Sciences, Rishikesh, Uttarakhand, India.
E-mail: motherprasanna@rediffmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Oct 13, 2021
- Manual Googling: Dec 14, 2021
- iThenticate Software: Dec 30, 2021 (14%)

ETYMOLOGY: Author Origin

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Oct 12, 2021**
Date of Peer Review: **Dec 08, 2021**
Date of Acceptance: **Dec 15, 2021**
Date of Publishing: **Feb 01, 2022**