

A Rare Case of Cavitating Lung Disease

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Abstract

Pantoea rarely causes infection in human lungs. Genus *Pantoea* is a rare pathogen causing opportunistic human infection in clinical practice. Among seven species of this rare organism, *Pantoea agglomerans* is the most common species in humans and is often associated with an occupational disease. Clinically, it may present with wound infection, abscess, catheter infection, pneumonia, and urinary system infection in immunocompromised individuals. However, there are no case reports of *P. agglomerans* causing cavitating lung disease in the past. Here, we report a case of a 67-year-old immunocompetent female who presented with cavitating lung lesions on chest imaging which were found to be associated with *P. agglomerans* leading to fatal outcomes. We emphasize on the unusual presentation, its course in the hospital, and importance of early recognition of this emerging bacterial infection causing fulminant human infections even in immunocompetent individuals.

Keywords: Immunocompetent, lung cavitation, *Pantoea agglomerans*

INTRODUCTION

Pantoea infections are scarce in humans. Only a few cases have been reported in the past worldwide causing bacteremia, septic arthritis, skin, and soft tissue abscess. *Pantoea agglomerans* is an environmental Gram-negative aerobic bacillus mainly associated with plants which is now emerging as human infection. Human infections usually follow wound infection with plant materials or hospital-acquired infection in immunocompromised individuals. There is little known about their clinical manifestations and usually, they are rarely responsible for cavitating lung disease. Here, we report a case of a 67-year-old smoker female who presented with cavitating lung lesions which were found to be associated with *P. agglomerans*.

CASE REPORT

A 67-year-old female presented to the emergency department with low-grade fever, irrelevant talk for 3 days and breathing difficulties, cough with expectoration, and generalized myalgia for 10 days.

As per her relative, she smoked hand-rolled tobacco products named bidi for the last 20 years. She had a progressive dyspnea. She did not have any history of seizure, headache, photophobia, vomiting, burning micturition, chest pain, orthopnea or

paroxysmal nocturnal dyspnea, hemoptysis, rashes, Raynaud's phenomenon, oral ulcers, numbness, or weight loss.

At presentation, she was drowsy. Her blood pressure was 140/88 mmHg, pulse – 123/min, respiratory rate – 30/min, and oxygen saturation of 79% on room air. On examination, there were no pallor, cyanosis, lymphadenopathy, edema, and icterus. On auscultation, bilateral wheezes and crepitation were heard in basal lung areas.

All relevant blood investigations including blood culture were advised [Tables 1 and 2]. Hemogram revealed neutrophilic leukocytosis and moderate anemia. Arterial blood gas analysis was suggestive of type 1 respiratory failure. Her chest X-ray showed a bilateral cavitating lesion [Figure 1]. For further evaluation, contrast-enhanced computed tomography (CECT) with high-resolution cuts of the chest and abdomen was done which showed multiple thick-walled cavitating lesions in both lungs [Figure 2] with the possibility of vasculitis or metastasis.

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How to cite this article: Gupta RK, Kumar A, Gangakhedkar M, Prakash V, Saini L, Sharma P, *et al.* A rare case of cavitating lung disease. *Indian J Respir Care* 2021;10:361-3.

Received: 14-06-2021

Revised: 06-08-2021

Accepted: 09-08-2021

Published: 13-09-2021

Access this article online

Quick Response Code:



Website:
www.ijrc.in

DOI:
10.4103/ijrc.ijrc_75_21

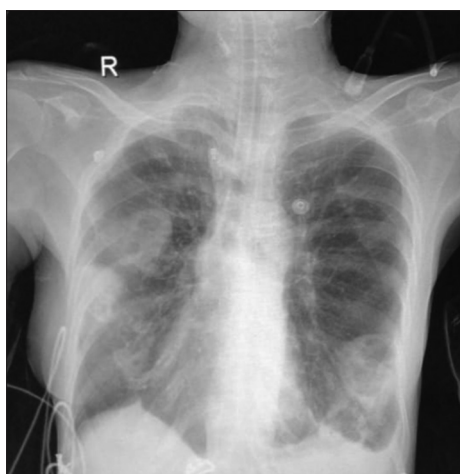


Figure 1: Chest X-ray showing bilaterally multiple cavitating lesions



Figure 2: Computed tomography chest at different levels suggesting bilateral cavitating lesions of different sizes

Table 1: Blood investigation reports at presentation and in intensive care unit

Investigations	At presentation	ICU
ABG (pH/PCO ₂ /PO ₂ /HCO ₃ /lactate)	7.41/31/38/20/2.1	7.24/79/37/34/3.4
WBC (cells/cumm)	31,000, n-88%	18,000 n-96%
Hemoglobin (g/dL)	7.3	8.3
Platelets (cells/cumm)	296,000	151,000
Urea/creatinine (mg/dL)	40/1.2	172/4.2
Sodium/potassium (mmol/L)	141/3.7	135/2.7
Total bilirubin/direct (mg %)	0.67/0.32	
SGPT/SGOT/ALP (U/L)	24/14/68	44/34/79
Viral markers (HIV, HBsAg, HCV)	Negative	
Albumin (g %)	4.84	1.65
International Normalised Ratio (INR)	1.2	1.5
Procalcitonin (mcg/L)	0.19	0.79
C-reactive protein (CRP) (mg/dL)	32	

ICU: Intensive care unit, ABG: Arterial blood gas, WBC: White blood cell, SSGPT: Serum glutamic-pyruvic transaminase, SGOT: Serum glutamic-oxalacetic transaminase, ALP: Alkaline phosphatase, HIV: Human immunodeficiency virus, HBsAg: Hepatitis B antigen, HCV: Hepatitis C virus

However, no abnormal findings were seen on the abdomen scan. In view of low mental status, CECT of the brain was obtained which was suggestive of age-related changes. She started on oxygen therapy, intravenous (IV) ceftriaxone and azithromycin, and deep-vein thrombosis prophylaxis with subcutaneous low-molecular weight heparin along with supportive management.

Her echocardiography showed an ejection fraction of 45%, severe tricuspid regurgitation, and dilated right and left ventricle with no vegetations. Her fundoscopic examination was normal. She was given IV vancomycin and acyclovir in view of suspected meningitis. Her cerebrospinal fluid was acellular with normal biochemistry. Her vasculitis workup was negative, blood and urine culture did not show growth of any organism.

At the 6th day of illness, the patient developed a generalised tonic-clonic seizure. Her trachea was intubated due to her inability to maintain airway and she was shifted to the intensive care unit (ICU). She further developed hypernatremia and acute kidney injury followed by bilateral pleural effusion which was of an uncomplicated exudative nature.

Due to high suspicion of malignancy, an ultrasound-guided lung biopsy was obtained from the cavity lesion. It was blood and necrotic tissue and was negative for any bacterial and fungal growth. Her bronchoalveolar lavage (BAL) fluid, however, grew *P. agglomerans* which was sensitive to minocycline, tigecycline, and colistin. Acid-fast bacilli stain and GeneXpert for tuberculosis infection were negative. Computed tomography-guided biopsy from left lung lesion was done, but it was negative for malignant cells and did not show any organism on special stains and tissue culture. She was started on IV meropenem and later on colistin based on the culture report. The patient developed septic shock and multiorgan failure during the course of illness. Unfortunately, she developed cardiac arrest and succumbed to death.

DISCUSSION

Cavitating lung lesions are caused by a variety of etiology. In our case, we considered various etiologies such as lung abscess, tuberculosis, fungal infection, vasculitis, malignancy, hydatid cyst, and pulmonary thromboembolism. Further on, CECT differential diagnosis was narrowed. Although we kept a strong possibility of metastasis or primary lung malignancy, these possibilities were ruled out on histopathology report of lung biopsy. Finally, her BAL fluid culture grew *P. agglomerans* which is a rare pathogen in humans.

Pantoea is a Gram-negative, nonencapsulated, nonspore-forming bacillus, previously known as *Enterobacter agglomerans* or *Erwinia herbicola*.^[1] They can be isolated from plants, soil, food, human feces, and the environment. *Pantoea* belongs to the family *Enterobacteriaceae*. It is different from

Table 2: Reports of other relevant investigations

Investigation	Report
CSF	Colourless, 25 cells/cumm; lymphocytes 100%, ADA-3.35, protein-31 mg/dL, sugar-68 mg/dL
Pleural fluid	Exudative bilateral ADA-3.67 IU/L (L) 4.54 IU/L (R)
D-dimer (mg/L)	0.45
Hydatid serology	Negative
Carcinoembryonic antigen (CEA) (ng/ml)	2.16
Cancer antigen 19-9 (CA 19- 9) (U/ml)	1.49
Alfa feto protein (ng/ml)	1.7
RA factor, C ANCA, P ANCA	Negative

CSF: Cerebrospinal fluid, ADA: Alanine deaminase; RA factor: Rheumatoid Arthritis factor, C ANCA: Cytoplasmic Antineutrophilic cytoplasmic antibody, p ANCA: Perinuclear Antineutrophilic cytoplasmic antibody

the *Enterobacter* genus. Rarely, they have been shown to cause human infections.^[2] Most commonly infection occurs following a penetrating injury by vegetation.

There are seven *Pantoea* species identified: *P. agglomerans* (prototype species), *Pantoea ananatis*, *Pantoea stewartii*, *Pantoea dispersa*, *Pantoea citrea*, *Pantoea terrea*, and *Pantoea punctata*.^[3] *Pantoea* presented as lung infection, catheter-related infection, cholecystitis, or neonatal sepsis in various case reports. Lung cavities due to *P. agglomerans* are rare. There are scarce reports available worldwide.

Schmid *et al.* described a case of respiratory infections in a 71-year-old immunocompromised female patient with acute myeloid leukemia and multiple myeloma who developed a cavity lesion in the left lung with pleural effusion. Culture of BAL in this patient grew *P. dispersa* and the patient improved after adequate antibiotic (imipenem and gentamicin) therapy.^[4]

Hagiya and Otsuka described *P. dispersa* bacteremia caused by central line-associated bloodstream infection in a female patient with diabetes and cardiomyopathy. The pathogen was sensitive to ceftazidime, cefepime, and levofloxacin. She recovered after 4 weeks of cefepime therapy without recurrence of infection.^[5]

In India, Sengupta *et al.* reported the case of 2-day-old neonate presenting with recurrent generalized tonic-clonic seizure in the neonatal ICU. Blood culture was positive for Gram-negative bacilli. The organism was identified as *P. agglomerans*, susceptible to ceftriaxone, cefepime, meropenem, and colistin. Seizures subsided and the child was discharged in a stable condition on the 11th day.^[6]

Sarika *et al.* reported the case of a 25-year-old, healthy male, who presented with indurated swelling over the posterolateral aspect of his right thigh after trauma from a plant thorn where

P. agglomerans was isolated on a culture of the excised muscle tissue.^[7]

In our case, the causative organism was *P. agglomerans* which is a rare pathogen in causing cavitating lung lesions. Although we were not able to identify causative sources, as the patient did not have any history of trauma by plant thorns even though he was a farmer. A high degree of suspicion is required in patients with lung cavities having a history of plant thorn injury. Early detection of the organism is the key to prevent fatal complications.

CONCLUSION

Pantoea species may be a rare cause of lung cavitation, but it can be a possible cause of fatal cavitory lung disease leading to severe sepsis and mortality. Early detection and appropriate antibiotic therapy can improve the overall outcome.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Acknowledgement

Patient for sharing data.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

- Cheng A, Liu CY, Tsai HY, Hsu MS, Yang CJ, Huang YT, *et al.* Bacteremia caused by *Pantoea agglomerans* at a medical center in Taiwan, 2000–2010. *J Microbiol Immunol Infect* 2013;46:187-94.
- Gavini F, Mergaert J, Beji A, Mielcarek C, Izard D, Kersters K, *et al.* Transfer of *Enterobacter agglomerans* (Beijerinck 1888) Ewing and Fife 1972 to *Pantoea* gen. nov. as *Pantoea agglomerans* comb. nov. and description of *Pantoea dispersa* sp. nov. *Int J Syst Bacteriol* 1989;39:337-45.
- Delétoile A, Decré D, Courant S, Passet V, Audo J, Grimont P, *et al.* Phylogeny and identification of *Pantoea* species and typing of *Pantoea agglomerans* strains by multilocus gene sequencing. *J Clin Microbiol* 2009;47:300-10.
- Schmid H, Schubert S, Weber C, Bogner JR. Isolation of a *Pantoea dispersa*-like strain from a 71-year-old woman with acute myeloid leukemia and multiple myeloma. *Infection* 2003;31:66-7.
- Hagiya H, Otsuka F. *Pantoea dispersa* bacteremia caused by central line-associated bloodstream infection. *Braz J Infect Dis* 2014;18:696-7.
- Sengupta M, Banerjee S, Das NK, Guchhait P, Misra S. Early onset neonatal septicaemia caused by *Pantoea agglomerans*. *J Clin Diagn Res* 2016;10:D01-2.
- Sarika J, Ishwar B, Rakesh M, Sonal J, Chugh TD. *Pantoea agglomerans* infection behaving like a tumor after plant thorn injury: An unusual presentation. *Indian J Pathol Microbiol* 2012;55:386-8.