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# A rare case of community-acquired pneumonia complicated by severe Acute Respiratory Distress Syndrome in an immunocompetent male: *Aeromonas jandaei*

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**Key words:** *Aeromonas jandaei,* ARDS, multidrug-resistant pathogen, immunocompetent patient.

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**Availability of data and materials:** all data underlying the findings are fully available.

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**Informed consent:** informed consent was obtained from the patient's relatives. The patient's relatives gave their written consent to use his personal data for the publication of this case report and any accompanying images.

## Abstract

*Aeromonas jandaei* is an oxidase-positive Gram-Negative (GN) motile rod and is usually found in aquatic environments. It is commonly associated with gastrointestinal infections, wound infections, and septicemia. While species like *A. veronii* and *A. hydrophila* are known to cause pneumonia, we report a rare incidence of severe pneumonia complicated by Acute Respiratory Distress Syndrome (ARDS) caused by *A. jandaei,* highlighting the challenges in diagnosing and managing infections caused by rare, multidrug-resistant pathogens. This report describes a case of pneumonia caused by *Aeromonas jandaei*, a bacterium previously less documented in respiratory infections.

## Introduction

Aeromonas species are Gram-Negative (GN) bacilli found ubiquitously in fresh and brackish water habitats. These bacteria are facultatively anaerobic, non-sporulating, carbohydrate fermenters, and beta-hemolytic on a blood agar culture.[1,2] Initially classified within the family Vibrionaceae, Aeromonas has been reclassified based on subsequent phylogenetic analyses.[2] It is more commonly associated with gastrointestinal infections, wound infections, meningitis, peritonitis, and septicaemia.[3] While species like *A. veronii* and *A. hydrophila* are known to cause pneumonia, Aeromonas *jandaei,* an opportunistic bacterium, rarely causes respiratory infections like community-acquired pneumonia in an immunocompromised individual.[4]

Aeromonas species are uniformly resistant to commonly used antibiotics such as ampicillin, ceftriaxone, imipenem, piperacillin, and ticarcillin.[5] This resistance is mediated through various Antimicrobial Resistance (AMR) strategies, including efflux pumps, inhibition of drug uptake, and horizontal gene transfer, making infections difficult to treat.[6] In this report, we present a rare case of severe Acute Respiratory Distress Syndrome (ARDS) caused by *Aeromonas jandaei* in a previously healthy, immunocompetent adult, highlighting the importance of awareness regarding this organism as a respiratory pathogen, its resistance patterns, and the appropriate management of such rare pathogens.

## Case Report

A 52-year-old male well-digger with a history of bronchial asthma for 20 years on irregular inhalers and chronic hepatitis B infection treated with tenofovir disoproxil fumarate for 9 years

presented to the emergency department at a private hospital with a history of fever associated with chills for 5 days, shortness of breath for 2 days which was acute in onset, which had progressed from Modified Medical Research Council (mMRC) grade I to IV in 2 days. Later, due to worsening respiratory failure, he was referred to our center on the same day. He had no history of previous stroke, diabetes, or hypertension. Family members didn't give any history of recent drowning.

On arrival, the patient was afebrile, conscious, and oriented. On initial assessment, the individual had tachypnea (30 breaths per minute), tachycardia (104 beats per minute), hypotension (80/60 mmHg; Mean Arterial Pressure, MAP, 67 mmHg), and a Room Air Oxygen Saturation ($SpO_2$) of 89%. Initial Arterial Blood Gas (ABG) analysis showed type 1 hypoxemic respiratory failure with a $PaO_2/FiO_2$ ratio of 285. Chest X-ray showed left-sided heterogeneous opacities in upper and mid zones with left costophrenic angle blunting **(**Figure 1**).** Point-of-care ultrasound demonstrated normal left ventricular ejection fraction, Inferior Vena Cava (IVC) with >50% respiratory variability, and a left upper lobe C profile with minimal pleural effusion consistent with pneumonia.

The patient was empirically started on intravenous ceftriaxone and azithromycin suspecting Community-Acquired Pneumonia (CAP) as per intensive care protocol in India.[7,8] Hypotension resolved with initial intravenous fluid resuscitation suggesting hypovolemic shock. The patient was transferred to the Respiratory Intensive Care Unit (RICU) for further management.

Routine blood and sputum cultures were obtained. Ultrasound (USG) guided diagnostic thoracocentesis was performed from left mild pleural effusion and 40 mL of brownish fluid was aspirated. Laboratory parameters revealed a white blood cell count of 15500/$mm^3$ (normal

range 4000-11000/mm$^3$), hemoglobin level and platelet count were normal. Liver and renal function parameters were within the normal range. Paired blood cultures were reported sterile, but serum procalcitonin was elevated (14.6 ng/mL; normal <0.05 ng/mL).

A Computed Tomography Pulmonary Angiogram (CTPA) was performed given persistent tachycardia and worsening hypoxemic respiratory failure with a $PaO_2/FiO_2$ ratio of 167. The patient's CT imaging results revealed no evidence of pulmonary thromboembolism. The CT scan demonstrated left upper lobe consolidation and surrounding ground-glass opacities with interlobar septal thickening, and bilateral lower lobe perihilar consolidation with minimal effusion was also noted, the imaging findings were consistent with ARDS, likely caused by an infectious etiology given the short history and fever (Figure 2). Chest radiography also showed an increase in heterogeneous opacities in the left upper, mid and lower zone with left-side costophrenic angle blunting (Figure 3). Notably, a previous CT scan performed the day before at an outside hospital showed consolidation limited to the apical-posterior segment of the left upper lobe, indicating rapid deterioration of the patient's condition. The antibiotic was escalated to meropenam considering the severity of pneumonia and the patient was intubated and placed on volume-assist controlled mechanical ventilation. Sedation and paralysis were administered as per the ARDSnet trial.[9] The designated settings included a tidal volume of 420 mL (6 mL per kg ideal body weight), Positive End-Expiratory Pressure (PEEP) set at 10 cm $H_2O$, a respiratory rate at 30/min, and $FiO_2$ at 45%, P plateau constantly maintained below 30 cm $H_2O$. A Pressure-Volume (PV) tool was implemented manually to assess PEEP responsiveness, and subsequent adjustments were made accordingly. Sputum pyogenic culture, fungal cultures, Acid Fast Bacilli (AFB) smear, Cartridge-Based Nucleic Acid Amplification Technique

(CBNAAT) for *Mycobacterium tuberculosis* (MTB) and COVID-19, influenza, Reverse Transcription Polymerase Chain Reaction (RT-PCR) testing were all negative. Pleural fluid analysis confirmed an exudative, neutrophilic effusion with an Adenosine Deaminase (ADA) level of 38.2 U/L. Pleural fluid cultures and CBNAAT for MTB were negative.

On day 2 of admission, oseltamivir was added for viral coverage because of worsening oxygen requirement with a p/f ratio of around 108. Mini Bronchoalveolar Lavage (BAL) was performed as sputum cultures were sterile and sent for pyogenic culture which grew multi-drug resistant *Aeromonas jandei* (Figure 4) identified by Matrix-Assisted Laser Desorption/Ionization-Time of Flight (MALDI-TOF) mass spectrometry technique**.** The sensitivity report indicated resistance to ceftriaxone, piperacillin-tazobactam, and meropenam, while the organism was sensitive to sensitive to amikacin, levofloxacin, and trimethoprim-sulfamethoxazole (Table 1). Despite treatment efforts, the patient's condition did not improve, on the third day of admission, he succumbed to severe ARDS and respiratory failure. A post-mortem lung biopsy subsequently confirmed the presence of bronchopneumonia changes (Figure 5).

## Discussion

The first documented human Aeromonas infection was reported in 1951 when the organism was isolated from cerebral spinal fluid during an autopsy in Jamaica.[8] Since then, the genus has expanded to include 36 species, with 19 identified as emerging human pathogens. *A.*

*jandaei*, named after J. Michael Janda was first isolated in the USA in 1991 from clinical samples such as blood, wounds, and stools.[1]

*Aeromonas hydrophila* is the most commonly implicated species in human infections, although rare cases of *A. caviae* and *A. sobria* have been reported, particularly in burn patients, where infections are associated with high mortality rates.[10] *A. jandaei,* isolated in our case, has traditionally been associated with skin, soft tissue, and gastrointestinal diseases. To our knowledge, this is the first reported instance of *A. jandaei* causing ARDS in an immunocompetent individual.

Risk factors for Aeromonas infection include immunocompromised states and exposure to environmental water or soil sources.[11] The gastrointestinal tract serves as the primary portal of entry. Soft tissue infections, including necrotizing infections, have been documented following contamination of open wounds with soil. *A. hydrophila* has also been identified in pneumonia cases associated with near-drowning incidents with reported mortality as high as 50%.[4]

Our patient's occupation as a well-digger suggests possible exposure to contaminated water sources harboring *Aeromonas jandaei.* The rapid and severe disease progression observed in our case was attributed to the organism's virulence factors and its intrinsic resistance to a wide range of antibiotics, including ampicillin, cefazolin, and imipenem.[12] Aeromonas species are known to produce various virulence factors such as hemolysins, cytotoxins, enterotoxins, proteases (like AspA and AhpB), lipases (Pla and Plc, Sat), DNAses, adhesins (type IV pili, polar flagella), capsule, and T3SS.[3] Hemolysins and proteases likely contributed to the rapid

disease progression observed in our patient. Poor prognostic indicators include shock, admission to the intensive care unit, and the need for mechanical ventilation.[13]

The overuse of antibiotics has accelerated AMR development in various bacterial pathogens, leading to treatment complexities, increased mortality rates, and prolonged hospital stays. Horizontal gene transfer is a common mechanism of AMR acquisition in Aeromonas, as demonstrated by the ability of environmental isolates to acquire and transfer resistance genes.[14] In our case, *Aeromonas jandaei* exhibited resistance to beta-lactams and carbapenems, which are frequently used empirical antibiotics for severe pneumonia.[8] Recommended antimicrobial therapies for *Aeromonas pneumonia* typically involve third or fourth-generation cephalosporins or quinolones,[10] the emergence of multi-drug resistant *Aeromonas jandei,* underlines the importance of starting appropriate antibiotics earlier in treating such infection.

## Conclusions

This case emphasizes the importance of clinical awareness regarding Aeromonas infections in patients presenting with rapidly progressing skin, soft tissue, gastrointestinal, or respiratory infections, particularly those with exposure to freshwater environments. Early recognition, appropriate antibiotic administration, and comprehensive supportive care are crucial for achieving favorable outcomes.

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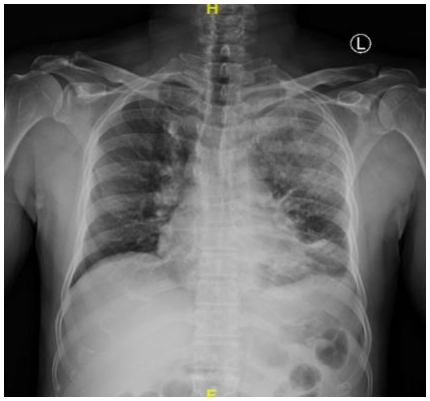
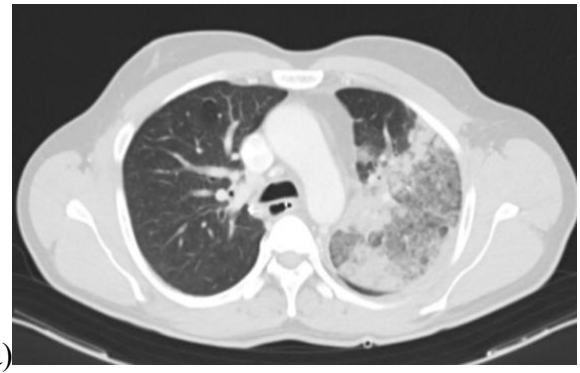


**Figure 1.** Chest radiography posteroanterior view taken on day 1 of admission showing heterogenous opacity in the left upper and mid-zone with costophrenic angle blunting consistent with pneumonia.

a) 

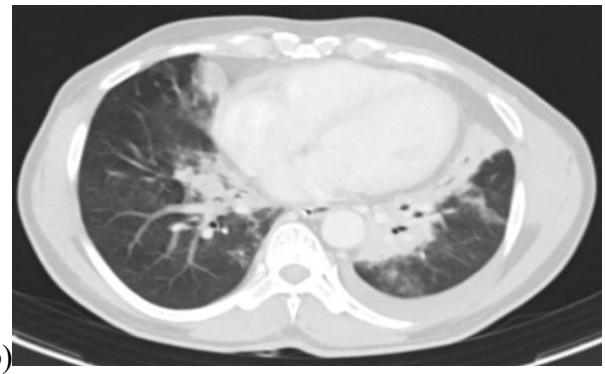
b) 

**Figure 2. a)** Computed Tomography Pulmonary Angiogram (CTPA) of the thorax (axial view) showing left upper lobe consolidation with surrounding ground glass opacities (suggested by arrows) and interlobar septal thickening. **b)** CTPA with axial view showing bilateral lower lobe perihilar consolidation with left minimal effusion consistent with acute respiratory distress syndrome.

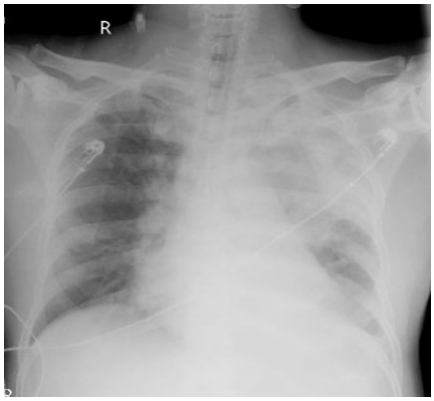



**Figure 3.** Chest radiography posteroanterior view showing an increase in heterogeneous opacities in left, upper, mid zone, and lower zone with left side costophrenic angle blunting indicating rapid progression.

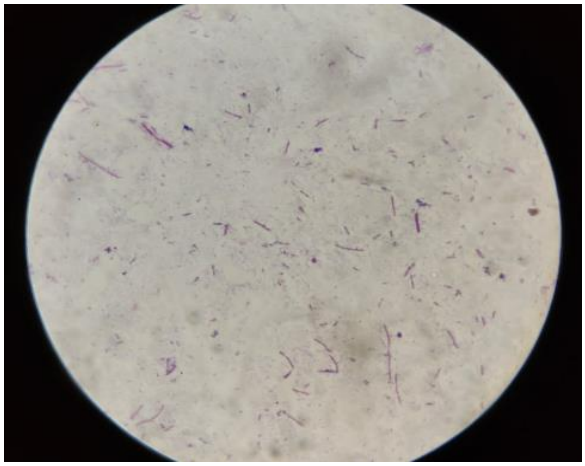


**Figure 4.** Gram staining of mini bronchoalveolar lavage showing gram-negative rods.

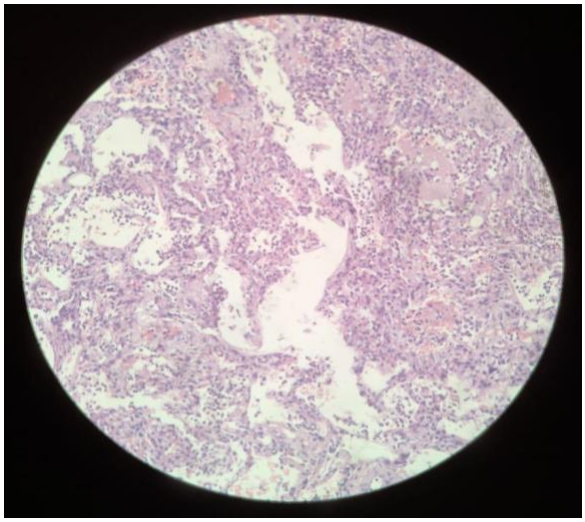


**Figure 5.** Post-mortem biopsy of left lung showing alveolar dilation with focal congestion in intra alveolar as well as interstitial neutrophilic infiltration suggestive of bronchopneumonia.

**Table 1.** Antimicrobial sensitivity pattern of *Aeromonas jandaei* isolated in our case.

| ANTIBIOTIC | MIC dilution (µg/mL) | MIC interpretation |
|---|---|---|
| Ceftriaxone | 4 | Resistant |
| Piperacillin tazobactam | >128 | Resistant |
| Meropenem | >16 | Resistant |
| Ciprofloxacin | <0/06 | Sensitive |
| Amikacin | 8 | Sensitive |
| Cotrimoxazole | <8 | Sensitive |

MIC, Minimum Inhibitory Concentration