

## Case Report

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# A Case of Fulminant Type Community-Acquired Pneumonia Found to be Caused by *Pseudomonas aeruginosa* at Autopsy

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## Abstract

The present case involved a 27-year-old woman, who presented to a nearby physician with chief complaints of fever and dyspnea and hospitalized with a diagnosis of right lobar pneumonia on the same day. The patient was treated with antibiotics (aminobenzylpenicillin/sulbactam); however, exacerbation of respiratory status occurred with hypotension, and she was transferred to the authors' hospital under tracheal intubation. The right superior lobe exhibited a wide range of infiltrative shadows with air bronchograms. Vasopressor and continuous hemodiafiltration were initiated to address overall exacerbation, and antibiotics (ciprofloxacin) were added. However, ventilation could not be maintained due to the large quantity of respiratory secretions; consequently, extracorporeal membrane oxygenation (ECMO) was initiated 5 h after admission to the intensive care unit. Due to the large quantity of Gram-negative bacilli confirmed by gram stain of respiratory secretions, amikacin was added. Although oxygenation was improved after initial ECMO induction, the patient died on day 2 of admission, despite being revived from sudden cardiac arrest. *Pseudomonas aeruginosa* was detected in respiratory secretions, blood cultures, and all lung tissues at autopsy, and the illness was believed to be a fulminant form of community-acquired pneumonia caused by the organism. This is a rare case and autopsy finding and, as such, contributes to the scarce literature.

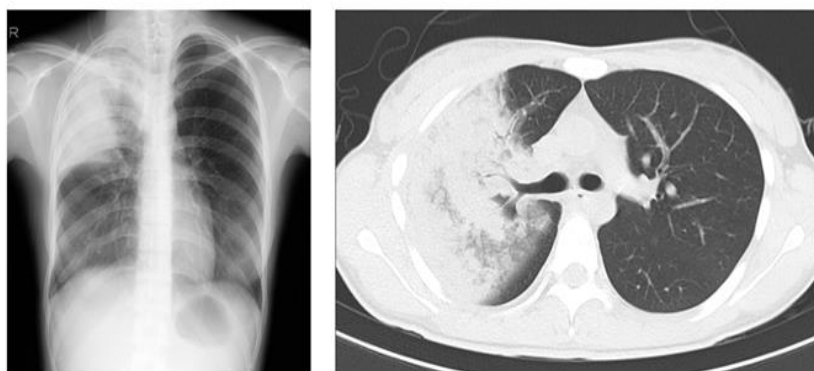
**Keywords:** *Pseudomonas aeruginosa*; Fulminant community-acquired pneumonia; Autopsy

## 1. Case Presentation

The patient was a 27-year-old woman who developed a fever 2 days before hospital consultation and received a prescription for an antipyretic analgesic by a nearby physician and sent home. However, because dyspnea appeared on the day before consultation, the hospital was consulted, and it was hospitalization by a diagnosis of the right lobar

pneumonia in an image in the hospital. Although she was treated with antibiotics (aminobenzylpenicillin [ABPC]/sulbactam [SBT]), her respiratory status became exacerbated, with a rapid reduction in blood pressure. She was transferred to the authors' hospital after intubation in the middle of the night.

The patient was a beautician, with a medical history of untreated, intermittent bronchial asthma. She smoked 20 cigarettes per day, but had no history of alcohol consumption or allergies, nor a history of bird exposure, hot-spring public bathing, or extensive foreign travel. On admission, her symptoms were G2VTM2 on the Glasgow Coma Scale, in an intubated state with 10 L of oxygen by mask. The patient exhibited tachycardia (heart rate, 110 beats/min), hypotension (blood pressure, 85/65mmHg) and decreased body temperature (35.7°C), with a respiratory rate of 24 breaths/min. Oxygen saturation (SpO<sub>2</sub>) was not measured for peripheral coldness. On auscultation, breath sounds were diminished in the right embryonate. An infiltrative shadow with air bronchogram was apparent at the top right corner embryonate on chest X-ray at the previous hospitalization. Similarly, simple chest computed tomography revealed an infiltrative shadow on the right superior lobe. These findings were absent in the right lower lung field, left embryonate (Figure 1).



**Figure 1:** Pulmonary imaging. Chest X-ray revealing a consolidation shadow in the right apex area. Chest computed tomography revealing consolidation with air bronchogram shadow in the right superior lobe.

CBC/Coagulation		Biochemistry		Infection	
WBC	2.2x10 <sup>3</sup> /μl	TP	2.4 g/dL	CRP	11.45 mg/dL
RBC	3.85x10 <sup>6</sup> /μl	Alb	1.2 g/dL	PCT	0.5 ng/mL
Hb	11.7 g/dL	LDH	569 IU/L	Urinary pneumococcus antigen (-) Urinary Legionella antigen (-) (Arterial blood gas Fi <sub>2</sub> 1.0)	
Hct	36.4%	γ-TP	5 IU/L		
Plt	108x10 <sup>3</sup> /μl	CK	140 IU/L		
APTT	120 s	BUN	35.8 /dL	pH	6.868
Fib	50 mg/dL	Cre	1.69 mg/dL	PCO <sub>2</sub>	130 mmol/L
FDP	1159.3 μg/dl	UA	4.8 mg/dL	PO <sub>2</sub>	136.6 mmol/L
D-dimer	14.5 μg/dl	Na	144 mEq/L	HCO <sub>3</sub>	23.11 mmol/L
AT III	22%	K	2.9 mEq/La	Lac	35.1 mg/dL

**Table 1:** Laboratory findings

A blood count revealed a low white blood cell (220/ $\mu$ l) and platelet (108,000/ $\mu$ l) counts. Coagulation tests revealed elevated levels of D dimer and fibrinogen/fibrin degradation products (14.5 and 1159.3  $\mu$ g/dl, respectively), and ATIII was decreased (22%). Biochemical tests revealed renal dysfunction and hypoalbuminemia. High levels of C-reactive protein (11.45 mg/dl) were also observed. Examination for the urinary antigen of pneumococcus, Legionella, was negative. Partial pressure of carbon dioxide ( $\text{PaCO}_2$ ) was 130 torr and partial pressure of oxygen ( $\text{PaO}_2$ ) was 136.6 torr, hypoxemia with hypercapnia was found with artificial breathing on 100% oxygen (pH 6.868). Blood gas analysis revealed elevated lactate levels (Table 1).

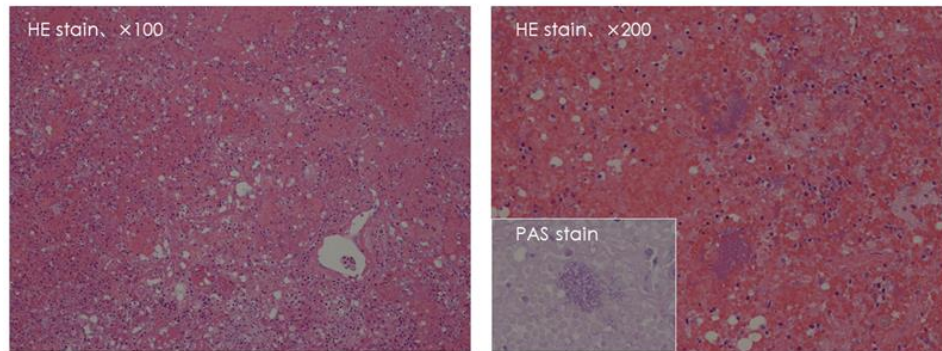
Corticosteroids, vasopressors and sustained hemofiltration dialysis were initiated. Ventilatory volume decreased, and suction of fluids secreted from the airway, which were significant and frequent, was required. Due to progression of acidemia, extracorporeal membrane oxygenation (ECMO) was initiated. ABPC/SBT and levofloxacin were continued, and amikacin (AMK) was added because a large quantity of Gram-negative bacilli was confirmed on sputum gram-stain of secreted airway fluids. Additionally, endotoxin adsorption therapy was administered because blood endotoxin levels increased to 25 pg/ml. Nevertheless, the patient experienced sudden cardiac arrest 46 h after hospitalization. Cardiopulmonary resuscitation was performed immediately, and a heartbeat was rescued once; however, blood pressure could not be maintained, nor could heart rate after catecholamine administration. Death was confirmed 90 min after the first cardiac arrest. A pathological autopsy was performed. A sectioned surface of the lung is shown in Figure 2, with an invasive entity found in the right superior lobe. Evidence of pulmonary hemorrhage was also found. As the main lesion, macroscopic findings indicated that multiple organ failure was not found in the hemorrhagic pneumonia of both lungs.



**Figure 2:** Pathological anatomy findings (macroscopic) from a sectioned surface of the lung. An invasive entity is apparent in the right superior lobe. General evidence of pulmonary hemorrhage is apparent.

Macroscopic imaging of the lung revealed that alveolar bleeding was the main evidence of hemorrhagic pneumonia with neutrophilic infiltration. Higher-power imaging suggested the presence of *Pseudomonas aeruginosa* supported by Gram-negative bacilli stained by periodic acid-Schiff staining (Figure 3). *P. aeruginosa* grew in all cultures of

secretions from the right superior lobe, blood cultures of the airway, and the pathological anatomy. Septic shock due to pseudomonas pneumonia was diagnosed.



**Figure 3:** Pathological anatomy findings (microscopic). A low-power image of the lung. Alveolar bleeding was supportive evidence suggesting hemorrhagic pneumonia with neutrophilic infiltration. With a higher-power imaging, *Pseudomonas aeruginosa* was suspected based on gram-negative bacilli on periodic acid-Schiff staining.

## 2. Discussion

Pneumonias that occur in community settings usually have little or no direct connection with hospitals. As such, so-called “community-acquired pneumonia” is significantly different from pneumonia due to nosocomial infections, and depend on the type of pathogen and the identification, differential diagnosis, and many points such as the timing of treatment and intervention(s). There are predominant pneumococci causing community-acquired pneumonia, and, among the primary causative organism(s), atypical pneumonia, including *Hemophilus influenzae* and mycoplasma, are also common [1-4]. In contrast to hospital-acquired pneumonia, even in the case of elderly individuals, *P. aeruginosa* is not usually included in the differential diagnosis of community-acquired pneumonia [3]. Takayanagi et al. reported the frequency of community-acquired pneumonia due to *P. aeruginosa* in Japan to be 6.3% [5].

Pneumonia caused by *P. aeruginosa* is a predominantly nosocomial infection [3], and is extremely rare and does not often occur without underlying disease and risk factor(s) such as those exhibited by the patient described in the present report [3, 6]. Similar to Legionella, the use of a humidifier and/or foaming bathtubs are known to cause *P. aeruginosa* pneumonia; however, few such cases have been reported [3, 6]. In community-acquired pneumonia, *P. aeruginosa* is not usually considered as a primary causative organism of community-acquired pneumonia. More often, primary causative pneumococcus organisms, such as *H. influenzae*, or primary causative organisms of atypical pneumonia, such as *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, and Legionella are suspected [1-4]. It is extremely difficult to predict the bacterial origin of pneumonia as *P. aeruginosa* before culture results are confirmed, unless pneumonia due to Gram-negative bacilli from gram staining of sputum is strongly suspected.

Pneumonias caused by nosocomial infection(s) and underlying respiratory disease are usually sudden. Despite the rarity of pseudomonal community-acquired pneumonias without respiratory underlying disease, many die from a fulminant course similar to this case [7, 8]. Henderson et al. [7], reported two cases of *P. aeruginosa* pneumonia without underlying disease, but who already exhibited shock and significant hypoxemia, similar to our case, at the

hospital visit, and died within several hours after hospitalization. *P. aeruginosa* was not suspected in these cases, but became clear after culture results and death, similar to the case described in the present report. One patient was a 52-year-old man, in whom *Legionella* was suspected as the primary causative organism. He died 1 h after hospitalization without responding to treatment. On autopsy, air vesicles were full of bloody decoction, and bleeding was found in both adrenal glands. Friedlander's bacillus was the suspected primary causative organism in other case, a 27-year-old woman, who died 10 h after hospitalization without responding to vasopressor and oxygenation. On autopsy, air vesicles were full of bloody decoction. The disease course in both individuals was extremely violent, and both died despite gentamicin susceptibility. The prognosis of the *Pseudomonas aeruginosa* community-acquired pneumonia is extremely poor. Rello et al. examined 204 cases of community-acquired pneumonia of the severe form that required an intensive care, eight case of those are *pseudomonas pneumonia* and reported that seven of eight case died [8].

### 3. Conclusion

In this case, the patient was complicated by disseminated intravascular coagulation, septic shock and acute respiratory distress syndrome, and was suddenly aggravated. Despite combined multi-modality therapy, the patient died on the fourth day of illness. There was no consultation with the medical institution, and underlying disease associated with smoking history in youth was not found. Additionally, the *P. aeruginosa* species detected had good chemical sensitivity to systemic carbapenem and quinolone, with no results indicating resistance. It is not apparent why *P. aeruginosa* pneumonia without underlying disease exhibits a violent course. Both patients described by Henderson et al. died < 36 h from onset and, even if diagnosis is difficult, the violent course cannot be explained solely by a delay in treatment. Because case 1 described by Henderson et al. exhibited bleeding in both adrenal glands, Waterhouse-Friderichsen syndrome should have been considered [9] due to similar meningococcal pathology. However, bilateral adrenal hemorrhage is not necessarily caused by *P. aeruginosa* pneumonia all cases. Evidence of adrenal hemorrhage was not found at autopsy in the present case.

*P. aeruginosa* is a bacterial species not commonly assumed to be the cause of community-acquired pneumonia; however, it should be considered in cases of severe pneumonia with rapid exacerbation, for which it is necessary to start a broad-spectrum antimicrobial agent at an early stage.

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