

# *Aeromonas* Pneumonia in a Trauma Patient Requiring Extracorporeal Membrane Oxygenation for Severe Acute Respiratory Distress Syndrome: Case Report and Literature Review

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## Abstract

**Background:** *Aeromonas* species, particularly *Aeromonas hydrophila*, cause a wide spectrum of diseases in human being such as gastroenteritis; soft tissue infections including necrotizing fasciitis, meningitis, peritonitis, and bacteremia; but pneumonia and respiratory tract infections are uncommon.

**Methods:** Case report and literature review.

**Results:** A 30-year-old victim of a motor vehicle crash sustained pelvic fractures and splenic injury. Delayed splenic rupture caused sudden cardiorespiratory arrest. The patient was resuscitated but suffered septic shock and severe hypoxemia refractory to advanced mechanical ventilatory strategies. *Aeromonas hydrophila* was isolated as the causative pathogen of severe bilateral pneumonia. Venovenous extracorporeal membrane oxygenation (ECMO) was used temporarily. The patient recovered uneventfully.

**Conclusion:** This is the first case, to our knowledge, of the use of ECMO in a trauma patient with severe fulminant *A. hydrophila* pneumonia. Clinicians should be aware of the characteristics of this pathogen and associated clinical infections.

**AEROMONAS** SPECIES are water-borne motile gram-negative non-spore-forming bacilli widely distributed in the aquatic environment, both fresh- and salt-water. The species has been implicated in rare opportunistic infections in humans involved in aquatic trauma, those with burns, and immunocompromised patients such as those with leukemia or other cancer [1].

*Aeromonas* species, particularly *Aeromonas hydrophila*, cause a wide spectrum of diseases in humans such as gastroenteritis, soft tissue infections including necrotizing fasciitis, meningitis, peritonitis, and bacteremia [2–9]. Although there are reports of extraintestinal human infections caused by *Aeromonas* in both immunocompromised and immunocompetent patients, respiratory tract infections remain uncommon [10–12]. To our knowledge, the present paper is the first report of the use of extracorporeal membrane oxygenation (ECMO) support for severe acute respiratory distress syndrome (ARDS) related to *Aeromonas* pneumonia.

We report a patient involved in a motor vehicle crash who sustained multiple injuries and developed severe pneumonia

with worsening hypoxemia and ARDS requiring ECMO. *Aeromonas hydrophila* was the causative pathogen.

## Case Report

A 30-year-old otherwise-healthy man was involved in a rollover motor vehicle crash as an unrestrained driver with ejection from the vehicle onto the pavement. The patient was neurologically and hemodynamically stable on the scene. He was airlifted to the University of Michigan Medical Level I Trauma Center. The initial examination confirmed hemodynamic stability, and the injuries included multiple pelvic fractures and a grade I splenic injury with no free fluid in the peritoneum by computed tomography (CT) imaging of the abdomen/pelvis. No thoracic injuries were identified by chest radiography (Fig. 1). He was admitted for non-operative management of his pelvic fractures and splenic injury.

On post-injury day 3, he was found in cardiac arrest with pulseless electrical activity. The Advanced Cardiac Life Support protocol was initiated, with endotracheal intubation

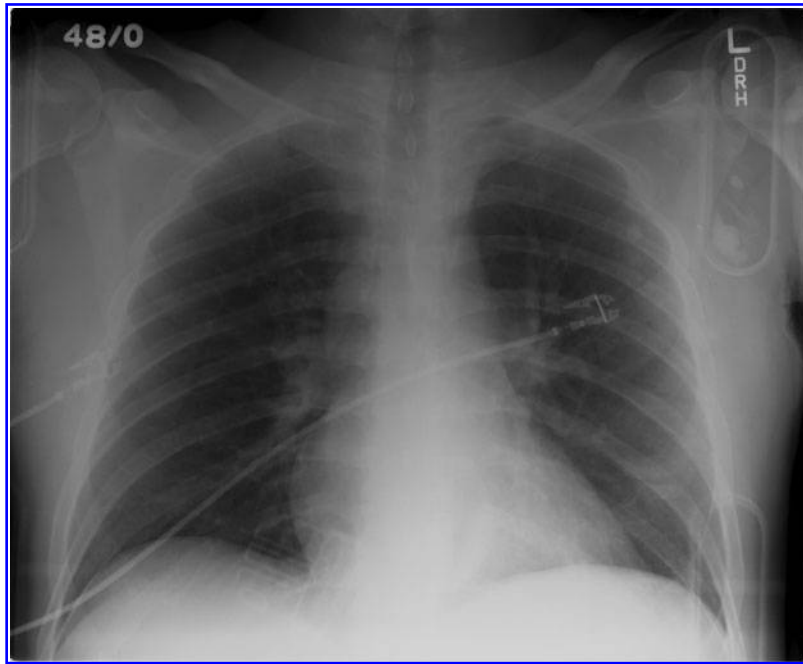


FIG 1. Chest radiograph on hospital admission post-accident revealing no traumatic injuries. Normal chest radiograph.

and resuscitation, leading to return of spontaneous cardiac rhythm and blood pressure, and he was transferred to the intensive care unit. He was intubated on first attempt, Mallampati Class I laryngoscopy view, with no evidence of aspiration. Delayed splenic rupture was confirmed by CT scan of the abdomen performed immediately after stabilization of hemodynamics (Fig. 2). Angiographic embolization was successful.

The patient subsequently developed severe hypoxemia with increasing oxygen requirement, and bilateral infiltrates were noted on the chest radiograph consistent with ARDS or bilateral bacterial or aspiration pneumonia (Fig. 3). A CT scan confirmed no evidence of pulmonary embolus. Evidence of hepatic ischemic injury with increased serum concentrations of his liver enzymes was noted on laboratory examination.

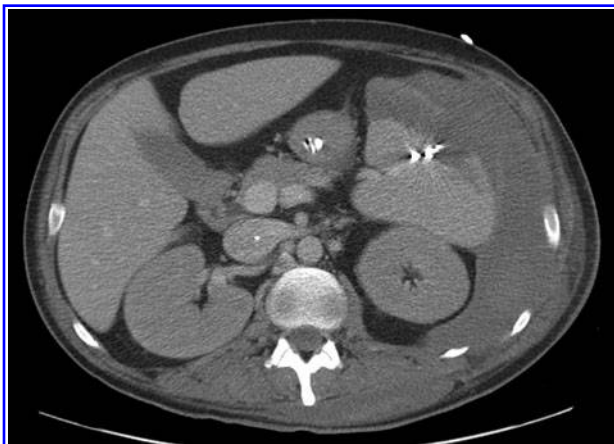
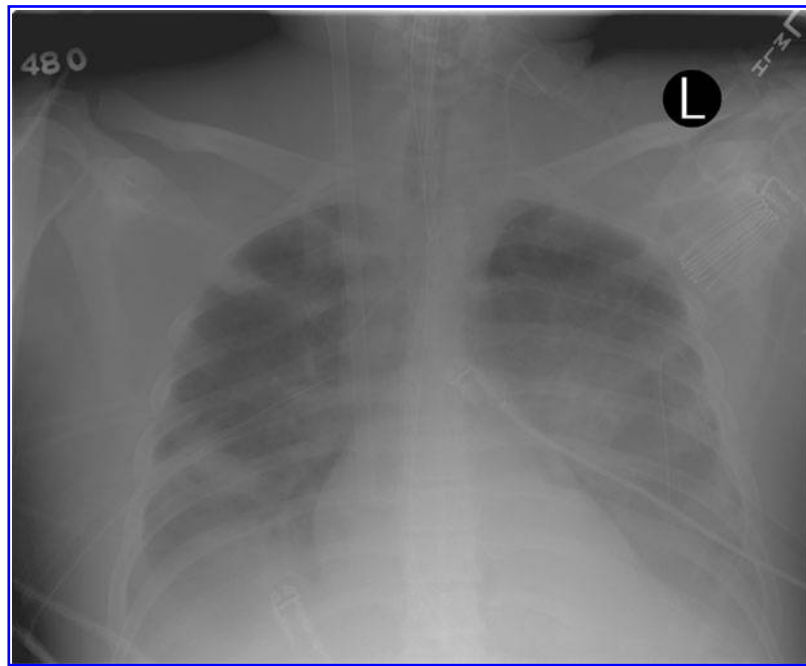


FIG. 2. Computed tomography scan of abdomen documenting splenic rupture, hemoperitoneum, and angiographic coil embolization.

Persistent fever and neutropenia developed, with a decrease in the white blood cell count to a nadir of  $1.0 \times 10^9$  /dL. At this time, he had evidence of septic shock with multiple organ dysfunction syndrome, including acute renal failure necessitating renal replacement therapy. Cultures of blood and urine were obtained, and diagnostic bronchoscopy with bronchoalveolar lavage and quantitative cultures were performed to evaluate for pneumonia as a sepsis source. Empiric antimicrobial management with intravenous piperacillin-tazobactam and vancomycin was initiated. Vasopressors (norepinephrine, vasopressin) and steroids were used for management of his septic shock. Granulocyte colony-stimulating factor (G-CSF) was given as treatment for severe neutropenia.

The patient's respiratory function continued to deteriorate, with persistent hypoxemia despite the provision of high  $FiO_2$  (1.0). Multiple mechanical ventilation strategies were used in attempts to improve oxygenation, including recruitment maneuvers, inverse-ratio pressure control ventilation, and high-frequency oscillatory ventilation (HFOV), but hypoxemia persisted.

An ECMO evaluation was requested for severe ARDS and refractory hypoxemia, and, given the likely reversible nature of his pulmonary disease and hypoxemia, he was cannulated for venovenous ECMO (VV-ECMO) with right internal jugular and right femoral cannulas. Full systemic anticoagulation with continuous infusion of unfractionated heparin was initiated for ECMO. Because he had undergone angiographic embolization of his splenic injury two days prior, it was recognized that he was at high risk for intra-abdominal bleeding, which would be difficult to diagnose. A diagnostic peritoneal lavage (DPL) catheter was inserted (open technique) after initiation of VV-ECMO to monitor for signs of intra-peritoneal bleeding while on ECMO and maintained sterile with an occlusive dressing. The catheter was interrogated daily by sterile saline lavage.



**FIG. 3.** Chest radiograph after cannulation for venovenous extracorporeal membrane oxygenation demonstrating bilateral infiltrates consistent with acute respiratory distress syndrome or bilateral pneumonia.

Cultures of bronchoalveolar lavage fluid confirmed *Aeromonas hydrophila* >10,000 colony-forming units (CFU)/mL and *Streptococcus pneumoniae* >10,000 CFU/mL (Table 1). All other cultures, including of blood, were negative. Levofloxacin was added, and the dose for piperacillin-tazobactam

was adjusted according to the minimum inhibitory concentration (MIC) values on the microbiology report.

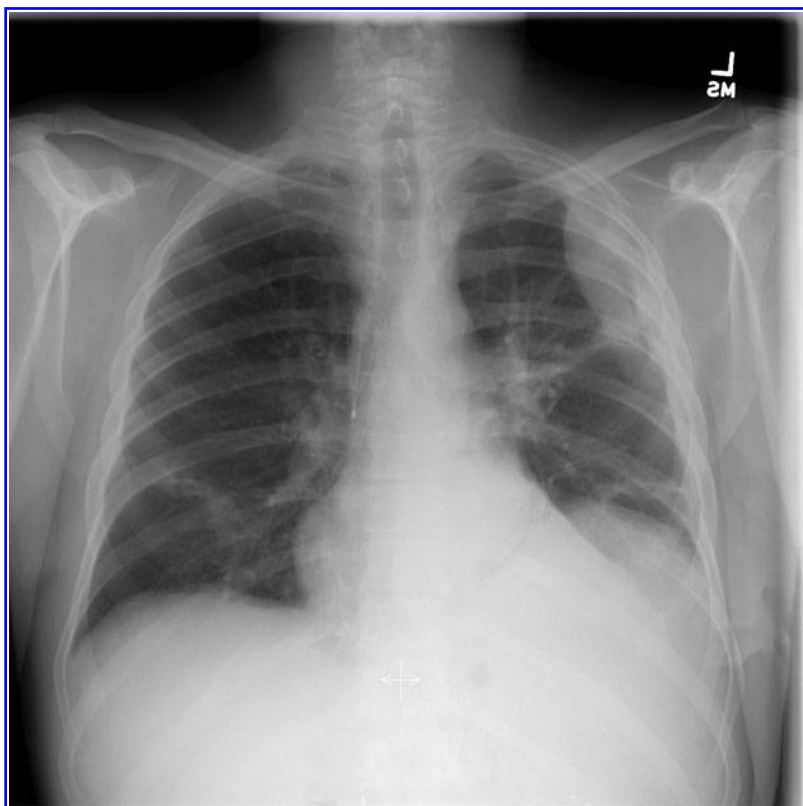
In anticipation of a prolonged need for ventilatory support, open tracheostomy was performed on VV-ECMO day two. The white blood cell count normalized, and G-CSF was discontinued. Hepatic enzymes normalized gradually. He was removed from ECMO after lung recruitment with HFOV. The ECMO was discontinued, and he was decannulated on postinjury day nine (VV-ECMO duration six days). An inferior vena cava (IVC) filter was placed just prior to decannulation with intravascular ultrasound guidance at the bedside in the surgical intensive care unit, with confirmation of IVC thrombus during the procedure. He was transitioned from HFOV to conventional ventilatory support with bilevel ventilation. He had full renal recovery, and renal replacement therapy was discontinued. Liberation from mechanical ventilation occurred on postinjury day 27, and his tracheostomy tube was removed (Fig. 4). The patient had a full uneventful recovery thereafter and was discharged home on postinjury day 51, able to perform all activities of daily living and with no neurologic sequelae from the cardiac arrest and severe hypoxemia.

## Discussion

*Aeromonas* species is a rare causative agent of human infection. It is associated with high mortality rates, as high as 70% [13]. *Aeromonas hydrophila* is the most common species implicated in human infections, with rare reports of *A. caviae* and *A. sobria* causing infections of burn patients, also with high mortality [14]. *Aeromonas hydrophila*, a member of the family *Vibrionaceae*, is a gram-negative bacillus, a catalase- and oxidase-positive, facultative anaerobe that is ubiquitous; it is common in aquatic media and has a worldwide distribution. It is isolated frequently from water, soil, and certain

**TABLE 1. ANTIMICROBIAL SUSCEPTIBILITIES (MINIMUM INHIBITORY CONCENTRATIONS) OF PATHOGENS IDENTIFIED IN BRONCHOALVEOLAR LAVAGE FLUID CULTURES**

	<i>Aeromonas hydrophila</i> >10,000 CFU/mL	<i>Streptococcus pneumoniae</i> >10,000 CFU/mL
Penicillin		<0.06
Cefepime	<8.00	
Piperacillin	<8.00	
Piperacillin-tazobactam	<8/4	
Cefuroxime		<0.25
Cefoxitin	<8.00	
Ceftriaxone	<8.00	<0.06
Aztreonam	<1.00	
Meropenem	<4.00	<0.03
Gentamicin	<4.00	
Tobramycin	<4.00	
Amikacin	<16.00	
Trimethoprim-sulfamethoxazole	<2/38	.12/2.37
Erythromycin		<0.06
Clindamycin		<0.06
Doxycycline	<4.00	<0.50
Vancomycin		<0.125
Levofloxacin	<2.00	<0.50
Ciprofloxacin	<1.00	



**FIG. 4.** Chest radiograph after extracorporeal membrane oxygenation decannulation, weaning from mechanical ventilation, and tracheostomy decannulation.

foods. In immunocompetent hosts, respiratory infection attributable to this pathogen occurs by ingestion of contaminated water [15].

*Aeromonas hydrophila* is generally susceptible to a wide range of antimicrobial drugs [16]. *Aeromonas* species can express  $\beta$ -lactamases and hence may be resistant to penicillin, first-generation cephalosporins, vancomycin, and clindamycin [17]. Despite the abundant knowledge about inducible, chromosomally-mediated  $\beta$ -lactamases among *Aeromonas* species, extended-spectrum beta-lactamase (ESBL)-producing *A. hydrophila* strains have rarely been isolated in clinical practice [18]. There are recent reports of *A. hydrophila* strains resistant to tetracyclines, cotrimoxazole, aminoglycosides, and extended-spectrum cephalosporins [19–21]. Apart from rare reports in the literature [22,23], *A. hydrophila* is consistently sensitive to third-generation cephalosporins, aztreonam, fluoroquinolones, aminoglycosides, and carbapenems [24–26].

Risk factors for *Aeromonas* infection include an immunocompromised state and an environmental water or soil source [27,28]. The gastrointestinal tract is the main portal of entry. Soft tissue infections, including necrotizing infections, have been reported in instances of injury and contamination of open wounds with soil [29,30]. Additional reports of sepsis in burn patients have documented contact with soil or water.

However, severe, even fatal, *Aeromonas* infections have been reported in immunocompetent normal children with pneumonia and sepsis [31,32]. *Aeromonas* also has been reported as a causative pathogen in cases of near-drowning-associated pneumonia [33].

The portal of entry of the *Aeromonas* organism was not readily identified in our patient, although aspiration may have occurred after ejection. Presumed aspiration pneumonia caused by *Aeromonas hydrophila* in immunocompetent patients has been described previously [34]. We also investigated the possibility that the patient acquired the infection through the hospital environment, either via the ventilator or as a cross-infection while in the trauma/burn ICU, but there were no reports of additional *Aeromonas* clinical infections in that ICU for the time period.

The severity of the *A. hydrophila* pneumonia infection in this patient necessitated the use of VV-ECMO as a bridging strategy to pulmonary recovery. The ECMO required full systemic anticoagulation with heparin, and, given the patient's underlying splenic injury, we utilized a DPL catheter as an inexpensive, reliable, and readily available monitoring device for signs of intraperitoneal bleeding, because transport of ECMO patients to a CT scanner can be challenging. This case highlights the potentially serious nature of *Aeromonas* pneumonia infection in trauma patients.

#### Author Disclosure Statement

No conflicting financial interests exist.

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