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Case report

An unusual case of *Salmonella* Enteritidis causing pneumonia, septic shock and multiple organ failure in an immunocompetent patient

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ABSTRACT

Salmonella species are facultative intracellular pathogens that most frequently cause self-limiting gastrointestinal disease, often acquired through the ingestion of contaminated food. We report the case of a 33-year-old otherwise healthy, not overtly immunosuppressed, man who was transferred to our facility with the chief complaint of respiratory failure and septic shock. Computed tomography of the chest revealed multifocal pneumonia in both lungs. A bronchial alveolar lavage was performed in the right middle lobe and cultures predominantly grew *Salmonella enterica* serovar Enteritidis. The patient received a prolonged course of antimicrobials, ultimately changing to oral levofloxacin. The etiology of the salmonella infection likely occurred through an aspiration event. Salmonella species are not a typical respiratory pathogen in immunocompetent hosts; however, clinicians should be aware of the possibility that salmonella species may be a pathogenic source of infection in the lungs; a prolonged course of antimicrobials may be warranted.

Introduction

Salmonellosis may present with varying clinical infections from gastroenteritis to enteric fever [1]. According to the Centers for Disease Control and Prevention (CDC), salmonellosis is estimated to cause one million cases of illness in the United States, with 19,000 hospitalizations and 380 deaths annually [2]. Pneumonia, however, is an atypical site for salmonellosis. Most individuals infected with the bacterium develop fever, abdominal cramps, and diarrhea between 12 and 72 h after infection. The illness manifests as gastrointestinal distress and generally lasts 4–7 days; however, more severe infections can invade into the blood stream and cause bacteremia with the possibility of disseminated focal infection. The severity and risk of translocation are often determined by the virulence of the isolate and host immunity [1]. In severe cases patients are at risk of death if not promptly treated with antimicrobials. Infants, elderly, and individuals who are immunocompromised are more likely to present with severe illness from salmonellosis [2].

Case report

A 33-year-old white male with an intellectual disability residing in a community group home was admitted to an outside hospital with nausea, vomiting, and diarrhea and was found to be hypotensive with acute kidney injury (AKI) and a serum creatinine of 5 mg/dL. Past medical history was significant for hypertension, schizophrenia, depression, anxiety, and seizures. The patient's medication history directly from the patient's group included metoprolol tartrate, mirtazapine, prazosin, sertraline, cyproheptadine, vitamin D supplement, multivitamin, divalproex sodium, quetiapine and pantoprazole. At the time of admission he was not known to have any medication allergies, except a possible intolerance to phenytoin.

While at the outside hospital, his respiratory status quickly declined requiring emergent intubation and transfer to the ICU. A computed tomography (CT) of his abdomen revealed diffuse edema in the rectum, colon, and small bowel, as well as vasoconstriction of the mesenteric arteries. Due to the patient's elevated lactate of 6 mmol/L an exploratory laparotomy was

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performed with no significant findings. The patient was started on intravenous linezolid, meropenem and metronidazole prior to transfer due to septic shock with suspected abdominal or pulmonary source. In addition, he underwent hemodialysis prior to transfer due to oliguric AKI with severe metabolic acidosis.

Two days after initial admission, the patient arrived at our facility and was found to be febrile, hypotensive with septic shock and multiple organ failure. Maximum recorded temperature on admission was 102.2 °F. The patient had a leukocytosis with a white blood cell count of 18.2 k/uL (normal range 3.7–10.3 k/uL) comprised of: neutrophils 81% (normal range 34–71%), lymphocytes 7%, (normal range 20–55%), and eosinophils 0% (normal range 1–7%). The immature granulocyte count was 2%, (normal range 0–1%). Renal function tests were found to be abnormal with serum creatinine 2.59 mg/dL (normal range 0.80-1.3 mg/dL) and blood urea nitrogen 23 mg/dL (normal range 7–21 mg/dL). Liver function tests were also abnormal with aspartate aminotransferase (AST) elevated at 2035 U/L (normal 12–40U/L), alanine aminotransferase (ALT) 704 U/L (normal 11–41 U/L), alkaline phosphatase normal at 79 U/L (normal 40–115U/L) and total bilirubin 0.4 mg/dL (normal 0.2–1.1 mg/dL). The patient's initial lactate level at our facility was elevated at 9.5 mmol/L, which was increased from the documented lactate at the outside hospital and the arterial pH was measured at 7.07. A procalcitonin level was substantially elevated at 157.8 ng/mL. His hypotension persisted with a mean arterial pressure (MAP) 62, on norepinephrine and phenylephrine infusions. He was also found to have rhabdomyolysis with a creatinine kinase of >20,000 U/L (normal 52–336 U/L). The patient had a partial pressure of oxygen to fraction of inspired oxygen (PaO2/FiO2) ratio of 71 on 100% oxygen.

Upon admission, the patient was also begun on continuous renal replacement therapy for treatment his acute oliguric renal failure and metabolic acidosis. He was continued on broad-spectrum empiric antimicrobials that included vancomycin, cefepime, metronidazole, and micafungin for possible pneumonia or abdominal source. A urinalysis was unremarkable. The patient was noted to have altered mental status, for which the differential diagnosis included possible seizures considering the patient's PMH, potential anoxic brain injury due to the respiratory arrest. A serum valproic acid level was drawn to assess possible toxicity and returned at 4 μ/mL (normal therapeutic range 50–100 μ/mL). CT of the head was negative for any acute changes. Intravenous acyclovir was added to the patient's empiric antimicrobial regimen on day 2 for possible viral encephalitis. Due to the severity of the patient's illness and reported hobbies of fishing and outdoor stream and river exposure, doxycycline was empirically added for possible tick-borne disease. Both acyclovir and doxycycline were discontinued after 2 days of therapy and no change in clinical status. A two-dimensional transthoracic echocardiogram was completed with no significant findings. Blood and urine cultures were negative.

On day 1 of the patient's hospital course at our institution, the patient had a severe pneumonia with elevated WBC, purulent tracheal secretions, PaO2:FiO2 71, and chest radiograph indicating very low lung volumes with significant atelectasis. CT of the chest was performed on day 2 and revealed multifocal bilateral pneumonia. Subsequent bronchoscopy identified mucus plugs in the right middle lobe, right lower lobe, and left lower lobe and a bronchial alveolar lavage (BAL) was done on the right middle lobe. A potassium hydroxide preparation was performed on the BAL specimen and revealed no fungal elements; micafungin was discontinued after 4 days of therapy. A Gram stain was performed on the BAL specimen and revealed rare epithelial cells, rare white blood cells and no micro-organisms. An acid fast bacterium stains on the BAL fluid and subsequent AFB culture was negative. On day 5 the culture from the BAL predominantly revealed a non-lactose fermenting Gram negative rod as well as some *K. pneumoniae*. The antimicrobial regimen was narrowed to cefepime plus a single dose of tobramycin while awaiting susceptibilities of the Gram-negative pathogen. At this time, the chest radiograph indicated persistent but improving right upper lobe airspace disease with mild worsening patchy left lung airspace disease. The non-lactose fermenting Gram-negative bacillus was identified as a salmonella and reference laboratory speciation found it to be *Salmonella enterica* serovar Enteritidis (*S.* Enteritidis).

Empiric antimicrobials were de-escalated on day 6 based on the results of susceptibility testing (Table 1). In total, the patient received 6 days of parenteral cefepime followed by 10 days of oral levofloxacin. It should be noted that according to the final susceptibility results of both the *K. pneumoniae* and *S.* Enteritidis

Table 1
Antimicrobial Minimum Inhibitory Concentration results for *S.* Enteritidis and *K. Pneumoniae* recovered via Bronchial Alveolar Lavage Culture.

Pathogen	Antibiotic	Minimum Inhibitory Concentration (mcg/ml)[a]
90% *Salmonella* Enteritidis	Ampicillin	>16 Resistant
	Trimethoprim/Sulfamethoxazole	≤0.5/9.5 Susceptible
	Ceftriaxone	0.047 Susceptible (ETEST)
	Levofloxacin	≤1 No CLSI Range
10% *Klebsiella Pneumoniae*	Amikacin	≤8 Susceptible
	Ampicillin	≥16 Resistant
	Ampicillin/Sulbactam	8/4 Susceptible
	Aztreonam	≤2 Susceptible
	Cefazolin	≤2 Susceptible
	Cefepime	≤1 Susceptible
	Cefuroxime	≤4 Susceptible
	Gentamicin	≤2 Susceptible
	Levofloxacin	≤1 Susceptible
	Meropenem	≤1 Susceptible
	Piperacillin/Tazobactam	4/4 Susceptible
	Tetracycline	≤2 Susceptible
	Tobramycin	≤2 Susceptible
	Trimethoprim/Sulfamethoxazole	≤0.5/9.5 Susceptible
	Ertapenem	≤0.5 Susceptible

[a] Susceptibility testing and isolate identification performed by BD PhoenixTM Automated Microbiology System.

found on the BAL, both were susceptible to cefepime. The patient was treated with cefepime for a total of 6 days prior to the de-escalation to levofloxacin. By the final day of antimicrobials, the patient was afebrile, his WBC had decreased to 13.6k/uL and was tolerating ventilation through a tracheostomy prior to weaning from the ventilator. Follow up respiratory cultures did not grow any salmonellae. No stool cultures were initially obtained and later stool cultures as well as stool comprehensive PCR testing did not show evidence of salmonellae or any other pathogens. The patient was transferred on day 28 to a long-term acute care rehabilitation facility. At the time of discharge the patient was afebrile, stable on room air, tolerating a speech valve through his tracheostomy stating that he felt well.

Discussion

Non-typhoid salmonellae (NTS) are present in industrialized nations and are associated with contaminated food products [3]. NTS infects and colonizes a wide range of animal hosts and human disease generally manifests as self-limiting gastroenteritis. Salmonella are acid susceptible and must survive the gastric acid barrier and traverse a mucosal barrier before invading intestinal epithelial cells [1]. Previous gastric surgery and medications that reduce gastric acidity increase susceptibility to NTS infection [4]. Once the bacterium has evaded the gastric acid and mucosal barriers, it invades intestinal epithelial cells through a process called bacteria-mediated endocytosis [5]. The innate immune system is activated by multiple pro-inflammatory pathways including activation of toll-like receptor (TLR)-4 in the presence of lipopolysaccharide on the cell surface, TLR-5 by bacterial flagellin, and secretion of interleukin (IL)-β [6]. Resultant intestinal inflammation is characterized by fluid secretion and diarrhea. Salmonellae enter macrophages in the submucosal space and Peyer's patches where it is able to survive and replicate [6]. Components of the cellular immune system that play a role in clearing salmonella from the macrophages include interferon- γ, IL-12, IL-23, and tumor necrosis factor-α [7]. Patients with HIV, disorders of oxidative cellular killing, and sickle cell disease are at higher risk for invasive NTS disease [8–11].

Pulmonary infections associated with non-typhoid *Salmonella* pneumonia in immunocompetent hosts are rare in the medical literature. A MEDLINE/PubMED (1966 to October 2016) search limited to the English language and humans using the keywords Salmonella Enteritidis AND pneumonia AND/OR immunocompetent revealed four detailed reports of *S.* Enteritidis pneumonia and are summarized in Table 2.

Hall et al. describe a case of a healthy 24 year old male who developed severe sepsis, bacteremia and possible pneumonia from *S.* Enteritidis acquired from contaminated food [12]. Two cases summarized by Knight et al. describe *S.* Enteritidis pneumonia as a complication of Salmonellosis of the GI track with bacteremia [13]. Neither patient had traditional risk factors for disseminated Salmonellosis, except one had prior exposure to undercooked food. Both were newly diagnosed with diabetes during their hospital stay and were discharged alive with treatment on oral ciprofloxacin. Many reports describe extra-intestinal Salmonellosis in patients with known risk factors such as immunosuppression, living in a developing country, or known exposure to contaminated food [14–17]. One series described in a Taiwanese hospital found an association between extra-intestinal salmonellosis (including pneumonia) and older age, diabetes and chronic lung disease [18]. Samonis et al. describe a 72 year old male with a

Table 2
Previous reports *S.* Enteritidis pneumonia in medical literature.

Patient	Presentation	Immune status/ Risk factors	Microbiology and Antibiotic Susceptibilities	Treatment/Outcome
Our case	Septic shock, pneumonia source	Previously healthy. Developmental disability	*S.* Enteritidis. Susceptible to cephalosporins, fluoroquinolones, sulfamethoxazole/trimethoprim. Resistant to ampicillin,	Empiric treatment with cefepime, convalescent treatment with levofloxacin for a total of 16 days. Discharged after 28 days to acute care rehab with tracheostomy
24 YO male [12]	Back pain, fevers, rigors	None, possible food-borne transmission	*S.* Enteritidis. Susceptible to ciprofloxacin, ceftriaxone, azithromycin	Gluteal abscess and blood cultures also positive. Received 14d of ceftriaxone followed by 6 weeks oral ciprofloxacin
65 YO male [13]	Diarrhea, cramps, vomiting	Ingested undercooked eggs, cigarette smoker. Undiagnosed diabetes	*S.* Enteriditis. Susceptibilities not reported	Developed bacteremia, stool cultures positive. Empiric treatment with cefuroxime and erythromycin. Discharged alive on oral ciprofloxacin
59 YO female [13]	Fever, cough	Undiagnosed diabetes	*S.* Enteriditis. Susceptibilities not reported	Developed bacteremia. Treated with ciprofloxacin, discharged alive.
72 YO male [19]	10 months post diagnosis of small cell lung cancer; admitted with low grade fever, dyspnea, tachypnea, chest discomfort, and a productive cough with purulent sputum	Lung cancer	*S.* Enteritidis. Susceptible to ampicillin, ceftazidime, ceftriaxone, cefotaxime, ciprofloxacin, sulfamethoxazole/ trimethoprim	empiric treatment started with sulfamethoxazole/trimethoprim, ceftazidime, and clindamycin; patient deteriorated, transferred to ICU, developed ARDS, and expired after 5 days
26 YO male [20]	2 day history of fever, non-productive cough, back pain and shortness of breath	Hodgkins lymphoma, concurrent chemo-therapy smoker	*S.* Enteritidis. Susceptible to cotrimoxazole, ceftriaxone, meropenem, chloramphenicol, and ciprofloxacin. Resistant to nalidixic acid and ampicillin.	Empiric treatment with ceftriaxone for 14d, then discharged home with 14 additional days of oral cefixime

previous diagnosis of small cell lung cancer who succumbed to a pulmonary infection of *S.* Enteritidis after five days of treatment with sulfamethoxazole/trimethoprim, ceftazidime, and clindamycin [19]. The most recent report comes from the Kingdom of Bahrain [20]. The author describes a case of *S.* Enteritidis pneumonia complicated by encysted empyema in a 26 year old male with Hodgkins lymphoma actively receiving chemotherapy. He was discharged alive with a 14 day course of a third generation cephalosporin.

The species isolated in our patient's case was a common non-typhoid serovar belonging to group D1 Enteritidis. The final serotyping of this sample was confirmed by Kentucky state laboratory testing. Other cases have described salmonellosis with delayed identification, serotyping and susceptibility testing of the pathogen, which can lead to worsening outcomes if proper antimicrobial therapy is delayed [20,21]. Other documented serovars isolated from pulmonary sources include Typhimurium, Derby, Virchow, and Rostock, Dingiri and Abony [15,22–26].

Susceptibility testing of our patient's *S.* Enteritidis isolate revealed resistance to ampicillin and susceptibilities to sulfamethoxazole/trimethoprim and levofloxacin. Susceptibility to ceftriaxone was confirmed via the E-test. Emerging resistance to sulfamethoxazole/trimethoprim, fluoroquinolones, and some extended-spectrum cephalosporins has previously been documented [27]. Our patient was given a 10-day course of levofloxacin after de-escalating from an empiric regimen of cefepime, metronidazole, and vancomycin and was eventually discharged from the ICU. The few detailed reports available on salmonella pneumonia report difficulties in treatment and antimicrobial durations of at least two weeks [8,19–22,27–30].

The underlying source of the patient's *S.* Enteritidis pneumonia was ultimately never discovered. Salmonellosis can be caused by contaminated food or handling animals such as reptiles or amphibians or the environments in which they live. Mermin et al. reported a population-based, case-control study with data suggesting that each year ~74,000 *Salmonella* infections may be associated with reptile and amphibian exposure [31]. The results of this study concluded that patients with reptile and amphibian exposure attributed the highest risk of infection of numerous assessed risk factors commonly thought to cause salmonellosis in people age 21 or less. The significant number of salmonella infections thought to be caused by amphibian or reptile exposure in this study coincides with our belief that aquatic animal exposure could have been a possible source of infection for our patient infected with *S.* Enteritidis. Our patient's primary daily hobby was fishing, with likely stream and river water exposure. It is unclear if he had direct contact with aquatic animals such as a turtles, as we were not able to directly ask the patient at any time during his ICU stay.

Overall, this case is noteworthy due to the absence of traditional risk factors associated with severe extra-intestinal disease in the lung from salmonellosis, including pharmacologic immune suppression, auto-immune disease, known exposure to pathogen-borne food and living in a developing country [20]. The patient was only 33 years of age, an age not commonly associated with pneumonia, septic shock and multi-organ failure. Additionally, the patient presumably had a functional immune system prior to admission and was not otherwise overtly immunosuppressed. It is questionable whether or not the diagnosis of intellectual disability could have contributed to increasing his risk of contracting the disease. Our patient did have other risk factors such as taking an acid suppressing medication (pantoprazole), and lower socioeconomic class [20]. Most other cases of salmonellosis-associated pneumonia have presented in immunocompromised patients or patients with a primary pulmonary disorder such as lung cancer or lupus [8,10,20–22,27–30]. The presumed method of exposure in this case was from an aspiration event, either prior to admission or at the time of intubation at the outside hospital.

Conclusion

We report a case of severe *S.* Enteritidis pneumonia causing septic shock in a patient with a functional immune system who was not otherwise immunosuppressed. Clinicians should be aware of the potential for *S.* Enteritidis to be a pathogen in the lung, even in patients without traditional risk factors for *S.* Enteritidis infection.

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