

INVASIVE SALMONELLOSIS PRESENTING AS A LUNG ABSCESS: A CASE REPORT

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Abstract. *Salmonella* spp are an uncommon cause of lung abscess. A 59 year old man presented to our hospital with a 1 month history of cough and low grade fever progressing to high grade fever for 1 week. He had a past medical history significant for diabetes mellitus type 2 and focal segmental glomerulosclerosis for which he was receiving prednisolone, initially at 60 mg daily tapering to 20 mg daily. On presentation he was febrile and had decreased breath sounds and dullness to percussion over the right lower lung field. A chest X-ray showed a cavitory lesion with an air-fluid level in the right lung. Computed tomography of the lung revealed 2 cavitory lesions in the right upper and lower lungs. Sputum culture revealed *Salmonella* spp group B. He was treated successfully with ceftriaxone intravenously for 1 month followed by oral cefdinir. A chest X-ray at 1 month showed significant improvement; he was treated conservatively without surgical drainage. *Salmonella* can cause lung abscesses, especially in the immune suppressed.

Keywords: *Salmonella* group B, extraintestinal salmonellosis, lung abscesses, focal segmental glomerulosclerosis, salmonellosis

INTRODUCTION

Salmonella is an enteric gram-negative bacillus that normally causes invasive disease in immunocompromized patients with a defect in cell-mediated immunity and rarely causes pulmonary infections (Crump *et al*, 2015). *Salmonella* infection in patients with a normal immune status usually manifests as enteric fever or typhoid fever (Pegues and Miller, 2015). Non-

typhoid salmonellosis usually presents with self-remitting acute gastroenteritis in immunocompetent individuals (Ispahani and Slack, 2000). Immunocompromized adults may present with primary bacteremia without gastrointestinal symptoms, suggesting the infection arises from bacterial reactivation to a dormant site, such as the reticuloendothelial system, rather than secondary to a gastrointestinal infection (Pegues and Miller, 2015). A lung abscess is localized necrosis of lung tissue caused by a microbial infection, usually as a complication of aspiration pneumonia (Walters *et al*, 2011). Another possible route of *Salmonella* infection is hematogenous spread and should be suspected in cases with multiple abscesses in the lungs (Yazbeck *et al*, 2014). Common causative

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pathogens of lung abscesses include mixed anaerobic bacteria, which colonize oral cavities and aerobes, such as *Staphylococcus aureus* (Yazbeck *et al*, 2014). We report here a rare case of multiple lung abscesses caused by *Salmonella* group B, which resolved with antibiotic therapy only without surgical intervention.

CASE REPORT

A 59-year-old Thai male presented to Siriraj Hospital, Bangkok, Thailand with fever and cough for 1 month. Initially, he had low-graded fever and non-productive cough. He denied chest pain or weight loss. He initially did not seek medical treatment.

One week prior to presentation, his symptoms worsened. He developed productive cough with yellowish phlegm and inspiratory pain in right side of his chest. His fever worsened and he took paracetamol orally but the symptoms persisted. He went to a private clinic and was diagnosed with having a respiratory tract infection. He was prescribed with 1 dose of intramuscular medication and then 5 days of oral medication. There were no medical records from the clinic and the patient could not remember the names of the medication. His symptoms improved slightly with that medicine.

Three days prior to presentation, he developed high-grade fever and shortness of breath. His symptoms worsened daily until he was admitted to our hospital.

The patient had a history of focal segmental glomerulosclerosis (FSGS) diagnosed 6 years previously. His FSGS presented as bilateral leg edema and he underwent a kidney biopsy. He was treated with prednisolone, tacrolimus and cyclophosphamide. Two years prior to the presentation at our hospital he had

remission of the FSGS and discontinued his medication. However, 6 months prior to presentation to our hospital he again developed bilateral leg edema again and was diagnosed with a relapse of his FSGS. He was again treated with prednisolone, initially at 60 mg daily tapering to 20 mg daily and was also given furosemide. He also given trimethoprim-sulfamethoxazole for pneumocystis pneumonia prophylaxis. He also had a history of diabetes mellitus type 2 which was poorly controlled despite taking medications regularly, probably due to corticosteroid use for his FSGS. He also had a history of essential hypertension which was well controlled. His medications of admission to our hospital were: prednisolone 15 mg/day, trimethoprim-sulfamethoxazole (80/400) 1 tab daily, glipizide 10 mg/day, vildagliptin 50 mg/day, manidipine 10 mg/day and vitamin D2 20,000 iu/week.

He had worked as a farmer for 40 years in Nakhon Pathom Province, Thailand where he owned a swine farm. He had a 20 pack-year smoking history and drank alcohol occasionally until he stopped 20 years previously. He denied any intravenous drug use. He denied any herbal or over-the-counter drug use. He reported no family history of chronic lung disease or malignancies. He denied contact with active tuberculosis patients.

Physical examination revealed a temperature of 36.9°C, a pulse rate of 110/min, a blood pressure of 130/90 mmHg, a respiratory rate 24/min and an oxygen saturation of 98% on room air. Oral examination showed no oral thrush or oral hairy leukoplakia. He did not have any skin rashes. Chest examination revealed a mid-line trachea, normal chest expansion and dullness to percussion in the right lower lung. On lung auscultation there were decreased breath sounds and decreased

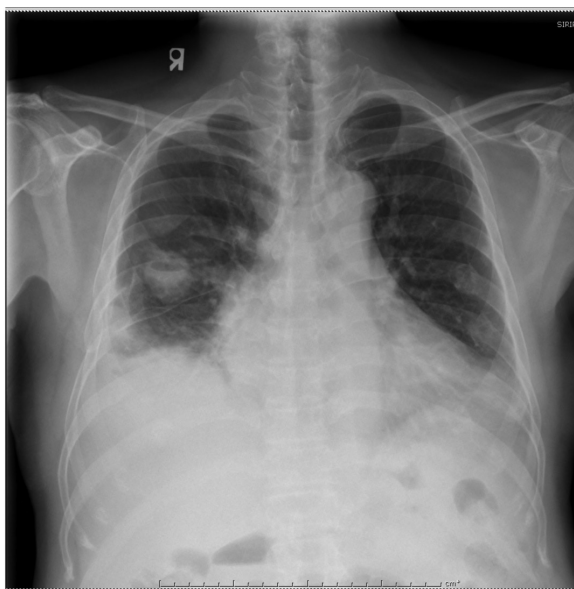


Fig 1—Chest X-ray posterior-anterior (PA) upright view showing a thick walled cavity with an air-fluid level in the right lung with blunting of the right costophrenic angle.

vocal resonance over the right lower lung area. He had no lymphadenopathy or hepatosplenomegaly. The neurological examination was unremarkable.

On laboratory testing he had a hemoglobin of 10.4 g/dl, a white blood cell count of 16,270/mm³ and a platelet count of 234,000/mm³. His creatinine level was 1.04 mg/dl, his aspartate aminotransferase (AST) level was 16 U/l, his alanine aminotransferase (ALT) level was 52 U/l, his albumin level was 2.5 g/dl and his globulin level was 5.6 g/dl. A human immunodeficiency virus (HIV) antibody assay was negative. His hepatitis B surface antigen and anti-hepatitis C virus antibody were negative.

An upright chest radiograph (Fig 1) showed a 2.2 x 2.3 cm thick-walled cavity with an air-fluid level and a blunt costophrenic angle in the right lung. A

computed tomography (CT) scan (Fig 2) showed two cavitory lesions, one in the anterior segment of the right upper lobe of the lungs and in the anterior basal segment of the right lower lobe of the lungs which showed an air-fluid level and rim enhancement; the 2 lesions measured 2.7x2.3 cm and 4.8x2.2 cm, respectively. A small right pleural effusion and multiple lymphadenopathy of the right hilar and right interlobar regions were also present. The findings were suggestive of hematogenously spread lung abscesses.

A sputum Gram stain revealed numerous polymorphonuclear (PMN) leukocytes and rare gram-negative bacilli. A sputum aerobic bacterial culture grew gram-negative bacilli on blood agar and MacConkey agar by 3 days. The biochemical and serological tests were compatible with *Salmonella* spp serogroup B. Drug susceptibility tests showed resistant to quinolones but susceptibility to third generation cephalosporins and to carbapenems.

The patient was empirically treated with intravenous imipenem and was later changed to intravenous ceftriaxone. His condition gradually improved. The total duration of intravenously antimicrobial therapy was 1 month after which he was changed to oral cefdinir 400 mg/day for eight more weeks. A chest X-ray performed after 1 month of treatment (Fig 3) showed a significant decrease in size of the abscesses.

DISCUSSION

The genus *Salmonella* contains two species, *Salmonella enterica* and *Salmonella bongori* (Eng et al, 2015). *S. enterica* subspecies *enterica* is responsible for salmonellosis in humans (Pegues and

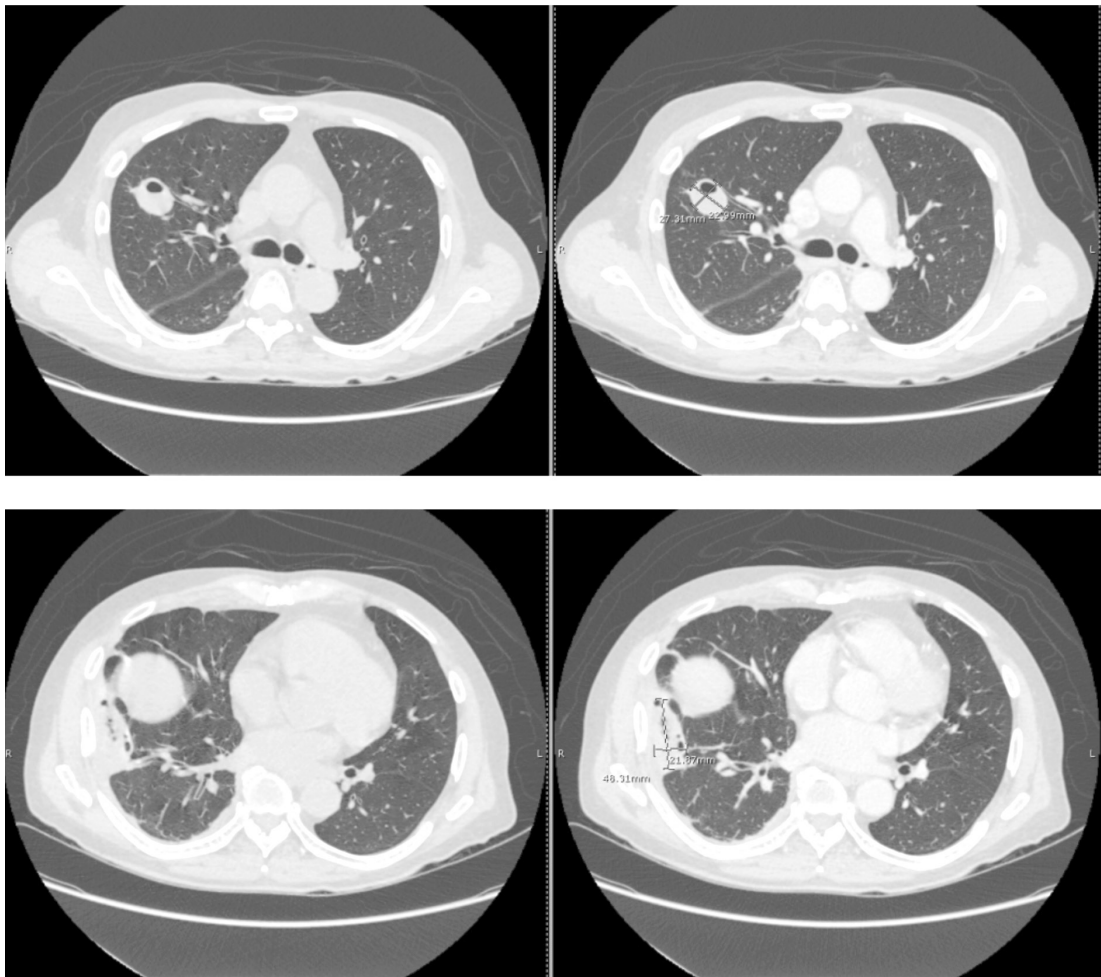


Fig 2—Axial contrast enhanced computed tomography (CT) scan, lung window view scan of the chest showing two cavitary lesions in the anterior segment of the right upper lobe and anterior basal segment of the right lower lobe of the lungs showing an air-fluid level and rim enhancement, measuring about 2.7 x 2.3 cm and 4.8 x 2.2 cm, respectively. Minimal pleural effusion in the right lung and multiple lymphadenopathies in the right hilum are also noted.

Miller, 2015). *Salmonella* infection spreads after ingestion of bacteria in contaminated food or water. Clinical manifestations of salmonellosis differ substantially by serotype and include acute gastroenteritis, bacteremia, enteric fever and an asymptomatic carrier state (Ispahani and Slack, 2000). Extraintestinal salmonellosis is an uncommon manifestation of non-typhoidal *Salmonella*; *Salmonella* bacteremia is

found in 10-15% (Pegues and Miller, 2015). *Salmonella* spp have a propensity to infect vascular sites (Crump *et al*, 2015). Other infected sites include soft tissue, bones, joints, the urinary tract and the central nervous system; but these are uncommon (Wilkins and Roberts, 1988; Ispahani and Slack, 2000).

Pulmonary involvement is uncommon, found in about 10% of all extrain-

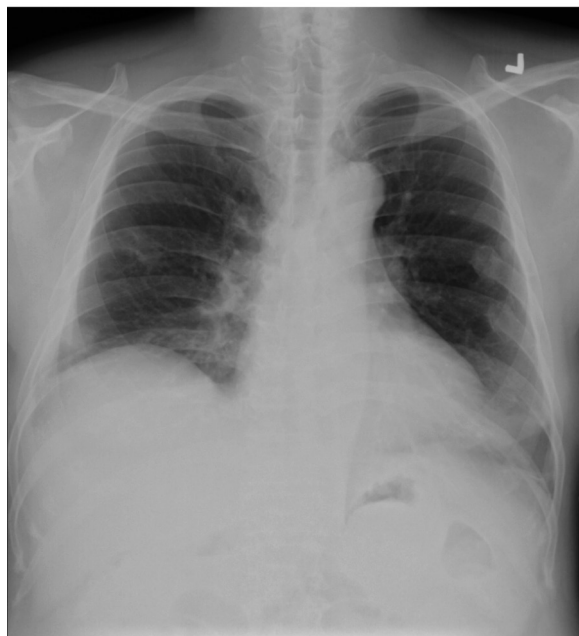


Fig 3—Chest X-ray posterior-anterior (PA) upright view performed 1 month after treatment onset showing significant improvement in the abscesses.

testinal *Salmonella* cases (Wilkins and Roberts, 1988). Patients with pulmonary involvement are likely to be immunocompromised by having human immunodeficiency virus (HIV) infection or by taking immunosuppressive agents such as corticosteroids (Kędzierska *et al*, 2008). Similar to other foci of extraintestinal salmonellosis, pulmonary infections occur due to hematogenous spread (Feasey *et al*, 2012). However, bacteremia may be transient. About 25-45% of patients presenting with pulmonary salmonellosis have negative blood cultures at presentation, similar to our patient (Cohen *et al*, 1987; Aguado *et al*, 1990). The most common *Salmonella* lung infection is pneumonia; abscesses are rare. To date, only 2 cases of *Salmonella* lung abscess occurring in adult non-HIV infected patient have been reported in the literature.

In the first case (Baird and Capper, 1949) was a 48-year-old with no history of co-morbid disease who presented to the hospital with a history of prolonged fever and cough for several weeks. A chest radiograph revealed a right upper lung abscess. Both blood and sputum cultures grew *S. Enteritidis*. He was treated with intravenous penicillin and sulfonamide. He later developed empyema thoracis and a brain abscess. Despite treatment, he died 4 weeks after being admitted to the hospital. The second case (Chan and Raffin, 1991) was a 45-year-old female diagnosed with having Wegener's granulomatosis who presented with fever and cough for 4 weeks. At that time, she was receiving prednisolone 60 mg/day and cyclophosphamide 2 mg/kg/day. Her chest radiograph showed a left upper lung abscess. Fluid from bronchoalveolar lavage grew *S. Cerro* while all her

blood cultures were negative. Treatment with ampicillin was successful and her condition resolved without complications.

The third case of *Salmonella* lung abscess in a non-HIV patient is the case we report here. Our case is similar to the second case discussed above in that he also had received an immunosuppressant and presented with prolonged fever and chronic cough. In our case, we did not perform the Widal test because it was unavailable and has poor diagnostic accuracy (Anduaem *et al*, 2014). The clinical characteristics, treatments and outcomes of the 3 cases discussed above are compared in Table 1.

The primary treatment for lung abscesses is a prolonged course of systemic antimicrobials and has a treatment success rate of 80-90% (Walters *et al*, 2011). Surgical drainage is an adjunct to antimicrobial

Table 1
Case reports of *Salmonella* lung abscesses among adult non-HIV infected patients.

No	Patient	Comorbidities and clinical presentation	Serotype	Treatment	Complications and outcome
1	Male, aged 48 years	No comorbid disease. Fever and right upper lung abscess.	<i>S. Enteritidis</i>	Penicillin plus sulfonamide	Brain abscess, deceased.
2	Female, aged 45 years	Wegener's granulomatosis and left upper lobe lung abscess.	<i>S. Cerro</i>	Ampicillin for 6 weeks	No complications. Resolved
3 (Current reported patient)	Male, aged 59 years	Focal segmental glomerulosclerosis. Multiple lung abscess.	<i>Salmonella</i> group B	Ceftriaxone then oral cefdinir	No complications. Resolved

treatment. Treatment of *Salmonella* lung abscess is the same as for any lung abscess (Yu, 2011). However, the duration of treatment is not well-defined. The minimum duration for antimicrobial treatment is 2 weeks but this should be extended until the abscess resolves (Pegues and Miller, 2015). We treated our patient for a total duration of 12 weeks, longer than previous cases, until the abscesses resolved completely.

In conclusion, we reported here a rare case of *Salmonella* lung abscesses in a male patient who has been taking corticosteroids for FSGS. He had no gastrointestinal symptoms and his blood cultures were negative. He was treated successfully with systemic antibiotics without surgery. No residual complications were observed.

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