Case Report of Disseminated Pseudomonas Infection with Superadded Burkholderia Infection - A Battle Lost!

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Abstract
Pseudomonas aeruginosa is a gram-negative pathogen, that often causes nosocomial pneumonia in hospitalized patients. Most of these patients have risk factors for pseudomonas infection. Although uncommon, there have been case reports of previously healthy individuals who developed community-acquired pneumonia (CAP) caused by P. aeruginosa. Such cases have often rapidly progressive course and prove fatal. We, hereby, report a case of pseudomonas pneumonia in a 29-year old immunocompetent patient, who developed disseminated infection and superinfection with yet another nosocomial pathogen, Burkholderia cepacia, eventually leading to septic shock and death, despite appropriate antibiotic therapy.

Keywords: Infection, bacteremia, pneumonia, antibiotics, healthy

Introduction
Community-acquired pneumonia (CAP) due to pseudomonas aeruginosa in a previously healthy individual is rare. Pseudomonas is a gram-negative, aerobic organism which generally infects patients with structural lung diseases like cystic fibrosis and chronic obstructive lung disease (COPD) and immunocompromised host [1]. We report a rare case of P. aeruginosa CAP in a young female without risk factors that progressed to necrotizing pneumonia, with formation of cavities. Further, there was dissemination of infection to other organs resulting in metastatic abscesses in liver and spleen, superinfection with Burkholderia cepacia, eventually leading to septic shock, multi-organ dysfunction syndrome (MODS) and death, despite being treated with appropriate antibiotics. Such a case of disseminated pseudomonas infection is hitherto not described in the literature.

Case report
A 22-year-old previously healthy lady was admitted to our hospital with history of high-grade fever for 7 days accompanied by cough, productive of scanty yellowish sputum for 5 days. She had also noticed swelling of both legs, three days prior to the admission. She had undergone caesarian section 3 months prior and remained asymptomatic till 1 week prior to hospital admission. She did not have past history of any significant medical ailment and addictions. On examination, she had pallor and bilateral pitting pedal edema. There was no cyanosis, clubbing or generalized lymphadenopathy. She was febrile, pulse rate was 124/minute, regular, blood pressure was 112/70 mm Hg and respiratory rate was 22/minute with accessories working. Breath sounds were vesicular type and were diminished at both bases. SaO₂ while breathing ambient air was 92%. Abdomen was fatty, non-tender and there was no organomegaly. Other systems were normal.

On admission, her hemoglobin was 6.4gm/dl, mean corpuscular volume (MCV) 77.8 fl, WBC count 2,300/µL (52% neutrophils with toxic granules, 36% lymphocytes, 12% monocytes) and platelet count 51,000/cu mm. Evaluation of the
cause of anemia revealed serum iron – 34.8 mcg/dl, serum ferritin – 564 mcg/L, serum folic acid – 5.6 ng/ml, serum B12 - 442 pg/ml, reticulocyte count of 0.8%, normal adult pattern hemoglobin electrophoresis and negative direct Coomb’s test (DCT). Renal function test was normal. Liver function test revealed serum bilirubin – 1.72 mg/dl, alanine transferase (ALT) - 27.9 U/L, aspartate transferase (AST) 40.8 U/L, alkaline phosphatase (ALP) - 284.4 U/L, total serum protein 6.1 g/dl, serum albumin - 2.07 g/dl, serum globulin – 4.03 g/dl. Her coagulation tests revealed prothrombin time–13.2, control-11 secs, international normalized ratio (INR) 1.54 and activated partial thromboplastin time – 26.9 sec, control-24.6. C- reactive protein (CRP) was 29.24 mg/dl. Her viral markers (HBsag, HIV 1 and ll and HCV antibodies) were non-reactive. Her antinuclear antibodies (ANA) and antibodies to double-stranded DNA (dsDNA) were negative. Mantoux skin test was negative. Urine routine examination showed 10 to 12 pus cells/ high power field and 274.6 mg/dl of protein. Her serial laboratory investigations were as tabled in the Table 1.

Table 1: Serial laboratory parameters from the day of admission

| Lab values          | Day 1 | Day 3 | Day 5 | Day 8 | Day 12 | Day 15 |
|---------------------|-------|-------|-------|-------|--------|--------|
| Hb (g/dl)           | 6.4   | 7.2   | 8.2   | 8.1   | 9.3    | 8.4    |
| TLC (/cu mm)        | 2,300 | 1,400 | 1,000 | 3,600 | 9,400  | 9,500  |
| (N 52%, L 36%, M 12%) |      |       |       |       |        |        |
| Platelets (/cu mm)  | 51,000| 52,000| 48,000| 54,000| 45,000 | 42,000 |
| Peripheral smear    | Toxic granules + | Toxic granules +, reactive lymphoid cells | Toxic granules + | Toxic granules + | Toxic granules + |
| Serum bilirubin (mg/dl) | 1.72 | 1.58 | 1.44 | 2.13 | 2.09  |
| ALT/AST             | 27.9/40.8 | 18.6/16.2 | 16.1/17.4 | 15.6/20.6 | 12.1/21.7 |
| ALP                 | 284.2 | 234.5 | 217.1 | 295.3 | 311.5  |
| Serum albumin (g/dl)| 1.91  | 1.96  | 2.08  | 2.14  | 2.25   |
| PT(INR)             | 1.56  | 1.4   | 1.34  | 1.5   | 1.4    |
| Creatinine (mg/dl)  | 0.85  | 0.82  | 0.93  | 0.98  | 1.1    |

Chest radiograph showed left mid zone pneumonia (Figure 1) while contrast enhanced computed tomography (CECT) of thorax showed multiple small nodules and cavities in both lungs with mild left pleural effusion (Figures 2a-2d). Echocardiography was normal. Abdominal ultrasound showed hepatosplenomegaly with imaging features suggestive of multiple abscesses in liver largest measuring 6 cm x 4 cm and largest splenic abscess measuring 4 x 3.5 cm with moderate ascites while contrast enhanced computed tomography (CECT) abdomen confirmed the above findings and additionally showed few small abscesses in the right kidney (Figures 3a-3d).

Figure 1a & b: Chest radiograph showing left mid zone pneumonia (on admission and 4 days later)
Her blood culture sent on the day of admission grew gram negative bacilli, pseudomonas aeruginosa (BACTEC method), which showed sensitivity to meropenem, imipenem, ciprofloxacin and piperacillin-tazobactum and resistance to amikacin, colistin, cefepime, cefaperazone-sulbactum and tigecycline. She was treated with intravenous piperacillin-tazobactum 4.5 g thrice a day and levofloxacin 500 mg once a day for 10 days with other supportive measures. However, she continued to get high grade fever spikes (104 to 105 F). A second blood culture sent on day 8 of admission grew Burkholderia cepacia sensitive to meropenem and ciprofloxacin. Her antibiotic therapy was escalated to intravenous meropenem 1 g tid and oral trimethoprim/sulfamethoxazole (80/400 mg). She, however, went into septic shock needing double vasopressor support. She later developed multiorgan dysfunction syndrome (MODS) and disseminated intravascular coagulation (DIC) and succumbed to her illness. A final diagnosis of community acquired pseudomonas pneumonia with dissemination with metastatic abscesses with superadded Burkholderia infection leading to MODS was made.

**Discussion**

This patient was diagnosed to have severe community-acquired pneumonia (CAP) due to P. aeruginosa, which was complicated by secondary hematogenous spread causing metastatic abscess in liver and spleen and MODS involving cardiovascular, hematologic and respiratory systems. Injury to the alveolar epithelium causes release of proinflammatory cytokines into the circulation which are ultimately responsible for septic shock. During treatment, she developed superadded secondary infection with Burkholderia cepacia. Pseudomonas aeruginosa is a gram-negative aerobic bacterium that causes several types of infections including wound,
Pseudomonas aeruginosa, can cause CAP. The disease, although described in immunocompromised patients and with underlying risk factors, can also occur in apparently healthy individuals. The initial symptoms may not be precise enough for a specific diagnosis. Though earlier studies have shown predilection for right upper zone, pseudomonas pneumonia can be bilateral and tend to become necrotizing with cavity formation. CT thorax can demonstrate coalescing of infectious foci into cavities and is recommended when the initial radiograph is not contributory. The infection can disseminate to other organs as in our case and ultimately lead to septic shock despite appropriate antibiotic therapy. Superadded infection with other pathogens can also occur. Physicians must, therefore, keep this possibility in mind while diagnosing CAP, especially if there is associated septic shock and treat the patient early with anti-pseudomonal antibiotics.

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Nil

**Conflict of interest**
Nil

**Consent**
Written consent was taken from the patient during the hospital stay for the publication of the case and pictures.
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