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STENOTROPHOMONAS MALTOPHILIA CAVITARY PNEUMONIA IN AN IMMUNOCOMPETENT PATIENT: A CASE REPORT

Respiratory Medicine

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ABSTRACT

Stenotrophomonas maltophilia (S. maltophilia) is a gram-negative pathogen, known to cause nosocomial infection in immunocompromised patients in intensive care units. Use of central venous catheters, history of prior hospitalization, chronic obstructive lung disease and use of broad-spectrum antibiotics especially carbapenems are known risk factors for development of ventilator associated pneumonia (VAP) due to S. maltophilia. We present an interesting case of S. maltophilia causing cavitary pneumonia in an immunocompetent patient, while on treatment with broad spectrum antibiotic therapy, with no prior history of hospitalization.

KEYWORDS

Cavitary pneumonia, Ventilator associated pneumonia, Biofilm

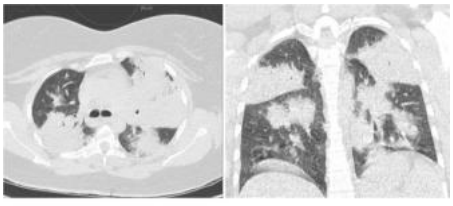
INTRODUCTION

Stenotrophomonas maltophilia (S. maltophilia) is an emerging gram-negative pathogen. It is frequently linked to infections of the respiratory tract in humans.[1] In immunocompromised patients, nosocomial infections are the primary mode of acquiring S. maltophilia. On rare occasions, patients with respiratory tract infection, urinary tract infection, and wound infection have been reported to have acquired S. maltophilia infection. Comorbidities such as chronic obstructive pulmonary disease, central venous catheter use, malignancy, trauma, prior hospitalization, human immunodeficiency virus infection, or other forms of immune suppression are frequently observed in patients infected with S. maltophilia.[2,3] To the best of our knowledge this is the first case of S. maltophilia ventilator associated pneumonia (VAP) causing cavitary pneumonia in an immunocompetent patient.

Case Description

A 31 year female with no prior comorbidities presented with chief complaints of fever with associated cough, throat pain, body pain and chest tightness for 7 days. She was a diagnosed case of Influenza A (throat swab positive) by another healthcare facility. She was on home treatment with oral Oseltamivir. She presented to our hospital with worsening of her symptoms with increasing episodes of fever and cough.

On initial evaluation, her Influenza A throat swab was negative. Her lab investigations showed high C-reactive protein (CRP) levels along with bilateral non-homogenous opacities on chest X-ray. A non-contrast computed tomography (NCCT) thorax showed bilateral multifocal air space nodular opacification with air bronchograms in bilateral upper lobes, right middle lobe and bilateral lower lobes along with a small left parapneumonic effusion (Figure 1a and 1b). She was managed as a case of community acquired pneumonia with acute respiratory distress syndrome (ARDS). After sending all appropriate cultures, she was continued on empirical antibiotics Intravenous (IV) Ceftriaxone 1 gm twice daily and oral Azithromycin 500 mg once daily. Since she had already completed a 5-day course of oral Oseltamivir, no further Oseltamivir was added. Her initial CURB-65 score was noted to be 2 and Pneumonia Severity Index (PSI) was noted to be 112 points. Her admission Acute Physiology and Chronic Health Evaluation (APACHE) II score was 11 points with predicted mortality of 15 %. Initial sputum culture was noted to be sterile but blood culture showed growth of Streptococcus pneumonia.



1a 1b

Figure 1: Initial non-contrast CT (NCCT) thorax suggestive of multifocal lobar consolidation secondary to Influenza A community acquired pneumonia with ARDS. 1a. Axial view 1b. Coronal view

Despite empirical antibiotic therapy, her general condition worsened further with worsening oxygenation and new onset shock. Need of increasing oxygen support with high-frequency nasal cannula (HFNC) and vasopressor support was also present. Concomitantly, there was worsening of infiltrates on chest radiography.

On evening of day 3 of intensive care unit (ICU) stay, she was intubated in view of worsening acute hypoxemic respiratory failure. She underwent 3 sessions of prone ventilation with each session lasting approximately 16 hours. A 2D-echocardiography done for raised serum N-terminal pro b-type natriuretic peptide (NT-proBNP) levels was found to be normal. She also received steroids with IV hydrocortisone 200 mg in daily divided doses for a total duration of 7 days. Euvolemic status was been maintained with strict fluid input and output charting. Lung protective ventilation was continued with regular monitoring of driving pressures. Her antibiotics were escalated to empirical IV Imipenem-cilastin 1gm thrice daily with continuation of her oral Azithromycin for a total duration of 7 days. Additionally, she was given coverage with IV Linezolid after her blood culture showed growth of Streptococcus pneumoniae. In view of sustained improvement in oxygenation status after 3rd session of proning, no further proning sessions were performed.

After this improvement, patient remained afebrile period for 4 days. However, on day 6, she developed new onset fever spikes. Concomitantly her WBC counts and CRP levels showed increasing trend (Table 1).

Table 1: White Blood Cell (WBC) counts and C - reactive protein (CRP) levels showing rising trend after day 6 of ICU stay

Day of ICU stay	WBC counts (10^3 /µL)	CRP (in mg/L)
Day 1	3.33	291.2
Day 3	7.96	262.8
Day 4	11.00	120.4
Day 5	9.71	46.6
Day 6	9.80	25.8
Day 7	12.27	51.9
Day 8	15.43	----
Day 9	15.79	163.1
Day 10	18.68	123.2
Day 11	14.80	118.2
Day 12	8.27	24.7

Repeat cultures (Endotracheal aspirate, urine and blood) were all sterile. Despite broad spectrum antibiotic coverage with IV Imipenem-cilastin and IV Linezolid, she continued to have persisting fever spikes. She was then started empirically on Levofloxacin 750 mg IV OD along with oral Trimethoprim–sulfamethoxazole (TMP-SMX) 160/800 mg twice daily on day 7, for ventilator associated pneumonia. Gradually her fever spikes reduced with down-trending of her inflammatory markers (Table 1). Remarkably, the endotracheal aspirate culture sent during the period of worsening showed growth of Stenotrophomonas maltophilia (sensitive to TMP-SMX and resistant to Levofloxacin). With improved oxygenation and afebrile status, she was extubated on day 9 of ICU stay.

Incidentally a routine chest radiograph done on day 10 showed a cavity of diameter 3.4 cm in left lower lobe (Figure 2). A follow up NCCT thorax done on day 14 confirmed the same with resolving areas of pneumonia and presence of thick-walled cavity lesions seen in lingula and lower lobe of left lung showing thickened septae suggestive of cavitating pneumonia (Figure 3a and 3b). She was continued on oral TMP-SMX for 10 days. Gradually her general condition improved further and she was sent to ward and later discharged home.

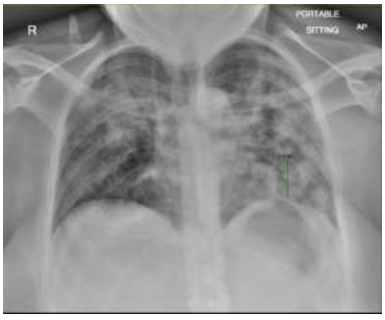
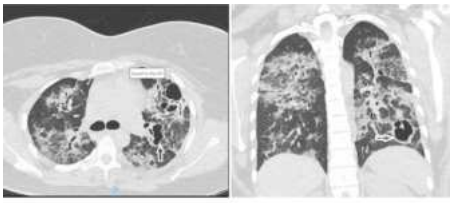


Figure 2: Chest radiograph showing a thick walled cavity of diameter 3.4 cm (green line) in left lower lobe



3a 3b

Figure 3: Follow-up non-contrast CT (NCCT) thorax suggestive of resolving areas of pneumonia and presence of thick-walled cavity lesions (white arrows) secondary to Stenotrophomonas maltophilia ventilator associated pneumonia. 3a. Axial View 3b. Coronal view

DISCUSSION

S.maltophilia is an aerobic, nonfermentative, gram-negative bacillus related to the Pseudomonas species.[4] It is frequently linked to infections of the respiratory tract in humans infecting immunocompromised patients with nosocomial infections. Pneumonia and bacteremia are the most common manifestations of infection. It has an inherent ability to adhere to foreign materials and form a biofilm. Infections primarily occur in individuals who have spent a prolonged period of time in critical care units, have undergone mechanical ventilation for more than 7 days, have a tracheostomy, or have been exposed to broad-spectrum antimicrobial drugs like carbapenems, extended-spectrum cephalosporins, and fluoroquinolones.[5-7] These organisms have a natural resistance to carbapenems, and being exposed to these drugs can lead to excessive growth and make it easier for a secondary infection to occur compared to other drugs.[8]

The uniqueness of our case lies in the occurrence of Stenotrophomonas cavitary pneumonia in a patient who was immunocompetent, with no prior history of hospitalization. Although a rapidly progressive and fatal hemorrhagic pneumonia associated with S.maltophilia has been reported in immunocompromised patients, our patient was immunocompetent.[9,10] While it is possible that our patient acquired cavitary pneumonia as a result of Streptococcus infection, it is extremely unlikely because our patient had sufficient antimicrobial coverage for Streptococcus. Additional aetiologies of cavitary pneumonia were also excluded, since tests for rheumatoid factor (RF), antinuclear antibodies (ANA), antiphospholipid antibodies (APLA), anti-cyclic citrullinated peptide (anti-ccp), anti-double stranded DNA (anti-ds DNA), and Covid-19 Reverse transcription polymerase chain reaction (RT-PCR) yielded negative results.

Another distinctive aspect of our case was the emergence of a S. maltophilia infection that was resistant to levofloxacin. With the growing problem of antimicrobial resistance, there have been multiple studies and case reports documenting the presence of Stenotrophomonas strains that are resistant to levofloxacin. Our patient improved because of administration of empirical TMP-SMX, in addition to Levofloxacin. The Infectious Disease Society of America (IDSA) guidelines recommend combination antimicrobial therapy being more effective than monotherapy for treatment of moderately severe or severe infections due to S. maltophilia.[11]

Retrospective investigations have found similar results when comparing the use of quinolone monotherapy to TMP-SMX, with no significant differences observed in terms of microbiological cure or 30-day mortality.[12] Cho et. al reported the emergence of quinolone resistance in patients who were originally treated with levofloxacin and later suffered recurrent bacteraemia. They found that 50% of the isolates had evolved resistance to quinolones.[13] Approximately 20% of patients with S. maltophilia pneumonia who fail to obtain complete elimination of the microorganism may experience the development of resistance to quinolone after being subjected to levofloxacin, compared to 7-8% of those exposed to TMP-SMX.[14-16]

One notable aspect of our case was that our patient experienced an early onset of ventilator-associated pneumonia (VAP) within a period of less than seven days, specifically caused by S. maltophilia. The primary understanding is that S. maltophilia can lead to early VAP, and intensive care specialists should consider the possibility of VAP caused by S. maltophilia, particularly when there is a persistent fever despite the administration of broad-spectrum antibiotics such as IV Imipenem-cilastin and IV Linezolid, as was the case in our situation.

An ideal situation would involve completely minimising the chances of infection by implementing antibiotic stewardship and careful administration of antibiotics, especially carbapenems. Multiple studies have consistently found that carbapenems are a significant risk factor for acquiring S. maltophilia.[5,6] Furthermore, it is crucial to prioritise fundamental source control concepts, such as promptly draining abscesses and strictly removing catheters and foreign body material, given of Stenotrophomonas' inclination towards biofilm development.[1]

CONCLUSION

Clinicians must be cognizant of the fact that infections caused by S. maltophilia may manifest in healthy, immunocompetent individuals presenting as early VAP with necrotizing cavitary pneumonia, as indicated by this unique case.

Clinical Significance:

S. maltophilia can cause cavitary pneumonia in an immunocompetent patient, while on treatment with broad spectrum antibiotic therapy, with no prior history of hospitalization. Combination antimicrobial therapy is recommended over monotherapy for treatment of moderately severe or severe infections due to S. maltophilia.

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