

## Case Report

# Haemorrhagic pneumonia caused by *Stenotrophomonas maltophilia* in two newborns

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### Abstract

Invasive procedures and antibiotic treatment increase the risk of nosocomial infections in neonatal intensive care units. Early identification and appropriate treatment is important. Herein we report two cases of massive hemorrhagic pneumonia caused by *Stenotrophomonas maltophilia*. The first case was diagnosed with congenital pneumonia; a chest tube was inserted because of pneumothorax on the third day of life. The second case had been referred with respiratory distress syndrome, and bilateral pneumothorax was present on admission. Upon follow up, the cases' clinical condition worsened; acute respiratory distress syndrome and massive pulmonary haemorrhage developed. After *Stenotrophomonas maltophilia* was isolated in blood cultures, the cases were treated successfully using a combination of trimethoprim/sulfamethoxazole and fluoroquinolone.

**Key words:** Haemorrhagic pneumonia; *stenotrophomonas maltophilia*; newborn.

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### Introduction

In recent decades, advances in neonatal intensive care have led to a significant decline in neonatal mortality. However, the use of invasive procedures, mechanical ventilation, and antibiotic treatment increase the risk of nosocomial infections in vulnerable neonates. The Gram-negative bacillus *Stenotrophomonas maltophilia* (SM) is an important agent of nosocomial infection in immunocompromised and long-term-hospitalized adults. Recently, nosocomial infection with SM has been reported in neonatal patients in the intensive care unit [1, 2]. SM infection is a critical concern as it can lead to serious respiratory morbidity and is resistant to most antibiotics. Therefore, early identification and appropriate treatment are important. We report two cases of significant haemorrhagic pneumonia caused by SM and its successful treatment using a combination of trimethoprim/sulfamethoxazole (TMP/SMX) and fluoroquinolone.

### Case 1

A male infant was born at full-term to a 30-year-old female by vaginal delivery in our hospital. The Apgar score was 10 at 5 min. He was admitted to the neonatal intensive care unit (NICU) and diagnosed with congenital pneumonia due to tachypnea,

retraction and fewer within the first few hours of life. Nasal continuous positive airway pressure was performed. Ampicillin and gentamycin treatments were initiated. On the third day of life, a chest tube was inserted because of pneumothorax, and breathing was assisted by mechanical ventilation. On day 6, the patient's condition worsened, with increased need for oxygen and suctioning. Clinical and laboratory assessments suggested nosocomial pneumonia. Vancomycin and meropenem treatments were initiated. Surfactant replacement therapy and high-frequency oscillatory mechanical ventilation was performed to treat the pneumonia. However, the pneumonia progressed to acute respiratory distress syndrome, and massive pulmonary haemorrhage developed. A red blood cell and fresh frozen plasma transfusion were administered. SM was detected in the blood culture. Susceptibility testing was performed against amikacin, amoxicillin/clavulanic acid, cefepim, ceftizidime, ciprofloxacin, gentamicin, netilmicin, imipenem, meropenem, and TMP/SMX. Based on the antibiotic sensitivity report the infant's treatment was changed to ciprofloxacin and TMP/SMX. At follow up, the patient's respiratory distress improved gradually. The patient received continued treatment for 14 days and was discharged on day 30.

## Case 2

A 2000 g, 33-week-old male infant was born to a 38-year-old female by vaginal delivery. Because of respiratory distress, the infant was intubated in the delivery room and transported to our NICU. Bilateral pneumothorax was evident by chest radiography upon admission. Chest tubes were inserted, and breathing was assisted by mechanical ventilation. An umbilical venous catheter was placed. After blood cultures were drawn, penicillin and amikacin therapies were introduced empirically. Echocardiographic examination revealed a mean pulmonary artery pressure of 45 mm Hg. After making the diagnosis of pulmonary hypertension, oral sildenafil was started.

On day 5, the patient's clinical course deteriorated progressively, and massive pulmonary haemorrhage was seen in the endotracheal tube. The chest X-ray revealed pulmonary opacities in both lungs. White cell and platelet counts were normal, but the CRP level was high. After taking blood cultures, the antibiotic treatment was changed to vancomycin and ciprofloxacin. Upon follow-up, the pneumonia progressed to acute respiratory distress syndrome, massive pulmonary haemorrhage ensued, and the haemoglobin level decreased. Despite administration of intravenous immunoglobulin, red blood cells, fresh frozen plasma and intensive cardiorespiratory support, pulmonary haemorrhage, severe hypoxemia, and acidosis continued and chest X-ray showed a worsening condition. On day 8, SM was isolated from blood cultures. Susceptibility testing was performed against amikacin, amoxicillin/clavulanic acid, cefoperazone/ sulbactam, gentamicin, imipenem, meropenem, levofloxacin, and TMP/SMX. Antimicrobial testing showed that the isolates were sensitive only to levofloxacin and TMP/SMX. The infant's treatment was changed to levofloxacin and TMP/SMX. After these treatments, the clinical course of the patient improved gradually. Microbiological response was achieved by day 14 of the treatment, and the patient was discharged in good health.

## Discussion

We presented two cases of newborns with massive haemorrhagic pneumonitis caused by SM infection that was successfully treated using a combination of TMP/SMX and fluoroquinolone. Limited numbers of neonatal cases with septicaemia, infections of the central nervous system, nosocomial pneumonia, and conjunctivitis due to SM have been reported. Cases of haemorrhagic pneumonia associated with SM have been reported only in patients with haematological

disorders. Mori *et al.* [3] reviewed all cases in the literature that reported patients with haemorrhagic pneumonia caused by SM. They emphasised that SM leads to fulminant and fatal haemorrhagic pneumonia. The mechanism underlying pulmonary haemorrhage is unknown, but a protease produced by SM has been considered to play a major role in pulmonary haemorrhage. The function of the protease might lead to the destruction of alveolar microvessels [4].

Mutlu *et al.* [1] reported that the most important risk factors for SM infection in neonates are invasive procedures, exposure to aminoglycoside and carbapenem, administration of total parenteral nutrition due to inadequate nutrition on enteral feedings to preterm infants, H2 blockers, and exposure to steroids, cholestasis, and prolonged hospitalization. Our cases were incubated and received chest tubes.

SM is highly resistant to various classes of antibiotics such as carbapenem and aminoglycoside, which are used empirically for nosocomial sepsis. TMP/SMX, ticarcillin-clavulanate, fluoroquinolone, colistin, and tigecycline are agents that show consistent therapeutic activity against SM. However, because resistant strains of SM have increased, combination regimens are recommended [5]. We have treated patients with a combination of TMP/SMX and fluoroquinolone with successful outcomes.

In conclusion, SM should be considered as a possible infectious agent in neonates with haemorrhagic pneumonia. Clinicians must be aware of cases with haemorrhagic pneumonia in the NICU, as appropriate antibiotic treatments to this multidrug-resistant agent can prevent disease progression and death in neonates with haemorrhagic pneumonia.

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