



Case Report

Volume 6 Issue 1 - July 2017
DOI: 10.19080/JGWH.2017.06.555677

J Gynecol Women's Health

Copyright © All rights are reserved by Ara Dadivanyan

Acute Intra Amnioti Infection Due to *Streptococcus pneumonia*



Ara Dadivanyan*, Mark G Martens and Antonio Santillan

Jersey Shore University Medical Center, USA

Submission: June 29, 2017; Published: July 17, 2017

*Corresponding author: Ara Dadivanyan, Jersey Shore University Medical Center, USA, Tel: 9176351098; Email: aradadiv@yahoo.com

Introduction

Streptococci are Gram-positive bacteria. Species of Streptococci are classified based on their hemolytic properties as alpha-, beta- or gamma-hemolytic. Beta-hemolytic streptococci are further classified as groups A, B, C, D. The beta-hemolytic streptococci groups A and B are a serious threat to the newborn, and are a frequent cause of neonatal meningitis and septicemia. In the medical settings the alpha-hemolytic streptococci including *S.pneumonia* can cause serious infection in adults.

Alpha-hemolytic streptococci are best known because of *Streptococcus pneumonia*- recognized as a major cause of pneumonia in the late 19th century. *Streptococcus pneumonia* remains the most frequent cause of pneumonia in adults, immune compromised patients, and children. *Streptococcus pneumonia* causes community acquired pneumonia, a severe disease with the mortality rate 12% or greater. Also, *Streptococcus pneumonia* is a leading cause of bacterial meningitis in adults and young adults in the USA with the mortality rate higher than from the disease caused by any other microorganism.

Streptococcus pneumonia rarely colonize the genital tract, but can cause maternal septicemia, and thus could also infect the fetus via hematogenous dissemination [1]. By its ability to colonize the lower female genital tract, the parturitional threat to the neonate is real and potentially serious. The purpose of this report is to describe a case of intra-amniotic infection and perinatal septicemia due to *Streptococcus pneumonia*, and present a diagnostic and therapeutic plan if suspected in the future.

Case Report

A 25 year old female Gravida 3 para 2, at 36-weeks gestation with uncomplicated prenatal care, presented to labor and delivery with a history of 18 hours of spontaneous rupture of membranes (clear amniotic fluid), and complete cervical dilatation. One gram of cefoxitin IV was given for the prolonged ruptured membranes followed by a precipitous vaginal delivery of a male infant. The infant Apgars were 9 at one minute, 9 at five

minutes, and 8 at ten minutes. There was no history of maternal fever, or colonization with group B streptococcus. Placental cultures from maternal and fetal sites were performed. Placental Gram stain from maternal site demonstrated a few Gram-positive cocci in pairs, with no organisms seen in the fetal site. Microscopic examination of the placenta revealed acute signs of infection consistent with chorioamnionitis. The mother had an uncomplicated postpartum course, and was discharged a febrile on postpartum day one. Placental culture subsequently returned as positive for *Streptococcus pneumonia*.

At day 1, the infant was transferred from a regular nursery to the neonatal care unit, secondary to tachypnea. White blood cell count was 7.1K/uL with 18% polymorphonuclear cells, and 46% band forms. Hematocrit was 45%. Examination of the infant demonstrated chest retractions requiring oxygen on admission. Blood cultures were drawn. Ampicillin and gentamicin were started. Supplemental oxygen was administered and a chest X-ray showed diffuse infiltrates bilateral, suggesting diffuse areas of pneumonitis. The infant had transient tachypnea, but demonstrated adequate improvement, and oxygen was discontinued the following morning. Blood cultures from the neonate were negative, presumably due to the antepartum intravenous antibiotics, but placental cultures both in maternal and fetal sides indicated growth of *Streptococcus pneumonia*, which was sensitive to penicillin. Considering the symptomatology and the abnormal blood count, the infant was treated for seven days with ampicillin for suspected *Streptococcus pneumonia* sepsis. Gentamicin was discontinued after four days. The infant improved rapidly with the antibiotic treatment, and was discharged on the fourth day in a good condition.

Discussion

Streptococcus pneumonia is a Gram-positive alpha-hemolytic streptococcus best known by its ability to cause community acquired pneumonia and bacterial meningitis. It also, though not frequently, colonizes the lower female genital tract posing a parturitional threat to the neonate.

Maternal carriage of *Streptococcus pneumoniae* has been reported to occur in 0.83% of all pregnancies [2]. Though rare, neonatal pneumococcal septicemia can be a highly lethal disease of the newborn. The incidence of the neonatal disease due to *Streptococcus pneumoniae* is higher than the maternal carriage rate, and has been found to cause significant disease in 1 to 2% of infants with early neonatal sepsis [2,3]. The mortality of this condition is as high as 50% with 13% incidence of neurological sequel in the survivors [4].

Urogenital colonization of pregnant women with pneumococci appears rare and is not considered as a part of the normal vaginal flora. Pneumococcal genital infection was more common in the pre-antibiotic era, with a high lethality rate of 26% for localized infection, and 74% for peritonitis [1]. Today, mortality appears to be improving over the 25-year period from 1965 to 1990, as all 24 patients reported worldwide survived their pneumococcal genital infection [4]. We suspect that improvement in universal screening for hemolytic streptococci has resulted in early detection of *Streptococcus pneumoniae* and thus the initiation of early and efficient treatment.

S. pneumoniae may reach the fetus or newborn and potentially lead to neonatal infection via four possible mechanisms: transplacentally secondary to maternal bacteremia; ascending infection from the maternal genital tract; passage through a colonized birth canal; or postpartum by respiratory spread.

This highlights the importance to collaborate with microbiology laboratories to be alert to identify and report *S. pneumoniae* when routinely screening for B-hemolytic streptococci. The clinical course of pneumococcal neonatal sepsis is similar to those seen with early onset group B streptococcal sepsis. It has been suggested that administration of penicillin prophylaxis during labor may prevent vertical transmission of pneumococcus to the newborn in patients with positive vaginal isolates [5], perhaps accounting for the drastic but welcome reduction in the neonatal incidence and mortality with *S. pneumoniae* recently.

Because of the high morbidity and mortality of pneumococcal septicemia, it has been proposed that maternal vaginal cultures

positive for pneumococcus should be regarded as pathogenic [1,2]. However, because reporting of pneumococcus from vaginal cultures in pregnant women is inconsistent, it is unknown how many infants are exposed to pneumococci. Research investigating neonatal host defenses to this specific bacteria are insufficient, and many become necessary if the incidence or severity increases [1,2,6].

Although perinatal infections associated with *Streptococcus pneumoniae* are rare, they may cause significant morbidity and mortality in both neonatal and puerperal patients. Given the reported poor outcomes of neonatal septicemia, maternal vaginal or infant cultures positive for pneumococcus should not be ignored, and these infants should be followed carefully. Based on the literature review, we also suggest that maternal carriage and neonatal colonization be monitored carefully to assess current trends. It should be aggressively treated with empirical antibiotic therapy and initiated during labor as is done with Group B beta-hemolytic streptococci [3].

Although most of the *S. pneumoniae* infected infants are successfully treated by the Group B streptococci protocols in place, it is important to understand the burden of each individual streptococcal species as resistance pattern may change in the future, as it has with the Group D streptococci.

References

1. Kaplan M, Rude sky B, Beck A (1993) Perinatal Infections with *Streptococcus pneumoniae*. *Am J Perinatol* 10: 1-4.
2. Singh J, deck J, Santosham M (2000) Colonization of the Female Urogenital Tract with *Streptococcus pneumoniae* and Implications for neonatal diseases. *Pediatr infect dis J* 19(3): 260-262.
3. Baltimore RS, Huie SM, Meek JI, Schuchat A, O'Brien KL (2001) Early-Onset Neonatal Sepsis in the Era of Group B Streptococcal Prevention. *Pediatrics* 108(5): 1094-1098.
4. Primhak RA, Tanner MS, Spencer RC (1993) Pneumococcal Infection in the Newborn. *Arch Dis child* 69(3): 317-318.
5. Joseph A Prats G, Cooper TR (2000) The Critically Ill Neonate with Infection: Management Considerations in the Term and Preterm Infant. *Seminars in Pediatric Infection Diseases* 11(1): 4-12.
6. Hugh HN, Arthur Hertig T, Mass B (1938) Pneumococcal Infection of the Genital Tract in Women. *American Journal of Obstetrics and Gynecology* 35(5): 782-793.



This work is licensed under Creative Commons Attribution 4.0 License
DOI: [10.19080/JGWH.2017.06.555677](https://doi.org/10.19080/JGWH.2017.06.555677)

Your next submission with Juniper Publishers will reach you the below assets

- Quality Editorial service
- Swift Peer Review
- Reprints availability
- E-prints Service
- Manuscript Podcast for convenient understanding
- Global attainment for your research
- Manuscript accessibility in different formats (Pdf, E-pub, Full Text, Audio)
- Unceasing customer service

Track the below URL for one-step submission
<https://juniperpublishers.com/online-submission.php>