Neonatal Serotype 8 Pneumococcal Invasive Disease; A Happy End in a Potentially Fatal Disease

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Summary

This paper aims to describe the case of an early-onset neonatal sepsis due to serotype 8 Streptococcus pneumoniae. This is a rare situation, generally associated with high mortality and morbidity outcomes, albeit with a good denouement in our case. We alert for the need for a high suspicion of pneumococcal etiology in a septic newborn with no risk factors for group B Streptococcus infection except vaginal delivery. Serotype 8 is not included in PCV13 vaccine, but it is of increasing incidence in Pediatric population. We discuss the need of immunizate women during the third trimester of pregnancy in order to protect child until 2 months immunization.

Abstract

Neonatal sepsis by Streptococcus pneumoniae (SP) is very rare but associated with high morbidity and mortality outcomes. The authors describe the case of a newborn male who developed eight hours after birth a septic syndrome with meningitis. A SP of serotype 8 was isolated in CSF and blood with no registered antibiotic resistance. He had a good clinical evolution and mother was always asymptomatic.

In early-onset SP sepsis cases, vertical transmission is the most probable mechanism of infection. Only vaginal delivery was considered to be a significant risk factor. Prophylactic strategies to neonatal GBS sepsis could contribute to the increase of SP infections and to higher resistance rates. It is discussed if it would be beneficial to immunize women during the third trimester of pregnancy in order to protect child until two months pneumococcal immunization.

Introduction

Neonatal sepsis by Streptococcus pneumoniae (SP) is rare (1-5,5% in USA and Europe until 11% in developing countries) [1], but associated with high morbidity and mortality outcomes.

There are few publications about this theme, mostly case reports [2-6] and small case series [1,7-10].

Authors describe a case of early-onset neonatal sepsis and meningitis due to serotype 8 SP and make a brief review of the literature about this theme, focusing on portuguese reality.

Clinical Case

Newborn male, first child of healthy young parents, born at term pregnancy, with poor medical supervision and no data about maternal rectovaginal colonization by group B Streptococcus (GBS). Rupture of membranes occurred five hours before eutopic delivery. Apgar score was 9 at 1st and 10 at 5th minutes and birth somatometry was appropriate for gestational age. Eight hours later he began groaning and refusal to eat. Physical examination revealed axial hypotonia, poor peripheral perfusion and polypnea. Laboratory found white cells count 3500/mm³ [3], with 60% neutrophils. C-reactive protein (CRP) was 11mg/dL increasing to 42mg/dL and 109mg/dL at 23 and 36 hours of life respectively.

Lumbar puncture issued clear and normotensive cerebrospinal fluid (CSF), with no cytochemical changes (leukocytes 15cell/mm³, CSF glucose 61mg/dL, blood glucose 78mg/dL, proteins 60mg/dL). He started intravenous ampicillin and gentamicin.

SP of serotype 8 was isolated in cultural examination of CSF and blood, and antibiotic therapy was changed to vancomycin and cefotaxime, with clinical improvement.

Antibiotic sensivity profile showed susceptibility to penicillin (minimum inhibitory concentration – MIC ≤ 0,6 μg/mL), cefotaxime (MIC ≤ 0,6 μg/mL) and vancomycin (MIC ≤ 1 μg/mL) as well as to all other tested antibiotics. Vancomycin was stopped and cefotaxime maintained for 21 days.

 Imaging studies confirmed existence of thymus and spleen. Transfontanellar ultrasound revealed no abnormalities. He had a good clinical evolution, without complications during hospitalization.

Mother was always asymptomatic and with no laboratory signs of infection. Maternal blood and vaginal search for SP were not performed.

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Discussion

*Streptococcus pneumoniae* (SP) is an uncommon ethiology of invasive infections in neonates. Newborn vertical transmission can occur by transplacental transmission of mother’s bacteremia or passage in birth canal, the most probable mechanism of transmission in early sepsis. Horizontal transmission occurs mainly from near contacts (parents, siblings attending nurseries), being more frequent in late-onset sepsis (after first week of life) [1,6,10,11].

A couple of reports substantiates the transmission during the birth canal passage, isolating the same pneumococci serotype in newborn blood specimens and mother vaginal exsudate [2,5,6,9]. Colonization of maternal genital tract by *SP* is rare (<0.03%) and transitory [3,5,9,12]. It seldom causes invasive disease in pregnant or puerperal women, suggesting a high invasion to colonization ratio. Clinically, *SP* neonatal sepsis is similar to GBS sepsis but with a significantly higher mortality (reaching 50%) and morbidity, mainly in early onset cases (first 48 hours) [1,2,4-6,8].

Low gestational age, low birth weight and prolonged rupture of membranes don’t seem to be risk factors for early-onset neonatal invasive pneumococcal disease (IPD) [1]. Most IPD cases occur in full-term babies with a good birth weight [4,8]. Only vaginal delivery was considered to be a significant risk factor for early *SP* infection [1].

In septic newborns, from mothers with negative GBS screening or with a full intrapartum prophilactic scheme, of full term and with no risk factors for GBS infection except vaginal delivery, *SP* infection shall be kept in mind [4].

Prophylactic strategies directed to neonatal GBS sepsis could contribute to the increase of infections caused by *SP* as well as to resistance rates to commonly used antibiotics directed against GBS infections [4].

According to new Clinical and Laboratory Standards Institute breakpoints [13], pneumococcal resistance to penicillin is less relevant in non-meningitis groups than it was previously thought, but it is higher in meningitis groups due to the lower capacity of crossing blood-brain barrier.

Most reported cases concern to implementation period of 7-valent antipneumococcal conjugate vaccine (PCV7) and are due to serotypes included in 13-valent antipneumococcal conjugate vaccine (PCV13), namely: 7F [6,7,11, 19] A [14], 3 [9,11], 5 [10], 1 [10,11,14] and 14 [14]. Serotype 8 is seldom related in neonatal sepsis [2,11,12]. It is rare in pediatric population and is not included in PCV13, but it is in pneumococcal polysaccharide vaccine (PPSV23).

In Portugal, PCV7 was introduced in 2001 and PCV10 became available in the mid 2009. PCV 13 is commercialized since the beginning of 2010, being recomended to children ≥2 months. PCV13 inclusion in national immunization program only occurred since the beginning of 2015 [14]. S. Aguiar et al [14] reported a decreased incidence of IPD in Portuguese children under 18 years in about one half of cases from 2008-2009 to 2011-2012. That fail resulted from significant decreases in additional serotypes included in PCV10 and PCV13 (as serotypes 3, 6A and 19A in children aged 0-11 months). Incidence of IPD due to PCV7 and non-vaccinal serotypes was maintained. In 2014 serotypes included in PCV13 remained the main responsible for children IPD in Portugal.

Portuguese pneumococcal resistance to penicillin was of about 0.8% in non-meningitis groups and 16/39 in CSF isolates considering the current CLSI breakpoints for parenteral penicillin [14]. Cefotaxime resistance remained very low and there was no resistance to vancomycin, levofloxacin an linezolid [9]. Portuguese guidelines for neonatal meningitis include empiric administration of ampicilin and cefotaxime, adding vancomycin if suspicion of pneumococcal etiologi.

Some authors [6] propose that while herd immunity is not achieved, it would be beneficial to immunize women during the third trimester of pregnancy in order to protect child until 2 months immunization. Theoretically, conjugate vaccines could induce greater IgG maternal response than pollysacaride ones. It is difficult to ensure a cost-benefit that favours this measure. Fothy et al [6] advocate this strategy applicability should only be profitable in risk-groups (asplenia, IPD in previous pregnancies).

In our case, being a non-PCV 13 serotype, we wonder if maternal immunization with PPSV23 could help to protect the newborn. Serotype replacement is a consequence of PCV introduction. Some authors show the actual serotype composition is not optimal and could be improved if new serotypes, with higher invasiveness, were included [15].

In Portugal, there were only two reported cases of neonatal IPD during 2014, of serotypes 14 (included in PCV13) and 8. IPD in the group of children <60 days has specific features important to be known in order to better adequate perinatal care.

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References


