Cervical Lymphadenitis as a Manifestation of Group B Streptococcus Infection in a 1-Month-Old Infant

Linfadenite Cervical como Manifestação de Infeção por Streptococcus do Grupo B em Lactente de 1 Mês

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Abstract

Group B streptococcus (GBS) remains an important cause of neonatal sepsis and meningitis. Although these are the most common clinical manifestations, GBS can cause a wide variety of focal infections. Isolated lymphadenitis is a rare form of presentation and may be the only sign of bacteremia. We describe a case of a two-month-old male with recurrent GBS infection and lymphadenitis.

Resumo

O *streptococcus* do grupo B (SGB) continua a ser uma causa importante de sépsis e meningite neonatal. Embora estas sejam as apresentações mais frequentes, este agente pode causar vários tipos de infeção localizada. A linfadenite isolada é uma forma rara de apresentação e pode ser o único sinal de bacteriemia. Descreve-se um caso clínico de um lactente de 2 meses com linfadenite isolada causada por SGB.

Keywords: Infant; Lymphadenitis; Neck; Streptococcal Infections; Streptococcus agalactiae

Palavras-chave: Infecções Estreptocócicas; Lactente; Linfadenite; Pescoço; Streptococcus agalactiae

Introduction

Group B streptococcus (GBS), also known as *Streptococcus agalactiae*, remains the most important cause of invasive early-onset infection of the newborn infant, its incidence being estimated at 0.27-1.4 per 1000 live births.[1-5] GBS is mainly responsible for early-onset sepsis (EOS), and less frequently for late-onset infection.[2]

EOS is typically related to perinatal vertical transmission, via GBS-contaminated amniotic fluid or vaginal secretions, whereas the origin of late-onset infection remains unclear. It has been theorized that transmission could occur both vertically and horizontally from the mother, but nosocomial sources have also been reported.[3] The routine use of intra partum antibiotic prophylaxis (IAP) has substantially reduced the incidence of

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early-onset GBS disease but has had no impact on the incidence of late onset disease. [4]

Late-onset group B streptococcal infection occurs between 7 and 90 days of life and affects otherwise healthy children.[1-4] The most common clinical manifestations are sepsis and meningitis. Nevertheless, GBS can cause a wide variety of focal infections such as conjunctivitis, osteomyelitis, empyema, ethmoiditis, and cellulitis-adenitis. Isolated lymphadenitis is a rare form of presentation and may be the only sign of bacteremia. Non-focal disease can also occur, ranging from asymptomatic bacteremia to fulminant septic shock.[5,6]

We describe a case of a two-month-old male with recurrent GBS infection and lymphadenitis.

Case Report

A previously healthy 50-day-old male infant was admitted for fever and irritability. The mother was a healthy 23-year-old primipara and he was born at 41 weeks of gestation, via vaginal route, after an uneventful pregnancy, rupture of amniotic membranes 12 hours before birth, weighing 3780 g at birth. Family history was irrelevant. Antenatal vaginal swabs and urine samples were positive for GBS and the mother was treated with ampicillin and gentamicin during labor. The infant was discharged from the hospital after 72 hours.

On admission, besides irritability and a rectal temperature of 38.4°C, the infant also presented with a left sided cervical swelling. There were no other remarkable physical findings including other signs of local infection, such as cellulitis, parotitis, or otitis media. Cervical ultrasonography revealed multiple enlarged lymph nodes, the largest one measuring 17 mm diameter in size, with no signs of abscess formation or cellulitis.

Laboratory evaluation revealed a white blood cell count (WBC) of 3.9 x 103/mm3 with 2.0 x 103/mm3 neutrophils and C reactive protein (CRP) serum concentration was 3.64 mg/L. A blood culture was obtained and empirical antibiotic therapy with intravenous ampicillin 150 mg/kg per day, IV, every 8 hours and gentamicin 5 mg/kg per day, IV, every 24 hours, was started. The blood culture was positive for *streptococcus agalactiae* (GBS), sensitive to penicilin, ampicilin, amoxicilin, erythromycin, clindamycin and azithromycin. The infant was discharged asymptomatic after 10 days of treatment with ampicillin and 3 days of gentamicin.

The fever reappeared 10 days after discharge as well as a right submandibular mass. The area surrounding the mass was painful when palpated, but no other signs of local infection were present. Otherwise, there were no other physical findings. Since the infant was clinically well and there were no signs of meningitis, lumbar puncture was not performed. Laboratory evaluation revealed WBC of 4.2 x 103/mm3, with 1.9 x 103/mm3 neutrophils; CRP was 4.0 mg/L, Procalcitonin serum concentration (PCT) was 1.70 ng/mL. Urinalysis was normal. He was admitted for surveillance and 12 hours later laboratory evaluation was repeated: WBC of 15.9 x 103/mm3, neutrophils 10 x 103/mm3 (63%) and lymphocytes 4.5 x 103/mm3 (28%), CRP was 140 mg/L and PCT 39.8 ng/mL. Thoracic X-ray was normal. Two blood cultures and urine cultures were obtained and empirical antibiotic therapy with ceftriaxone 75 mg/kg per day, IV, every 24 hours, was started. A buccal swab near the parotid duct was also performed. Ultrasonography revealed multiple enlarged cervical right lymph nodes without any abscess formation, signs of cellulitis or parotitis. Urine culture yielded no bacterial growth and blood culture once again showed the growth of GBS with the same antibiogram testing as before. Treatment with parenteral penicillin G was started, 50 000 units/kg per dose, IV, every 8 hours, and within 24 hours of antibiotic therapy the infant was afebrile, and the adenitis had a rapid resolution. Buccal swab was also positive for GBS. The infant was discharged after 5 days of ceftriaxone and 10 days of penicillin G.

As for the mother, a sample of breast milk was analyzed even though there were no signs of mastitis, but cultures yielded no bacterial growth. No further studies were performed.

Discussion

Invasive Group-B streptococcal disease is a leading cause of infant mortality and morbidity worldwide. GBS colonises the maternal rectum and vagina, and transmission of bacteria from a colonized mother to her infant at birth is an important risk factor for GBS disease.[7]

It is also important to note that IAP greatly diminishes neonatal exposure to GBS during labor and delivery, therefore reducing the incidence of EOD by GBS. It does not, however, have any effect in altering the colonization status, having little impact in posterior horizontal transmission.[4] In this particular case, with appropriate IAP, horizontal transmission in a later period from a still colonized mother has to be considered.

Another hypothesis is that the infant could be colonized since early infancy, through a low inoculum of bacteria during labour, even though antenatal antibiotic therapy was administered. This is important data, since it has been hypothesized in other reports that after colonization, bloodstream invasion of a pathogenic bacterial strain could occur and lead to seeding in the neck area.[5]

GBS disease has also been associated with case reports of transmission via infected breast milk.[6] This has raised questions

about the mode of acquisition and transmission of this enteric pathogen and the development of neonatal disease. However, most breastfed infants remain unaffected by GBS in breast milk. Mechanisms associated with transmission of GBS in breast milk and potential factors that may protect the infant from transmission remain poorly understood.[6,7] In the presented case, there were no signs of mastitis, and culture of the mother's breast milk yielded no bacterial growth, making it an unlikely source of infection.

The most common presentation of late-onset GBS infection is bacteraemia with or without focal infection, such as meningitis, pneumonia, cellulitis-adenitis, osteomyelitis, or arthritis.[5]

Meningitis accounts for 25%-30% of cases of late-onset GBS disease and its clinical presentation is typically indistinguishable from sepsis in small infants, being irritability, temperature instability and lethargy the predominant symptoms. Many of the late-onset lymphadenitis cases coursed with subsequent meningitis. For this reason some authors have suggested that a lumbar puncture should be performed in young infants with cellulitis/adenitis and the empiric antibiotics should include coverage for SGB meningitis until CNS infection is excluded especially towards a persistent/recurrent SGB late infection.[8] In this particular case, since the infant was well appearing and there was a lack of high fever at presentation, as well as a good clinical response to the antibiotics, the hypothesis of meningitis was ruled out, and lumbar puncture was not performed.

Cellulitis-adenitis have accounted for less than 5% of SGB LOD and most commonly involve the face or submandibular area and SGB bacteremia is present in almost all of this cases. Albeit rare, isolated lymphadenitis as well as cellulitis may be a useful indicator of underlying bacteraemia, and therefore can serve as a valuable clinical clue.[8,9]

Conclusion

Invasive Group-B streptococcal disease is still a leading cause of infant mortality and morbidity worldwide. Albeit a rare manifestation of SGB infection, isolated lymphadenitis may be the only sign of bacteremia.

Routine use of IAP has substantially reduced the incidence of early-onset GBS disease but has had no impact on the incidence of late onset disease. It has been hypothesized that early colonization can lead to future late-onset infections.

Physicians need to be aware of unusual forms of late neonatal GBS infection, such as the one presented, in order to more accurately diagnose and treat what could rapidly become a serious disseminated infection, such as sepsis.

We believe further studies should be conducted in order to better understand the pathophysiological mechanisms behind this.

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