**Streptococcus agalactiae Acute Neonatal Parotitis: a rare form of late-onset neonatal infection**

Catarina de Abreu Amaro¹, Constança Soares Santos¹, Ricardo Jorge Costa¹

### Abstract

Parotid masses in pediatrics are rare clinical situations nowadays, being acute parotitis one of its differential diagnosis, rare in neonates. This pathology has a clinical diagnosis, the most common pathogen being *Staphylococcus aureus*. Female newborn, 24-day-old, admitted to the hospital with fever and irritability, who developed inflammatory signs of the right parotid region. Without pathognomonic sign, an acute parotitis was confirmed by ultrasound and the *Streptococcus agalactiae* was isolated in blood culture. She was treated with ampicillin during 14 days, with no complications or recurrences. Though rare in the neonatal period, an acute parotitis should be suspected in newborns with parotid region swelling. Towards this diagnosis, an antibiotic that covers not only the main pathogenic agent, but also the *Streptococcus agalactiae*, should be administered, minimising the risk of complications and recurrences.

### Keywords:
- Parotitis
- Infant
- Newborn
- Diseases
- Neonatal Sepsis
- Infant, Newborn
- Streptococcus agalactiae
INTRODUCTION

Parotid swelling is an uncommon finding in the pediatric population that has been linked to a number of conditions\(^1\) including acute parotitis, a disease rarely described in newborns.\(^2,5\)

The signs and symptoms of acute neonatal parotitis include edema, erythema, pain in the area of the affected parotid gland,\(^1,3,4,6,7\) and systemic findings such as irritability, refusal to feed, and fever.\(^5,6,8,9\) The most common bacterial agent involved,\(^1-3,5,6,8\) *Staphylococcus aureus* is found in approximately 55% of the patients with acute parotitis.\(^3\)

*Streptococcus agalactiae* is the most common agent in cases of early neonatal infection (occurred between birth and day 6 after birth), most of which manifest in the form of sepsis.\(^10-12\) Late-onset neonatal infection by *S. agalactiae* - disease occurring from day 7 to day 90 after birth - is less frequent and manifests primarily as bacteremia without an apparent focus.\(^1,11\)

CASE REPORT

A female newborn aged 24 days was admitted to the emergency unit with irritability and fever progressing for 12 hours. She was born after an uneventful pregnancy. Her mother’s vaginal and rectal screening tests for *Streptococcus agalactiae* were negative. The baby was born via cesarean section at 39 weeks of gestation as her mother met the criteria for labor arrest after ten hours of prelabor rupture of membranes. The fetus was in meconium-stained amniotic fluid and required orotracheal intubation to clear the airways. Resuscitation maneuvers were not needed. The newborn’s 1- and 5-minute Apgar scores were 5 and 10, respectively. She was born weighing 2600g. No additional adverse events were recorded in immediate neonatal life. The newborn was on partial breastfeeding, showing good weight gain, and had been immunized as per the requirements of the National Immunization Program.

The patient weighed 3390g on admission. Other findings included axillary temperature: 37.1°C; heart rate: 181 bpm; blood pressure: 91/58 mmHg; and SpO2: 100%. She was generally well, although she was crying vigorously and showing signs of irritability. She had rosy and well-hydrated skin and mucosa, cutis marmorata, and a capillary refill time of less than two seconds. Muscle tone was adequate; the anterior fontanelle was pulsating and pressure was normal; and her physical examination had no signs of alteration.

Her test results were as follows: white blood cell count: 6800/μL; neutrophils: 4800/μL; lymphocytes: 900/μL; Hb: 13.7 g/dL; PCR: 3.51 mg/dL; procalcitonin: 12.38 ng/mL. Urinalysis was normal and urine culture came back negative. A blood culture was ordered. The patient was hospitalized and started on intravenous ampicillin and cefotaxime.

A preauricular edema was observed close to the angle of the right jaw along with signs of inflammation 12 hours after admission. The edema measured about 5 cm in the larger diameter and covered the jaw angle (Figure 1). The parotid duct did not show clear signs of obstruction. No signs of effusion were seen. Physical examination did not reveal signs of oral cavity inflammation or additional alterations.

Ultrasound examination showed a heterogeneous right parotid gland with increased vascularity probably tied to inflammation consistent with early-stage parotitis and reactive lymph nodes. No signs of expansive process or abscess collections were seen (Figure 2). New blood tests were ordered and revealed the following signs of infection progression: white blood cell count 19,400/μL; neutrophils 9800/μL; lymphocytes 6600/μL; PCR 11.11 mg/dL; procalcitonin 20.28 ng/mL. Therapy with ampicillin was discontinued. The patient was prescribed vancomycin and kept on cefotaxime. She was also given intravenous paracetamol for pain management.

Workup parameters and clinical signs improved 24 hours after the start of antibiotic therapy. Blood culture was positive for a multisensitive strain of *Streptococcus agalactiae*, and the prescription was changed to monotherapy with intravenous ampicillin.

![Figure 1. Newborn with parotid edema and erythema in the right jaw angle.](image)
Acute parotitis starts mostly by retrograde spread from the oral cavity into the parotid gland through the parotid duct in a process facilitated by salivary stasis. In the case described herein, a blood culture positive for *Streptococcus agalactiae* and the absence of effusion through the parotid duct supported the choice of hematogenous dissemination as the primary pathogenic mechanism.  

*Streptococcus agalactiae* has been rarely correlated with acute neonatal parotitis. In our case and in the few published in the literature, parotid infection occurred in contexts of late-onset sepsis.  

As in previously reported cases, diagnosis was formed based on the presence of signs of inflammation in the area close to the parotid gland and findings such as irritability, fever, and systemic signs and symptoms. However, the patient did not present the telltale sign of the condition (purulent effusion through the parotid duct), which called for confirmation via ultrasound examination.  

Acute neonatal parotitis is eminently diagnosed based on clinical findings. Workup is unspecific and may show white blood cell counts greater than 15,000/µl with a predominance of neutrophils. Serum amylase is not useful in diagnosis. Increased levels are seen in approximately half of the cases, probably as a result of the immaturity of the isoenzyme in the saliva of newborns. Meningitis has been described in association with acute neonatal parotitis. In these cases, lumbar puncture followed by cerebrospinal fluid culture is the recommended course of action. Since our patient was in good general condition and showed no sign of central nervous system infection, we chose not to perform a lumbar puncture on her.  

Blood culture was extremely relevant in determining the etiology of the disease and adjusting antibiotic therapy based on the sensitivity of the isolated bacterial agent. In the presence of pathognomonic signs, exudate culture plays a key role in the identification of the etiological agent.  

Imaging tests and ultrasound examination in particular are valuable diagnostic tools, particularly in patients without the characteristic signs of the disease. Ultrasound examination allows the identification of enlargement, hypoechogeticity, heterogeneity, and hypervascularity of the parotid gland, as seen in this case report. Ultrasound examination may also be useful in the differential diagnosis against other conditions including cervical and preauricular lymphadenopathy, cellulitis, soft part abscesses, osteomyelitis and non-infectious diseases such as tumors, hemangiomas, and facial trauma, to name a few. Predisposing factors for parotid infection such as anatomic anomalies of the parotid duct, salivary duct obstruction, and parotid tumors, may be equally detected via ultrasound examination.  

The treatment of acute neonatal parotitis consists of intravenous antibiotic therapy for seven to 21 days, fluid replacement, and pain management. Initial empirical antibiotic...
therapy must include an antistaphylococcal drug combined with an aminoglycoside or a third-generation cephalosporin.\textsuperscript{1,5,8,9,12,13} In the case described, suspicions resided initially around community-acquired sepsis and the patient was prescribed adequate empirical therapy. When parotitis was considered, the patient was switched to an antistaphylococcal drug. Nonetheless, flucloxacillin would have been a more adequate choice, with vancomycin left for cases suspected for infection by methicillin-resistant strains of \textit{Staphylococcus aureus}.\textsuperscript{1,6,8}

Surgery is required\textsuperscript{1} only in the unlikely appearance of an organizing abscess.\textsuperscript{2,13}

The prognosis for acute neonatal parotitis is encouraging,\textsuperscript{4,11} although complications such as parotid abscesses,\textsuperscript{1,2} facial palsy,\textsuperscript{1,2} salivary gland fistulae,\textsuperscript{1,2} mediastinitis, and infection of the ipsilateral ear\textsuperscript{1} may occur. Prompt administration of antibiotics significantly decreases the chance of complications.\textsuperscript{1} In most cases, proper treatment results in clinical improvement within the first 24-48 hours and gradual parotid edema resolution,\textsuperscript{1,2} as seen in our patient. Poor clinical response to treatment prompts ultrasound examination to identify a possible case of organizing abscess.\textsuperscript{1,6} Our patient progressed well, but additional ultrasound examination was performed at the end of treatment.

Unlike early neonatal infections, the incidence of late-onset neonatal infection by \textit{Streptococcus agalactiae} is not decreased with the introduction of prophylactic care to mothers during pregnancy. The mode of transmission of this bacterial agent in late-onset neonatal infection has not been completely elucidated.\textsuperscript{15} Transmission is considered to occur via infected family members,\textsuperscript{11} breast milk,\textsuperscript{14} and vertical mother-to-child transmission after early bowel colonization followed by infection.\textsuperscript{15}

This report described a case of acute bacterial parotitis associated with sepsis in a rare form of late-onset neonatal infection by \textit{Streptococcus agalactiae}.

Additional studies are required to clarify the mode of transmission of \textit{Streptococcus agalactiae} in late-onset neonatal infection and establish the basis for the implementation of effective prevention strategies.

\section*{REFERENCES}