

Neonatal bacterial suppurative parotitis: a rare cause of bacterial infection in preterm infants

Abstract

Neonatal suppurative parotitis is a rare entity in the neonatal period, whose etiology is associated with prematurity, dehydration and feeding with an orogastric tube and is mainly caused by *Staphylococcus aureus*.

Objective: To present the case of a 31-week-old preterm infant, fed with an orogastric tube, who at 15 days of age presented bilateral suppurative parotitis, isolating *Staphylococcus aureus*, methicillin-resistant, treated with vancomycin and surgical drainage, with excellent clinical evolution and without complications.

Conclusion: Although this entity is rare and has a good prognosis, we propose to monitor neonates with orogastric tube feeding, mainly preterm, in addition to early stimulation of breastfeeding, performing stimulation of the sucking swallowing mechanism in premature infants and neonates with alteration of this, to reduce feeding times through orogastric tube.

Keywords: neonate, premature infant, suppurative parotitis, orogastric tube, *Staphylococcus aureus*

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What is known

Neonatal suppurative parotitis is a rare event, more frequent in premature infants fed with an orogastric tube, and caused mainly by *Staphylococcus aureus*. Diagnosis is based on clinical and physical examination supported by parotid ultrasound.

What is new

This is the first and only autochthonous case of bilateral neonatal suppurative parotitis in a 31-week preterm infant presented by our department after a 2-week hospitalization and which was directly associated with the orogastric feeding tube. Her evolution was favorable, with no immediate or late complications.

Introduction

Acute suppurative parotitis is a rare diagnosis in pediatric services and neonatal units that has been reported since the 1970s. Several case reports have already been published in the pediatric literature, being an event that can appear in the first week of life,¹ about the second week of life,² and others in the third and fourth weeks of life.³⁻⁵ It has been associated with prematurity and dehydration and is frequently caused by *Staphylococcus aureus*, although other bacterial microorganisms can cause it.^{2,5,7,8} Over the years, this event has been increasingly reported.⁹ The diagnosis is clinical, supported by ultrasound of the parotid¹⁰ and antibiotic treatment should include therapies against Gram-positive cocci and Gram-negative bacilli, with recurrence being rare.¹⁰ In this report, we intend to report the case of a 31-week-old preterm infant fed with an orogastric tube who, at 15 days of age, presented bilateral suppurative parotitis. Isolated from *Staphylococcus aureus*, resistant to methicillin, treated with vancomycin and drainage of the purulent collection, with excellent clinical evolution and no complications.

Case report

Background

A male newborn at 31 weeks of gestational age, who was born weighing 1,495 g, height 42 cm and head circumference of 30 cm. First child of a 25-year-old father and a 23-year-old mother, healthy, with a controlled pregnancy, whose only history was antenatal diagnosis of intrauterine growth restriction and a history of urinary tract infection with hospital antibiotic treatment. She was admitted to preterm labor without rupture of ovular membranes, chorioamnionitis or other infectious risk factors.

The pregnant woman had rectal and vaginal screening for *negative Streptococcus agalactiae*. The patient was born vaginally, with an Apgar score of 1-5-10 minutes of 8-9-9 respectively, without requiring resuscitation and without alterations to the physical examination. Due to the risk of early sepsis, she received ampicillin and amikacin for 48 hours until a negative blood culture was obtained. She also received parenteral nutrition for 8 days, enteral feeding was started at 24 hours of age, reaching 150 cc/kg/day at 9 days of age, always administered by orogastric tube. At 72 hours of age, due to the presence of apneas, caffeine citrate is administered at an initial dose of 10 mg/kg and then 5 mg/kg per day intravenously. At the age of 7 days, speech therapy was started by speech therapy to stimulate the coordination of the sucking-swallowing mechanism, which was still immature.

Clinical findings

At 15 days of age, she presented bilateral parotid induration, erythema, heat and edema. Purulent material drainage through Stenon ducts was assessed and found bilaterally, associated with irritability, hypoactivity, tachycardia with heart rates ranging from 168 to 185 beats/minute (not associated with fever), and a single episode of

thermal elevation of 38.4°C. Blood tests, cultures, and studies of cerebrospinal fluid and 2 blood cultures are performed. Ultrasound identified an increase in bilateral parotid volume, with hypoechoic areas and regional adenitis.

Laboratories

Blood count: Leukocytes 34,100/mm³, absolute neutrophils 23,393/mm³, lymphocytes 6,684/mm³, hemoglobin 9.92 g/dL, platelets 416,000/mm³; c-reactive protein: 122.37 mg/L (normal range 0-5 mg/L); cerebrospinal fluid cytochemical: Clear, colorless, pH 7, glucose 67.1 mg/dL, total proteins 68.27 mg/dL, LDH 32.2 U/L, white blood cell count 2/mm³, neutrophils 2/mm³; Gram cerebrospinal fluid: Negative for bacteria; serum amylase: <3.0 U/L (normal range: 3-50 U/L); Gram purulent secretion: Gram-positive cocci in moderate amount; blood cultures 1 and 2 (120 hours of incubation) and cerebrospinal fluid culture (72 hours of incubation): Negative for bacterial growth; culture of purulent secretion: *Staphylococcus aureus*. Antibiogram and minimum inhibitory concentration (MIC): Resistant oxacillin (MIC >2), positive cefoxitin screening (MIC > 4), resistant to methicillin; sensitive to clindamycin (MIC ≤0.5), sensitive to erythromycin (MIC ≤0.5), sensitive to trimethoprim sulfa (MIC ≤0.5/9.5), sensitive vancomycin (MIC 1).

Antibiotic treatment

Vancomycin 10 mg/kg/dose every 8 hours was started intravenously, and amikacin was started at a dose of 15 mg/kg/day intravenously. At 19 days of age, left parotid drainage was performed, extracting abundant purulent material, from which samples were taken for Gram and culture. At 20 days of age, right parotid drainage is performed, and abundant purulent material is also extracted.

The neonate evolved satisfactorily to antibiotic treatment for 10 days and parotid drainage, with evident reduction of inflammatory signs and disappearance of secretions through Stenon's ducts.

Discussion

This event has been the only one that we have diagnosed in our neonatal unit after 20 years, and more than 10,000 newborns attended in our history. The reported case meets the characteristics of the diagnosis, such as pain, heat, edema, and erythema in the parotid area, purulent exudate through the Stenon duct and the recovery of *Staphylococcus aureus* in the parotid secretion.

This type of infection is rare in the neonatal period. Despite the low incidence of bacterial suppurative parotitis, parotids are a site susceptible to infection due to their type of serous secretion that lacks bacteriostatic properties in their mucoid component. Sabatino et al.,¹² describe an incidence of 3.8/10,000 neonatal admissions, with a risk of developing suppurative parotitis in neonates admitted to a neonatal unit of 5.52 (range: 0.62-49.35). Makhoul et al.,¹³ have an incidence of 2.5/1000 hospitalizations in this age group. Spiegel et al.,¹ report 13 publications with 32 cases of purulent parotitis, of which 38% were premature, 71.9% were male, 21.9% required surgical drainage, and 56.3% were caused by *Staphylococcus aureus*.

Several factors have been involved in the occurrence of purulent parotitis in neonates. Stasis of secretions is described as a serous, non-mucous and non-bacteriostatic secretion, aggravated by dehydration that worsens this stasis. Other factors described are primary sepsis with bacterial seeding in the parotids and congenital malformations.^{14,15} It is more frequent in premature infants due to reasons such as prolonged hospitalization with increased bacterial colonization, trauma of the oral cavity associated with the orogastric feeding tube of

prolonged use (2-6 weeks), and dehydration associated with feeding problems,¹⁶⁻¹⁸ as occurs in the case of exclusive breastfeeding with insufficient breastfeeding.¹⁹ Contaminated breast milk or formulas can also be linked.²⁰ Neonatal involvement, especially in premature infants, may be related to the immaturity of salivary isoenzyme activity. Isoamylases are found with very low activity in the serum of fetuses at 14 weeks of gestation, increasing with age, reaching 80 U/L at 5 years of age.²¹ Other factors linked to suppurative parotitis include breastfeeding in women with breast abscess²² and cytomegalovirus infection.²³ Hematogenous dissemination is the least frequent, but cases have been reported in early sepsis with pneumonia, multiple congenital malformations, multiple skin abscesses, and late-onset sepsis.¹⁰

The clinical history and physical evaluation are essential to making the diagnosis, finding local inflammatory signs, purulent secretion through the Stenon duct when compressing the gland (pathognomonic), and growth of a pathogenic germ in this material.^{9,12,24} The submandibular salivary gland is rarely affected, whose secretion is richer in IgA and lysozymes.²⁵ Laboratory findings are nonspecific, except for hyperamylasemia, which is very rare in neonates due to enzyme immaturity.⁹

Our patient manifested local symptoms at the bilateral parotid level, but also showed systemic manifestations such as irritability, hypoactivity, tachycardia and thermal elevation. The literature describes clinical manifestations such as apneas, bradycardia, tachycardia, increased need for oxygen, lethargy, thermal instability, tachypnea, decreased peripheral perfusion, food intolerance, poor sucking, irritability, poor clinical appearance, fever that can exceed 39°C, while on physical examination it is found increased volume, swelling, redness, heat, pain, purulent drainage through the Stenon duct, as occurred in our case.^{6,26,27}

The patient in this report presented significant leukocytosis associated with neutrophilia and increased C-reactive protein. The literature describes that between 69% and 71% of neonates have an elevation of leukocytes >15,000/mm³ and 44% when the cut-off point is >20,000/mm³.^{9,10} Our patient's serum amylase was normal, however, this laboratory was increased in 25% of reported cases.^{8,23,28-30}

Our case was corroborated in the parotid ultrasound. Parotid ultrasound may show an enlarged gland, edema, hypoechoic areas, and increased vascularization. Reactive lymph nodes can sometimes be detected.^{1,2,16,25,29,30-34} However, in order to make an accurate diagnosis of the degree of parotid gland involvement and the associated extension through bone structures or facial nerve involvement, some authors have indicated the study of images such as computed tomography.^{3,26} When diffusion-weighted magnetic resonance imaging is performed, it may show multiple regions of punctuate hyperintensity with a reduced apparent diffusion coefficient, suggesting the formation of microabscesses in the affected gland.⁸

No other imaging studies were performed in our patient because the evolution was favorable with no suspicion of complications.

As in our case, the etiological diagnosis is made by obtaining purulent material from the orifice of the duct of Stenon, or by needle puncture of the gland directly to avoid contamination of the oral cavity with saprophytic germs.^{27,31}

The microorganism isolated in our patient was *Staphylococcus aureus*. The different reports record that *Staphylococcus aureus* is the most frequently isolated microorganism (56-61%), followed by other Gram-positive cocci (25%), Gram-negative bacilli (16%) and

anaerobic bacteria (11%). Among the other microorganisms associated with purulent parotitis are described *Streptococcus mitis*, *Klebsiella pneumoniae*, *Streptococcus viridans*, *Bacteroids*, *Prevotella sp.*, *Peptostreptococcus*, *Streptococcus pyogenes*, *Escherichia coli*, *Pseudomona*, *Moraxella catarrhalis* and *coagulase Staphylococcus negative*. Other reported isolates include *Prevotella*, *Bacteroides melaninogenicus*, and *intermedia*, *Fusobacterium nucleatum*. Although our blood culture was negative for bacterial growth, the literature reports that cultures can isolate some microorganisms in 25%-35% of cases.^{1,5,8,10,16,31,35-37}

Our antibiotic treatment included vancomycin and amikacin seeking to cover *Staphylococcus aureus* and other Gram-negative bacilli for 10 days. The literature suggests administering antibiotics based on the epidemiology and behavior of antimicrobial susceptibility patterns to cover oral germs such as *Staphylococcus aureus*, *Streptococcus*, and anaerobic. In antibiotic therapy, antibiotics should be selected for penicillin-resistant microorganisms, first-generation cephalosporins, vancomycin for methicillin-resistant *Staphylococcus aureus*, clindamycin to cover anaerobic bacteria, if *Pseudomonas aeruginosa* is suspected, a third-generation cephalosporin such as ceftazidime, or an aminoglycoside such as gentamicin and amikacin. The duration of treatment is not agreed upon, but for the treatment of *Staphylococcus aureus*, it can range from 7-14 days. Some recommend completing 10 days.^{8,24,25,30} In the case of suppurative parotitis in preterm infants, when there is involvement of several organs or when the isolation is an anaerobic germ, treatment should be extended up to 21 days.^{7,30,31}

Differential diagnoses should be based on ultrasound findings such as cellulitis of the face, preauricular adenitis, angioma,³¹ trauma, lymphadenitis, adenoma, lipoma, abnormalities of the parotid gland duct, intra glandular abscesses,¹⁶ myositis, hemangiomas and lipomas.³⁸

Our patient evolved favorably, being discharged alive and in good clinical conditions. The literature describes that the prognosis is excellent with current antibiotic therapy, with reduction of the inflammatory process in 24-48 hours and complete recovery with antibiotics in 80% of cases, reducing the need for surgical drainage to less than 25%.^{12,15,31,38,39}

Despite the fact that the literature mentions cases of poor clinical prognosis,¹⁴ Complications are rare, and include intra parotid abscess (most common), facial paralysis, salivary fistula, mediastinitis, sepsis, and meningitis. No cases of mortality have been reported.^{10,38}

Conclusion

Neonatal suppurative parotitis is a rare event in neonatal medicine, more common in preterm neonates, male, who have been hospitalized for several days, fed by orogastric tube. The diagnosis is clinical, and cultures of purulent material obtained from the drainage point of the Stenon duct or by needle puncture of the parotid allow the etiological diagnosis to be made and to better target antimicrobial therapy. Antibiotic therapy should be started early and cover Gram-positive and Gram-negative isolates, and even anaerobic germs, with high effectiveness. Complications are rare.

Ethical responsibilities

Protection of people and animals

The authors declare that the procedures followed conformed to the ethical standards of the committee for responsible human experimentation and in accordance with the World Medical Association and the Declaration of Helsinki.

Data confidentiality

The authors state that they have followed their workplace's protocols on the publication of patient data.

Right to privacy and informed consent

The authors have obtained informed consent from the parents (guardians) of the patient and/or subjects referred to in the article. This document is in the possession of the corresponding author.

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None

Conflicts of interest

The authors declare that they have no conflicts of interest.

References

- Megged O, Baskin E. Neonatal parotitis. *J Pediatr*. 2018;196:319.
- Gupta A, Kingdon T, McKernan A. Neonatal parotitis: a case report. *Clin Pract Cases Emerg Med*. 2021;5(2):218–221.
- El Omri M, Jemli S, Belakhder M, et al. Neonatal suppurative parotitis: case report and review of literature. *Ear Nose Throat J*. 2024;1455613241234281.
- Velkoski A, Amoroso S, Brovedani P, et al. Presentation of acute suppurative parotitis in a newborn with incessant crying. *Arch Dis Child Fetal Neonatal Ed*. 2017;102(2):F125.
- Ichinose M, Matsushima T, Hataya H. Purulent discharge from stensen duct in neonatal suppurative parotitis. *J Pediatr*. 2022;243:230–231.
- Deepak K, Garima G, Jhamb U. Bilateral acute neonatal suppurative parotitis: a rare finding in neonatal age. *J Neonatal Perinatal Med*. 2015;8(1):63–65.
- Ray S, Nadeem L. Suppurative parotitis in a preterm infant. *BMJ Case Rep*. 2023;16(1):e253713.
- Mori T, Shimomura R, Ito T, et al. Neonatal suppurative parotitis: case reports and literature review. *Pediatr Int*. 2022;64(1):e14762.
- Spiegel R, Miron D, Sakran W, et al. Acute neonatal suppurative parotitis: case reports and review. *Pediatr Infect Dis J*. 2004;23(1):76–78.
- Ismail EA, Scoudi TM, Al-Amir M, et al. Neonatal suppurative parotitis over the last 4 decades: report of three new cases and review. *Pediatr Int*. 2013;55(1):60–64.
- Costa L, Leal LM, Vales F, et al. Acute parotitis in a newborn: a case report and review of the literature. *Egypt J Otolaryngol*. 2016;32:236–239.
- Sabatino G, Verrotti A, de Martino M, et al. Neonatal suppurative parotitis: a study of five cases. *Eur J Pediatr*. 1999;158(4):312–314.
- Makhoul J, Lorrot M, Teissier N, et al. Acute bacterial parotitis in infants under 3 months of age: a retrospective study in a pediatric tertiary care center. *Arch Pediatr*. 2011;18(12):1284–1289.
- Leake D, Leake R. Neonatal suppurative parotitis. *Pediatrics*. 1970;46(2):202–207.
- Salaria M, Poddar B, Parmar V. Neonatal parotitis. *Indian J Pediatr*. 2001;68(3):283.
- Avcu G, Belet N, Karli A, et al. Acute suppurative parotitis in a 33-day-old patient. *J Trop Pediatr*. 2015;61(3):218–221.
- Fathalla B, Collins D, Ezhuthachan S. Acute suppurative parotitis: uncommon presentation in a premature infant. *J Perinatol*. 2000;20(1):57–59.
- Lindgren C, Lucovic VB. Aseptic sialadenitis in preterm infants associated with long-term oro-gastric tube feeding. *Eur J Pediatr*. 1998;157(12):1014–1016.

19. Moritz ML, Manole MD, Bogen DL, et al. Breastfeeding-associated hypernatremia: are we missing the diagnosis? *Pediatrics*. 2005;116(3):e343–e347.
20. Tapisiz A, Belet N, Ciftçi E, et al. Neonatal suppurative submandibular sialadenitis. *Turk J Pediatr*. 2009;51(2):180–182.
21. Skude G. Sources of the serum isomylases and their normal range of variation with age. *Scand J Gastroenterol*. 1975;10(6):577–584.
22. Singh K. Bilateral parotid abscess in a neonate. *Indian Pediatr*. 2006;43(11):1009–1010.
23. Todoroki Y, Tsukahara H, Kawatani M, et al. Neonatal suppurative parotitis possibly associated with congenital cytomegalovirus infection and maternal methyl dopa administration. *Pediatr Int*. 2006;48(2):185–186.
24. Curiel JA, del Río PG, del Val CP, et al. Neonatal acute suppurative parotitis. *An Pediatr (Barc)*. 2004;60(3):274–247.
25. Schwab J, Baroody F. Neonatal suppurative parotitis: a case report. *Clin Pediatr (Phila)*. 2003;42(6):565–566.
26. Chevalier J, Jadcherla SR. Parotid swelling in a premature neonate. *Am J Perinatol*. 2002;19(8):435–438.
27. Isfaoun Z, Radouani MA, Azzaoui S, et al. Acute neonatal suppurative parotitis: about three clinical cases and review of the literature. *Pan Afr Med J*. 2016;24:286.
28. Akgun C, Peker E, Akbayram S, et al. A 3-day-old boy with a right preauricular swelling. *Eur J Pediatr*. 2010;169(5):637–638.
29. Guerra AAH, Osguthorpe RJ. Acute neonatal parotitis caused by *Streptococcus pyogenes*: a case report. *Clin Pediatr (Phila)*. 2010;49(5):499–501.
30. Özdemir H, Karbuz A, Ciftçi E, et al. Acute neonatal suppurative parotitis: a case report and review of the literature. *Int J Infect Dis*. 2011;15(7):e500–e502.
31. de Suremain N, Marteau E, Leruste A, et al. Neonatal suppurative parotitis: a case report and review of the literature. *Arch Pediatr*. 2014;21(2):223–225.
32. Walter C, Noguera A, Gene A, et al. Group B streptococcal late-onset disease presenting with parotitis. *J Paediatr Child Health*. 2009;45(12):764–766.
33. Khan SU, O’Sullivan PG, McKiernan J. Acute suppurative neonatal parotitis: case report. *Ear Nose Throat J*. 2010;89(2):90–91.
34. Miranda A, Pereira KD. Neonatal suppurative parotitis. *Ear Nose Throat J*. 2010;89(10):488–489.
35. Cabezón R, Kreft J, Ramírez C, et al. Parotiditis aguda en recién nacido. *Rev Otorrinolaringol Cir Cabeza Cuello*. 2010;70:65–70.
36. Özdil M, Erçin D. Acute suppurative parotitis in a 22-day-neonate with sepsis: a rare case report. *Cam and Sakura Med J*. 2024;4(1):36–38.
37. Hadizadeh T, Uwaifo OO. Neonatal acute suppurative parotitis. *Clin Pediatr (Phila)*. 2020;59(11):1019–1021.
38. Palacín PS, Gil RM, Vilella LC, et al. Group B *Streptococcus* late-onset disease presenting as cellulitis-adenitis syndrome. *An Pediatr (Barc)*. 2004;60(1):75–79.
39. Brook I. Suppurative parotitis caused by anaerobic bacteria in newborns. *Pediatr Infect Dis J*. 2002;21(1):81–82.