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Case Report

Acute neonatal suppurative parotitis: a case report and review of the literature

Halil Özdemir [a,*], Adem Karbuz [a], Ergin Çiftçi [a], Suat Fitöz [b], Erdal İnce [a], Ülker Doğru [a]

[a] Department of Pediatric Infectious Diseases, Ankara University Medical School, Dikimevi, Ankara, Turkey
[b] Department of Radiology, Ankara University Medical School, Ankara, Turkey

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SUMMARY

Neonatal suppurative parotitis (NSP) is an uncommon disease. Information about the etiopathogenesis and management of the disease is very limited. Here, we describe a newborn who developed NSP due to *Pseudomonas aeruginosa* and who was treated successfully with antibiotics.

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1. Introduction

Neonatal suppurative parotitis (NSP) is an uncommon disease with a prevalence of 3.8/10 000 admissions in one report from Italy.[1] Only 32 cases of NSP have been described in the English language literature over the last 35 years, and the causative agent in most cases was *Staphylococcus aureus*.[2] Here, we describe a newborn who developed NSP due to *Pseudomonas aeruginosa* and who was treated successfully with antibiotics.

2. Case report

A 20-day-old breast-fed male newborn presented with a 1-day history of restlessness and right pre-auricular swelling. On the morning of admission the swelling had increased in size, turned red, and was tender; the parents then brought him to our hospital. He was born by caesarean section at 35 weeks of gestation and his birth weight was 2980 g. Oligohydramnios was detected in the prenatal period, and after delivery, the Department of Pediatric Surgery determined the cause to be a posterior urethral valve. A urethral catheter was placed to allow for bladder drainage and he was discharged with this urethral catheter and antibiotic prophylaxis 3 days before the beginning of his complaints. The parents reported no history of trauma to the infant's face or head, and the mother denied any history of breast tenderness or recent skin infection.

On admission the baby was irritable, but otherwise appeared healthy. His weight was 2950 g, and axillary temperature was 38.5 °C. Examination revealed erythema and fluctuant swelling of the right parotid gland (Figure 1). Pus exuded from the right Stenson's duct when pressure was applied to the gland. The rest of the physical examination was unremarkable.

Laboratory tests revealed: hemoglobin 12.0 g/dl, white blood cell count 18.2 × 10^9/l with a differential of 60% neutrophils, 28% lymphocytes, 10% monocytes and 2% stabs, erythrocyte sedimentation rate 18 mm/h, C-reactive protein 0.89 mg/dl, and serum alpha amylase concentration 7 IU/l (normal range 30–100 IU/l). The renal and liver function tests, serum electrolytes, and urine analysis were normal. Ultrasonography of the parotid glands demonstrated an enlarged right parotid gland with hypoechoic area compatible with acute suppurative parotitis, which was progressing to abscess formation. Also, intraparotid lymph nodes of millimeters in diameter were detected (Figure 2).

Parotid pus, blood, and urine cultures were obtained, and therapy with intravenous ampicillin–sulbactam 150 mg/kg/day was started. The blood and urine cultures were sterile, however *P. aeruginosa* grew in the parotid pus culture on the second day after admission. Ampicillin–sulbactam was then changed to intravenous ceftazidime (100 mg/kg/day). After 1 day of parenteral ceftazidime therapy the fever resolved and on the fourth day of treatment the parotid swelling resolved. Antibiotic treatment with ceftazidime was continued for 10 days. The patient's serum immunoglobulin (Ig) levels were within normal limits (IgG 849 mg/dl, IgA 5.7 mg/dl, and IgM 43.2 mg/dl). Follow-up examination demonstrated no residues or abnormalities of the gland and he did not show chronic recurrent parotitis.

* Corresponding author. Tel.: +90 312 5956539; fax: +90 312 3191440.
E-mail address: doktorhalil@gmail.com (H. Özdemir).

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Figure 1. Neonate with parotid gland swelling.

3. Discussion

NSP is an uncommon infection. Common predisposing conditions include prematurity, dehydration, and duct stasis. In infants, infection of the parotid glands appears to be more common than infection of the submandibular glands.[3,4]

Spiegel et al. reviewed the cases of patients with NSP during the past 35 years, mostly from case reports. NSP was unilateral in most cases, and swelling, with or without redness of the parotid region, was the most prevalent sign at the time of admission. Thirty-eight percent of patients with NSP were born prematurely and, because 11% of births in the general population are preterm, prematurity should be considered as a major risk factor for the infection.[2,5] The increased risk of NSP among preterm babies has been attributed to their increased risk of dehydration, which may reduce salivary secretion. NSP is reported as more prevalent among boys, with a rate of almost 3:1.[2,6] Our patient was male and premature.

Although bacterial seeding of the parotid can occur hematogenously, infection is more common from oral flora tracking in a retrograde fashion into the gland. Possible etiologies of retrograde flow from the oral cavity into the parotid include dehydration with resultant decrease in saliva production and stasis, dilatation of the ducts (sialectasis) through scarring or obstruction by a stone or mass, and congenital variations in ductal structure. Other causes of facial swelling that may be confused with parotid enlargement include maxillary infections, trauma, lymphangiomas, hemangiomas, lipomas, and adenomas.[4] Transmission of bacteria during breastfeeding or through contaminated formula can be a potential cause of sialadenitis.[7] In the presented case, the infant was breastfed, but the mother had no signs of mastitis.

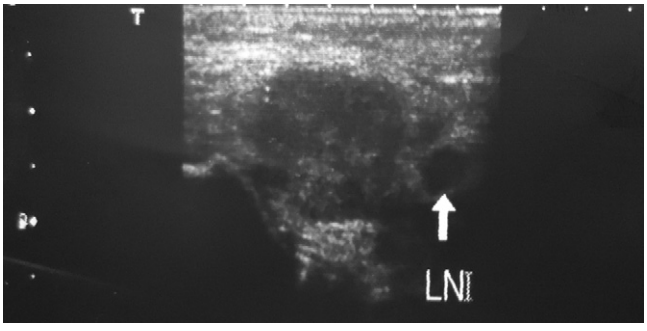


Figure 2. Sonogram of right parotid gland shows increased gland size with hypoechoic area and intraparotid lymph nodes of millimeters in diameter.

The most common presentation of NSP is fever and swelling and erythema in the pre-auricular area beginning at between 7 and 14 days of life. The infection may be bilateral.[4] Laboratory findings in NSP have usually been nonspecific. Leukocytosis above 15×10^9/l with neutrophil predominance was found in 71% of the cases, and the erythrocyte sedimentation rate was elevated in only 20% of the patients. Serum amylase levels were elevated in 45% of the newborns.[2] This phenomenon may be related to the immaturity of this salivary isoenzyme activity in newborns.[2,8] Our patient had leukocytosis with neutrophil predominance, but his serum amylase level was within the normal range.

Purulent drainage from Stenson's duct is pathognomonic of this condition, and culture of the exudate will both confirm the diagnosis and help guide treatment. Parotid ultrasound may reveal a diffusely enlarged gland with a coarse echo pattern.[4] Examination with ultrasound is non-invasive, cheap, and useful for diagnosis, differential diagnosis, and excluding the other predisposing factors like anatomical abnormalities of Stenson's duct, mechanical salivary duct obstruction secondary to a sialolith, and infection related to a parotid gland neoplasm. Our patient fulfilled the diagnostic criteria of suppurative parotitis: a combination of parotid swelling, purulent exudation from the Stenson's duct, and growth of pathogenic bacteria in culture of the pus.[2,9]

S. aureus is the most common pathogen isolated from infants with NSP. Other potential isolates include viridans Streptococcus species and *Escherichia coli*. Gram-negative organisms such as *Klebsiella pneumoniae* and *P. aeruginosa* have been implicated in nosocomial and hematogenous infections secondary to sepsis. Recent reports have called attention to the presence of anaerobic species, such as *Bacteroides melaninogenicus* and *Fusobacterium nucleatum*, which can also be isolated from the parotid exudate found in NSP. As the source of infection in NSP is commonly ascending bacteria from the oral flora, the presence of such anaerobes is not entirely surprising.[4] Additionally, in the report of Spiegel et al., the most common pathogen was *S. aureus*, which was found in 55% of patients. Other less frequent agents were other Gram-positive cocci, i.e., viridans streptococci, *Streptococcus pyogenes*, Peptostreptococcus and coagulase-negative Staphylococcus (22%), and Gram-negative bacilli (16%) and rarely anaerobic bacteria. In our patient *P. aeruginosa* grew in the parotid pus culture. We believe the source was the oral flora, which was colonized with *P. aeruginosa* during hospitalization in the Department of Pediatric Surgery.

The mainstay in the treatment of NSP is the appropriate selection of antibiotics to cover the causative organism. A penicillinase-resistant penicillin or first-generation cephalosporin to effectively cover *S. aureus*, along with clindamycin or a similar medication to cover possible anaerobic infection are good initial choices until better direction can be obtained from the study of cultures of expressed material from Stenson's duct. A treatment period of 7–10 days appears to be adequate.[3,4] Incision and drainage of the affected parotid is occasionally performed for abscess formation, but the need for such a procedure has declined as antimicrobial treatments have improved.[1,4] We started only ampicillin–sulbactam because it is effective for streptococci and *S. aureus*, and in our population methicillin-resistant *S. aureus* is rare. However, we then had to change this to ceftazidime because of the growth of *P. aeruginosa*.

In conclusion, although NSP is rare, it should be suspected in newborns presenting with an erythematous pre-auricular mass with or without any predisposing factors. Although *S. aureus* is the most common pathogen isolated from infants with NSP, Gram-negative bacilli such as *P. aeruginosa* can be isolated from the parotid pus.

Conflicts of interest: No conflict of interest and no funding source to declare. Ethical approval and informed consent were obtained.

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