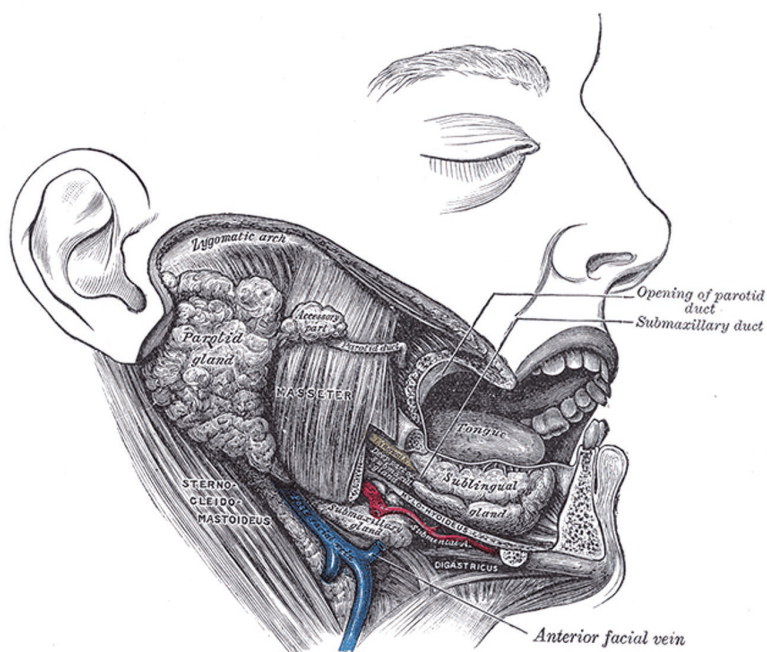


## Acute suppurative parotitis in two preterm infants

September 2014





This female infant was born at 32 0/7 weeks to a 32-year-old G1/P1 by Caesarean section because of HELLP syndrome. Apart from cholecystolithiasis, pregnancy had been unremarkable up to this point. Arterial umbilical cord pH was 7.33 and the baby adapted well with Apgar scores of 8, 8, and 9 at 1, 5 and 10 minutes, respectively. Birth weight was 1420 g (P 20). Apart from mild respiratory distress, requiring nasal CPAP for the first 12 hours of life, the postnatal course was uneventful until the 18th day of life when the baby developed signs of infection with repeated apneas, tachycardia and poor peripheral perfusion. A CBC revealed a left shift of 25%, and CRP and IL-6 were slightly elevated (10 mg/l and IL-6 61 pg/ml, respectively). Late-onset sepsis was suspected and antibiotic therapy with amikacin and amoxicillin/ clavulanic acid was initiated.

Within a few hours, swelling and erythema of the left cheek were noted (Fig. 1). There was induration of the skin and the regional lymph nodes were enlarged. No purulent secretion from the parotid duct was seen. Ultrasonography of the parotid and preauricular region showed edematous swelling, hypoechogenicity and hyperperfusion of the left parotid gland (Fig. 2,3). No abscess was seen. Several enlarged cervical lymph nodes measuring up to 5 mm in diameter were noted. The parotid duct was slightly dilated, but no stone was seen within the duct.

The clinical and ultrasound findings were consistent with acute suppurative parotitis, which is a rare finding in preterm infants. Hematogenous spread was the most likely mechanism of infection. After 4 days, enoral cultures grew penicillin-resistant, but amoxicillin/clavulanic acid sensitive *Staphylococcus aureus*. Blood cultures remained negative. Antibiotic therapy was continued with intravenous amoxicillin/clavulanic acid for a total of 10 days. At this point, ultrasonography revealed persistent hyperperfusion, but decreased swelling. The infant was discharged home at the age of 5 weeks.



*Swelling with mild erythema of the left cheek.*

**Fig. 1**

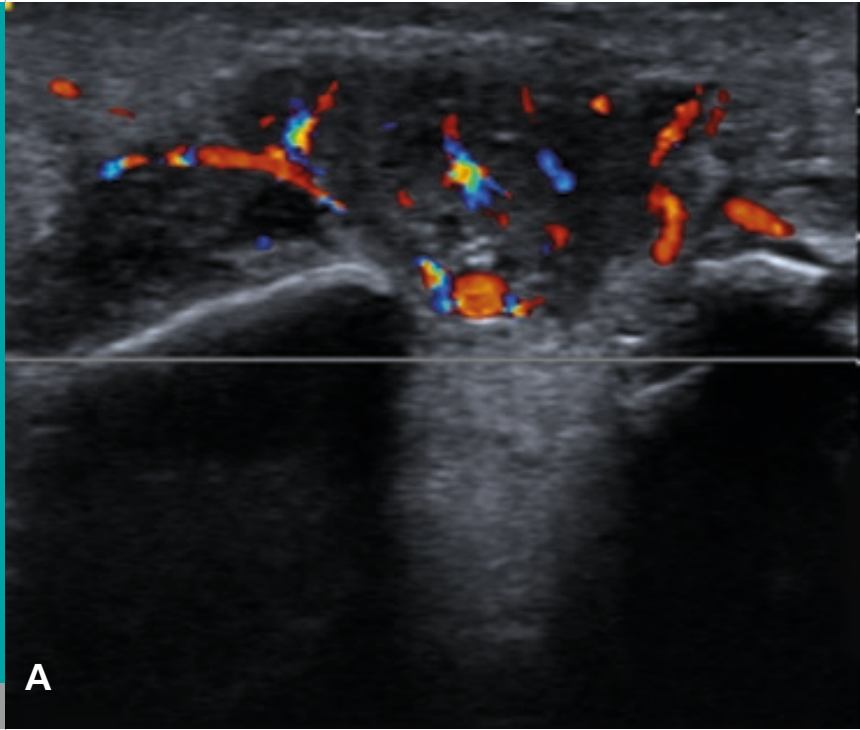
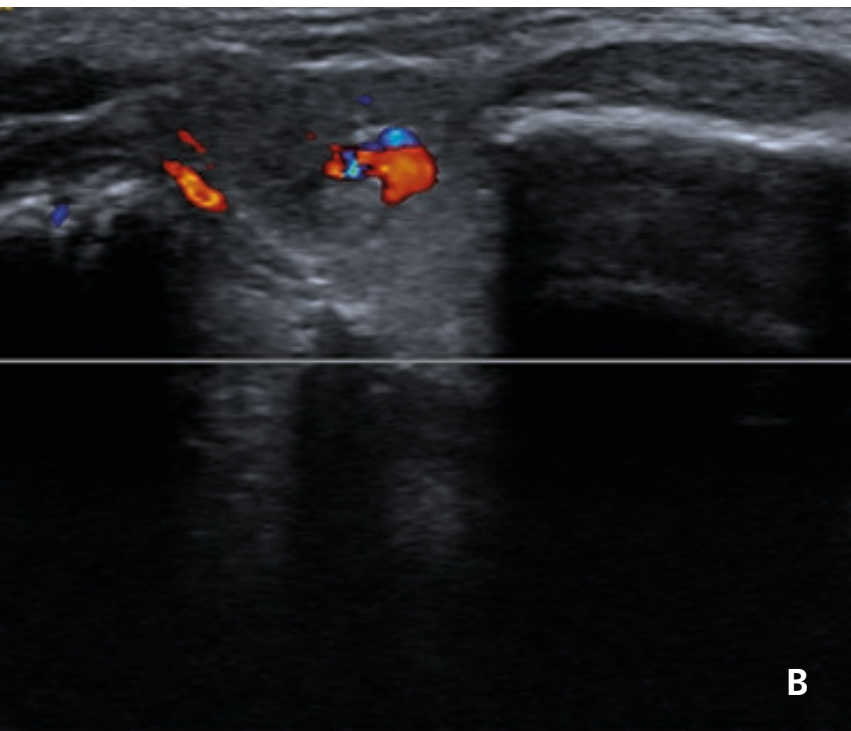
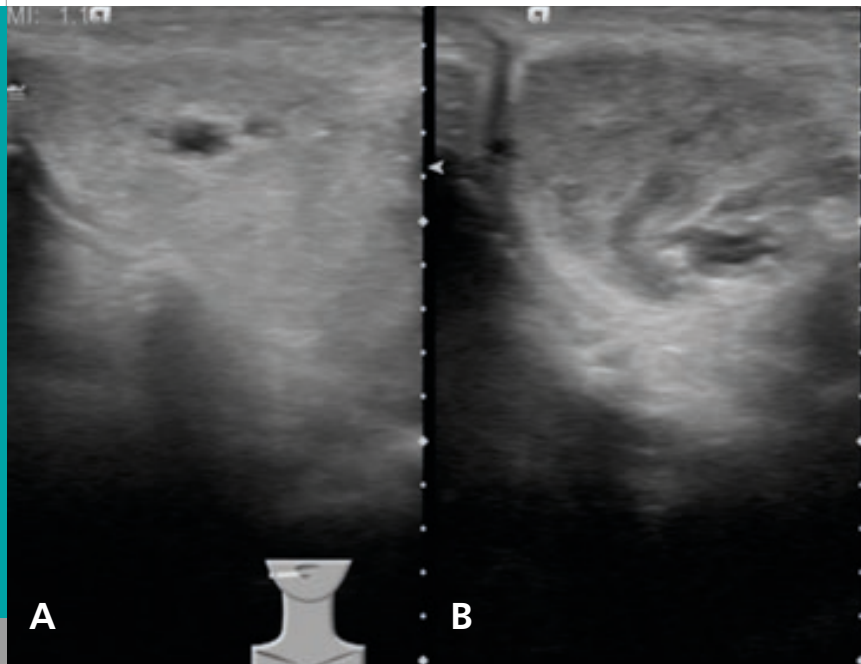


Fig. 2

*Doppler sonography: hypoechogenicity and hyperperfusion of left parotid gland (A); in comparison, normal perfusion of right parotid gland (B).*





**Fig.3**

*Comparison of the right-sided normal parotid gland (A) and the left-sided enlarged, hypoechogenic parotid gland (B).*



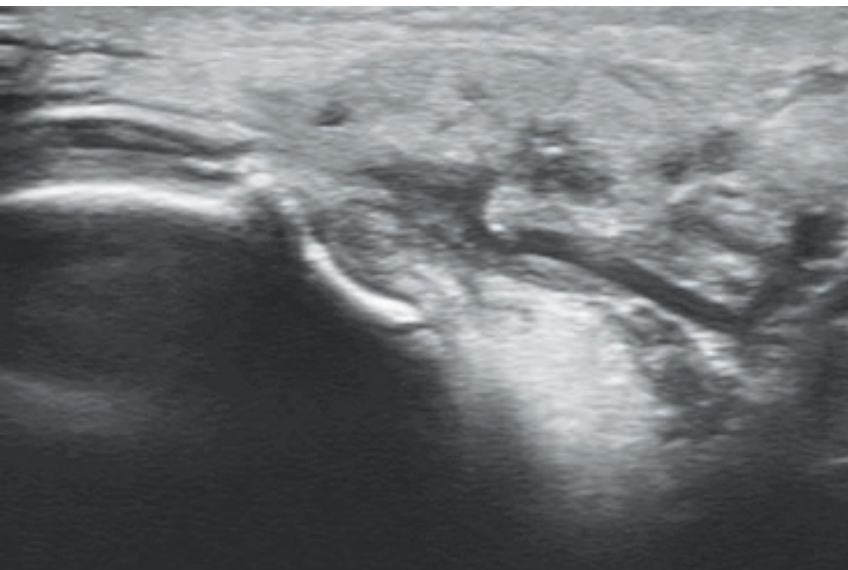
This female preterm infant was born to a 34-year-old G3/P1 at 34 4/7 weeks of gestation by Caesarean section because of an abnormal CTG. Pregnancy was complicated by intrauterine growth restriction and suspected aortic stenosis (the latter was not confirmed postnatally). The prenatal karyotype was 46, XX. Apgar scores were 6, 7, 9 at 1, 5 and 10 minutes, respectively, and arterial umbilical cord pH was 7.29. There was symmetrical growth restriction with a birth weight of 1245 g, length of 37.0 cm and head circumference of 29.0 cm (all < P3).

The postnatal course was uneventful until the 38th day of life, when the baby developed an elevated temperature of 38.5°C. The infant appeared ill with bilateral conjunctivitis and a left-sided preauricular swelling, induration and tenderness (Fig. 4). Inspection of the oral cavity was normal. Ultrasonography demonstrated an enlarged parotid gland with hyperperfusion; an abscess, however, was ruled out (Fig. 5). CBC was normal, but the CRP was elevated at 68 mg/l. Acute suppurative parotitis in combination with conjunctivitis was diagnosed and the baby was treated with intravenous cefuroxime for 10 days. Blood culture and conjunctival swab were positive for penicillin-resistant but cefuroxime-sensitive *Staphylococcus aureus*. The infant recovered rapidly and was discharged home on the 49th day of life.



**Fig. 4**

*Left-sided preauricular swelling and erythema of the skin*



**Fig. 5**

*Enlarged parotid gland with slightly dilated duct without evidence of an abscess.*

## DISCUSSION

Neonatal suppurative parotitis is a rare condition. The prevalence is estimated to be 3.8-14 per 10'000 hospital admissions (1). It is more common in boys than in girls with a ratio of 3:1. Preterm infants account for 32-38% of the cases. Various published articles consist of case reports or case series and literature reviews, with a total of 48 cases reported in the English literature over the last 42 years (1, 2).

Typical clinical signs are rapid swelling of the parotid gland with induration, erythema and warmth of the overlying skin. Purulent secretions draining from the parotid duct are not evident in all cases, but can sometimes be seen with gentle pressure on the parotid gland.

*Staphylococcus aureus* is the most frequently found pathogen (61%) followed by other gram-positive (25%), gram-negative (16%) or anaerobic bacteria (11%) (1, 3). Blood cultures are positive in only 35% of the cases (4), and laboratory investigations are generally not very helpful. An ascending route of infection via the parotid duct has been proposed, particularly when dehydration causes reduced salivary flow (3). The second route is hematogenous spread as part of septicemia (3). The exclusively serous secretions of the parotid gland lack bacteriostatic mucoid components, rendering the gland more susceptible to infections than other salivary glands. Additional risk factors are low birth weight, immune suppression, obstruction of

the parotid duct, oral trauma and infected or contaminated nutrition (1). Usually the diagnosis can be made easily, but mandibular osteomyelitis, cervical lymphadenitis, and infected lymphangiomas or hemangiomas should be ruled out.

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