

Bilateral Parotid Abscess in a Neonate

Suppurative salivary gland diseases are rare in neonates. Only 22 cases of neonatal suppurative parotitis are described in the English literature in the last 35 years(1). During a 9-year study by Sabatino, *et al.*, five cases of neonatal suppurative parotitis were detected in 3624 hospital admissions. The relative risk of developing neonatal suppurative parotitis in admitted infants was 5.52 (0.62 - 49.35)(2). *Staphylococcus aureus* is the causative organism in most cases, which reaches parotid glands via Stensen's duct. We report a term breastfed neonate with bilateral parotid abscess.

A 14-day-full term male neonate, second in birth order, delivered at home by trained health worker was brought with bilateral parotid swellings for last 7 days. The baby was exclusively breastfed. Examination revealed a 2.8 kg lethargic, toxic looking neonate with poor neonatal reflexes. There were bilateral, symmetrical and fluctuant parotid swellings, 2.5 × 4.5 cm each (*Fig. 1*). Overlying skin was red, hot and necrosed in the central part of the swelling. Aspiration of the swelling revealed



Fig. 1. Bilateral Symmetrical Parotid Swellings.

thick pus. This was followed by excision and drainage on both sides. *Staphylococcus aureus* was isolated from pus and blood. HIV serology was negative. The neonate was treated with parenteral ampicillin and cloxacillin for 14 days resulting in complete recovery.

Reappraisal of history revealed maternal breast abscess on left side noted on 4th post partum day. Pus and breast milk culture, both isolated *Staphylococcus aureus*.

Acute suppurative infection of salivary gland is rare in neonatal period and occur more frequently in pre-term newborns(3). Dehydration, congenital anomalies, prolonged orogastric feeding and septicemia have also been associated with suppurative parotitis in newborn(1,4,5). Protective role of exclusive breast-feeding in prevention of bacterial sialadenitis has not been defined. Various organisms known to cause suppurative parotitis are *Staphylococcus aureus*, *Streptococcus pyogenes*, *Streptococcus viridens*, *E. coli*, *Pseudomonas aeruginosa* and *N. catarrhalis*(3). Suppurative parotitis is usually bilateral and may progress to abscess formation. In the reported case, bilateral parotid abscess also followed suppurative parotitis. The probable source of infection, in the present case, was maternal breast milk, which reached parotid glands via Stensen's ducts. Isolation of *Staphylococcus aureus* from breast and parotid abscesses as well as breast milk, supports a cause-effect relationship.

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Persistent Thrombocytopenia after Dengue Hemorrhagic Fever

We report two children with persistent thrombocytopenia after Dengue hemorrhagic fever (DHF). On literature review we could locate only one similar case(1).

Case 1

A 9-month-old boy presented with history of high grade fever for five days, hypotension, soft hepatomegaly and malena. Investigations revealed a hematocrit of 53.4% and platelet count of 58,000 per cubic mm. DHF was confirmed serologically by dengue specific IgM and IgG antibodies by ELISA test. The child recovered in 10 days; however, thrombocytopenia persisted (platelet count: 12,000/mm³). A bone marrow aspirate, performed on 26th day of illness, suggested adequate megakaryocytes.

Case 2

A 9-year-old girl presented with history of high grade fever for four days, myalgia, hypotension, epistaxis and petechiae. Investigations revealed a platelet count of 40,000 per cubic mm, and a hematocrit of 48.6%. DHF was confirmed serologically by ELISA for IgG and IgM antibodies. She had persistent thrombocytopenia of 13,000 per

cubic mm of blood at 30 days. A bone marrow aspirate revealed adequate megakaryocytes.

Persistent thrombocytopenia in both cases responded to intravenous methylprednisolone. Thrombocytopenia is known to occur in DHF, which promptly recovers by 9-10 days of illness(2). The pathogenesis of persistent thrombocytopenia in these two cases is not clearly understood. Following are possible mechanisms that have been postulated for thrombocytopenia in DHF;

- (a) Dengue virus induces bone marrow suppression(2);
- (b) Dengue virus can bind to human platelets in presence of virus specific antibody and immune mediated clearance of platelets(3);
- (c) Spontaneous aggregation of platelets to vascular endothelial cell pre-infected by virus inducing aggregation, lysis and platelet destruction(4).
- (d) Anti-platelet antibodies generated after dengue virus infection causes destruction of platelets(5).

In the two patients that we are reporting, the presence of adequate megakaryocytes, confirmed by bone marrow aspirate, with co-existent thrombocytopenia at a time when the virus was normally expected to have been cleared and prompt response to methyl prednisolone suggests immune mediated