Natal and Neonatal Teeth: A Review of the Literature

SUMMARY
Normal eruption of primary teeth into the oral cavity begins at about 6 months of child’s age. Teeth that erupt prematurely have occasionally been reported in the medical and dental literature and have been referred to as congenital teeth, foetal teeth, pre-deciduous teeth and dentitio praecox. The most affected teeth are lower central incisors and only 1-10% of them are supernumerary teeth. The incidence of natal and neonatal teeth ranges from 1:2000 to 1:3500. The exact etiology has not been proved yet, but there is a correlation between natal teeth and hereditary, environmental factors and some syndromes. The management of the case depends on clinical characteristics of the natal or neonatal teeth, as well as on complications they might cause.

The aim of this text is to present a literature review on important aspects of natal and neonatal teeth concerning prevalence, etiology, clinical and histological characteristics, differential diagnosis, complications and management.

Keywords: Natal Teeth; Neonatal Teeth

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Introduction
Typical eruption of primary teeth begins at about 6 months of age. Teeth observed at birth are considered as natal teeth, while teeth observed within the first 30 days as neonatal teeth, based on the classification given by Massler and Savara in 1950 according to the time of eruption. In 1966, Spouge and Feasby categorized these teeth based on clinical features as mature and immature. Mature are those which are fully developed in shape and comparable in morphology to the deciduous teeth; immature are the teeth whose structure and development are incomplete. Finally, Hebling in 1997 presented 4 clinical categories:
- Shell-shaped crown loosely attached to the alveolus by gingival tissue and absence of a root;
- Solid crown loosely attached to the alveolus by gingival tissue and little or no root;
- Eruption of the incisal margin of the crown through gingival tissue;
- Mucosal swelling with the tooth non-erupted but palpable.

The rare occurrence of natal and neonatal teeth was associated in the past with superstition and folklore. Today this phenomenon creates great interest and concern, not only to parents but to clinicians as well. This is due to their clinical characteristics (small size, conical shape, and great mobility) which are the cause of certain complications (laceration of mother’s breasts, sublingual ulceration, and danger of aspiration of the teeth).

History
The rare occurrence of natal and neonatal teeth has led to association with superstition and folklore. Some cultures have believed that children born with teeth were favoured, particularly in Western Europe and Malaysia, whereas other considered natal teeth as an ill omen. In England it was believed that natal teeth showed that the children would grow into famous soldiers, in France and Italy that they ‘would get on in the world’ and in Sweden that they could cure an injured finger if it were placed in...
their mouth. In many places like Poland, India, and Africa, superstition still prevails considering these children to be monsters or evil children\textsuperscript{14}. Among several native African tribes, such as in urban Bariba in Benin West Africa, one of the most dangerous signs suggesting a witch child is to be born with teeth and if that happened the child was either abandoned or killed. Precautions in the form of a purification ritual are still taken today in such cases, and sometimes the teeth will be extracted\textsuperscript{101}. In China a child born with teeth suggests misfortune for the family: if the child is male then the father will die and if it is a female the mother. Many historic personalities, like Hannibal, Cardinal Richelieu, Broca, Zoroaster, Napoleon, English King Richard the III and King Louis XIV of France are said to be born with teeth. Also many proverbs and apothegms are made up for natal teeth, such as ‘The one whose teeth grow early, will early sink into the grave’\textsuperscript{14}. Due to these superstitions it is suggested that a trans-cultural approach be adopted in managing cases in which the parents feel particularly anxious and uncomfortable about prematurely erupted teeth in order to cater for the social well-being of the child and family\textsuperscript{88}.

### Prevalence

Many authors have reviewed the incidence of natal and neonatal teeth (Table 1). The estimated prevalence ranges from 1:10 to 1:30,000. It is accepted by many authors that the ratio of natal and neonatal teeth is somewhere between 1:2000 and 1:3500\textsuperscript{14,23,24,78,110,123}.

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Location of study</th>
<th>Natal and neonatal teeth</th>
<th>Total births</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Magicot (1883)\textsuperscript{71}</td>
<td>Paris, France</td>
<td>3</td>
<td>17,578</td>
<td>1:6,000</td>
</tr>
<tr>
<td>Howkins (1932)\textsuperscript{45}</td>
<td>Birmingham, England</td>
<td>1</td>
<td>10,000</td>
<td>1:10,000</td>
</tr>
<tr>
<td>Massler and Savara (1950)\textsuperscript{78}</td>
<td>Chicago, USA</td>
<td>7</td>
<td>9,400</td>
<td>1:2,000</td>
</tr>
<tr>
<td>Allwright (1958)\textsuperscript{33}</td>
<td>Hong Kong, China</td>
<td>2</td>
<td>6,817</td>
<td>1:3,400</td>
</tr>
<tr>
<td>Mayhall (1967)\textsuperscript{80}</td>
<td>Juneau, Alaska (Tlingit Indians)</td>
<td>8</td>
<td>90</td>
<td>1:11.25</td>
</tr>
<tr>
<td>Gordon and Langley (1970)\textsuperscript{41}</td>
<td>Oklahoma, USA (American Indian)</td>
<td>4</td>
<td>407</td>
<td>1:100</td>
</tr>
<tr>
<td>Jarvis and Gorlin (1972)\textsuperscript{50}</td>
<td>Alaska, USA (Eskimo)</td>
<td>16</td>
<td>1,571</td>
<td>1:98</td>
</tr>
<tr>
<td>Anderson (1982)\textsuperscript{52}</td>
<td>Columbia, USA</td>
<td>13</td>
<td>18,155</td>
<td>1:3,667</td>
</tr>
<tr>
<td>Kates et al (1984)\textsuperscript{52}</td>
<td>Boston, USA</td>
<td>13</td>
<td>18,155</td>
<td>1:3,667</td>
</tr>
<tr>
<td>Leung (1989)\textsuperscript{65}</td>
<td>Alberta, Canada</td>
<td>15</td>
<td>50,892</td>
<td>1:3,392</td>
</tr>
<tr>
<td>King and Lee (1989)\textsuperscript{57}</td>
<td>Hong Kong, China</td>
<td>17</td>
<td>22,500</td>
<td>1:1,324</td>
</tr>
<tr>
<td>Gladen et al (1990)\textsuperscript{59}</td>
<td>Taiwan</td>
<td>13</td>
<td>128</td>
<td>1:10</td>
</tr>
<tr>
<td>Rusmah (1991)\textsuperscript{100}</td>
<td>Kuala Lumpur, Malaysia</td>
<td>4</td>
<td>9,600</td>
<td>1:2,325</td>
</tr>
<tr>
<td>To (1991)\textsuperscript{117}</td>
<td>Hong Kong, China</td>
<td>48</td>
<td>53,678</td>
<td>1:1,118</td>
</tr>
<tr>
<td>Diaz-Romero et al (1991)\textsuperscript{30}</td>
<td>Mexico</td>
<td>31</td>
<td>1,200</td>
<td>1:387</td>
</tr>
<tr>
<td>De Almeida and Gomide (1995)\textsuperscript{27}</td>
<td>Brazil</td>
<td>47*</td>
<td>1019**</td>
<td>1:22</td>
</tr>
<tr>
<td>Alaluusua et al (2002)\textsuperscript{22}</td>
<td>Finland</td>
<td>34</td>
<td>34,457</td>
<td>1:1,013</td>
</tr>
<tr>
<td>Liu and Huang (2004)\textsuperscript{70}</td>
<td>Taipei, Taiwan</td>
<td>2</td>
<td>420</td>
<td>1:140</td>
</tr>
<tr>
<td>Freudenberger et al (2008)\textsuperscript{36}</td>
<td>Mexico</td>
<td>50</td>
<td>2182</td>
<td>2.3:100</td>
</tr>
</tbody>
</table>

* 14 with complete unilateral cleft lip and palate and 33 with bilateral cleft lip and palate
** 692 with complete unilateral cleft lip and palate and 327 with bilateral cleft lip and palate
The prevalence of occurrence of natal and neonatal teeth in males and females is controversial, with some authors giving a higher ratio for females. Kates et al reporting a 66% proportion for females against a 31% proportion for males, and others suggesting that there isn’t any correlation with gender.

Natal teeth are more common than neonatal teeth. Zhu and King (1995) have reported natal teeth as a familiar trait in 8-62% of cases. Bondenhoff and Gorlin (1963) reported family association in 14.5% of cases, while Kates et al (1984) found a positive family history in 7 out of 38 cases of natal and neonatal teeth. A hereditary transmission of an autosomal dominant gene has also been suggested.

The hereditary factor is assumed a possible cause of natal teeth. Zhu and King (1995) have reported natal teeth as a familiar trait in 8-62% of cases. Bondenhoff and Gorlin (1963) reported family association in 14.5% of cases, while Kates et al (1984) found a positive family history in 7 out of 38 cases of natal and neonatal teeth. A hereditary transmission of an autosomal dominant gene has also been suggested.

Etiology

The exact etiology of natal and neonatal teeth has not been elucidated yet. Many theories have been expressed regarding the cause of the occurrence of these teeth. One of them includes dietary deficiencies or hypovitaminosis due to poor maternal health, endocrine disturbances and pyelitis during pregnancy. Another theory refers to hormonal stimulation, meaning the excessive secretion of pituitary, thyroid or gonads. It is also significant to mention that congenital syphilis seems to have varying effect; in some cases premature eruption was noticed, while in others the eruption was retarded. Moreover, febrile states can affect the normal eruption of teeth, for example fever and exanthemata during pregnancy can cause premature eruption.

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Table 2. Syndromes and developmental disturbances related to natal and neonatal teeth

<table>
<thead>
<tr>
<th>Syndromes and developmental disturbances</th>
<th>Author(s)</th>
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<tbody>
<tr>
<td>Patent ductus arteriosus and intestinal pseudo-obstruction</td>
<td>Harris et al (1976)45</td>
</tr>
<tr>
<td>Optiz (G/BBB) syndrome</td>
<td>Shaw et al (2006)103</td>
</tr>
<tr>
<td>Van der Woude syndrome</td>
<td>Hersh and Verdi (1992)46</td>
</tr>
<tr>
<td>Steatocystoma multiplex</td>
<td>King and Lee (1987)56</td>
</tr>
<tr>
<td>Wiedermann-Rautenstrauch neonatal progeria</td>
<td>Pivnick et al (2000), Arboleda (1997); Byung-Duk and</td>
</tr>
<tr>
<td></td>
<td>Jung-Wook (2006); Castiñeira et al (1992),</td>
</tr>
<tr>
<td></td>
<td>Korniszewski et al (2001)117,18,22,62</td>
</tr>
<tr>
<td>Pfeiffer syndrome type 3</td>
<td>Alvarez et al (1993)4</td>
</tr>
<tr>
<td>Walker Warburg syndrome (Congenital hydrocephalus with congenital</td>
<td>Mandal et al (2002)13</td>
</tr>
<tr>
<td>Hyper IgE syndrome</td>
<td>Hennekam and Van Doorne (1990)45</td>
</tr>
<tr>
<td>Rubinstein-Taybi syndrome</td>
<td>Darwish, Sastry and Ruprecht (1987)26</td>
</tr>
<tr>
<td>Bifid tongue and profound sensorineural hearing loss</td>
<td>Boyd and Miles (1951)16</td>
</tr>
<tr>
<td>Cyclopia</td>
<td>Koklu and Kurtoglu (2007)61</td>
</tr>
<tr>
<td>Pierre Robin syndrome</td>
<td>Kharbanda et al (1985)34</td>
</tr>
<tr>
<td>Short rib-polydactyly syndrome type II (Saldino-Noonan syndrome )</td>
<td>Stromme Koppang, Boman and Hoel (1983)113</td>
</tr>
<tr>
<td>Soto’s syndrome</td>
<td>Callanan, Anand and Sheehy (2009)20</td>
</tr>
<tr>
<td>Adrenogenital syndrome</td>
<td>Leung (1989)67</td>
</tr>
<tr>
<td>Epidermolysis bullosa simplex</td>
<td>Liu, Chen and Miles (1998)69</td>
</tr>
<tr>
<td>Cleft lip-palate</td>
<td>Cabate et al (2000)19</td>
</tr>
<tr>
<td>Odonto-Tricho-Ungual-Digital-Palmar Syndrome</td>
<td>Mendoza and Valiente (1997)81</td>
</tr>
<tr>
<td>Teebi hypertelorism syndrome</td>
<td>Koenig (2003)59</td>
</tr>
<tr>
<td>Clouston syndrome</td>
<td>Reynolds, Gold and Scriver (1971)96</td>
</tr>
</tbody>
</table>
Another theory explaining the premature eruption is considered to be the abnormal position of the germ during its development in the alveolar bone\textsuperscript{8,97}. Furthermore, Clergueau-Guerithault proposed that the eruption of natal and neonatal teeth could be dependent on osteoblastic activity within the area of the tooth germ\textsuperscript{102}.

As far as environmental factors are concerned, some environmental toxins are considered to be causative factors. Gladden et al (1990)\textsuperscript{39} reported that 13 of 128 newborns, whose mothers where exposed to polychlorinated binephyls and dibenzofurans during the Yusheng environmental accident in Taiwan, had natal teeth. Also, 2 out of 12 live-borns from parents poisoned by PCBs in Kyushu, Japan were reported to have natal teeth\textsuperscript{82}. Another report by Alaluusua et al (2002)\textsuperscript{2} supports that there is no association between milk levels of polychlorinated binephyls, and dibenzofurans and the occurrence of natal teeth. They suggest that the prevailing levels of polychlorinated binephyls and dibenzofurans are likely below the threshold to cause prenatal eruptions of teeth.

Moreover, the presence of natal and neonatal teeth has been associated with many syndromes and developmental disturbances but there is no conclusive evidence of a correlation with these systemic conditions\textsuperscript{25}. The conditions that are related with the appearance of natal teeth are shown in the table 2.

Natal and neonatal teeth have also been reported in cutis gyratum and acanthosis nigricans\textsuperscript{10}, Turnpenny ectodermal dysplasia\textsuperscript{119}, in association with primary congenital glaucoma\textsuperscript{72}, in a case of an encephalic infant with cleft palate\textsuperscript{74}, in association with giant congenital nevocellular nevus\textsuperscript{53}, in a case of restrictive dermopathy\textsuperscript{79}, in a case of multiple joint dislocations with metaphyseal dysplasia\textsuperscript{90}, in a case of multiple anomalies: natal teeth, palatal cyst, bilateral lymphangiomas of the alveolar ridge and median alveolar notch\textsuperscript{21}, in a case of complex craniofacial anomalies\textsuperscript{12}, in Mohr syndrome\textsuperscript{9} and in association with syringomas and oligodontia\textsuperscript{35}. It is suggested that tooth abnormalities are dysmorphic markers of earlier developmental abnormalities, and could give warning signs in a syndrome diagnosis\textsuperscript{13}.

### Clinical and Histological Characteristics

Regarding clinical characteristics, the most affected teeth are the lower primary central incisors (85%), followed by the maxillary incisors (11%), mandibular canines and molars (3%) and maxillary canines and molars (1%)\textsuperscript{123}. Another characteristic of natal teeth is that they occur in pairs\textsuperscript{65,123}. The eruption of more than 2 teeth is rare. Despite that, Masatomi et al\textsuperscript{77} in 1991 reported an 18-month-old Japanese boy with 14 natal teeth, Gonçalves et al\textsuperscript{40} in 1998 presented the case of a newborn with 12 natal teeth and Portela et al\textsuperscript{52} in 2004 reported a newborn with 11 natal teeth.

Natal teeth are described as conical or normal in size and shape, yellowish, with hypoplastic enamel and dentin, and poor or absent root development\textsuperscript{37,100,123}. The hypoplastic enamel might be related to gingival covering\textsuperscript{52} and has a tendency to discolor. The incomplete root formation is the reason for the great mobility of the natal and neonatal teeth.

As far as histological characteristics are concerned, despite the normal basic structure of the natal teeth, early eruption is associated with hypo-mineralization of the enamel, which is usually described as dysplastic\textsuperscript{52}, reduced in thickness and covering only the two thirds of the crown\textsuperscript{6,42}, but has a normal ultrastructure\textsuperscript{111}. Complete absence of enamel is noted rarely\textsuperscript{3,78}. The enamel for the age of the child is normal but since the tooth erupts prematurely the matrix of the non-calcified enamel wears off in time and this is probably the reason why their crowns look small in size and appear yellow brown in colour\textsuperscript{52}. The dentino-enamel junction seems irregular\textsuperscript{42}.

Dentin and predentin appear normal coronally, but become irregular and with reduced number of dentinal tubules and large inter-globular spaces with abnormal cell inclusions\textsuperscript{14,16,42} cervically and bonelike apically resembling osteodentin, which is attributed to stimulation by movement of the teeth. It has been further suggested that the mobility may cause degeneration of Hertwig’s sheath, thus preventing root development and stabilization\textsuperscript{109}. Increased mobility causes histological changes in the cervical dentin and cementum\textsuperscript{4,62,109}. Cementum is either absent\textsuperscript{4} or, if present, shows variation in thickness covers the cervical third of the crown and is usually acellular\textsuperscript{42}. The pulp tissue has a normal appearance but the pulp cavity and the radicular canals are wider\textsuperscript{6,42,100}.

In neonatal teeth the differences from normal primary dentition are less pronounced due to their more mature state at the time of eruption\textsuperscript{6}. Root formation in natal and neonatal teeth is grossly deficient\textsuperscript{14}.

### Differential Diagnosis

Most of the teeth that occur in the oral cavity at birth or during the first days of life represent the early eruption of the normal primary deciduous dentition\textsuperscript{44,65}. The prevalence of supernumerary teeth has been suggested to range from 1-10\%\textsuperscript{17,37,123}. At this point, it is important
to mention the need of radiographic examination, in order to differentiate the premature eruption of a primary deciduous tooth from a supernumerary tooth\textsuperscript{15,25,65}. Moreover, radiographic verification reveals the root development of the tooth, adjacent structures and the existence of a relative germ in the primary dentition.

There are also 3 types of inclusion cysts that might be confused with natal teeth: Epstein’s pearls, Bohn’s nodules and dental lamina cysts. Epstein’s pearls are located along the mid-palatine raphe in the line of fusion of embryonic palatal processes. They are true cysts derived from residual ectodermal cells covering these processes. The cysts are lined by stratified squamous epithelium and the lumen contains keratin\textsuperscript{24}. Bohn’s nodules are usually multiple and located along the buccal and lingual aspects of the mandibular and maxillary ridges\textsuperscript{68}. They represent remnants of minor mucous salivary glands. They are true cysts comprised of stratified squamous epithelium lining a dense fibrous connective tissue wall that contains mucous acinar cells and well-formed ducts. The clinical appearance of Epstein’s pearls and Bohn’s nodules is similar. They are both small white-gray, raised nodules, 0.5-3 mm in diameter and no treatment is necessary\textsuperscript{24}.

The third type of cyst is dental lamina cyst which appears as single or multiple swellings on the maxillary or mandibular ridges. These cysts, also known as gingival cysts of the newborn, are lined by thin epithelium and show a lumen usually filled with desquamated keratin, occasionally containing inflammatory cells. It is believed that they are created by fragments of dental lamina that remain within the alveolar ridge mucosa after tooth formation. Most of them degenerate and involute or rupture into the oral cavity within two weeks to five months of postnatal life\textsuperscript{63}.

Furthermore, natal teeth should be discriminated from epulis and odontogenic hamartomas. Epulis are tumour-like growths of the gum that might be either sessile or pedunculated, and are reactive rather than neoplastic lesions\textsuperscript{68}. Odontogenic hamartomas are tumour-like lesions, without the growth characteristics of a neoplasm, and develop during the time dental structures remain capable of further development and maturation\textsuperscript{38}.

Complications

Problems that arise from the presence of natal and neonatal teeth include interruption in breastfeeding\textsuperscript{95} either by pain on suckling or by ulceration of the mother’s nipples, but the infant’s tongue usually overlies the lower incisors while nursing and any trauma will be to the infants tongue rather than mother’s breast\textsuperscript{106}, inflammation of the surrounding tissues, pain associated with mobility, which all may lead to refusal to nurse\textsuperscript{52}. Although no case is reported, there is usually a concern about aspiration or swallowing of the teeth due to excessive mobility or spontaneous exfoliation\textsuperscript{95}. Furthermore there can be teething symptoms just as with eruption of the primary teeth\textsuperscript{52} or even infantile diarrhea, drooling and malaise\textsuperscript{106,110}. The development of an abscess, probably due to the loss of attachment, has also been reported\textsuperscript{52,51}.

A complication that is common with natal teeth is ulceration of the tip or the ventral surface of the tongue, known as Riga-Fede disease. The ulceration occurs after repetitive tongue thrusting not only in newborns but also to elder infants with the eruption of the primary mandibular central incisors and in children with familiar dysanatomia\textsuperscript{107}. There has also been a report of prenatal ulceration of the tongue due to natal teeth\textsuperscript{58}. The lesion begins as an ulcerated area and with repeated trauma it may progress to an enlarged fibrous mass with the appearance of a granuloma. The pain occurring from the ulceration often results on dehydration, feeding difficulties and discomfort. It also may lead to bleeding and in a child with other medical problems a potential of infection is added to the concerns\textsuperscript{107}. Periapical abscess is possible because enamel breakdown may lead to carries\textsuperscript{52}. Another complication in children with cleft lip-palate is the potential interference in naro-alveolar moulding\textsuperscript{124}.

There have also been reported a case of reactive fibrous hyperplasia by a natal tooth\textsuperscript{106}, hypoplasia of primary and permanent teeth following osteitis due to infection by neonatal tooth\textsuperscript{55} and also microdontic teeth succedaneous to natal teeth, suggesting that there might be some unknown developmental influence common to the occurrence of natal teeth and abnormally small (mesiodistal dimension) permanent successors\textsuperscript{52} and in neonatal orthopaedics\textsuperscript{31}.

Management

The treatment plan for natal and neonatal teeth has many factors to consider. If the tooth is not interfering with the nutrient intake of the child and is otherwise asymptomatic no intervention should be made\textsuperscript{95}. Although it is difficult to determine initially whether root formation will occur in natal or neonatal teeth\textsuperscript{104} those teeth that are stable beyond 4 months have a good prognosis\textsuperscript{52}. The retention of a natal tooth, which is part of the normal primary dentition, is suggested because of possible space loss, although the opinions differ\textsuperscript{23,32,38}. If the tooth is supernumerary or has an excessive mobility, if it is poorly developed or is associated with soft tissue growth\textsuperscript{106} or if
it interferes with naso-alveolar moulding\textsuperscript{124} or presents an abscess, the treatment of choice is extraction\textsuperscript{32,51,52}. Before extraction, a dental radiograph should be obtained in order to inform the parents of possible complications and to get their consent. It is suggested to leave the tooth in the mouth as long as possible in order to decrease the possibility of removing permanent tooth buds with the natal tooth or risk defecating them\textsuperscript{16}. The possibility of hypoprophthrombinaemia should be taken into consideration as the commensal flora of the intestine might not have been established until the child is 10 days old. Since vitamin K is essential for the production of prothrombin in the liver it should be administered before extraction (0.5-1.0 mg, intramuscularly) if the routine postnatal injection is not given\textsuperscript{32}. Also, haemophilia should be investigated\textsuperscript{38}. The extraction is usually done under local anaesthesia but can also be done without anaesthesia depending on the gingival attachment, with the use of gauze as a pharyngeal guard\textsuperscript{52}. After the extraction, it is advised to curette the socket to prevent the cells of the dental papilla from continuing to develop and erupting as odontogenic remnants\textsuperscript{11,25,108}. If curettage is to become the routine treatment, then the injection of local anaesthetic to provide adequate anaesthesia would be required\textsuperscript{32}. Residual natal teeth have been reported with a risk of formatting without curettage about 9.1\textsuperscript{.}\textsuperscript{32} 86. Myxoid calcified hamartoma\textsuperscript{1}, pulp polyp as erupted remnants\textsuperscript{121}, pyogenic granuloma due to trauma during extraction\textsuperscript{84} and peripheral ossifying fibroma\textsuperscript{60}.

Riga-Fede disease is another complication of natal teeth and neonatal teeth but it’s not an indication for extraction\textsuperscript{79}. The treatment options include smoothing off the incisal edges of lower incisors with an abrasive instrument, modifying feeding behaviour or feeding devise, treatment of symptoms with oral triamcinolone acetonide in orabase applied on the lesion (Kenalog\textsuperscript{®} in Orabase\textsuperscript{®} Triamcinolone Acetonide Dental Paste USP, APOTHECON\textsuperscript{®} A Bristol-Myers Squibb Company), or placement of composite over the edges of the incisors\textsuperscript{89,107}. As many natal and neonatal teeth have hypo-mineralised enamel and are difficult to access and keep adequate moisture control, the bonding of the resin is questionable and presents the risk of swallowing or inhaling it. In cases of mild-to-moderate irritation to the tongue, such treatment may suffice. If the ulcerated area is large, however, even the reduced incisal edge may still contact and traumatize the tongue during suckling to such an extent that would delay healing\textsuperscript{86,118}. The fact that the lesion could reoccur should also be taken into consideration\textsuperscript{89,107}. If none of the more conservative measures is effective, the option is extraction of the tooth or even excision of the lesion\textsuperscript{107}.

Paediatric dentists should educate parents and medical community about the preferred treatment and should conduct any necessary extraction in order to prevent trauma. The child should be re-evaluated periodically to ensure oral health. Management of natal and neonatal teeth should consist of concern to avoid any complication, to make early diagnosis and provide adequate treatment.

**Conclusion**

Natal and neonatal teeth are rare conditions in infancy. Most commonly involved teeth are the mandibular central incisors. Despite the fact that the exact etiology is still unknown, superficial position of the tooth germ with association of hereditary factors is the most accepted possibility. Many complications may occur with the nursing problem most commonly reviewed. Treatment and periodic follow-up should be conducted by a paediatric dentist.

**References**


