REVIEW ARTICLE

Precocious Teeth: Review of Literature

Aanchal Jaiprakash Mohta¹, Anand Tavargeri²

ABSTRACT

This paper reports a recent update on natal teeth. It highlights the prevalence, various etiological factors and syndromes it can be associated with. The paper focuses on the clinical and histological features. The presence of natal teeth can lead to various complications ranging from affecting the health of the child to risk of aspiration of the teeth due to high mobility and sublingual ulceration. It is vital for a clinical practitioner to counsel the parents concerning the condition in addition to appropriate managing approaches required to establish well being of the child.

Keywords: Clinical features, etiology, histological features, management, natal teeth.

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INTRODUCTION

Teething is one of the most awaited moments in a parent’s life for their children. It is necessary to create awareness among the parents about the eruption of tooth which ordinarily occurs at 6 months of life whereas enlightening about premature eruption, its circumstances and also the various complications which stress on the need for a correct diagnosis as well as its management. One such complication is eruption of natal teeth.¹ (Figure 1) This paper emphasizes on a refreshed and recent knowledge on natal teeth.

Figure-1: Natal tooth in 5 days old baby

BRIEF HISTORY

The natal teeth had always been a subject of interest which was associated with various superstitions and beliefs. In 23 B.C Caius Plinius Secundus was of the opinion that males had a splendid future whereas it was a bad omen in girls. According to Titus Livius in 59 B.C it was a forecast of disastrous events. These superstitions lasted for a long time in countries like Poland, India and many African tribes where the children were killed soon after birth as they were considered to bring bad luck to the family. The Chinese believed the presence of teeth a bad omen as they thought the natal teeth would bite one of the parents leading to their death. The English believed these babies would grow up to become famous warriors. In France and Italy people regarded they would grow up to conquer the world. Historical figures such as Zoroaster, Hannibal, Luis XIV, Mazarin, Richelieu, Mirabeau, Richard III, and Napoleon may have been blessed with bright futures credited to natal teeth.³ Even though terms of “natal” and “neonatal” teeth described by Massler and Savara are accepted worldwide, they are broadly characterized as teeth that have erupted at birth or shortly after that. Dentitia praecox, dens connatalis, congenital teeth, fetal teeth, infancy teeth, predeciduous teeth, and precocious dentition are some of the terminologies used previously but discarded due to inaccuracy in describing this condition.⁴

CLASSIFICATION

Massler and Savara gave the definition taking only the time of eruption as reference; natal teeth are those observable in the oral cavity at birth and neonatal teeth
are those that erupt during the first 30 days of life and not to the anatomical, morphological and structural characteristics.²

Spouge and Feasby⁵,⁶ recognized the need to organize them into:

- Mature- when they are fully developed in shape and comparable in morphology to the primary teeth with good prognosis.
- Immature- when their structure and development are incomplete and holds a poor prognosis.

On the basis of literature data, Hebling classified natal teeth into 4 categories:³

1) Shell-shaped crown poorly fixed to the alveolus by gingival tissue and absence of a root.
2) Solid crown poorly fixed to the alveolus by gingival tissue and little or no root.
3) Eruption of the gingival margin of crown through gingival tissue.
4) Edema of gingival tissue with an unerupted but palpable tooth.

Natal teeth are classified as (1) and (2), if the degree of mobility is more than 2 mm; such teeth usually need extraction.⁷

PREVALENCE

Authors have reviewed the prevalence of natal and neonatal teeth as 1:700 to 1:30,000⁶,⁸

Etiology:
The etiology of natal and neonatal teeth still remains a mystery although various authors have presented their views and theories regarding the same.

Etiology is multifactorial:

- Infection- Congenital syphilis; where eruption can be early or delayed.⁹,²⁰
- Malnutrition- Dietary deficiencies viz. hypovitaminosis.²⁰
- Febrile states- Fever, rashes during pregnancy - tend to advance their eruption.⁹
- Endocrinial disturbances and Hormonal stimulation- Tooth eruption is influenced by pituitary growth hormone, thyroid hormone and parathyroid hormone-related protein; all are required for normal tooth eruption.²¹ Venkatesh C and Adhisivam B reported a nonsyndromic case of congenital hypothyroidism.²⁶
- Hal’s stated that “occurrence of natal and neonatal teeth is due to an abnormally superficial position of the tooth germs which, in turn, is due to a hereditary factor.”²²
- Osteoblastic activity within tooth germ as reported by Jasmin and Clergeau-Guérithault is responsible for eruption of natal and neonatal teeth.²

- Pregnant mothers accidentally exposed to PCBs (polychlorinated biphenyl) and PCDD/ Fs (PCDF-polychlorinated dibenzofuran, PCDD- polychlorinated dibenzo-p-dioxin) have increased prevalence for natal teeth.²³ They have been associated with skull abnormalities representing irregular calcification, dystrophic finger nails, hyper pigmentation. Natal teeth appear as the bone of the mandible is easily penetrated.²⁴
- Natal teeth have also been associated with various syndromes⁸ (Table 1).

Fauconnier and Gerardy reported “early eruption” due to alteration in endocrine system featuring normal eruptive pathway with intact gingiva and “premature eruption” as pathologic phenomena like trauma in delivery leading to ulceration and source of infection resulting in exfoliation of a root less tooth in a short time later defined as “expulsive Capdepont follicle” featuring rapid tooth eruption with extreme mobility, turgidity and inflamed gingiva.³

CLINICAL FEATURES

- Clinically, these teeth can be hypoplastic, conical or normal size and shape, rarely with opaque yellow- brownish color depending on the degree of maturity. Mobility is due to degeneration of hertwigs root sheath leading to incomplete development of roots and loose attachment to the alveolar ridge. (Figure 2) Most commonly occurring are mandibular central incisors (85%), maxillary incisors (11%), mandibular canines and molars (3%) and maxillary canines and molars (1%).²

Figure-2: Ground sectioning in 40X magnification showing:
A) Enamel with fish scale pattern; B) Absence of normal scalloping of dentin

HISTOLOGICAL FEATURES

- Hawkins first described the histological features of natal teeth. Commonly natal teeth reveal dysplas-
tic or hypomineralized enamel due to premature exposure to the oral cavity explained by Friend et al. Soni et al described presence of irregular dentin and osteodentin cervically and coronally interglobular dentin with high vascularity of pulp due to dilatation of the blood vessels often increased in number with inflammatory cells. The decalcified section displayed the pulpal soft tissue with the peripheral odontoblastic cell layer and absence of Weil’s and cell rich zone. The pulp core is represented by the loosely arranged fibrocytes with collagen, blood capillaries, defense cells like plasma cells and foamy histiocytes in the pulpal stroma.

- The enamel thickness for natal teeth (300mm) differs from normal primary teeth which is between 1000 and 1200mm. The enamel structure can display a cross striation (fish- scale pattern) in a disorganized pattern (figure 2), whereas the dentin revealed the primary curvatures of the dentinal tubules, enamel spindles at the cusp tip, interglobular

<table>
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<tr>
<th>Syndromes and developmental disturbances</th>
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<tr>
<td>Ellis van creveld syndrome</td>
<td>Himelhoch(1988); Kurian et al(2007)</td>
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<tr>
<td>Patent ductus arteriosus and intestinal pseudo-obstruction</td>
<td>Harris et al (1976)</td>
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<td>Opitz (G/BBB) syndrome</td>
<td>Shaw et al (2006)</td>
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<td>Van der Woude syndrome</td>
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<td>Steatocystoma multiplex</td>
<td>King and Lee (1987)</td>
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<td>Pfeiffer syndrome type 3</td>
<td>Alvarez et al (1993)</td>
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<td>Hyper IgE syndrome</td>
<td>Roshan et al (2009)</td>
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<td>Rubinstein-Taybi syndrome</td>
<td>Hennenkam and Van Doorne (1990)</td>
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<td>Bifid tongue and profound sensorineural hearing loss</td>
<td>Darwish, Sastry and Ruprecht (1987)</td>
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<td>Cyclopia</td>
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<tr>
<td>Transient Pseudohypoparathyroidism</td>
<td>Koklu and Kurtoglu (2007)</td>
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<tr>
<td>Short rib-polydactyly syndrome type II</td>
<td>Stromme Koppang, Boman and Hoel (1983)</td>
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<td>(Saldino-Noonan syndrome )</td>
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<td>Soto’s syndrome</td>
<td>Callanan, Anand and Sheehy (2009)</td>
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<td>Adrenogenital syndrome</td>
<td>Leung (1989)</td>
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<td>Epidermolysis bullosa simplex</td>
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<td>Odonto-Tricho-Ungual-Digital-Palmar Syndrome</td>
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<td>Goltz syndrome</td>
<td>Dias et al (2010)</td>
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<td>Clouston syndrome</td>
<td>Reynolds, Gold and Scriver (1971)</td>
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Table-1: Natal teeth and associated syndromes
dentin at the circumpulpal dentin and dead tracts. The dentinoenamel junction doesn’t show a normal scalloped pattern with a gradual decrease in the number of dentinal tubules from crown to the cervical region as it shows an atypical arrangement of dentinal tubules showing spaces and enclosed cells with calciospherites and predentin of varying thickness. Deciduous teeth and natal teeth show similar unscalloped dentinoenamel junction.10

Radiological findings
The RVG revealed disorganized enamel, dentin and pulp chamber and absence of root. (Figure 3).

Figure-3: Digital Radiovisiography of natal teeth

COMPLICATIONS

- Difficulty in feeding due to ulceration leading to nutritional deficiencies.2
- Riga-Fede disease or syndrome- first identified by Riga an Italian physician and described by Fede who performed the histological studies of the lesion, is a reactive mucosal disease caused due to the continuous rubbing of the anterior lower teeth with the ventral surface of the tongue. The expression “traumatic ulcerative granuloma with stromal eosinophilia” (TUGSE) was coined by Elzay, referring to a chronic but self-limiting reactive ulcer of the oral mucosa.11 It is also recognized as “Riga’s papilloma, Riga’s boil, Riga’s apthae, sublingual ulcer, sublingual granuloma” and also known as Cardarelli’s apthae, Cardarelli’s disease or Cardarelli’s syndrome.13 Domingues–Cruz et al14 classified Riga-Fede as:
  - Precocious Riga Fede disease- seen in first 6 months of life defined as those occurrences associated with (neo)natal teeth, unrelated with neurologic disorders.
  - Late Riga Fede disease- seen after 6-8 months of life referring to those instances which typically start with the first dentition, usually the lower incisors.
  - Zaenglein et al stated clinically that these lesions are erythematous with a centrally removable, yellow, fibrinopurulent membrane depicting a rolled white hyperkeratotic border immediately adjacent to the ulceration occurring on the buccal mucosa, palate, lips and commonly on ventral surface of tongue. Histological examination reported a mixed inflammatory reaction under the ulcerated surface, T cells, large mononuclear cells and eosinophils suggesting as a subtype of eosinophilic ulcer.15 Jariwala et al suggested biopsy for lesions that remain for 2 weeks after removal of the cause.16
  - Possibility of aspiration due to extreme mobility.2
  - Whether the tooth is supernumerary or deciduous dentition can be clarified by viewing a radiograph of the same region.2
  - Hamper with breast feeding and causing injury to the breast. But certain authors like Hals, Zhu and King and Walter debated this possibility as the tongue lies in between the nipple and the natal tooth.2
  - Ulceration below the tongue due to suckling was first reported by Kinirons.2
  - Residual natal tooth - Tsuborne et al introduced the term for the calcified structure earlier referred as “tumourlike masses, “toothlike structures,” “irregular mass (es) of dentin,” “odontogenic remnants” and “pearls of hard tissue.” Southam hypothesized that after losing the coronal tooth structure of natal and neonatal teeth, the exposed surface of the papilla was likely to become infected and necrotic together with the odontoblasts and remnants of the HERS.17 After the natal tooth is extracted it should be made sure to remove the HERS and dental papilla by gentle curettage as such remnant structures can continue to develop if left in situ.13

MANAGEMENT

The essential objective should be to eliminate the source of trauma to initiate healing and to avoid any above mentioned complications as far as possible. Slayton18 and Pujar P19 have given various treatment options as:

- Smoothening of incisal edges of lower incisors by using a finishing bur or a sandpaper disk.
- Modify feeding behavior or feeding device so as to reduce trauma to the tongue. Parents can be advised to enlarge the hole of the nipple of the bottle or using a sippy cup which requires reduced vigorous sucking or by feeding using a spoon depending
on the age of the infant.

- Treat symptoms— use Kenalog in Orabase to relieve the painful symptoms and distressing of the child.
- Place an increment of composite over the incisal edges by bonding in minimal increments.
- Do nothing.
- Extract lower incisors if symptoms don’t subside conservatively.
- Apply liquid nitrogen to the lesion.
- Use stomahesive wafers.

Also use of breast pumps or a storing device can be used to feed the child if the mother insists on breast feeding.

CONCLUSION

As these conditions are unusual and their existence can cause various complications these children should be supervised by clinical professionals by attaining definite history, clinical and radiographical examination. Management involves diagnosis of the condition followed by immediate measures to restore the functions without affecting the general health of the child.

REFERENCES