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Puberty Induced Gingival Enlargement

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Editorial

Gingival hyperplasia is an atypical increase in the number of healthy cells in normal arrangement in a tissue. Gingival hyperplasia can be the result of unusual tissue response to the inflammation caused by local irritants, such as plaque and calculus; systemic disturbances, such as hormonal changes; or medications specifically cyclosporine, nifedipine and phenytoin.

The homeostasis of the periodontium involves complex multifactorial relationships, in which the endocrine system plays an important role [1]. Hormones are specific regulatory molecules that modulate function reproduction, growth and development and the maintenance of internal environments as well as energy production, utilization and storage [1]. As well as being the regulators of reproductive functions, sex steroid hormones have potent effects on the nervous and cardiovascular system, and on major determinants of the development and integrity of the skeleton and oral cavity including periodontal tissues [2]. Under the broad category of dental plaque induced gingival diseases that are modified by systemic factors, those associated with the endocrine system are classified as puberty, menstrual cycle and pregnancy associated gingivitis [3]. Researchers have shown that changes in periodontal conditions may be associated with variations in sex hormones [4].

Enlargement of the gingival can sometimes occur during puberty. It occurs in both male and female adolescents and appears in areas of plaque accumulation. The size of the gingival enlargement greatly exceeds that usually seen in association with comparable local factors. It is marginal and interdentally and is characterized by prominent bulbous interproximal papilla. Often, only the facial gingival are involved and the lingual surfaces are relatively unaltered, the mechanical action of the tongue and the excursion of food present a heavy accumulation of local irritants on the lingual surface.

Puberty marks the initiation of changes from maturation into adulthood [5]. It is associated with a major increase in the secretions of the sex steroid hormones: testosterone in males and estradiol in females. Several cross-sectional and longitudinal studies have demonstrated an increase in gingival inflammation without accompanying an increase in plaque levels during puberty. Increased gingival inflammation was positively correlated with an increase in serum estradiol and progesterone, and was not accompanied by a significant change in the mean plaque index [1].

Gingival tissues and the subgingival microflora respond with a variety of changes to the increasing hormone level at the onset of puberty. Microbial changes have been reported during puberty and can be attributed to changes in the microenvironment seen in the gingival tissue response to the sex hormones, as well as the ability of some species of bacteria to capitalize on the higher concentration of hormones present [6]. Clinically, during puberty there may be a nodular hyper plastic reaction of the gingival in areas where food debris, material alba, plaque, and calculus are deposited. The inflamed tissues are deep red and may be lobulated, with ballooning distortion of the interdentally papillae. Bleeding may occur when patients masticate or brush their teeth. Histologically, the appearance is consistent with inflammatory hyperplasia.

Gingival enlargement during puberty has all the clinical features associated with chronic inflammatory gingival disease. It is the degree of enlargement and the tendency to develop massive recurrence in the presence of relatively scant plaque deposits that distinguish pubertal gingival enlargement from uncomplicated chronic inflammatory gingival enlargement.

At puberty, the production of sex hormones (estrogen and progesterone) increases to a level that remains relatively constant throughout the normal female reproductive phase. A number of studies [7] have shown that increased sex-hormone levels correlate with an increased prevalence of gingivitis. Gingival tissues and the subgingival microflora respond with a variety of changes to the increasing hormone level at the onset of puberty. Microbial changes reported during puberty can be attributed to changes in the microenvironment seen in the gingival tissue response to the sex hormones, as well as the tendency of specific bacteria to proliferate due to the higher concentration of hormones present [7]. This is seen with Prevotella intermedia (*P. intermedia*), a gram-negative anaerobe that can substitute estrogen and progesterone for vitamin K, an essential growth factor [8].

In addition, Capnocytophaga species, another gram-negative bacterium, increases in incidence as well as in proportion. Both organisms are believed responsible for the increased bleeding tendency noted in puberty. A longitudinal study [9] of 127 children

11 to 17 years of age showed a high initial prevalence of gingival enlargement that tended to decline with age. When the mean number of inflamed gingival sites per child was determined and correlated with the time at which the maximum number of inflamed sites was observed and with the oral hygiene index at that time, a pubertal peak in gingival inflammation unrelated to oral hygiene factors clearly occurred. Gingival enlargement in puberty is treated by performing scaling and curettage, removing all sources of irritation and controlling plaque. Surgical removal may be required in severe cases.

Conclusion

It is clear that endogenous sex steroid hormones play significant roles in modulating the periodontal tissue responses and may alter periodontal tissue responses to microbial plaque, and thus directly may contribute to periodontal disease. They can influence the periodontium at different life times such as puberty, menstruation, pregnancy, menopause and postmenopause. Thus a better understanding of the periodontal changes to varying hormonal levels throughout life can help the dental practitioner in the diagnosis and treatment.

References

1. Mariotti A (1994) Sex steroid hormones and cell dynamics in the periodontium. Crit Rev Oral Biol Med 5(1): 27-53.
2. Lorenzo J (2003) A new hypothesis for how sex steroid hormones regulate bone mass. J Clin Invest 111(11): 1641-1643.
3. Armitage GC (1999) Development of a classification system for periodontal diseases and conditions. Ann Periodontol 4(1): 1-6.
4. Plancak D, Vizner B, Jorgic-Srdjak K (1998) Endocrinological status of patients with periodontal disease. Coll Antropol 22: 51-55.
5. Ferris GM (1993) Alteration in female sex hormones their effect on oral tissues and dental treatment. Compendium 14(12): 1558-1570.
6. Amar S, Chung KM (1994) Influence of hormonal variation on the periodontium in women. Periodontol 2000: 79-87.
7. Bimstein E, Matsson L (1999) Growth and development considerations in the diagnosis of gingivitis and periodontitis in children. Pediatr Dent 21(3): 186-191.
8. Nakagawa S, Fuji H, Machida Y (1994) A longitudinal study from prepuberty to puberty of gingivitis. Correlation between the occurrence of Prevotella intermedia and sex hormones. J Clin Periodontol 21(10): 658-669.
9. Sutcliffe P (1972) A longitudinal study of gingivitis and puberty. J Periodontol Res 7(1): 52-58.