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Review article

Dental Fluorosis: An update

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ABSTRACT

Dental fluorosis is a major problem identified all over the world. High levels or prolonged ingestion of fluoride from various sources, primarily drinking water, certain high fluoride containing foods and beverages etc. can lead to fluorosis of teeth and skeletal tissues if ingestion occurs till 8 yrs of age. Dental fluorosis is not only a major esthetic concern but also poses a danger of damage to the enamel causing its severe loss as well. It becomes the moral responsibility of the dental professional to identify the causes of dental fluorosis and provide aesthetic and restorative treatment. Apart from this the dental professional should also stress upon the recommendations to prevent any further loss in individuals affected and minimize exposure to fluorides in those not affected especially the ones in the susceptibility age range. This review aims at consolidating various details on the development, clinical presentation, risk factors and ultra-structural features of dental fluorosis. The mechanisms of its development, various indices used for its clinical evaluation and assessment as well as recommendations for use of fluorides in the community have also been enumerated.

Key words: dental fluorosis, fluoride, hypoplasia, enamel

INTRODUCTION

Developmental defects of enamel are visible deviations from the normal translucent appearance of tooth enamel resulting from enamel organ dysfunction. Hypoplasia is a defect involving the surface of the enamel and associated with a reduced thickness of enamel which may be translucent or opaque. It is usually seen in the form of pits, grooves, or larger areas of missing enamel. Environmental and genetic factors that interfere with tooth formation are thought to be responsible for enamel hypoplasia such as trauma to the teeth and jaws during early periods of life, intubation of premature infants, infections during pregnancy or infancy, poor pre-natal and post-natal nutrition, hypoxia, exposure to toxic chemicals and a variety of hereditary disorders [1]. Dental fluorosis is characterized by a hypoplasia of the enamel resulting from long term ingestion or exposure to high levels of fluoride during the phase of tooth mineralization. It can be described as a diffuse

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symmetric hypo-mineralization disorder of ameloblasts. The results of prolonged exposure to such high levels of fluoride in humans are first recognizable in children after the age of six years. Precise estimates of the most severe effects of fluoride can be obtained only when pre-molars and second molars have erupted, because the later in life the teeth undergo mineralization, the more severely they are affected [2].

Clinical Presentation

Clinically, enamel fluorosis is defined by the presence of characteristic enamel opacities. The greater the dose of fluoride ingested during the period of mineralization, the more severe are the clinical manifestations. Instead of being a normal creamy white translucent color, fluorosed enamel is porous and opaque and may exhibit objectionable secondary staining. The teeth can resemble ghastly white chalk colour and light refractivity is greatly reduced because the enamel's prism structure is defective. Cloudy striated enamel, white specks or blotches; 'snow capping', yellowish brown spots or brown pits on teeth are all characteristics of dental fluorosis.

In its mildest forms, enamel fluorosis appears as a few faint white flecks scattered across the dentition that would generally go unnoticed by all except a trained examiner. With increasing severity the areas of white flecking or snow flaking become more pronounced and cover an increasingly greater proportions of the enamel thus becoming more noticeable. *(Figure 1,2)* The fluorotic lesions are not just confined to enamel but can also be seen extending into dentine microscopically. In its most severe forms, enamel fluorosis is characterized by dark brown staining and pitting of enamel surface with large enamel defects occurring in some cases *(Figure 3a and 3b)*. In its most severe forms, The fluorosed enamel in these cases is structurally weak (brittle) and prone to erosion and breakage especially when drilled and filled. Because of the bone and tooth seeking quality of fluorine, it also deposits bone or bone like materials externally on the roots of the teeth and internally in the pulp chamber causing

Figure 1. Clinical photograph depicting mild fluorosis

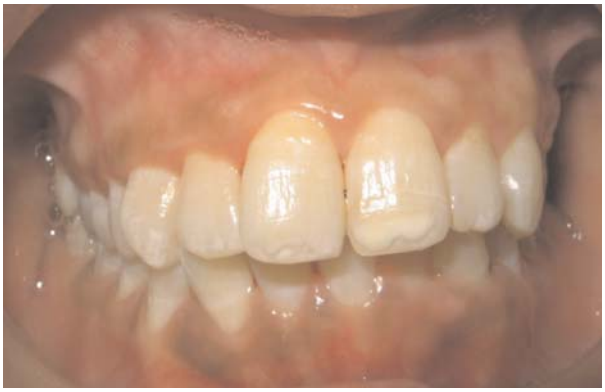


Figure 2. Clinical photograph depicting moderate fluorosis

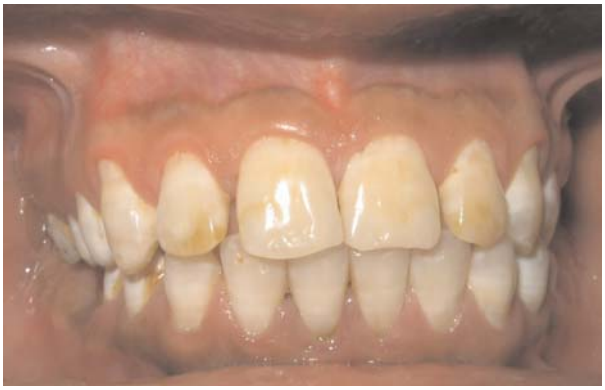
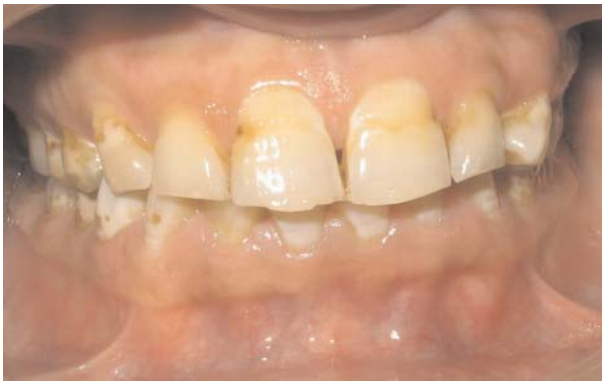
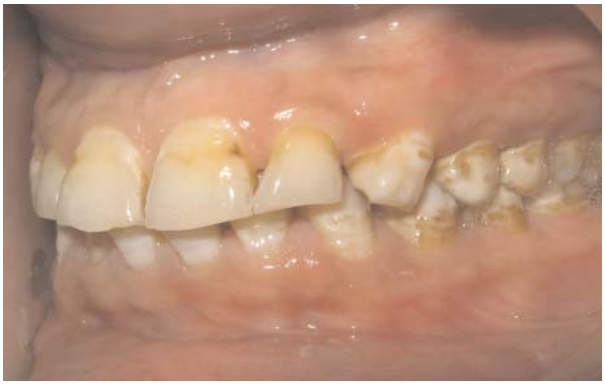


Figure 3a and 3b. Clinical photograph depicting moderately severe fluorosis





narrowing and interfering with tooth nutrition [3,4].

Role of fluoride ion

Fluoride is an essential nutrient and has been recognized to be physiologically essential for the normal growth and development of human beings. A level of 0.05–0.07 mg/kg body weight is often thought of as "optimal"; however, lower levels of intake have been associated with fluorosis. The optimal level is virtually impossible to calculate because of variations in fluoride levels in all sorts of foods and beverages. It cannot be assumed that because a person resides in a community with non-fluoridated water, he or she is receiving low levels of fluoride. People can get fluoride from water while at locations other than home (e.g., child care setups, school, workplace etc.) or from drinking substantial amounts of soft drinks or juices, which often have fluoride levels close to the optimal range for drinking. Based on an extensive research, the U.S. public health service in 1986 established that the optimum concentration of fluoride in water for human consumption to be in the range of 0.7-1.2 ppm [4]. This range effectively reduces tooth decay while minimizing the occurrence of dental fluorosis. In India, the fluoride levels in ground water vary substantially in different regions. High concentrations of fluoride (>1.5 mg/l) have been reported in some of the areas of states of Haryana, Delhi, Rajasthan, Karnataka, Uttar Pradesh, Maharashtra, Gujarat, Madhya Pradesh, Andhra Pradesh, Tamil Nadu, Kerala, J& K, Punjab, Orissa, Himachal Pradesh and Bihar [5].

Fluoride is usually available for use in two forms: topical and systemic. Topical fluorides strengthen teeth already present in the mouth by being incorporated on to the surface and preventing tooth decay or dental caries thus, providing local protection to the tooth surface. Topical fluorides include: toothpastes, mouth rinses, and professionally applied gels and rinses. Systemic fluorides are those ingested into the body and become incorporated into forming tooth structures. They provide a longer lasting as well as topical protection due to their levels in the saliva which serves as a reservoir of fluoride ions [6]. Fluoride also gets incorporated into the dental plaque and facilitates further remineralization [7]. Sources of systemic fluorides include drinking water, dietary fluoride, supplements in the form of tablets, drops or lozenges and fluoride present in food and beverages. Fish such as sardines may contribute to higher fluoride intake if bones are ingested. Brewed teas also have 1-6ppm of fluoride in them. Turmeric used in Indian cooking is also a rich source of fluoride.

The re-mineralization effect of fluoride is of prime importance as fluoride ions in the enamel surface result in a fortified enamel that is not only more resistant to decay but an enamel that can repair or remineralize early dental decay caused by acids from decay causing bacteria.

Metabolism of Fluoride

On systemic ingestion of fluoride, primary absorption occurs from the stomach and small intestine into the blood stream. This causes a short term increase of fluoride levels in the blood which increase quickly and reach a peak concentration within 20-60 min., the concentration declines rapidly usually within 3-6 hrs due to uptake of fluoride by hard tissues of the body and its efficient removal by the kidneys. Approximately 99% of the fluoride present in the body is associated with hard tissues. The amount of fluoride taken up by bone and retained in the body is inversely related to age with more fluoride being retained in young bones [8].

Risk factors for the Development of Dental Fluorosis

The reason for the increase in fluorosis in fluoridated and non-fluoridated communities was investigated in several studies, and was in fact found to correlate with the unintentional ingestion of fluoride from sources other than drinking water. Fluoride sources included early use of fluoridated toothpastes [9], use of fluoride supplements 10,11], and prolonged formula intake in infants [9].

Fluorosis is a toxic manifestation of chronic (low-dose, long term) fluoride intake. To prevent fluorosis from occurring in the most prominent and or most susceptible teeth, the most critical time to avoid fluoride exposure is the first 3-6 years of a child's life. The timing of the exposure to fluoride is also important relative to its use as a biomarker. There are various metabolic factors that also influence the formation of dental fluorosis. Body weight in relation to total fluoride intake is an important factor. Fluoride exposure would be most accurately defined as dose per body weight. The activity level of a child could also clearly affect the total amount of water ingested, resulting in an increased ingestion of fluoride in drinking water. Nutritional factors, such as the amount of protein in the diet, may be related to the amount of fluoride available to mineralizing tissues such as enamel and bone, with the subsequent formation of enamel fluorosis. The high-protein diet also results in an increased glomerular filtration rate, resulting in a loss of fluoride greater than the additional amount which was absorbed under acidic conditions. Other factors which may affect the plasma levels of fluoride, and hence the degree of enamel fluorosis, include the rate of skeletal growth and periods of bone remodeling. Fluoride is rapidly absorbed from the plasma by the forming young bones of the growing skeleton [12].

Ultra-structural morphological & biochemical analysis of development of dental fluorosis

The earliest manifestations of dental fluorosis appear as an increased porosity along the striae of Retzius. This enamel porosity, when observed in microradiographs is mainly in the subsurface enamel, and the extent and degree of hypo-mineralization increases with increasing fluoride exposure. Thus, in the most severe forms the hypo-mineralized lesion extends throughout the enamel to the enamel-dentin junction in the cervical third of the crown, whereas in the occlusal two-thirds of the teeth, the band of hypo-mineralization extends about halfway through the enamel. The most severe degree of hypo-mineralization or porosity is located immediately deep to the surface layer, which is relatively well-mineralized at the time of eruption. It is generally accepted that fluorotic changes of the enamel can only occur during the development of the enamel. Once the enamel has formed, it is no longer at risk for dental fluorosis. This period of enamel formation lies between birth and approximately the eighth year of life for the permanent dentition (with the exception of the third molars). The development of enamel can be divided into three principal stages: the secretory phase, during which an organic matrix is formed; a maturation phase, in which most of the mineralization of the enamel occurs; and a transitional phase, which is between the other two phases [2, 13].

Processes during amelogenesis which may be affected by fluoride [2]

Enamel Matrix Secretion

- Impaired cell function
- Quantity of matrix proteins
- Composition of matrix proteins
- Proteolytic enzymes
- Nucleation and/or crystal growth
- Transport of calcium to sites of mineralization

Enamel Maturation

- Impaired cell function
- Reduced length of zone of ameloblast
- Modulation reduced number of cell modulations
- Impaired removal of matrix proteins
- Proteolytic enzymes
- Crystal growth
- Transport of calcium to sites of mineralization

Basic Processes Involved in Bio-mineralization

- Availability of Ca/P
- Ca/P precipitation/crystal growth
- Calcium metabolism

Effect of fluoride on apatite nucleation and crystal growth

Numerous studies have been conducted to explore the effects of fluoride on apatite nucleation and crystal growth [14, 15]. Depending on the experimental conditions, fluoride has been shown to have the following effects:

(a) Increase the driving force toward apatite nucleation and growth

(b) Facilitate conversion of Dicalcium Phosphate Dihydrate (DCDP) or Octacalcium Phosphate (OCP) to Hydroxylapatite

(c) improves the crystallinity of apatite

(d) increases the size of apatite crystals; and

(e) increase the Ca/P ratio approximating stoichiometric apatite. Increasing evidence has been presented in support of the hypothesis that excessive levels of fluoride in the extracellular fluids result in delay of the cleavage and removal of amelogenin matrix proteins during enamel maturation. Fluoride may complex ionic Ca2+ in the matrix fluid and inhibits an enzyme involved in hydrolysis of amelogenins. It has been shown that fluoride concentrations in developing enamel peak in the early-maturation stage of enamel development, at the time when amelogeninase activity would be expected to be most intense [16].

These studies make a strong case for the hypothesis that excessive concentrations of fluoride in developing enamel partially inhibit the proteinases that cleave the larger-molecular-weight amelogenins, resulting in retention of amelogenins and effects on apatite growth. It appears that the effect is temporary, and that amelogenin removal is complete, or nearly so, by the time the enamel erupts. Nevertheless, temporary inhibition at the critical stage could have significant effects on the structural characteristics of fully formed enamel.

Post-eruptive changes in fluorosed human enamel

Once erupted into the oral cavity, fluorosed teeth are subject to chemical and physical changes. The more hypo-mineralized the tooth is at time of eruption, the more likely it is to develop post-eruptive changes. The milder forms are subject to surface attrition. In particular, occlusal surfaces are rapidly worn, often to such an extent that the hypo—mineralized porous layer is abraded away. In more severe cases, the hypo-mineralization is so extensive that the outermost well-mineralized surface layer is rather brittle, and chewing forces may result in formation of surface enamel defects. In their mildest form, these defects appear as single pits along the perikymata, or the surface enamel may get chipped away, corresponding to the incisal edges or cusp tips. When observed under the scanning electron microscope, these pits appear as punched-out areas of the enamel, with the walls of the pits composed of enamel rods. However, the walls of the pits may get gradually rounded off due to abrasion. Depending on the severity of fluorosis at the time of eruption, the surface damage may occur immediately following exposure to the oral environment and the damage increases in the subsequent months and years which may influence severity scores with age. In the most severe cases, extensive abrasion of the very porous and soft exposed lesions occurs. Exposed porous fluorotic lesions may take up stain from the oral environment, which explains the brownish-dark discoloration often seen in severely fluorotic teeth. However, uptake of stain may also be seen in less advanced forms of dental fluorosis with no apparent surface damage. In conclusion, the effect of fluoride on human dental enamel results in hypo-mineralization, the severity of which is a direct reflection of the degree of past fluoride exposure having happened during the period of enamel matrix laying and maturation stages [2].

Clinical Assessment of Dental Fluorosis

The changes in enamel induced by fluoride were first described by Black and McKay (1916) [17] as mottled enamel, who suggested that the cause of mottled enamel was related to exposure to trace elements. In 1931, several independent laboratories compared the amounts of trace elements in water from areas with high levels of enamel mottling, and determined that the water had a relatively high concentration of fluoride [18,19]. Following these laboratory investigations, Dean (1934) [20] developed an index of dental fluorosis and used this index to relate the severity of dental fluorosis to the level of fluoride exposures of the individual especially due to drinking water. This was followed by the development of a modification of this index and further the Community Fluorosis index (CFI) was introduced based upon this system (Dean 1942). Subsequently, the Thylstrup and Fejerskov Index (1978) and its modification (Fejerskov et al 1988), the Fluorosis Risk Index, described by Pendrys (1990) came into use [21,22,23]. Amongst all these, Dean's index has the most widespread use over an extended period, because it serves as a standard of comparison for all subsequent indices. The acceptance of these fluorosis indices rests in large measure on the ability of an examiner to distinguish fluoride induced changes in the enamel from those that are not fluoride-induced. Till date there existed no continuous scale for measuring the severity of dental fluorosis. All these indices that have been mentioned used ordinal scales and therefore the scores should be considered only arbitrary points along a continuum of change. A preferred method would be to express the severity of dental fluorosis in a continuous scale by showing the position of different observations relative to each other and the extent to which one observation differs from another. These properties are extremely important when the correlation between two factors is studied, as well as when evaluating the cause-and-effect relationship between two variables. Thus, the importance of creating a continuous scale for assessing the severity of dental fluorosis was evident. Hence a visual analogue scale was devised for which photographs were taken of patient's anterior teeth in areas of endemic dental fluorosis, as well as from dental textbooks [24]. The examiners were asked to grade the amount of discoloration and malformation in the photographs on a 100-mm VAS. The main advantages of the VAS for dental fluorosis over the ordinal scales for dental fluorosis (such as TFI, DI, and Tooth Surface Index of Fluorosis) are the continuity of the scale, its simplicity and its precision. The continuity of the scale favors its use in correlations between DF severity and other parameters, such as fluoride concentration and tooth properties (for example, tooth micro hardness, hydroxyl-apatite crystal size, ultrasound velocity), and it allows the use of more robust parametric statistical tests. Analysis of the data showed that the VAS for DF is valid, has excellent reliability and is ready to be used on a large-scale basis in clinical trials.

Recommendations on the use of fluorides for prevention of Dental Caries

In view of the various ill effects of higher doses of fluoride in drinking water on the dental tissues especially in the critical time period of tooth development, sends a caution to analyze and assess before implementing or prescribing fluoride supplementation. The center for disease control (CDC), USA has laid down certain guidelines for public health and clinical practice to be followed which must be strictly adhered to [2]. These include:

- Continue and extend fluoridation of community drinking water at 1ppm level.
- Counsel parents and caregivers regarding use of fluoride toothpaste by young children, especially those < 6 years of age and advocate on use of fluoridated toothpaste for children and adults above the age of 6 years.
- Target fluoride mouth-rinsing to persons at high risk for dental caries.
- Judiciously prescribe fluoride supplements as these when administered despite reducing dental caries have been found to be significantly associated with mild to moderate degree of fluorosis [25].

• Apply or use of the high-concentration fluoride products such as fluoride varnish etc. to persons at high risk for dental caries.

There exists an imminent need to carry out further research on assessment of levels of fluoride in the community drinking water, conduct metabolic studies of fluoride and study the effect of fluoride supplements on causation of fluorosis and prevention of dental caries and guide the public at large accordingly.

CONCLUSION

This article aims at updating the details on dental fluorosis, highlighting the importance of identifying and understanding such cases in the routine dental clinic, and make efforts to guide the patients regarding its prevention as well as remedial measures to take care of the immediate esthetic and functional concerns.

Conflicting Interest: Nil

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