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Fluoride Toxicity: A Review on Dental Fluorosis and Its Prevalence in India

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Abstract:*Fluoride, when added to community water in optimum quantity, is an important public health measure. At concentrations of 0.7 – 1.2 ppm, fluoride significantly reduces the prevalence of dental caries. It does so by promoting remineralization of lesions, reducing the solubility of enamel by forming fluorapatite crystals. Additionally, it may have a direct effect on the enzymes produced by cariogenic bacteria. However, fluoride is often regarded as a 'double edged sword'. Chronic ingestion of significantly high levels of fluoride (greater than 3 ppm), most commonly through community water supplies, can cause dental fluorosis and rarely skeletal fluorosis. Dental fluorosis occurs in developing teeth due to the exposure of high concentration of fluoride during stages of tooth development, particularly the dentinogenesis phase. The histopathological appearance of such teeth shows areas of enamel porosity and hypomineralization, According to its severity, dental fluorosis can be classified as mild, moderate and severe. Dental fluorosis is highly prevalent in 24 countries of the world, including India. As of 2017, 62 million Indians are affected by dental fluorosis. The best treatment for fluorosis is its timely prevention. Teeth with mild to moderate dental fluorosis can be treated by several esthetic procedures like bleaching, microabrasion etc. Severe dental fluorosis is further treated by esthetic restorative procedures like veneers or crowns. This paper focuses on providing a comprehensive knowledge of the diagnosis, histopathological appearance, treatment and prevention of dental fluorosis. Additionally, it aims to analyse the prevalence of dental fluorosis in India.*

Keywords*: fluoride, dental fluorosis, prevalence*

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I. Introduction

Fluoride is the 13th most abundant element and constitutes 0.08% of the Earth's crust. Fluoride is excreted mainly by renal tubular secretion. Excess fluoride is stored in the body in skeletal tissues.The stable plateau level of plasma fluoride in a healthy adult is around 1.5 ml/L, and it has long been believed that it is the frequent 'spikes' in increase rather than increase in plateau level, which leads to fluorosis.

Fluoride is used in community water supplies in the concentration of 0.7-1.2 parts per million (ppm) [1].At this concentration, fluoride is known to reduce dental caries by 50-60% in permanent teeth.It protects teeth from demineralization by inhibiting bacterial enzymes at low concentrations, and further aids in remineralization. However, an increase in the concentration of fluoride to 3 ppm or higher can cause fluorosis. Fluorosis may manifest as dental or skeletal fluorosis. Such adverse effects of fluoride are more often caused due to chronic exposure to high levels of fluoride.

Dental fluorosis is the first clinical sign of a toxic effect of fluoride in children. It is important to appreciate that the initial changes in enamel although not readily visible to an inexperienced examiner, represent the first biological sign of an elevated intake of fluoride during tooth formation.Long term intake of fluoride during enamel calcification results in 'minute white flecks, yellow or brown areas scattered irregularly or streaked over the surface of a tooth, or it may be a condition when the entire tooth surface is of dead paper white colour.'

Skeletal fluorosis occurs due to chronic ingestion of water with a high concentration of fluoride, since excess fluoride in the body accumulates in skeletal tissues. It manifests as intermittent joint and bone pain, toughness of joints, stomach ache, headache and muscular weakness. Apart from the ingestion of fluoridated water, skeletal fluorosis may also occur due to consumption of fluoride-rich foods, inhalation of airborne fluoride or occupational exposure in industries and factories. Skeletal fluorosis results in osteosclerosis which causes calcification of bones resulting in impairment of major joints, muscles, spine and nervous system.

Fejerskov (1990), defined dental fluorosis as "the condition of permanent hypomineralised change with increased surface and sub-surface porosity resulting from excess fluoride reaching developing tooth prior to eruption." [2]

In 1901, Dr. Frederick McKay in Colorado, USA, noticed that many of his patients had an apparently permanent stain on their teeth, which was known by local inhabitants as 'Colorado stain'. He called the stain as 'mottled enamel' and said that it was characterized by minute while flecks or yellow, brown spots or areas scattered irregularly or streaked over the tooth surface.

In 1931, US Public Health service appointed Dr. Trendly H. Dean to determine the severity of mottled enamel. He conducted a survey among 22 cities in 10 States of USA on a total population sample of 5824 children. This study, popularly called the 'Shoe Leather Survey' established the cause of mottled enamel. Thus, after 30 years, in 1931 the mysterious factor responsible for enamel mottling was identified – element fluoride [3].

II. Pathogenesis of Dental Fluorosis

Proteoglycans (PG) and glycosaminoglycans (GAG) have been implicated in the mineralization of bone and teeth [4]. Dermatan sulfate is present in fluorotic teeth and its presence impairs the mineralization process leaving decalcified loci, and this major factor is responsible in disturbing the deposition of calcium phosphate in the tooth. The interaction of ground substance with the mineral phase is inhibited by fluoride and phosphate, but enhanced by calcium. The fluoride competes with calcium binding sites preventing the binding of GAG to hydroxyapatite. The implication is that in in vivo presence of fluoride affects the interphase between ground substance and mineralization front by stearic hindrance [5].

2.1 Timing of Fluoride Intake in Relation to Dental Fluorosis

The major etiological factor for dental fluorosis is due to excessive concentration of fluoride in drinking water. However, it may also occur due to early use of toothpaste, unnecessary consumption of fluoride supplements, and prolonged formula intake in infants.

Dental fluorosis results due to exposure to fluoride in children during the time of tooth formation, that is, mineralization of individual teeth. Mineralization of permanent incisors occurs by 24 months of age, and the 2nd permanent molars and premolars mineralize by 6-7 years of age. For example, the maxillary central incisors are most susceptible to dental fluorosis during a period of 22 to 26 months of age [6]. Therefore dental fluorosis does not occur when a child of 7 years or older is exposed to high concentrations of fluoride.Critical period of exposure is between 1-4 years. Also, this explains why dental fluorosis is seldom reported in deciduous teeth.

It is apparent that teeth which mineralize early in life develop less fluorosis. This means that the later any tooth undergoes mineralization, the greater will be the prevalence and severity of dental fluorosis of that particular tooth type. This is why the incisors and first molars are least affected, whereas the second molars and the premolars are most affected.

III. Diagnosis of Dental Fluorosis

Long term intake of fluoride during enamel formation results in a continuum of clinical changes of enamel varying from fine, white lines in enamel to severely chalky, opaque enamel which breaks apart soon after eruption. The severity of changes depends on the amount of fluoride ingested during the long lasting period of tooth formation.

3.1 Examination History

- A historic record of the patient's residence is important as it helps to have an idea of the level of fluoride present in the respective water sources.
- Any supplementary fluoride history should also be determined.

3.2 Diagnosis and Clinical Features

When undertaking a clinical diagnosis of enamel defects, the teeth should be dry and observed with cheek retractors in place and under good lighting.

Clinical features with increasing severity:

3.2.1 Mild Fluorosis

- The first sign of dental fluorosis appears as thin, white striae across the enamel surface. The fine, opaque lines follow the perikymata pattern and can only be distinguished after cleaning the tooth surface. Even at this stage of dental fluorosis, the cusp tips, inicisal edges and marginal edges may appear totally opaque white, which has been designated as 'snow cap phenomenon' [7].
- In slightly more affected teeth the white lines become broader and pronounced. Occasional merging of several lines occurs to produce smaller, irregular, cloudy or paper white areas scattered over the surface [8].

- The opaque white flecks are more visible near the incisal edge of anterior teeth, superimposed on a greater lack of translucency.These enamel changes would not be visible to the untrained eye at a normal conventional distance.

3.2.2 Moderate Fluorosis

- With increasing severity, the entire tooth surface exhibits distinct, irregular, opaque or cloudy, white areas. Between these opacities, perikymata lines are often visible.
- In the case of moderate fluorosis, the disturbances result in porosity and over a period of time following eruption, stains are taken up and trapped within the enamel, making these areas more obvious.
- There is a range of dietary chromogenic substances that can be taken up and retained with superficial enamel giving these areas a more distinct appearance. Thus, it is not uncommon for the teeth to erupt with the porous, white, opaque areas which then become discoloured over time.
- Discolouration can also occur before eruption, but in most cases it becomes more obvious following eruption of the teeth.
- There may be irregular opaque areas that merge until the entire tooth surface appears chalky white. At eruption, this stage may vary clinically from a white, opaque tooth which feels relatively hard on probing, to a totally chalky tooth, which immediately following eruption, exhibits surface damage

3.2.3 Severe Fluorosis

- In more severe form of fluorosis, the impact on the physical properties of enamel is more dramatic. A common sequel to this problem is that at various times following eruption, small areas of enamel are lost spontaneously, giving the appearance of enamel hypoplasia.
- In severe stages, there is a focal loss of outermost enamel. Such enamel defects are usually designated as 'pits'. The pits may vary in diameter and occur scattered over the surface, although most frequently they occur along incisal/occlusal half of the tooth. With increasing severity, these pits merge to form horizontal bands and in more severely affected teeth, confluence of the pitted areas produces larger 'corroded' areas [9].
- Ultimately, the most severely fluorotic tooth exhibits an almost total loss of surface enamel. The loss of surface enamel may be so extensive that only a cervical intact rim of markedly opaque enamel is left. The remaining part of the tooth exhibits dark brown discoloration [10].
- When teeth are highly opaque at the time of eruption, they are very susceptible to attrition and extensive occlusal abrasion is observed in high fluoride areas, even in young individuals.

3.3 Histopathology of Teeth with Dental Fluorosis

The changes in fluorotic human enamel have been described using light and electron microscopy.In principle, increased exposure to fluoride during tooth formation leads to an increase in enamel porosity.Porosity may increase along the Striae of Retzius in the outer enamel surface as seen in ground sections when observed under polarized light microscope [11].They may form a narrow, continuous zone of porosity along enamel surface.Thin cervical enamel, due to its structural character may often exhibit a more uniform porosity. This may explain why this part of the crown may appear clinically more homogenously opaque. The degree of porosity and depth of lesions vary which explains the chalky appearance of the tooth at the time of eruption.The porous areas are highly hypomineralized. The hypomineralization or increased porosity is a result of an increased intercrystalline space both in rod and inter-rod enamel, but is particularly pronounced along the arcade shaped rod boundaries. The width, thickness and cross sectional shape of the individual enamel crystals are within normal range.

It is evident that the more extensive the zone of hypomineralization deep to a mineralized thin surface layer, the more susceptible will the enamel be to post eruptive damage. The pits in severe fluorosis histologically represent 'punched out' areas in the enamel surface when observed in scanning electron microscope [12].In the microradiographs, significant changes are observed in the porous enamel which has been exposed to oral environment.In severe cases, the porous enamel has become exposed to the oral environment due to breakdown of surface layer and this results in highly irregular distribution in mineral content of enamel.In some cases, dentin is also affected. These are reflected as an enhancement of lines of Von Ebner and are particularly evident in pulpal part of dentin.

IV. Fluoride Levels in India

Dental fluorosis is prevalent in over 24 countries of the world, including India. In India, areas with high fluoride minerals are extensive. Based on reports of 2017, a large population of about 62 million, including 6 million children are affected by fluorosis in India [13]. According to reports by UNICEF, about 65% of India's rural population have direct exposure to fluoride-related health issues [14]. Fluorosis is endemic in over 20

states of India [15]. The problem of fluorosis is more alarming in the states like Andhra Pradesh, Tamil Nadu, Rajasthan, Punjab, Bihar, Uttar Pradesh, Madhya Pradesh and West Bengal, where fluoride concentration in drinking water is much higher than the safe limit [16]. The main fluoride bearing areas are Gujarat, Rajasthan and Andhra Pradesh where about 50-100% of the districts are affected by fluorosis. Only about 10-30% districts are affected in the states of Jammu and Kashmir, Kerala, Chhattisgarh and eastern India.

Table 1: Distribution of fluorosis affected states in India

Severe Districts Affected (70-100%)	Moderate Districts Affected (40-70%)	Mild Districts Affected (1-40%)
Rajasthan	Punjab	Jammu & Kashmir
Gujarat	Haryana	West Bengal
Andhra Pradesh	Madhya Pradesh	Orissa
	Maharashtra	Kerala
	Karnataka	
	Uttarakhand.	
	Tamil Nadu	
	Chhattisgarh	
	Jharkhand	
	Bihar	
	Delhi	
	Assam	

V. Treatment And Prevention

The dental fluorosis classification criteria developed by Thylstrup and Fejerskov Index (TFI) [26] is very appropriate to determine the kind of treatment, based on biological aspects of dental fluorosis, and classifying individuals into categories: mild (TFI = 1-3), moderate (TFI = 4-5) and severe (TFI = 6-9) [16].

For mild to moderate fluorosis (TFI 1-5), conservative techniques like enamel microabrasion and bleaching can be employed with minimal loss of sound dental tissues. Microabrasion may be carried out along with bleaching, where former treatment is done with microabrasion, and bleaching is performed in the subsequent appointment.Microabrasion causes elimination of white surface discolouration and the enamel surface becomes brighter.However, it is very technique sensitive.

Microabrasion involves the use of acid with abrasive particles to remove stains from the tooth structure superficially, with minimal loss of enamel. The earliest form of treatment was given by Dr Walter Kane in 1916, which involved the use of 18% hydrochloric acid (HCl) with pumice stone without heat, until the desired shade was obtained [17]. In 1986, Croll and Cavanaugh developed a technique of microabrasion using a thick paste of 18% HCl with finely powdered pumice for 15 seconds, followed by 10 seconds water wash [18]. 37% phosphoric acid may also be used instead of HCl, but the former results in increased surface roughness and lower depth of enamel demineralization, when compared to HCl [19].

Some studies indicate that bleaching agents are sufficient for treatment of dental fluorosis. These include application of carbamide peroxide (10-20%) and hydrogen peroxide (1-10%) for vital teeth. Home bleaching under professional supervision or in-office bleaching may be carried out [20]. Bleaching may also be carried out in-office using McInne's solution (1 part anaesthetic ether, 5 parts of 36% HCl and 5 parts of 30 % hydrogen peroxide). The treatment is repeated multiple times until satisfactory esthetic result is obtained. Vital bleaching is more successful in younger patients presenting with opaque to orange stains rather than older patients with darker brown stains [21]. Bleaching may cause post-operative sensitivity in patients with exposed dentin. However, this sensitivity disappears over a period of time without intervention [22].

Severe dental fluorosis presents an aesthetic concern for the patient and hence aesthetic restorative techniques are advocated.Restorations like resin-modified glass ionomer or composite restorations may be used for localized discoloured areas of mild fluorosis (TFI 1-3). Moderate fluorosis may also be treated with veneers. Prosthetic full ceramic or ceramometal crowns may be required in cases of severe fluorosis (TFI greater than 5). In case of loss of vertical dimension with severe fluorosis, full mouth rehabilitation with metal ceramic crowns may be required [23].

5.1 Methods of minimizing toxicity

Fluoride is most commonly used in mouth rinses, dentifrices, topical gels or solutions and tablets. When products containing proper amount of fluoride are used as recommended, very little chances of acute

toxicity exists. For example, weekly rinsing with 0.2% NaFmouthrinse (1mg/ml F) will not produce toxicity.However, ingestion of 50 mL of the same mouth rinse will lead to toxicity in a one-year old child.

The danger of fluorosis is high in cases of topical fluoride treatment where the teeth are treated with high fluoride containing solution or gel, one at a time. Therefore, a small amount of fluoride gel should be dispensed in each tray and a salivary ejector system should be used to remove excess. However, ingestion of 10 mL and 20 mL of 1.1% sodium fluoride gel of 5000 ppm of fluoride will lead to toxicity in a one-year old and five-year old child respectively [24].

The extent of toxicity further depends on the type of fluoride product being used. Studies have indicated that stannous fluoride preparations are slightly more toxic than sodium fluoride or sodium monoflurophosphate preparations [25]. Based on reports analysed by Dukes (1980) [26] and Bayless and Tinanoff (1985) [27], it was concluded that the 'Probable toxic dose' (PTD) of fluoride for children is 5.0 mg F/kg. Therefore, an 8.2 ounce dentifrice of 1500ppm of fluoride would contain nearly 360 mg of fluoride, which is nearly 7 times greater the PTD of a one-year old child.

The major brands of fluoride containing toothpastes manufactured in India have about 1000 ppm or 1mg F/g. For example, Colgate gel (Sodium monofluorophosphate) has a concentration of 935 ppm fluoride, while Sensodyne (Sodium monofluorophosphate) has a concentration of 970 ppm [28].

Fluoride tablets may be required to supplement dietary fluoride intake in children living in areas with low water fluoridation levels. Such supplements are given to children between 6 months-16 years of age. They are prescribed only if the water fluoride levels are lesser than 0.6 ppm. Therefore, if the water contains 0.7 ppm or more of fluoride, such supplements are not required. The American Dental Association (ADA) Council on Dental Therapeutics have recommended that not more than 264 mg of sodium fluoride (120 mg of fluoride) be dispensed at a time [29].

Another area of concern is prescribing home fluoride treatment at concentration of 0.05% F^-. Some preparations are packaged in quantities of 120 mL, totalling 600 mg of fluoride. This may be a lethal dose for a young child. Thus, no more than 30-40 mL of 0.05% APF home fluoride treatment should be dispensed or prescribed at a time.

Therefore, methods of preventing fluorosis include-

a. Parental supervision is compulsory during the use of dentifrice by younger children (under 6 years), so that they ensure that the child does not swallow the dentifrice.
b. A pea sized amount of fluoride toothpaste should be used for children.
c. Infant formulas should be mixed with water that is fluoride free or has low levels of fluoride.
d. Fluoride supplements should be dispensed only when required. These supplements should be prescribed considering the fluoride concentration of drinking water, daily fluoride intake and other factors that may indicate increased caries risk, for example, patients undergoing radiotherapy.
e. The use of mouth rinses with very high concentration of fluoride should be supervised by a dental professional.
f. Keep all fluoride products out of reach of children.

VI. Conclusion

Dental fluorosis is a developmental phenomenon due to excessive fluoride ingestion during dentinogenesis. Ingestion of water with fluoride concentration up to 3 times greater than recommended amount leads to fluorosis commonly. Overwhelming evidences exist for safety of fluorides at low concentration, but when high concentration of fluorides are used the possibility of toxic overdose exists. The first sign of fluoride overdose is enamel mottling.

Therefore, fluoride is regarded as 'double edged sword'. When used properly, fluoride is an ideal public health measure for prevention of dental decay. However, excessive intake of fluoride may have deleterious effects on the teeth and bones leading to dental and skeletal fluorosis.

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