Clinical Diagnosis Of Dental Fluorosis

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Abstract

Dental fluorosis is the most convenient biomarker of excessive exposure to fluoride. Dental flourosis leads to disruption of the structural integrity of enamel which causes pits to develop which are susceptible to caries. Hence frequently it is seen that mottled enamel is quite susceptible to dental decay because of being porous in nature. It has a great emotional impact on patient because he/she feels himself handicap due to esthetic and functional reasons. Though flourosis can be treated with several non invasive methods, the most important is to correctly diagnose at the earliest.

Key Words: Dental flourosis, Dental decay, Mottled enamel

INTRODUCTION

There has been a decline in dental caries prevalence and incidence during the last two decades, both in developed as well as in economically developing countries. Since its serendipitous discovery by McKay at the turn of the century, the story of fluoride in drinking water has arguably been a case of scientific progress for public good. Simultaneously, with the decline in caries, an increase in the prevalence of dental fluorosis has been noticed.[1] Dental fluorosis occurs because of the excessive intake of fluoride either through fluoride in the water supply, naturally occurring or added to it; or through other sources. It has been postulated that the people affected by fluorosis are often exposed to multiple sources of fluoride, such as food (brick tea), water, air (due to gaseous industrial waste), and excessive use of fluoride containing toothpaste. However, drinking water is typically the most significant source. The major damage occurs in tooth development stages only between the ages of 3 months to 8 years; children over age 8 are not at risk. Although it is usually the permanent teeth which are affected, occasionally the primary teeth may be involved. The spots of dental fluorosis are permanent and darken over age. Dental flourosis may be the first indication of systemic fluoride poisoning. The condition further deteriorates if the affected teeth get superimposed infection in term of dental caries.[2]

The clinical appearance of mild dental fluorosis is characterized by bilateral, diffuse (not sharply demarcated) opaque, white striations that run horizontally across the enamel. These may be invisible to the individual and the clinician but often can be seen after the enamel has been dried. The opacities may coalesce to form white patches.

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In the more severe forms the enamel may become discolored and/or pitted[3]. Upon eruption into the mouth, fluorosed enamel is not discolored – the stains develop over time due to the diffusion. The increase is in the *mild* and *very mild* forms of fluorosis, both in fluoridated and in nonfluoridated areas. A large amount of epidemiological data demonstrates that the occurrence of dental fluorosis is associated with excessive fluoride intake throughout the period of tooth development. Multiple sources of fluoride intake have been identified. This review describes the condition and summarizes the recent literature on the risk factors for dental fluorosis. Four major risk factors have been consistently identified: use of fluoridated drinking water, fluoride supplements, fluoride dentifrice and infant formulas. In addition, some manufactured children foods and drinks may also be important contributors to total daily fluoride intake leading to leeching of exogenous ions (eg, iron and copper) into the abnormally porous enamel. Clinical studies of dental fluorosis have demonstrated that the most critical period for development of fluorosis is during the post-secretory or early maturation phase of tooth development [6]. Fluorosis is less prevalent and less apparent in primary teeth than in permanent teeth, and, in any case, fluorosis of the primary teeth has only short term rather than long-term consequences. Therefore, the major concern about fluorosis is with the permanent teeth.

CASE REPORT

CASE 1

A 17 year old male patient, an engineering student, reported to Department of Public Health, with the chief complaint of dirty yellowish discoloured teeth since childhood with sensitivity and pain in posterior teeth.

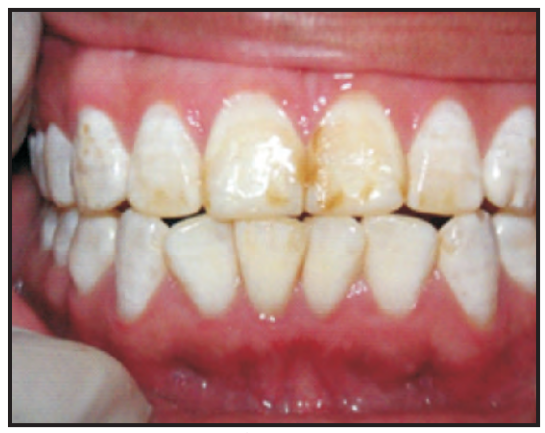


Figure 1

- Childhood residence – Jaipur (Fl endemic area, 2.2-4 ppm)
- Family history of Fluorosis- present
- Chief complaint of poor esthetics (mother)
- Brown discoloration, flecks present (<50% of tooth surface area)
- Brown staining visible

On examination, he had poor oral hygiene and moderate fluorosis with brown stains on teeth. Treatment plan involved oral prophylaxis and direct composite resin veneer for the patient.

The procedure involved:

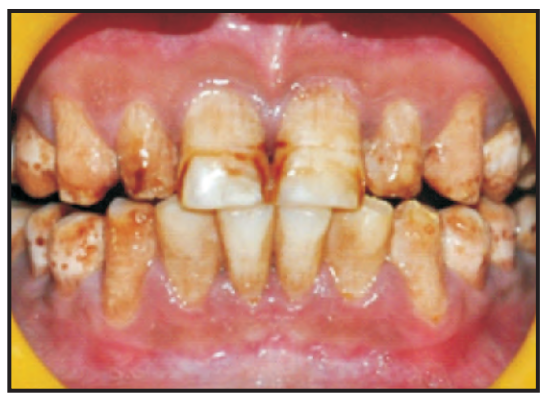
- Teeth were cleaned
- Shade was selected
- Teeth were isolated
- Tooth was prepared with a round ended diamond instrument.
- Veneer preparation with window design was done.
- Margins were not extended subgingivally as it was not involved.
- Incisal margins were not included as

they were not involved.

- Teeth were restored one at a time.
- After acid etching, rinsing and drying, resin bonding agent was applied and cured.
- Filtek Z 250 (3M ESPE, USA) Composite resin was placed in increments and cured.

Polishing was done. Patient was satisfied with treatment outcome.

CASE 2



- 23 yr old male.
- Childhood residence - Gorakhpur (Fl endemic area, 4-8 ppm).
- Family history of Fluorosis- present (younger brother).
- Chief complaint - dirty yellowish teeth since childhood with sensitivity and pain in posterior teeth.
- Introvert and feels shy on smiling.

On clinical examination patient had generalized enamel fluorosis affecting all the permanent teeth. Confluent pitting was present on most of the surfaces of the teeth with wide spread yellow brown stains (Figure 2).

Occlusion was in a class 1 relationship. Oral hygiene was good and the gingival tissue was in a healthy condition. Radiographic examination showed no caries or alveolar bone loss. Diagnosis of moderate dental fluorosis was made, based on history, clinical findings and dean's index. Given the age of patient and severity of fluorosis porcelain laminates were given as treatment option and was accepted by the patient.

DISCUSSION

The mechanism underlying the development of dental fluorosis has not been conclusively determined. It was believed previously that excessive fluoride intake interfered with the function of ameloblasts, perhaps inhibiting the secretion of, or altering the composition of enamel matrix proteins. It now appears that this is unlikely for several reasons including the fact that the risk of dental fluorosis is lowest during the secretory stage of enamel development [4]. Microscopically, the structural arrangement of the crystals appears normal, but the width of the intercrystalline spaces is increased, causing pores. The degree and extent of porosity depends on the concentration of fluoride in the tissue fluids during tooth development [5]. In fact, the risk of dental fluorosis, based on animal studies, is directly related to the interaction of circulating fluoride concentrations and time, i.e., the area under the time concentration curve. Thus it appears that dental fluorosis can result from a range of plasma fluoride concentrations provided that they are maintained for sufficiently long periods [6]. With increasing severity of fluorosis, the fluoride concentration throughout the enamel, the depth of enamel involvement, and the degree of porosity also increases[7].

Unlike extrinsic discolorations that occur on teeth surfaces, intrinsic stains are attributable to incorporation of chromogenic materials into enamel and dentin either before eruption

(during odontogenesis) or after eruption. Examples of preeruptive stains are dental fluorosis, tetracycline stain, inherited developmental defects of enamel or dentin without systemic features, and hematologic disorders. Posteruptive intrinsic stains result from pulp necrosis, iatrogenesis, and aging. Causes of intrinsic discolorations are localized or generalized. Localized discoloration can be caused by trauma to developing teeth, extraction of primary teeth, periapical infection of primary inadequate endodontic treatment, amalgam staining . Tetracycline-stained teeth are diagnosed from the history, clinical appearance, and fluorescence under ultraviolet light. Stained teeth display a bright yellow fluorescence when exposed to ultraviolet light of about 360 nm. As the teeth become brown on exposure to sunlight, owing to the degradation of the tetracycline, the fluorescent properties progressively decline. Consequently, the labial surfaces of the anterior teeth usually are the first to darken, whereas the more protected posterior teeth retain their yellow color for longer periods. Hemolytic diseases of the newborn, previously called erythroblastosis fetalis and icterus gravis neonatorum, may produce severe jaundice in the newborn, resulting in yellow green discoloration. Sickle cell anaemia and thalassemia may result in similar discoloration, owing to the presence of blood pigments within the dentinal tubules. Congenital erythropoietic porphyria results in the deposition of porphyrin pigments in dentin and bone, making primary and permanent teeth appear purplish-red or reddish-brown in color. Affected teeth will fluoresce red with ultraviolet light. Tooth discoloration associated with amelogenesis imperfecta ranges from white opaque through yellow, with a tendency to darken with age or even stain black in newly erupted teeth owing to exogenous staining. Dentinogenesis imperfecta, or hereditary opalescent dentin, is a heritable dental disease characterized by abnormal dentinogenesis affecting both the primary and permanent dentitions. The teeth have bulbous crowns, short blunt roots, and obliteration of the pulp chambers and root canals. The enamel is poorly attached and tends to chip away from the dentin. The color of the teeth may vary from a light blue to a dark brown. It should be noted that some intrinsic discolorations, caused by environmental or genetic factors, are associated with enamel hypoplasia and opacities.[8]

Fluorosis disturbs enamel significantly and affects esthetics quite adversely which can cause psychological distress to the affected person. The etiology of intrinsic discoloration of enamel is commonly associated with fluorosis. The treatment of enamel fluorosis usually ranges from ceramic veneer to free hand bonding restorations[6] Although vital bleaching does improve the esthetics to certain extent it has only met with partial success in regard to moderate to severe fluorosis[1]

The concept of veneering was first described in dental literature sometimes ago, although it is only with the advent of efficient bonding of resins to enamel and dentine and the use of etched, coupled porcelain surfaces that esthetically pleasing, durable and successful restorations can be made.[9]Porcelain veneers have traditionally been made from aluminous or reinforced feldspathic porcelains, which have relatively poor strength in themselves but produce a strong structure when bonded to enamel.

A key element in success with porcelain veneer is carefully controlled but appropriate tooth tissue reduction. Veneers are generally

prescribed for the buccal aspect of maxillary anterior teeth, but there are numbers of nonstandard applications, like the palatal/lingual aspect of teeth which have been worn or fracture, diastema elimination using slips restricted to the proximal aspects of teeth, lower incisors, and posterior occlusal onlays. Incisal coverage of porcelain has to be sufficiently thick to be durable under continuing rubbing contact with the opposite tooth. [10]In this case porcelain laminates were chosen for its esthetic performance, biocompatibility, durability and the translucency provided by the restoration which allows light transmission through to the underlying tooth which minimizes gingival shadowing and yields an appearance of vitality[11].

CONCLUSION

Dental fluorosis and dental caries both are dental ailments affecting esthetic and functional demands of the patient. Unfortunately in severe cases both go hand in hand as presence of one leads to superimposition of the other. Fortunately present treatment modalities in modern dentistry helps to regulate it and hence prevent further deterioration if attended timely. The treatment may vary from conservative bleaching to invasive procedure of crowns and bridges depending on severity of the disease. But a successfully accomplished treatment provides better esthetics to patient with full competence in function.

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