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Review Article

Effects of Dental Fluorosis on Bond Strength of Orthodontic Bracket, A Review of *In-vitro* Studies

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Abstract

Dental fluorosis is a developmental disturbance of dental enamel, caused by successive exposures to high concentrations of fluoride during tooth development, leading to enamel with lower mineral content and increased porosity. Bonding brackets to fluorosis enamel remains a clinical challenge because of frequent bracket failures at the compromised enamel interface. Increased fluoride content has been shown to resist acid etching due to the presence of fluorapatite at the outer enamel surface. This reduction in the effectiveness of acid etching can be superseded if the etching time is increased. Thus the present review focused attention on *in-vitro* studies and made attempt to discuss the effect of dental fluorosis on shear bond strength of orthodontic bracket.

Keywords: Dental Fluorosis; Acid Etching; Orthodontic Brackets; Shear Strength; Dental Bonding

Introduction

Dental fluorosis

Definitions of dental fluorosis are based on the widespread evidence, which links a specific pattern of opacities and exposure to excess fluoride at the time of tooth development. This association has become an assumed "cause and effect relationship" that is, identify one and assume the other, this is correct only if a unique relationship exits [9].

Definitions of dental fluorosis include

"Hypomineralization of tooth enamel or dentin by the long continued ingestion of excessive amounts of fluoride during tooth development" [15]. "A specific disturbance of tooth formation caused excessive intake of fluoride during the formative period of the dentition [27]. "Developmental defects of enamel induced fluoride. Clinically, it is characterized by a pattern of white opacities affecting homologous teeth. The opacities can vary from minor white striations to small or extensive areas of (lusterless) opaque enamel; post-eruptive staining or pitting of enamel may occur [21]. The severity and distribution depend on the fluoride concentration, duration of exposure to fluoride, the stage of ameloblasts activity and individual variation in susceptibility" [15].

Dental fluorosis affects not only enamel, but also dentin. In dentin, it results in an enhancement of the lines of Von Ebner particularly evident in the pulpal part of the dentin [19]. As compared to normal dental enamel, an outer hypermineralized acid resistant layer and retention of porous enamel in areas of the subsurface exemplify fluorosed enamel. Water and enamel secretory proteins that are retained due to the effect of the excessive fluoride levels on ameloblasts occupy the pores. The severity of dental fluorosis depends on when and the duration of the overexposure to fluoride occurs, the individual response, weight and degree of physical activity, nutritional factors and bone growth. In the milder forms, fluorosed enamel may also be characterized clinically by white opaque line corresponding to the position of perikymata; under severe circumstances, the enamel is composed of distinct irregular, opaque or cloudy white areas [21]. As a consequent of post-eruptive trauma, the formation of subsurface enamel defects may appear as single pits or the surface may be flaked off predominantly from the incisal edges or cusp tips. Consequently, such patients require tooth colored restorations including composite veneers [21]. The damage caused by fluorosis is permanent, making prevention very important in regions of the world where the problem is endemic. Fluorosis appears to be specifically a common manifestation in the

developing world (especially in many South Asian countries), for a variety of reasons, but it has not spared even the Western world where its prevalence has increased over the past five decades [12].

Prevalence of dental fluorosis

The global prevalence of fluorosis has increased from 7.7% to 80.9% (in areas with fluoridated water) and from 2.9% to 42% in areas without fluoridated water [13,26,33]. In San Luis Potosí, Mexico, the fluorosis prevalence was 69% where the levels of water fluoride were less than 0.7 ppm and increased to 98% for a fluoride level of 2 ppm [22]. In the United States of America, 23% of individuals aged 6-49 years had dental fluorosis in 1999–2004 [10]. Approximately, 2% had moderate dental fluorosis and less than 1% had severe dental fluorosis. Dental fluorosis was most prevalent among children aged 12–15 years, and less prevalent among the older age groups [10]. Al-Shammary., et al. [5] reported that 77% of the population surveyed in Unaiza, Al-Qaseem had no fluorosis, while only 2.5% were suffering from moderate to severe fluorosis. Akpata [2] showed that the fluoride level in different parts of AlQaseem region is ranging from 2-3 ppm. Almas., et al. [4] found that fluorosis is more prevalent in rural subjects than in urban population of Al-Qaseem province, 59% of 12 years in rural and 26% of 12 years old urban population are affected with fluorosis of teeth, 34% of 15 years old in urban area have fluorosis, while highest 67% of 35-44 years in rural areas have fluorosis [4]. Ramires., et al. [34] conducted a study to evaluate the prevalence of dental fluorosis in school children aging 12 to 15 years old, residents in the City of Bauru, State of São Paulo, Brazil. In this study, more than 1000 volunteers were enrolled in this study and examined in 18 public schools of the State of São Paulo. They concluded that the prevalence of dental fluorosis in Bauru is within the expected range, based on previous studies. Although fluoride is an important resource for caries control, its use must be adequate to the needs of each specific population [34].

Mechanism of dental fluorosis

Throughout the years, many studies aimed at understanding the mechanism by which dental fluorosis occurs [14,19,20,24,27,37, 38]. In more recent years, several investigators have studied the nature of fluorotic enamel and proposed biologic mechanisms responsible for its occurrence. Robinson and Kirkham [36] concluded that fluoride appears to affect enamel in a number of different developmental stages. At concentrations in the physiological range, however, few effects have been observed. Cell proliferation and differentiation are not obviously affected, nor the chemical nature of the secreted matrix. Control of matrix deposition dose seemed to be affected. However, deregulation of growth in existing crystallites and absence of crystals in newly secreted matrix have been reported. They suggest that interferences with the mineral-matrix interface occurred directly or via some reversible alteration in the 3 dimensional arrangements of the matrix proteins. Retention of prolife-rich matrix may result from this initial interference. A more direct effect on matrix breakdown or its removal by the cells during secretion or maturation may also be important. They also suggested that inhibition of final maturation is perhaps due to an effect on matrix-mineral interactions, although a direct effect upon the modulation of ameloblasts could not be ruled out. Denbesten and Thariani [16] concluded that enamel fluorosis is characterized by retention of amelogenins in the early-maturation stage, and by the formation of highly porous enamel with subsurface hypomineralization. The mechanisms by which fluoride affects enamel development include specific effects on both the ameloblast and on the developing enamel matrix. Maturation-stage ameloblast modulation is more rapid in fluorosed enamel when compared with control enamel, and proteolytic activity in fluorosed early-maturation enamel is reduced when compared with controls. Secretory enamel appears to be more susceptible to the effects of fluoride following acute fluoride exposure. However, both human and animal studies show that the transition/early-maturation stage of fluorosis could be produced even when fluoride exposure commences after the secretory and transition stages of enamel development.

The mechanism by which fluoride acts to induce fluorosis may be categorized as follows [9]:

1. Effects secondary to changes in systemic calcium metabolism
2. Effects on the composition of matrix proteins when secreted
3. Effects on apatite nucleation and crystal growth
4. Effects on enamel matrix protein hydrolysis and removal from the developing enamel.

Effect of fluorosis on bracket bond strength

Surface enamel fluorosis differs from non-fluoride-induced opacities, which are generally well demarcated and asymmetrically distributed. Fluorosed enamel shows various degrees of hypomineralization [30]. Teeth with a higher concentration of fluoride are generally considered more resistant to acid etching than normal teeth and may require an extended etching time. An orthodontic bracket bonding has been in use for over four decades and the success of fixed appliance therapy depends on adequate bond strength and low failure rates [30]. Orthodontic brackets are subjected to large number of forces in the mouth resulting in complex distribution of stresses within the adhesive and its junction with enamel (tooth surface). The bond strength depends on large number of factors including the nature of enamel surface, enamel conditioning (preparation) procedures, types of adhesives used and the shape and design of bracket base. The bond failure

impairs the progress of the treatment and can be costly in terms of materials and time. In order to reduce the bond failures adhesion boosters were introduced in orthodontics. They incorporate hydrophilic resins as a component, which helps to reduce interfacial porosity and therefore adhesive defects. Noble., *et al*. [31] compared the shear bond strength of fluorosed enamel surface treated with sandblasting or acid etching, or a combination of both acid etching and micro etching. The groups were treated as follows: in group I, acid etching was followed by bonding with Transbond XT®; in group II, micro-etching and acid etching was followed by bonding with Transbond XT®; and in group III, micro-etching and acid-etching was followed by bonding with Enlight LC®. They found sandblasting followed by acid-etching provides significantly higher bond strength values compared to acid-etching alone, irrespective of the bonding material employed. Noble., *et al.* [31] compared fifty-two teeth with severe dental fluorosis bonded *in vivo* using a split-mouth design where the enamel surfaces of 26 teeth were micro abraded with 50 µm of aluminum silicate for 5 seconds under rubber dam and high volume suction. Thirty-seven percent phosphoric acid was then applied to the enamel, washed and dried, and followed by placement of Scotchbond Multipurpose Plus Bonding Adhesive. Finally, precoated 3M Unitek Victory brackets were placed and light cured. The remaining teeth were bonded using the same protocol but without micro abrasion. They found bonding orthodontic attachments to fluorosed enamel using an adhesion promoter is a viable clinical various stages of the bonding procedure. One hundred extracted human third molar teeth were randomly separated into 2 basic groups (normal or fluoridated teeth), then divided into 5 subgroups. Group A specimens were not contaminated. After etching, enamel surfaces were dry and clean. Group B was left with wet surfaces after etching. Group C specimens were contaminated with artificial saliva and then dried. Group D specimens were contaminated with artificial saliva, rinsed, and then dried. In Group E, enamel surfaces were left contaminated with saliva after the etching procedures (with maleic acid or phosphoric acids). Adhesive resins were applied to all enamel surfaces according to the manufacturer's instructions. They found that saliva contamination may not be a risk factor for successful bonding between bonding agent and dental tissues for normal or fluoridated enamel surfaces if they are rinsed and dried immediately after contamination. Etching of normal enamel surfaces with phosphoric acid in the presence of contamination may provide higher shear bond strength than etching with maleic acid. Opinya and Pameijer [32] studied the effect of fluorosis on the tensile bond strength of composite material to enamel. They found no significant effect of fluorosis on bond strength. However, grinding of the surface layer of fluorosed enamel and then acid etching resulted in an increase in tensile bond strength. No statistically significant difference was also reported between the mean values for bond strength of orthodontic brackets in fluorosed and non-fluorosed teeth [25]. Ateyah and Akpata [3] demonstrated in an *in vitro* study that the severity of fluorosis had no statistically significant effect on shear bond strength while etching time affects the shear bond strength of composite material to fluorosed human enamel. procedure that does not require the additional micro-mechanical abrasion step. Gungor., *et al*. [23] compared the shear bond strength of enamel, the groups were treated as follows in groups I (non-fluorosed teeth) and II (fluorosed teeth), standard etching protocol was used and brackets were bonded with Light Bond. In groups III (non fluorosed teeth) and IV (fluorosed teeth), Transbond Plus SEP was used and brackets were bonded with Transbond XT Light Cure Adhesive. All specimens were cured with a halogen light. They concluded that when standard etching protocol was used, enamel fluorosis significantly decreased the bond strength of orthodontic brackets. Duan., *et al.* [18] compared the failure rates of brackets bonded to severely fluorotic teeth using three different methods of enamel preparation. A total of 324 severely fluorotic teeth were included in the study. These were randomly divided into three groups according to the method of enamel preparation. Group A – each surface to be bonded was brushed clean with plain, non-fluoridated pumice and water. Group B - after the surface to be bonded was cleaned as in Group A, it was polished by evenly removing. 1 - .2 mm of enamel with a carbide drill. Group C - each surface to be bonded was cleaned and polished as in Group B. Next, a toothcolored. 2-.3mm layer of Transbond Plus Self- Etching Primer* was affixed to the surface, according to the manufacturer's instructions, to form a veneer. They found that bond failure rates after 12 weeks were 74.0% for Group A, 25.9% for Group B, and 1.7% for Group C. Benderii., *et al.* [11] examined the shear bond strength of bonding agents to normal or fluoridated enamel following use of weak or strong acids to prepare enamel surfaces and after contamination with a measured amount of saliva at various stages of the bonding procedure. One hundred extracted human third molar teeth were randomly separated into 2 basic groups (normal or fluoridated teeth), then divided into 5 subgroups. Group A specimens were not contaminated. After etching, enamel surfaces were dry and clean. Group B was left with wet surfaces after etching. Group C specimens were contaminated with artificial saliva and then dried. Group D specimens were contaminated with artificial saliva, rinsed, and then dried. In Group E, enamel surfaces were left contaminated with saliva after the etching procedures (with maleic acid or phosphoric acids). Adhesive resins were

applied to all enamel surfaces according to the manufacturer's instructions. They found that saliva contamination may not be a risk factor for successful bonding between bonding agent and dental tissues for normal or fluoridated enamel surfaces if they are rinsed and dried immediately after contamination. Etching of normal enamel surfaces with phosphoric acid in the presence of contamination may provide higher shear bond strength than etching with maleic acid. Bakhadher., *et al.* [7]. Fluorosis had no influence on the SBS of brackets, whereas it had a negative influence on retaining adhesives onto the tooth surfaces using microcomputed tomography technology. Marure PS., *et al.* [25] conclude the use of microabrasion method increases micromechanical orthodontic bracket retention of severely fluorosed human teeth and provides a clinically successful adhesive bonding protocol. Alhamadi W., *et al*. [1] reported that the application of the caries infltrant following 37% phosphoric acid etching on sound enamel prior to orthodontic bonding could be an alternative to be used as an additional preventive measure against WSL formation. It was concluded that the surface infltrated by Icon (DMG) did not interfere negatively on the bond strength to the resin composite. Basunbul A., *et al.* [8] conclude that fluoride varnishes should be considered as a preventive adjunct to reduce enamel demineralization adjacent to orthodontic bracket.

Conclusion

Our literature review conclude that When standard etching protocol was used, enamel fluorosis significantly decreased the bond strength of orthodontic brackets. Satisfactory bond strengths were obtained when SEP was used for bonding brackets to the fluorosed fluorosis of teeth reduce bracket bond strength to enamel, but the bond strength with these still exceed the minimum 6 to 8 MPa required to expect adequate clinical performance. Public awareness campaign both at School and Community level is to be mobilized. Children and adults inflicted with fluorosis and should be given treatment priority, as to minimize or disguise their staining stigma and pitting abnormalities of the teeth. More research is required to develop our understand of that factor in influencing the shear bond strength of orthodontic brackets.

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