Fluorides and Their Role in Demineralization and Remineralization

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Abstract: Demineralization and remineralization begins with historical perspective on caries. Caries were identified as a major public health problem in 1940s. Demineralization is a process of removal of minerals from dental enamel. Remineralization on the other side is the process of restoring minerals to hydroxyapatite lattice. The battle to keep teeth strong and healthy is dependent upon ratio between demineralization and remineralization. In this scientific era new advances have changed our idea from “cure” to “prevention”. Remineralization can mainly be achieved by mineral or ionic technology. Ionic technology mainly includes fluorides. Fluorides works primarily via topical mechanism which includes, inhibition of demineralization at crystal surface, enhancement of remineralization at crystal surface, and at high concentration inhibition of bacterial enzymes. This article deals with various aspects of fluorides in management of De/Remineralization.

Key words: Demineralization, Remineralization, Fluorides.

INTRODUCTION

In early 1960 Massler, Fusayama and Branstorm dealt with the science of De/Remineralization. Earlier dental caries was thought to consist of one-way progressive demineralization of enamel crystallite followed by degradation of dentin leading to cavity formation. Later with increased knowledge dental caries was found to be a dynamic process with demineralization of the hard dental tissue by the acidic products of bacterial metabolism that alternates with periods of remineralization.1 When the two processes are in balance no net mineral loss occurs at the tooth surface, but when the magnitude of one exceeds the other it leads to net demineralization or alternatively to remineralization.2 The notion that loss of tooth mineral can be compensated by mineral deposition has considerable consequences in operative and preventive dentistry. It implies that non-restorative clinical strategies have become a realistic option.

DEMINERALIZATION

Demineralization is the process of removing minerals, in the form of mineral ions, from dental enamel. In another words, Demineralization is “dissolving the enamel.” A substantial number of mineral ions can be removed from hydroxyapatite latticework without destroying its structural integrity. When too many minerals are dissolved from an area of the hydroxyapatite’s latticework, results in a cavity that is the loss of the hydroxyapatite’s crystalline latticework structure. The latticework can be strengthened and restored through the process of remineralization.3

REMINERALIZATION

Remineralization is the process of restoring minerals in the form of mineral ions to the hydroxyapatite latticework structure. Remineralization should be three-dimensional and must be replaced with same shape, size and the same electrical charge as those lost from the lattice.

Rationale for De/Remineralization

The solubility of the hydroxyapatite depends on both the presence of impurities and the pH of the environment. pH is the driving force for dissolution and precipitation of hydroxyapatite.4 At low pH the saturation concentration of the calcium and phosphate ions with respect to apatite is higher than at high pH. At neutral pH saliva and plaque fluid are super-saturated with respect to hydroxyapatite. Consequently mineral will precipitate if a suitable precipitation nucleus is available. The consumption of fermentable sugars leads to acid production in the plaque and the resulting decrease in pH increases the calcium and phosphate concentration needed for saturation. The decreasing pH also slows down the fermentation (rate of acid formation) by oral bacteria.5

The calcium and phosphate content and in particular the pH of these liquids determine whether enamel and dentin will dissolve or alternatively whether mineral will precipitate. The acid ions react principally with the phosphates in saliva and plaque, until the critical pH for the dissociation of hydroxyapatite is reached at approximately pH 5.5 - 5.2. Further decrease in pH results in progressive interaction of acid ions with phosphate groups of hydroxyapatite causing partial or full dissolution of the surface crystallites.

The stored fluoride released in this process reacts with Ca++ and HPO4- ion breakdown products, forming fluorapatite, or fluoride enriched apatite. If the pH decreases further below 4.5 that is the critical pH for fluorapatite dissolution, even fluorapatite will then dissolve. If acid ions are neutralized, and the Ca++ and HPO4- ions are retained, then the reverse process of remineralization occurs. The composition of the apatite then formed depends on the composition of the solution from which it is precipitated, in this case the plaque fluid. This periodic cycling of pH results in a step-by-step modification of the chemical composition of the outer layers of enamel that becomes somewhat less soluble with time. This process is known as the post-eruptive maturation of the enamel.6
De – Remineralization Cycle

It is apparent that the pH cycle depends on the strength of the acid that is present, the frequency and duration of its production and the remineralization potential in each particular situation, any one of the following sequelae can occur.

1. The enamel may continue to mature
2. Chronic caries may develop – slow demineralization with active remineralization
3. Rapid (rampant) caries may arise – rapid demineralization with inadequate remineralization
4. Erosion may occur – very rapid demineralization with no remineralization at all

The chemical basis of the demineralization – remineralization process is similar for enamel, dentin and root cementum. However the different structures and relative quantity of mineral and organic tissue content of each of these materials causes significant differences in the nature and progress of the carious lesion.

Enamel lesion

The initial enamel lesion results when the pH level at the tooth surface exceeds that which can be counter-balanced by remineralization but is not low enough to inhibit surface remineralization. The acid ions penetrate deeply into the prism sheath porosities, leading to sub-surface demineralization. The tooth surface may remain intact through remineralization, which occurs preferentially at the surface due to increased levels of calcium, phosphate, fluoride ions and buffering by salivary products.

The clinical characteristics of such lesions are

1. Loss of normal translucency of enamel with a chalky white appearance on dehydration
2. A fragile surface layer susceptible to damage from probing particularly in pits and fissures.
3. Increased porosity particularly of the sub-surface with potential for uptake of stain.
4. Reduced density of the sub-surface detected radiographically or with Transillumination
5. A potential for remineralization with an increased resistance to further acid challenge

The advancing coronal lesion

If the demineralization - remineralization imbalance continues the surface of the incipient lesion collapse through the dissolution of apatite or fracture of the weakened crystallite resulting in cavitations. Plaque can now be retained within the depths of the cavity and the remineralization phase is rendered more difficult and less effective. The dentin-pulp complex will become involved at this point but there can still be fluctuations in the degree of activity.

Demineralization into dentin

The process of demineralization continues to be driven by dietary substrate after bacteria have invaded dentin. The acid production by bacteria dissolves the hydroxyapatite of deeper dentin so there is a front of demineralization in advance of the bacterial invasion.

The texture and color of dentin changes as demineralization advances. The color will darken because of bacterial products and stains from foods and beverages. If the lesion is left to extend through the dentin the enamel will become progressively undermined and weakened resulting in a wide-open cavity that is relatively self-cleansing. The caries process may then slow down leading to the development of a hard leathery floor on the cavity that is more or less inactive.

Factors Influencing De-Remineralization

A high level of acid concentration and a high frequency of contact will lead to demineralization of the tooth surface, however natural protective factors and repair mechanisms can be enhanced and the problem controlled at least to a degree. There is a delicate balance between health and disease, involving acid arising from bacteria laden plaque competing with protective factors that are provided through normal salivary flow and good hygiene.

Role of Fluorides

There have been many schools of thought over the years as to the relative importance of different ways in which fluoride acts to reduce dental caries. It is now well accepted that the primary mode of action is the inhibition of demineralization and enhancement of remineralization. Fluoride acts by inhibiting mineral loss at the crystal surfaces and by enhancing the rebuilding or remineralization of calcium and phosphate in a form more resistant to subsequent acid attack.

Mechanism of action of fluoride

The most probable mechanism through which fluoride prevents dental caries is by stabilizing the enamel crystal i.e. by preventing enamel demineralization from the acid produced by the microflora or by favoring recrystallization of dissolved enamel surfaces or both. Preferably the fluoride should be bound permanently to the enamel crystal in the form of fluorapatite.

Fluoride ion substitutes for the hydroxyl ion in the apatite structure giving rise to a reduction of crystal volume and a concomitant increase in the structural stability. Under the influence of fluoride, large crystals with fewer imperfections are formed thus stabilizing the lattice and presenting a smaller surface area/unit volume for dissolution. Also enamel, which mineralizes under the fluoride influence, has lower carbonate content, thus giving a reduced solubility.

Fluoride can be firmly bound when it is incorporated in the crystalline lattice of hydroxyapatite or loosely bound when it is adsorbed to apatite forming calcium fluoride deposits. In the research on the cariostatic effect of fluoride, considerable emphasis is placed on the role of free fluoride ions in the oral fluid. Calcium fluoride is formed during treatments with high concentration fluoride solutions. It can act as a fluid reservoir on the tooth surface and release fluoride ions at low pH. This fluoride ion along with calcium and phosphate diffuses into the lesion and precipitates as fluorhydroxyapatite. The acid cycle thus contributes to the conversion of loosely to firmly bound fluoride.

The fluoride ion (F-) inhibits the bacterial enzyme enolase, thereby interfering with production of phosphoenolpyruvate (PEP). PEP is a key intermediate of the glycolytic pathway and, in many bacteria, is the source of energy and phosphate needed for sugar uptake. The presence of 10-100 ppm of F-, inhibits acid production by most plaque bacteria (Fig. 99-4). These levels are delivered easily by most prescription fluoride preparations; of equal interest is the finding that at acidic pH values (5.5 or below), low levels of F- (1-5 ppm) inhibit the oral streptococci. These levels are found in plaque, especially in individuals who drink fluoridated water or who use fluoridated dentifrices. If this plaque fluoride is derived from the tooth, an antibacterial mode of action, which involves a depot effect, can be postulated for systemic (water) and topical fluoride administration.

The depot effect comes about in this manner. Water fluoridation promotes the formation of fluorapatite, whereas topical fluorides cause a net retention by the enamel of fluoride as fluorapatite or as more labile calcium salts. Microbial...
acid production in the plaque may solubilize this enamel-bound fluoride, which at the prevailing low pH in the plaque microenvironment could become lethal for the acid-producing microbes. Such a sequence would discriminate against *S. mutans* and lactobacilli because they, as a result of their acidic nature, are most likely the numerically dominant acid producers at the plaque-enamel interface. The fluoridated tooth thus contains a depot of a potent antimicrobial agent that is not only released at an acid pH value but is most active at this pH value. This hypothesis, then, attributes some of the success of water fluoridation and topical fluorides to an antimicrobial effect. It further suggests that judicious use of topical fluorides would be effective in patients with highly active caries. The most effective dose schedule and fluoride preparation have not been determined.19

**SOURCES OF FLUORIDES**

**Fluoride containing dentifrices**

The use of fluoride containing toothpaste has been proven to reduce the incidence of caries in numerous clinical studies. During a typical one minute brushing period fluoride rapidly permeates the tooth and is taken up by the enamel as fluorapatite, calcium fluoride or even free fluoride. Rinsing the mouth after brushing rapidly drops the salivary fluoride concentration to 1 ppm or less within 15 minutes. However the treated tooth enamel and perhaps the oral mucosa acts as a sink for fluoride and subsequently release it to the oral cavity.7

The FDA as safe and effective for use in dentifrices approves three sources of fluoride. They are Sodium Fluoride, Sodium Monofluorophosphate and Stannous fluoride. Sodium fluoride directly provides free fluoride. It is generally not found in toothpaste formulations containing calcium-based abrasives because of its potential to irreversibly bind to the abrasive and form insoluble calcium fluoride on storage. Sodium Monofluorophosphate is the fluoride of choice when calcium-containing abrasives are used. The Monofluorophosphate ions releases free fluoride when it hydrolyses on exposure to phosphatase enzymes naturally present in the mouth.

Stannous fluoride provides fluoride and stannous ions which act as an antimicrobial agent. It can also produce stannous phosphate fluoride precipitates which slows down the caries process but has staining as a side effect.14

**Fluoride mouth rinses**

They raise the concentration of fluoride in saliva for several hours after use. Even though the residual concentrations of fluoride in plaque and saliva are small, the modest elevations in fluoride concentration may be sufficient to boost the rate of remineralization and help inhibit caries development. Use of 0.05% sodium fluoride mouth rinses has been shown to be better than brushing with conventional fluoride toothpaste.

**Fluoride releasing dental materials**

Resin modified GIC, conventional GIC and fluoride releasing composites have been postulated to protect against secondary caries in enamel and dentin. They have a synergistic effect with fluoride rinses or dentifrices in inhibiting demineralization.

**Pit and fissure sealants**

They are effective in preventing pit and fissure caries. The currently available sealants are second and third generation which are polymerized with chemical catalyst or require visible light to initiate a auto-catalytic reaction. It has been suggested that fluoride released from sealants may have its great effect at the base of the sealed groove helping remineralization of incipient enamel lesion.8

**CONCLUSION**

Fluorides have antacaries effect and it also prevents demineralisation, promotes remineralisation of early caries. Fluoride is most commonly used remineralising agent. As the pH rises, new and larger crystals that contain more fluoride forms are formed, thereby reducing the enamel demineralisation by forming fluorhydroxyapatite. crystals and enhancing remineralisation.

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