Minimally Invasive Demineralized Hard Tissue Repair: Remineralization

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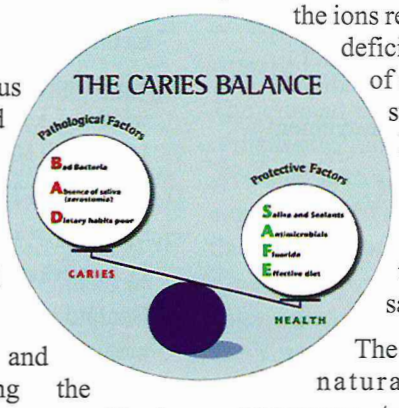
Abstract:

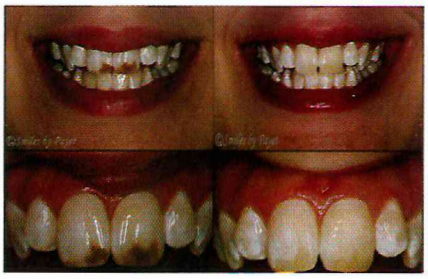
Caries is a disease caused by a multifactorial process and it remains a major public health problem in most communities even though the prevalence of the disease has decreased. One of the recent trends in caries control includes minimally invasive treatment of the lesions by reversal of mineral loss by remineralization. Restorative treatment options are advised only when the carious process has resulted in more extensive damage, and form, function or esthetics need to be restored. This article reviews the various remineralization strategies and their role in remineralizing tooth structure.

Introduction:

Traditionally, the management of a carious lesion involves invasive operative and surgical approach which can result in tissue loss and patient discomfort. One of the most important concepts that have evolved in cariology in the past several decades is the demineralization and remineralization of enamel.[1]



The ratio between demineralization and remineralization is crucial, determining the hardness and strength of tooth structure. If the demineralization phase continues for a long period of time, due to fall in pH, excessive loss of minerals results, which leads to loss of enamel structure and cavitation leading to caries. When the pH rises, the reverse takes place, resulting in deposition of minerals back to the tooth structure.[2,3] Fig 1.



The ability of saliva to remineralize demineralized enamel crystals stems from its ability to supply bioavailable calcium and phosphate ions to the tooth. At physiological pH, unstimulated and stimulated parotid, submandibular, and whole saliva are supersaturated with respect to most solid calcium phases. However, precipitation of calcium phosphate phases in saliva normally does not occur, due to the presence of salivary proteins, particularly statherin and proline-rich phosphoproteins. The proposed mechanism of action is that the segments of the proteins containing phosphoseryl residues, in particular the statherin sequence, bind to calcium and phosphate ion clusters, preventing growth of the ion cluster to the critical size required for precipitation and transformation into a crystalline phase. This critical stabilization of calcium and phosphate ions by salivary phosphoproteins ensures that the ions remain bioavailable to diffuse into mineral deficient lesions to allow for remineralization of demineralized crystals, while preventing surface deposition in the form of calculus. However, net remineralization produced by saliva is small and is a slow process, with a tendency for the mineral gain to be in the surface layer of the lesion due to the low ion concentration gradient from saliva into the lesion.[4]

The question now becomes how one can help naturally occurring processes to arrest lesion activity, repond to mineral dissolution by remineralization effort, and thus potentially reverse early carious lesions.[5] Where individual lesions or cavities are concerned, the treatment options are to restore or not to restore. If the decision is made not to restore, the question arises, can the lesion or cavity be cured, or at least prevented from further extention; in other words, can it be arrested?[6]

The best way of managing early carious lesion involves remineralization of the enamel with the aid of remineralization products. A variety of these products are available. This article reviews the various remineralization strategies and their role in remineralizing tooth structure.

Various Remineralizing Agents And Methods:

Fluorides: Fluoride ions promote the formation of fluorapatite in enamel in the presence of calcium and phosphate ions produced during enamel demineralization by plaque bacterial organic acids.[7]

Application of fluoride varnish

This is now believed to be the major mechanism of fluoride ion's action in preventing enamel demineralization. Fluoride ions can also drive the remineralization of previously demineralized enamel if enough salivary or plaque calcium and phosphate ions are available when the fluoride is applied. The non-invasive treatment of early caries lesions by remineralization has the potential to be a major advance in the clinical management of the disease. However, for every two fluoride ions, 10 calcium ions and six phosphate ions are required to form one unit cell of fluorapatite. Hence on topical application of fluoride ions, the availability of calcium and phosphate ions can be the limiting factor for net enamel remineralization to occur and this is highly exacerbated under xerostomic conditions.[8] When adequate levels of calcium and phosphate ions are together with the fluoride ions, it has been shown in vitro that this combination can produce substantial remineralization of lesions of enamel and even those penetrating the underlying dentin.[9]

Calcium based products:

Fluoride alone cannot achieve remineralization; calcium and phosphate ions are necessary for remineralization to occur. The calcium and phosphates in saliva are the primary source for remineralization. Remineralizing agents seek to promote remineralization through increase of bioavailable calcium and phosphate ions that become incorporated in the tooth structure. Supplementing calcium and phosphates is likely to have a positive effect, in particular when effective fluoride levels are available at the same time.[5]

Various calcium based systems are:

Crystalline calcium phosphate remineralizing systems: Calcium phosphate can exist in one of numerous crystalline phases. Each of these crystalline phases has different solubilities, and many have been tested as potential methods of delivering calcium and phosphate ions to subsurface enamel lesions. The problem with applying crystalline material to the oral cavity to promote enamel remineralization is the poor solubility of the calcium phosphate phases, such that the calcium and phosphate ions are unavailable for remineralization. These crystalline calcium phosphate phases must be released from the product on contact with saliva and then dissolve in that fluid to liberate ions capable of diffusing into the enamel subsurface lesion. The dissolution of the calcium phosphate phase in saliva requires that saliva be undersaturated with respect to that crystalline phase. Based on some typical concentrations of calcium, phosphate, and fluoride ions in saliva, the pH at which the various crystalline phases will dissolve has been calculated. These calculations show that, at the normal pH range of saliva, these crystalline calcium phosphate phases would not dissolve. Furthermore, localization of significant quantities of solid calcium phosphate phases at the tooth surface is problematic.[4]

Casein phosphopeptides (CPP): The casein phosphopeptides (CPP) are approximately 10% (w/w) of the protein casein. They are tasteless, have low antigenicity, and can be purified as CPP-ACP(casein phosphopeptide – amorphous calcium phosphate) complexes from a casein enzymic digest by filtration.[4] Four major bovine CPPs containing the sequence –Ser(P) – Ser(P) – Ser(P) – Glu – Glu –, where Ser(P) represents a phosphoseryl residue, have been shown to stabilize high concentrations of calcium and phosphate ions in metastable solution supersaturated with respect to the calcium phosphate solid phases at acidic and basic pH.[10] This interaction prevents growth of the calcium and phosphate ion clusters to the critical size required for nucleation and phase transformations.[11]

Potential benefits have been shown for casein phophopeptides, amorphous calcium phosphates, and other approaches. Inhibition of enamel and dentin demineralization, promotion of remineralization, and a slow – down of the caries process as well as regression of subsurface lesions have been reported for casein phosphopeptide – amorphous calcium phosphate(CCP – ACP).[12]

The main function of casein phosphopeptides is to modulate bioavailability of calcium phosphate levels by maintaining ionic phosphate and calcium supersaturation to increase remineralization. The role of ACP is also said to control the precipitation of CPP with calcium and phosphate ions.[13]

Combined with fluoride, CPP – ACP has an additive effect on caries activity. Use of CPP – ACP along with fluoride – containing dentifrice has proved to be beneficial in reducing the demineralization around orthodontic brackets and remineralizing white spots caused by demineralization. Adding CPP – ACP to soft drinks can reduce their erosion capacity.[2]

Mechanism of action for CPP – ACP : CPP-ACP has been determined to be amorphous electroneutral nanocomplexes with a hydrodynamic radius of 1.53 + 0.04 nm and 2.12 + 0.26 nm, respectively. From the size and electroneutrality of the nanocomplexes, it would be

expected that they would enter the porosities of an enamel subsurface lesion and diffuse down concentration gradients into the body of the subsurface lesion. Recently, it has been shown, with confocal laser microscopy and fluorescently labeled anti-CPP antibodies, that CPP was present inside a CPP-ACP remineralized enamel subsurface lesion. Once present in the enamel subsurface lesion, the CPP-ACP would release the weakly bound calcium and phosphate ions, which would then deposit into crystal voids.[4]

Calcium sodium phosphosilicate : It is another new agent that reacts with an aqueous environment and releases calcium and phosphate ions. It is used as a desensitizer and approved as hypersensitivity agent. Off–label use as remineralizing agent is promoted, but simultaneous delivery of the right amounts of calcium, phosphate, and fluoride ions at the same time and location might be problematic and cause undesired adverse effects.[14]

Sugar substitutes:

A non-fermentable sugar alcohol acts as a carrier or reservoir for calcium phosphates. A sugar free gum containing xylitol produces superior remineralization. The addition of fluoride to xylitol is said to provide additional benefit, assuming the fluoride concentration is more than 0.8 ppm. Besides fluoride, calcium lactate also enhances remineralization when added to xylitol. Sorbitol is another sugar substitute that is used as an artificial sweetener. The abilities of xylitol and sorbitol to remineralize early enamel caries seem to be almost similar. Isomalt is a non cariogenic sweetener that is widely used as a sugar substitute. Adding isomalt to a demineralizing solution has shown to significantly reduce tooth mineral loss.[2]

Ozone: ozone is a chemical compound consisting of three oxygen atoms. Ozone therapy is usually advocated in dentistry for sterilization of cavities, root canals, periodontal pockets, and herpetic lesions. Ozone therapy is also proposed to stimulate remineralization of incipient caries following treatment for a period of about 6 to 8 weeks.[2,15]

Hydroxyapatite: Carbonate hydroxyapatite nanocrystals, having size, morphology, chemical composition, and crystallinity comparable to that of dentin, are said to mineralize enamel. Hydroxyapatite has been used in toothpastes and pit and fissure sealants.[2]

Future Trends:

Various other approaches that have been suggested include : microabrasion[16], acid etching, bleaching/deproteination, or a combination approach such as bleaching and etching.[17] Bleaching appears to be an effective method of deproteinating the lesion surface to increase porosity inter-prismatically without the need for acid etching. Another approach to improve current remineralization systems is to improve the biomimetic peptides used to stabilize, deliver, and control remineralization.[4]

Conclusion:

Minimally invasive treatment of non – cavitated carious lesions involves remineralization of the tooth structures with flourapatite or fluorhydroxyapatite. In individuals at risk of disease, these procedures should be instituted to prevent the prevent the onset of disease and in those in whom disease is already present, for the treatment. At the individual patient level, there is a lot of variation between the factors involved in lesion development. Thus, it is important to assess the caries risk of the patients for successful management strategy.

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