Non-Destructive Caries Management : Reviewing Novel Approach in M.I.D.

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Abstract

The most conventional method for treatment of dental caries remains largely focused on use of surgical intervention i.e. restorative treatment without consistent and individualized consideration of underlying disease process for that patient. Evidence regarding effectiveness of restorative care in preventing and managing dental caries disease process is limited. A new approach in minimal invasive dentistry is to use non-destructive risk based caries management strategies by applying best scientific evidence available rather than to continue using traditional restorative only approach based on irreversible procedure. In order to accelerate the implementation of this approach for diagnosis and management of dental caries, it is very important to review our understanding of the caries process, the dynamic process of demineralization and remineralization of tooth structure and strategies for non-invasive demineralized tissue repair in precavitated/non-cavitated lesion.

Introduction

Worldwide spread, dental caries, is one of the predominant bacterial disease of the oral cavity. According to current glossary it is defined as the localized destruction of susceptible dental hard tissues by acidic byproducts from bacterial fermentation of dietary carbohydrates.[1] This multifactorial dynamic disease resulting from imbalance in the physiologic equilibrium between tooth mineral and the plaque fluid can be arrested at any point in time. Proper diagnosis of dental caries whether active or arrested not only aids in management and decision making but also guides the risk of developing new lesions in future.[2,3]

Etiologic Factors for Dental Caries Process

Diverse but characteristic oral microflora varies in composition significantly at distinct surface within mouth due to difference in environmental conditions.[4] Saliva plays a major role in maintaining oral pH at approximate neutrality which is optimal for growth of majority of microorganisms associated with oral health.

Role of Dental Plaque in Caries Disease Process

Various hypothesis regarding etiology of caries process have been listed to till date. Dental caries is viewed as a consequence of an imbalance in resident microflora due to enrichment within microbial community of potentially more highly cariogenic bacteria due to frequent conditions of low pH in biofilms. Conditions of low pH favor the proliferation of acidogenic bacteria tipping the balance towards demineralization, thereby disrupting the ecology of the biofilm.[5,6] Strategies which prevent the caries process via the principles of ecological plaque hypothesis including the extended version augment the conventional effective oral hygiene practices/maintenance.

Role of Saliva, Pellicle, Diet and Hard Tissue of Tooth in Caries Process

Saliva's protective role is mediated by its ability to clear cariogenic food substances from mouth, dilute, neutralize and buffer organic acids formed by biofilm microorganisms, and reduce demineralization rate enhancing remineralization by providing Ca, phosphate and fluoride in fluid phase of biofilm which is closely associated with tooth surface. Salivary proteins are suggested to be used as key biomarkers for several systemic diseases including dental caries.[7]

Acquired pellicle an acellular, bacteria free organic film occupies a position between enamel surface and dental biofilm. When pellicle reaches complete thickness approximately of 1micron m in 2 hrs there still exists a continuing maturation process modifying its characteristic as a diffusion barrier to ionic conductivity on the enamel surface.[8] So, the enzymatic activity promotes structural remodeling of acquired pellicle during maturation process is important for stabilizing and creating a more acid resistant pellicle.[9] The protein component contributes to protection of enamel tissues from acid induced demineralization.[10]

Diet i.e. fermentable carbohydrates still remains the main driver of the caries process. Simple sugars are considered to be more cariogenic as compared to complex carbohydrates with sucrose being most. Therefore, it has been implicated as an important determinant of dental caries disease.[11] Today, change in the dietary pattern and increase in use of fluoride has definitely modified the old relationship of diet with dental caries.[12]

Multiple factors like location, morphology, composition and structural composition of tooth dictates the carious process. Caries resistance is also affected by enamel crystal size, shape and proximity. Large and more uniform crystals having less specific surface area are less acid soluble. The crystals which are closely packed have less space for water diffusion thereby reducing acid solubility.

Enamel undoubtedly is most at-risk surface for caries lesion but of clinical importance is the fact that dentin is more soluble than enamel with a clinical pH value of 6.2 to 6.7.

De-Mineralization /Remineralization Thermodynamics in Enamel and Dentin

When there is amount of mineral loss for a significant amount of time, a caries lesion tends to develop in enamel surface. Caries lesion develops at the subsurface because crystals there are more susceptible to under saturation conditions created by diffusion of H+ from plaque fluid. Fluoride also has impact on surface layer by forming crystal areas of less soluble fluoridated hydroxyapatite being acid resistant and requiring low saturation levels before it starts to dissolve.[13,14] Caries lesion therefore develops into dentin without breakage of the surface.[15] Since bacteria's are too large to fit into diffusion spaces of intact surface layer, such lesions are bacteria free.

Under favourable conditions remineralization of non-cavitated lesion surface is more likely to occur first leading to blockage of communication channels that allowed demineralization of subsurface of lesion. These particular dynamics lead to formation of remineralized surface that is highly resistant to demineralization leaving a sealed partially demineralized subsurface lesion. As long as saturation conditions in the biofilms covering those lesions do not return to cariogenicity, lesions do not progress and teeth are fully functional. So, conditions leading to growth of undisturbed biofilms create site-specific, high risk situations which might lead to development of carious lesions.

Dentinal crystals are small and more reactive than those in enamel. Tubular sclerosis that starts to develop before clear enamel demineralization has reached DEJ is the first sign of dentinal reaction to caries process as seen at microscopic level.[16] Caries lesion spread laterally after reaching DEJ but evidences suggest that lesions follow axial direction of the enamel lesion though lateral spread might happen after cavitations. Due to diffusion related dynamics, path of diffusion typically is direction of enamel rods and dentinal tubules. Lesions can advance to significant levels of demineralization in dentin without cavitations of the surface.[17]

Management Strategies for Non-invasive Tissue Repair

Therapies that focus on rebalancing the interplay between demineralization and remineralization, tipping the balance toward an overriding mineral uptake in the tissue not only results in repair of the damage done but concurrently assist in preventing new lesions

of forming. Treatment and management of non invasive tissue repair strategies should be based on interpretation of activity of lesion and future caries risk of the patient .Lesion activity can be halted with several means like diet modification, general/targeted antibacterial strategies, plaque removal and plaque reducing strategies, stimulation of salivary flow, and sealing of lesions leading to reduction or elimination of acids attacks on the tooth surface. By assisting these natural processes in the presence of normal saliva, dynamics of ion exchange between hard tissues and ambient plaque fluids changes and there is automatic reduction in demineralization resulting in remineralization. Another preventive approach to halt the caries process involves disturbing/reducing the living environment of living microflora. The basic tooth cleaning mechanism is helpful in keeping the biomass of acidogenic and acid tolerant microflora under control. Increase in amount of bio available ions in saliva also drives the remineralization process. Several caries management strategies rely on providing additional fluoride, Calcium or phosphate ions to saliva with intention to deliver ample supply of ions to immediate caries active environment.

Management Strategies by Fluoride

The mechanism by which fluoride inhibits demineralization is facilitating re-precipitation of dissolved calcium and phosphate ions on the remaining crystals. This mechanism prevents the tissue ions from being leached out to environment into plaque and saliva. The precipitated ions at the tooth surface decrease pores in the enamel, obstructs the diffusion pathways for plaque acids and hamper acid penetration into the enamel. When the ambient pH is higher than 5.5, fluoride will facilitate remineralization promoting lesion to arrest and enhance repair.[18]

Topical Home Fluorides

Fluoridated toothpaste remains an efficient tool and a cost effective measure for caries control. As a basic preventive method, the concentration in over the counter fluoride toothpastes in India ranges from 1-2.5mg/gm. In caries active patient it is important to increase the fluoride therapy by intense use (at least 3 times/day) of fluoridated toothpaste until caries rate is under control. Studies have shown that no-rinse method after use of fluoridated toothpaste results in 26% reduction in proximal caries incidence.[19] Therapeutic use of toothpaste can also be attained by applying the paste locally on cleaned active caries lesion before going to bed. At home fluoride rinses like 0.02% NaF used daily for 1 minute may benefit adults with active caries who have difficulty cleaning their teeth adequately.

In-office Topical Fluoride Application

Professional application of topical fluoride is an effective approach to caries control. The ADA council on scientific affairs developed evidence based clinical recommendations for professionally applied topical fluoride as summarized in table-1.[20] Fluoride gel and foams should be applied for 4 minutes containing 10,000-12,300 ppm. The council on scientific affairs found insufficient evidence to address whether there is difference between NaF and acidulated phosphate fluoride gels. A strict control over remaining plaque after everyday brushing is very crucial for positive outcome. To attain this twice daily fluoride (0.05% NaF) rinses or dual rinsing strategy of Fluoride and CHX gluconate (0.05% NaF with 0.2% CHX) rinses could be helpful to suppress the oral microflora. Concentration of fluoride in recommended fluoridated mouth rinses is 230ppm.[21]

Calcium Based Strategies

Calcium and phosphate in saliva are primary source for recrystallizing minerals. Remineralizing agents seek to promote remineralization by increase of bioavailable calcium and phosphate ions which get incorporated in tooth structure. Calcium and phosphate supplements have positive effect particularly when effective fluoride levels are available at same time. Promotion of remineralization, inhibition of enamel and dentin demineralization, slow down of caries process as well as regression of subsurface lesions is reported for Casein phospho-peptide-amorphous Calcium phosphate (CCP-ACP).[22] Calcium sodium phospho-silicate bioactive glass is another agent which reacts with an aqueous environment and releases calcium and phosphate ions. It is used as a desensitizer and hypersensitivity agent. Researches and further studies are required to support its claims of caries prevention and remineralization. Another emerging calcium based formulation of calcium phosphate solution are entering in market which mark to reduce root caries and ACP technologies which can remineralize hard tooth tissues or slow down the process of demineralization.[22]

Resin Sealants

They remain the proven and new effective management strategy for dental tissue repair.[23] Their effectiveness in managing non-cavitated and cavitated lesions is overwhelming. Sealants protect underlying surface by blocking renewed and continuous attacks by plaque acids, by preventing plaque accumulation and dissolution of minerals from dental hard tissues. They show benefit after placement even when partially lost. Caries risk in formerly sealed cavities is found to be less as compared to those which were never sealed.[24] Sealing of cavitated lesions significantly reduced bacterial levels (50 to 99% of mean bacterial counts) and this effect increased with time.[25] Cavitated but sealed frank caries lesions have shown not to progress over a period of 10 years follow up.[26] Centers for disease control and prevention group (CDC) has recently recommended sealing sound surfaces and non cavitated lesions to provide sealants to children even if follow up cannot be ensured. As strongly sealing the caries process and sealing of restorations appear highly effective conserving sound tissues and protecting them against caries progression.[27]

Resin Infilteration of The Caries Lesions

This is a recent development in which subsurface porosity in lesion is filled with resin to strengthen the area. Eventually, after sealing the caries lesion the progression of lesion is halted. Once porous, demineralized enamel is filled with resin its refractory index gets changed. Lesion becomes less opaque and less visible as it gains translucency. Superficial surface layer gets eroded using hydrochloric acid so that underlying pores are opened up and this low viscosity resin is able to penetrate in demineralized tissue. Drawback of this technique is the need of erosion of intact surface layer which gets replaced by resin. This infiltrated lesion makes natural remineralization therapy impossible. Though the short term results which used this enticing concept is overwhelming but until proper clinical trials are presented this novel approach should be cautiously used in conjunction with other preventive strategies.

Management Strategies by use of Lasers

Since last decade, lasers are known application in caries research. Carbon-di-oxide laser irradiation in combination with fluoride treatment has shown to be effective in inhibiting caries like lesions. This technology is still emerging and there is no strong evidence of anti-caries efficacy. More clinical trials and research are needed before implementation of this novel technique for effective caries control.

Summary

With increasing number of technologies aimed at enhancing tooth remineralization, fluoride remains most widely used agent for managing caries process supported by strong evidences. Management strategies should be based upon interpretation of lesion activity and future caries risk of patient. Active monitoring of caries lesion activity, patient compliance with behavioral modification and co-operation remain the essential tools of successful caries management. New emerging technologies should always be welcomed in adjunct to fluoride treatments with evidenced caries preventive and therapeutic efficacy.

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In office topical fluoride

Evidence-Based Clinical Recommendations for Professionally Applied Topical Fluoride

Risk Category	Age category for recall patients					
	<6 years		6-18 years		18+ years	
	Recommendation	Strength	Recommendation	Strength	Recommendation	Strength
Low	May not receive additional benefit from topical fluoride application.	B	May not receive additional benefit from topical fluoride application.	B	May not receive additional benefit from topical fluoride application.	D
Moderate	Varnish application at 6-month intervals	A	Varnish application at 6-month intervals Or Fluoride gel application at 6-month intervals	A A	Varnish application at 6-month intervals Or Fluoride gel application at 6-month intervals	D D
High	Varnish application at 6-month intervals Or Varnish application at 3-month intervals	A D	Varnish application at 6-month intervals Or Varnish application at 3-month intervals Or Fluoride gel application at 6-month intervals Or Fluoride gel application at 3-month intervals	A A A D	Varnish application at 6-month intervals Or Varnish application at 3-month intervals Or Fluoride gel application at 6-month intervals Or Fluoride gel application at 3-month intervals	D D D D

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