REVIEW ARTICLE

Root Caries: Etiopathogenesis and Management

Jogikalmat Krithikadatta[1], Aruna Krishnan[2]

Received on: 01 March 2023; *Accepted on*: 01 December 2023; *Published on*: 03 February 2024

Abstract

Root caries is a type of dental decay that affects the roots of teeth. Unlike traditional dental caries, which typically affect the enamel and dentin of the tooth, root caries occurs when the roots of teeth become exposed due to receding gums or other factors. This can leave the softer, more vulnerable root tissue exposed to bacteria and acid, leading to decay and eventual tooth loss if left untreated. Root caries is a common problem for older adults, as well as people with certain medical conditions or medications that can affect oral health. Treatment for root caries typically involves removing the decayed tissue and restoring the tooth with a filling or crown. However, prevention is the best approach, and good oral hygiene, a healthy diet, and regular dental checkups can all help to reduce the risk of developing root caries. In recent years, researchers have also been exploring new approaches to preventing and treating root caries, such as using fluoride varnishes or other topical treatments to strengthen the tooth enamel and reduce the risk of decay. With proper care and attention, it is possible to prevent and manage root caries, helping to maintain healthy teeth and gums throughout life.

Keywords: Biofilm, Etiology, Management, Prevention, Root caries, Root decay.

Journal of Operative Dentistry and Endodontics (2022): 10.5005/jp-journals-10047-0124

Introduction

What is Root Caries?

By 2050, the World Health Organization predicts that there will be a considerable increase in the elderly population due to longer life expectancies, with almost 1.5 billion people—roughly 15–20% of the world's population—being 65 years of age or older.[1] The quality of life of older persons is significantly influenced by their medical and dental conditions. In the realm of dentistry, periodontal disease and dental caries, particularly dental root caries, are regarded as the key issues in older persons. Root caries has received more attention in the last two decades as a result of research demonstrating the high prevalence of root caries in populations all over the world. It is now known that one-third of the elderly population bears the majority of the root caries burden. With the increase in life expectancy and the retention of natural teeth among older adults, root caries is expected to become a significant public health problem. Sumney et al. defined root caries as "a cavitation below the cemento-enamel junction, not usually including the adjacent enamel, usually discolored, softened, ill-defined, and involving both cementum and underlying dentine." The retention of natural teeth among elders is longer due to advancement and development of oral healthcare and treatments. Accordingly, the elderly's oral health issues are moving away from full tooth loss and toward untreated carious lesions. Additionally, elderly individuals typically experience gingival recession, and the exposed root surfaces raise the possibility of developing root carious lesions.

Root caries is a multifactorial disease with risk factors such as poor oral hygiene, patients with gingival and periodontal problems, xerostomia, medication, lifestyle factors such as tobacco and alcohol consumption, the frequency of carbohydrate consumption, low fluoride exposure, proximity to dentures, limited manual dexterity for plaque control, and so on. Root surface is more susceptible to caries formation than crown because of the difference in the composition. Inorganic content of dentin is approximately 70% whereas that of enamel is 90%. This compositional difference accelerates the progression of cavities in the root dentin. Preventive methods for root caries focus on the topical application of fluoride agents such as silver diamine fluoride (SDF), sodium fluoride and fluoridated toothpaste. However, the limited effect of fluoride in preventing root caries has led to the exploration of new preventive therapies, including recently developed functional peptides, phosphate-based technologies, and biomodification of dentin extracellular matrix (ECM).

Once a cavitated root caries lesion is formed, treatment strategy is mainly based on the location of the lesion, access to the lesion, proximity to the pulp tissue, and moisture control. For restoring the cavitated lesion materials like glass ionomer cement (GIC), resin modified GIC, and composite can be used. Since dentin has higher moisture content than enamel, resin-based adhesive materials are not the best choice for restoration. This review focuses on the epidemiology, etiopathogenesis, clinical features, preventive methods, conventional intervention methods, and recent strategies in the management of root caries in brief.

Epidemiology

Many authors have reported an increase in the prevalence of root caries as a result of increase in life expectancy and dentition. Given the variation and heterogeneity among the studies included, the presented estimates should be interpreted with caution. The studies' high heterogeneity could be attributed to geographical

[1,2]Department of Conservative Dentistry and Endodontics, Saveetha Dental College and Hospital, Chennai, Tamil Nadu, India

Corresponding Author: Jogikalmat Krithikadatta, Department of Conservative Dentistry and Endodontics, Saveetha Dental College and Hospital, Chennai, Tamil Nadu, India, e-mail: Krithikadatta.sdc@saveetha.com

How to cite this article: Krithikadatta J, Krishnan A. Root Caries: Etiopathogenesis and Management. J Oper Dent Endod 2022;7(2): 38–43.

Source of support: Nil

Conflict of interest: None

differences or different criteria used to assess the prevalence of root caries and difference in the duration of follow up across the studies that were included.[2] Numerous variations in the prevalence of root caries (25–100%) and the mean Root Caries Index have been observed in published studies (9.7–38.7).[3] We can conclude that the prevalence of root caries was 41.5% overall.[4] Future studies on the prevalence of root caries should follow the standard guidelines of "Strengthening the Reporting of Observational Studies in Epidemiology (STROBE)" and widely accepted criteria. The incidence of root caries refers to the proportion of people who develop new root caries over a given time period. Annual root caries incidence has been observed to range from 10.1 to 40.6%.[3]

Etiopathogenesis

The over-65 age group is predisposed to a higher prevalence of root caries due to an increase in exposed root surfaces compared to younger population. The cementum layer is frequently abraded away in a population that is frequently subjected to scaling by dental health professionals, exposing the dentine. Dentine and root cementum have structural differences from enamel and respond to cariogenic challenges in distinct ways. For example, the critical pH of dentine and root cementum is roughly 6.4,[5] whereas that of enamel is 5.5.[6] Etiology of root caries is multifactorial includes anatomical, iatrogenic, and gingivitis and periodontitis related variables. Gingival recession, poor oral hygiene, inadequate biofilm control procedures, presence of fermentable carbohydrates and cariogenic bacteria on the root surface are the cause of the onset and progression of root caries. Numerous factors influence the onset and progression of root carious lesions, including old age, medications, comorbidities such as xerostomia, lifestyle factors such as tobacco and alcohol consumption, the frequency of carbohydrate consumption, low fluoride exposure, proximity to dentures, limited manual dexterity for plaque control, and so on.[7] Root caries was positively correlated with age, gingival recession, and tobacco use, while it displayed negative correlation with social status, the use of fluoride toothpaste, and oral hygiene status.[8] The relationship with the number of teeth and dental visit behaviors showed mixed results in the findings. The emergence of root caries has been linked to *Lactobacillus*, *Streptococcus mutans*, and *Actinomyces*.[6,9] However, not all research on these microbes have discovered a strong link between their presence in saliva and root caries. Root caries progression happens in two stage. First stage is characterized by dissolution of minerals and the second stage is degradation of organic matrix. Cementum contains 45–50% inorganic substance mainly calcium and phosphate in the form of hydroxyapatite. It contains 50% organic substance which primarily has type 1 collagen and protein polysaccharide. In comparison with enamel, root hard tissues have less mineral content, more magnesium, and carbonate accounting for their increased crystal solubility and smaller size of hydroxyapatite crystals.

Demineralization

After gingival recession, the areas where Sharpey's fibers are attached is gradually exposed and converted into canals paving way for the microbial penetration to occur. In the presence of high cariogenic diet, the equilibrium between oral fluids and tooth surface is disrupted resulting in more demineralization. The critical pH for root hard tissue range from 6.8 to 6.0 which is higher than enamel critical pH of 5.4.[10] Similarly, it has been proposed that starch product digestion by dental biofilm bacteria is sufficient to cause root caries but not enamel caries.

Degradation of Organic Matrix

Next stage of caries progression is degradation of collagen by the endogenous and exogenous collagenase and proteases. Root dentin contains matrix metalloproteinases which are secreted as inactive proenzymes (zymogens), with the prodomain preventing the catalytic domain from functioning. When the prodomain bridge with the catalytic zinc (the "cysteine switch") is disrupted by other MMPs, cysteine cathepsins, or other proteinases, or chemically, such as pH changes, activation occurs.[11] Another important family of proteases, the cysteine cathepsins, was recently discovered in dentin.[12,13] These enzymes are involved in degradation of ECM. Demineralized collagen acts as a scaffold for bacteria colonization. Pathologic stimuli on dentino-pulp complex produces tertiary dentin. Milder stimuli produce reactionary dentin with traces of dentinal tubules. Severe pathologic stimuli produces reparative dentin which is atubular, may have a bone-like structure (osteodentin), and may have traces of cells in its matrix due to its accelerated deposition.

Nevertheless, inadequate oral hygiene coupled with increased sugar consumption and decreased saliva production can trigger a series of developments where the biofilm accumulates and matures, leading to the acidification of the local environment and the proliferation of acid-producing and acid-resistant bacteria. As a consequence, this disrupts the natural balance between demineralization and remineralization, tipping the scales in favor of overall demineralization.[14] This leads to exposure of collagen and other host proteins which serve as substrates for proteolytic bacteria such as gram-negative and obligate anaerobic bacteria. Together, these species collaborate to break down both the inorganic and organic elements of the dental tissues.[14]

Why is Root Caries Different?

Clinically, Caries on the root surface ranges from small, softened, and discolored spots to extensive, brownish or very dark soft areas encircling the entire root surface and it spreads laterally giving a saucer-shaped appearance. Root caries lesions are classified as "active" and "inactive" lesions for treatment purposes.[15] Active lesion appear yellowish to lightish brown with a dull or matte surface typically covered by a biofilm. On gentle probing, it feels soft and leathery, and the margins are sharply demarcated. Inactive lesion appears yellowish, dark brown or black with a smooth and shiny surface. On gentle probing, it feels hard and margins of cavity are smooth. For the purpose of root caries restoration, it is important to understand cavitated lesion which presents itself in two forms. In the first form, many lesions are shallow and saucer-shaped, encircling much of the root surface. However, in the second form, the lesions may occasionally have a deeper portion that is difficult to cleanse namely a distinct cavity. These lesions are frequently found on the proximal surfaces of molars, just below the gingival margin, where a toothbrush cannot reach the lesion, especially the deepest parts with a high probability of pulp exposure. Visual and tactile detections are frequently difficult, and cavitated lesions may only be discovered after viewing a bitewing radiograph. The primary method for detecting and tracking root caries lesions in clinical practice relies on a combination of characteristics such as hardness, location, cavitation, and shine, which serve as indicators for both the presence and the current state of these lesions.[16]

Histopathology

In contrast to dentin from young teeth, which contains tubule lumens that are clearly open, older teeth's dentin has a higher rate of occluded tubules (dentin sclerosis). The development of

secondary dentin and tubule sclerosis cause a significant reduction in fluid flow inside the dentin tubules,[17] which in turn causes an age-related decline in pulp sensitivity. Pathologic stimuli like caries lesions stimulate the synthesis of tertiary dentin. Depending on the intensity of pathologic stimuli tertiary dentin can be reactionary or reparative. Milder stimuli initiate reactionary dentin formation by stimulating primary odontoblast in dentin-pulp complex. This leads to deposition of tertiary dentin onto the pulpal walls. Due to the fact that odontoblasts make reactionary dentin, it also exhibits signs of dentin tubules. Conversely, severe pathologic stimuli initiates reparative dentin formation by destruction of primary odontoblast layer and stimulation of mesenchymal cells in the pulp. As a result of its rapid deposition, reparative dentin lacks dentin tubules, may resemble bone (osteodentin), and may have cell remnants in its matrix. The cementum layer is the narrowest in the cervical areas of the tooth varying from 54 μm (at ages between 11 and 20 years) to 128 μm (between 51 and 76 years).[18] The dentin tubules contain bacteria at the earliest stages of the formation of root caries due to the thin layer of cementum at the cervical areas. If cementum is still present during the early phases of RC lesion formation, tiny clefts appear along this tissue, which becomes filled with bacteria. The demineralization causes the clefts to widen as the microorganisms ingest deeper into the cementum, exposing the underlying dentin. Consequently, a subsurface demineralization that can be seen as a radiolucent area on radiographs is another characteristic of the early RC lesions. About 150 μm below the cemento-dentinal junction, this immediate subjacent dentin primarily consists of sclerotic/atubular dentin. The early demineralization results in the loss of minerals from the atubular dentin but largely preserves the collagen-containing organic matrix. In advanced carious lesion, microorganism penetrates deeper resulting in the intertubular dentin's mineralization being lost and the inner portion of the peritubular dentin being destroyed. In inactivated lesions, the dentin tubules exhibit intratubular mineralization with an uneven precipitation pattern, and microbial ghost cells are found in between these crystals.

Management Strategies

Preventive Methods

The presence of fermentable carbohydrates in the thick biofilm is the primary etiological factor of dental caries. The removal of this colony on a regular and systematic basis using an effective biofilm control should result in the prevention of caries lesions or the arrest of the local carious process. Cervical active root caries is often characterized as a relatively rapid process that results in the loss of protective enamel near the cement-enamel junction at the crown of the tooth. The presence of overhanging enamel in this area encourages the accumulation of biofilm, making it necessary to conduct regular check-ups to reencourage patients to maintain proper oral hygiene as the primary approach for managing Root Caries Lesion.[19] Root caries progress shallowly rather than deeply giving a saucer-shaped configuration which makes it easier for the mechanical removal and control of biofilm.[20] Mechanical biofilm control by means of effective tooth brushing and by using fluoridated toothpaste. In 6–8 month clinical trials, there is strong evidence that 5000 ppm F toothpaste is more effective in arresting root canal length (RCLs) (by increasing hardness) and preventing new lesions (PF of 51%) compared to 1100–1450 ppm F toothpastes.[21] The use of 5000 ppm F toothpaste twice a day increases fluoride concentrations in saliva and biofilm, as well as the formation of CaF2 on teeth, while decreasing biofilm formation and *S. mutans* and *Lactobacillus* levels. It has been demonstrated that 5% NaF varnish and 38% silver SDF can prevent the emergence of new RCLs in 64 and 71% of cases, respectively. The effectiveness of SDF might be attributed to its antibacterial properties and its ability to promote remineralization.[22] Furthermore, SDF is effective in preventing the breakdown of collagen in dentin by inhibiting the actions of collagenases and cysteine-cathepsins.[23] Additionally, SDF is recognized for its capacity to alleviate tooth sensitivity in cases of hypersensitivity.[23] The use of SDF solution was determined to be more cost-effective in comparison to other alternatives such as chlorhexidine varnish and fluoride rinse.[22] A systematic review and meta-analysis pointed out that the professional application of SDF and chlorhexidine prevents and arrests root caries in elderly.[21,24] The main criticism leveled against SDF is that the precipitation, oxidation and dark staining of teeth. The use of 1% chlorhexidine varnish, 5% NaF varnish (both every 3 months), and 38% SDF solution (once a year) for the prevention of RCLs in institutionalized elderly people was also compared.[21] Chlorhexidine, NaF, and SDF have been shown to reduce the development of new RCLs by 56–57, 64, and 71–72%, respectively, with no significant differences observed after 3 years. In a systematic review and meta-analysis, it is concluded that the SDF application prevents and arrests root caries in elderly.[25] Noninvasive management of root caries lesion includes application of fluoride in various forms like dentifrices, mouthwash, varnish, and milk or even incorporating fluoride alongside other active components like triclosan, arginine, casein phosphopeptide-amorphous calcium phosphate (CPP-ACP), calcium sodium phosphosilicate, or supersaturated mineralizing ions. Fluoride's ability to modify the ionic saturation of tooth mineral contributes to its anticaries effect by supporting remineralization and preventing demineralization. Fluoride has the capability to halt and even reverse initial dentin lesions by facilitating the return of calcium and phosphate into the affected area as a less soluble compound known as fluorapatite [Ca5(PO4)3F].[26] The success of fluoride toothpaste is influenced not only by its fluoride content but also by how often it is used. For its impact to be maximized, fluoride needs to be consistently present in the oral fluids.[26]

Conventional Intervention Methods

Mechanical Methods

The management ethos must be centered on minimal intervention techniques aimed at maximum tooth tissue conservation. Cavitated lesions, even up to a depth of about 2 mm, should be evaluated to see if a toothbrush can reliably remove the biofilm on the entire lesion surface. If a patient can consistently remove biofilm, this noninvasive method may be the best form of lesion management. Regular biofilm removal using a high fluoride content toothpaste, such as 5000 ppm, as well as the use of other remineralizing agents such as Ca and PO_4 containing crèmes should be implemented.[24] Furthermore, brushing the softened lesion surface exposes the underlying harder, higher mineral content tooth tissue. The only disadvantage is that the lesion's color will frequently darken over time as the mineral content on the lesion's surface increases and possibly extrinsic stains are incorporated into the arresting lesion. In some cases, where lesions have shallow cavitation but extend beneath the enamel, a minimal surgical approach can be used to expose the dentin lesion by removing only the overlying unsupported enamel with a fine-grit tapered or flame-shaped diamond bur under air-water spray in either a low or intermediate

speed handpiece. Following dentin lesion exposure, the patient is given lesion-specific oral hygiene instructions using high fluoride toothpastes. The exposed dentin lesion could also be treated with a SDF solution; however, the resulting compromised aesthetics must be considered.

Deep Carious Lesion

The goal of treating these deeper lesions should be to preserve dentin that may still contain bacteria in a cavity beneath a sealed restoration. Care should be taken to avoid pulp exposure while excavating deep caries lesion with slow speed burs or hand instruments. Lesions on the labial/buccal surface of a tooth are typically managed conservatively by excavating only the deeper, non-cleansable portion of the lesion. For lesions located on the proximal surface, careful examination of the lesion should be made. If the lesion is visible from buccal or lingual embrasure, caries excavation can be accessed from the embrasure. If the lesion is not visible, it may be necessary to access the lesion through the marginal ridge from the occlusal surface. This results in destruction of the tooth structure and the gingival floor is usually quite narrow, the restoration is frequently not well supported at the gingival margin. Matrix positioning and placement is frequently difficult, making material insertion into the deep narrow cavity challenging. The root's curvature and frequently indistinct lesion borders make restoration placement and completion difficult.

Chemical Methods

Although chemomechanical caries excavation is not widely used in the profession, it is a useful and simple option. There are two broad types of solutions available. The first one is Carisolv, a sodium hypochlorite-based material. Carisolv has been available for many years, and its formulation has gone through several iterations. The second is Papacarie, an enzyme-based solution from papaya tree. Recent research has shown that both of these materials can be effective in removing denatured carious dentin, frequently leaving a thicker layer of mineral depleted carious dentin than rotary instrument excavation.[24] As a result, the chemomechanical method can provide a more conservative cavity preparation with a lower risk of pulp exposure. This method does not necessitate the use of specialized equipment such as handpieces. Both are gels that allow for easy placement into a cavity and removal of dentin with hand instruments without the need for local analgesia. This method is ideal for treating caries in patients who are unable to visit a dental office, such as home-bound special needs patients or the elderly.

Restorative Material Selection

The most commonly used restorative materials for root caries are glass ionomer cements (GIC), either the conventional or resin-modified GIC, or resin-based materials with the use of an enamel/dentin bonding agent. The gingival margin position, likelihood of moisture contamination, and potential for gingival bleeding must all be considered when restoring a root lesion. The use of a resin-based system is not recommended where moisture control is difficult to manage. The GIC should be used in such cases. The dentin surface should be treated with polyacrylic acid (PAA) to prepare it to bond to the GIC, as PAA begins to interact with the calcium in the tooth mineral via an ionic interaction, resulting in better adhesion. Polyacrylic acid conditioning also aids in cleaning the cavity and removing any blood or saliva contamination that may inhibit dentin adhesion. Cavity depth is critical for light-cured resin-modified GICs because light intensity attenuation may result in the resin component not curing to its full extent for deep posterior root lesions. Dentin has a lower mineral content and a higher moisture content, resin-based adhesive restorative materials are not the best option. Therefore, for patients with normal saliva flow or where the gingival floor of an approximal cavity extends more than 4.5 mm below the occlusal surface of the tooth, current fast-setting conventional GICs with good strength and higher fluoride release are the material of choice. The application of SDF to the cavity walls prior to restoration placement may be an adjunct treatment to possibly reduce caries formation around the margins of these restorations.[24] However, the application of SDF on cavity walls has yet to be clinically evaluated. A recent laboratory study comparing the progression of artificial dentin caries lesions restored with either a CPP-ACP containing or conventional GIC, with and without the application of 38% SDF to the cavity walls prior to GIC placement, found that both SDF treatment and incorporation of CPP-ACP into the GIC restorative material were able to slow deterioration and demineralization around the restoration margins.[24] However, for patients with reduced salivary flow, GIC because of their susceptibility to surface dissolution appears to be a poor choice. To overcome this GIC has been coated with proprietary resin-coating, for example, G-Coat Plus to slow down the process of dissolution and surface wear. For the deep posterior lesion, sandwich technique can be used with GIC in bottom third and composite in occlusal two-third. Resin composites require pre-treatment with adhesive systems. Presently, there are three types of adhesive systems available: etch-and-rinse adhesives (ER), self-etch adhesives (SE), and universal adhesive systems. Resin composites for restoring cervical lesions can enhance the even distribution of masticatory forces in the cervical area, potentially minimizing additional dental structure wear.[27] But the disadvantage of composite resins are lower retention rate. Based on available literature, the retention of composite resins can be enhanced by active (vigorous) application of adhesive (for both SE and ER adhesives), increasing the number of adhesive coatings (SE and ER), placing a hydrophobic coating in one-step SE adhesives, selective enamel etching for SE adhesives, and dentin pre-treatment with EDTA for SE adhesives.[27] Approaches that have the potential to enhance the quality and longevity of the composite resin restorations, yet require additional research to be fully validated are dentin roughening, use of MMP-inhibitors after acid etching for ER adhesives and enamel beveling.[27]

Recent Strategies

The exploration of new preventive therapies, including recently developed functional peptides, phosphate-based technologies, has been spurred by the limited effectiveness of fluoride on root caries. The majority of the specific peptides under investigation are based on the protective actions of salivary proteins which includes

- Statherin-derived peptides **StN21** and **StN15**
- Enamel- and dentin-derived peptide **8DSS**
- A self-assembling peptide P11-4
- Antimicrobial peptide **(AMP)**
- Specifically targeted antimicrobial peptides **(STAMPs)**
- Casein phosphopeptide **(CPP)** combined with amorphous calcium phosphate **(ACP)** known as **CPP-ACP** technology or **Recaldent**
- Dentonin peptide
- **RAD/KLT** peptide

These peptides either promote remineralization or prevent demineralization. The mechanism of action of peptides include binding and nucleation of hydroxyapatite, attraction of calcium ions, buffering actions to neutralize the plaque pH, and antimicrobial actions. The addition of polyphosphate salts to fluoridated vehicles was shown to have a synergistic effect in promoting enamel remineralization and preventing demineralization. However, these effects are mainly based on the results obtained from in vitro studies.

Phosphate-based Technologies

Among polyphosphates, sodium trimetaphosphate (TMP) has been shown to promote synergistic effects against hydroxyapatite dissolution, forming a protective layer that limits acid diffusion and enhances calcium and fluoride diffusion into enamel, phosphorylation of type I collagen in demineralized dentin, and induces intrafibrillar collagen remineralization.[28] Although these therapies appear to be promising alternatives to root caries prevention, more clinical evidence is needed before they can be widely used.

Biomodification of Dentin

Dentin has 70% inorganic, 20% organic content, and 10% water. The extracellular matrix (ECM) of dentin mainly consists of type I collagen scaffold interspersed with non-collagenous components such as proteoglycans, phosphoproteins, and proteases. Dentin biomodification for preventing root caries progression through site modifications of the mature extracellular dentin matrix. Synthetic and biosynthetic compounds can reduce the biodegradability of the dentin ECM and possibly play a role in the effective remineralization of caries-affected root dentin.[29] Host-derived enzymes, endogenous matrix metalloproteinase and cysteine cathepsins play an important role in degradation of dentin ECM.

Synthetic Biomodification of Collagen

Physical or chemical agents can be used to mediate collagen cross-linking with synthetic collagen modifiers. Physical methods rely on photo-oxidation, which is facilitated by light exposure, particularly ultraviolet radiation. The photo-oxidative method necessitates the presence of singlet oxygen, and riboflavin (vitamin B2) is one of the most potent producers of these oxygen radicals. Glutaraldehyde being the most commonly used chemical agent that cross link collagen. One significant disadvantage of this agent is its high cytotoxicity which limits its clinical applicability. Carbodiimide hydrochloride (EDC) is less hazardous than aldehyde and because of its ability to cross-link peptides in collagen without introducing additional linkage groups, EDC is known as a "zero-length" agent.

Biosynthetic Modification of Collagen

Genipin, an iridoid compound, is a naturally occurring biomodifiers. Their slow collagen cross-linking reaction and deep blue tissue staining are disadvantages, limiting clinical applications.[29] Polyphenols are secondary plant metabolic compounds classified into four classes: phenolic acids, flavonoids, stilbenes, and lignans. Flavonoids proanthocyanidins (PACs) are of particular interest for dental applications. Proanthocyanidins have a highly hydroxylated structure, so they can form insoluble complexes with carbohydrates and proteins.[29] Their dental applications have expanded due to their strong inhibition of proteolytic activity, effect on proteoglycans, and mediate reinforcement of the dentin collagen.

Conclusion

The prevalence of root caries in older adults is generally found to be positively correlated with the presence of more retained teeth at older ages.[30] Given that this trend has been demonstrated to be widespread, it is projected that the predicted increase in the aged population will lead to a significant increase in the number of seniors who need access to effective root surface caries prevention and treatment in the near future. So, it is important to understand predisposing factors, risk factors, and etiopathogenesis of root caries for prevention. However, if root caries has occurred, thorough clinical examination of the lesion for diagnosis is necessary. The management of root caries should focus on effective biofilm control procedures and preventing further spread of caries. Efforts should be made to inactivate the active carious lesion. After excavation of the carious lesion, it is to be restored with suitable restorative material. Newer preventive therapies like peptides and phosphates to decrease demineralization and improve remineralization of dentin are to be studied and experimented further. Biomodification of dentin to reduce dissolution of the organic matrix are recent advancements in preventive methods for root caries.

References

1. [No title] [Internet]. [cited 2023 Nov 15]; Available from: https://www.nia.nih.gov/sites/default/files/2017-06/global_health_aging.pdf.
2. Hariyani N, Setyowati D, Spencer AJ, et al. Root caries incidence and increment in the population - A systematic review, meta-analysis and meta-regression of longitudinal studies. J Dent 2018;77:1–7. DOI: 10.1016/j.jdent.2018.06.013.
3. Hayes M, Burke F, Allen PF. Incidence, prevalence and global distribution of root caries. Monogr Oral Sci 2017;26:1–8. DOI: 10.1159/000479301.
4. Pentapati KC, Siddiq H, Yeturu SK. Global and regional estimates of the prevalence of root caries – Systematic review and meta-analysis. Saudi Dent J 2019;31(1):3–15. DOI: 10.1016/j.sdentj.2018.11.008.
5. Mellberg JR. Demineralization and Remineralization of Root Surface Caries. Gerodontology 1986;5(1):25–31.
6. Stephan RM, Miller BF. A quantitative method for evaluating physical and chemical agents which modify production of acids in bacterial plaques on human teeth [Internet]. J Dent Res 1943;22(1):45–51. DOI: 10.1177/00220345430220010601.
7. Doméjean S, Banerjee A. Assessing the risk of developing carious lesions in root surfaces. Root Caries: From Prevalence to Therapy 2017;26. Chapters 55–62. Available from: http://dx.doi.org/10.1159/000479343.
8. Zhang J, Sardana D, Wong MCM, et al. Factors associated with dental root caries: A systematic review. JDR Clin Trans Res 2020;5(1):13–29. Available from: http://dx.doi.org/10.1177/2380084419849045.
9. Hayes M, Burke F, Allen PF. Etiology, risk factors and groups of risk. Monogr Oral Sci 2017;26:9–14. DOI: https://doi.org/10.1159/000479302.
10. Damé-Teixeira N, Parolo CCF, Maltz M. Specificities of caries on root surface. Root caries: From prevalence to therapy 2017;26. pp. 15–25. Available from: http://dx.doi.org/10.1159/000479303.
11. Boukpessi T, Menashi S, Chaussain C. Endogenous enzymes in root caries. Root caries: From prevalence to therapy 2017;26. pp. 35–42. Available from: http://dx.doi.org/10.1159/000479305.
12. Tersariol IL, Geraldeli S, Minciotti CL, et al. Cysteine cathepsins in human dentin-pulp complex [Internet]. J Endodon 2010;36(3):475–81. Available from: http://dx.doi.org/10.1016/j.joen.2009.12.034.
13. Nascimento FD, Minciotti CL, Geraldeli S, et al. Cysteine cathepsins in human carious dentin. J Dent Res 2011;90(4):506–511. Available from: http://dx.doi.org/10.1177/0022034510391906.

14. Do T, Damé-Teixeira N, Naginyte M, et al. Root Surface Biofilms and Caries. Root Caries: From Prevalence to Therapy 2017;26. pp. 26–34. Available from: http://dx.doi.org/10.1159/000479304.
15. Fejerskov O, Nyvad B. Root surface caries – Rationale behind good diagnostic practice. Root caries: from prevalence to therapy 2017;26. pp. 43–54. Available from: http://dx.doi.org/10.1159/000479306.
16. Pretty IA. Monitoring of root caries lesions. Monogr Oral Sci 2017;26:70–75. DOI: 10.1159/000479347.
17. Carvalho TS, Lussi A. Assessment of root caries lesion activity and its histopathological features. Monogr Oral Sci 2017;26:63–69. DOI: 10.1159/000479346.
18. Zander HA, Hurzeler B. Continuous cementum apposition. J Dent Res 1958;37(6):1035–1044. DOI: 10.1177/00220345580370060301.
19. Meyer-Lueckel H, Machiulskiene V, Giacaman RA. How to intervene in the root caries process? Systematic review and meta-analyses. Caries Res 2019;53(6):599–608. DOI: 10.1159/000501588.
20. Maltz M, Alves LS, do Amaral Zenkner JE. Biofilm control and oral hygiene practices. Root caries: From prevalence to therapy. 2017;26. pp. 76–82. Available from: http://dx.doi.org/10.1159/000479348.
21. Magalhães AC. Conventional preventive therapies (fluoride) on root caries lesions. Monogr Oral Sci 2017;26:83–87. DOI: 10.1159/000479349.
22. Zhang J, Sardana D, Li KY, et al. Topical fluoride to prevent root caries: Systematic review with network meta-analysis. J Dent Res 2020;99(5):506–513. DOI: 10.1177/0022034520906384.
23. Oliveira BH, Cunha-Cruz J, Rajendra A, et al. Controlling caries in exposed root surfaces with silver diamine fluoride: A systematic review with meta-analysis. J Am Dent Assoc 2018;149(8):671–679.e1. DOI: 10.1016/j.adaj.2018.03.028.
24. Burrow MF, Stacey MA. Management of cavitated root caries lesions: Minimum intervention and alternatives. Monogr Oral Sci 2017;26: 106–114. DOI: 10.1159/000479352.
25. Grandjean ML, Maccarone NR, McKenna G, et al. Silver Diamine Fluoride (SDF) in the management of root caries in elders: A systematic review and meta-analysis. Swiss Dent J 2021;131(5): 417–424. PMID: 33515230.
26. Cai J, Palamara J, Manton DJ, et al. Status and progress of treatment methods for root caries in the last decade: A literature review. Aust Dent J 2018;63(1):34–54. DOI: 10.1111/adj.12550.
27. Reis A, Soares PV, de Geus J, et al. Clinical performance of root surface restorations. Root caries: From prevalence to therapy 2017;26. Chapter 115–124. Available from: http://dx.doi.org/10.1159/000479353.
28. Buzalaf MAR, Pessan JP. New preventive approaches part I: Functional peptides and other therapies to prevent tooth demineralization. Monogr Oral Sci 2017;26:88–96. DOI: 10.1159/000479350.
29. Bedran-Russo AK, Zamperini CA. New preventive approaches part II: Role of dentin biomodifiers in caries progression. Monogr Oral Sci 2017;26:97–105. DOI: 10.1159/000479351.
30. Carrilho MRO. Concluding remarks. root caries: From prevalence to therapy 2017;26. pp. 125–132. Available from: http://dx.doi.org/10.1159/000479355.