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Review Article

CEMENTUM IN DISEASE - A REVIEW

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A B S T R A C T

A calcified tissue which covers the surface of dentin and the roots of teeth forms the 'cementum'.[1] It has some properties similar to that of alveolar bone and provides a support to the tooth by anchoring the principal fibres to the root surface.[2,3] Alterations in the physical characteristics, as well as chemical composition of cementum may be observed in periodontal disease conditions.[4] Resorption of the cementum causes or removal of the tissue substance, as a result of any pathology, may lead to the exposure of the dentin, thereby leading to dentinal hypersensitivity.[5,6] Hence, damage to the cementum must be prevented, by preventing the periodontal diseases and minimizing the inadvertant removal of the tissue during periodontal therapy.[7,8]

INTRODUCTION

'Cementum' is a specialized calcified tissue of mesenchymal origin which covers the root surface and provides for the attachment of the periodontal fibres to the surface of the root.[1,2]Cementum has been described as a "bone-like structure" and it differs from bone in that it is non-vascularized, non-innervated and does not undergo continuous turn over.[2]It also anchor the principal collagen fibres of the periodontal ligament to the surface of root.[3]

Normally healthy periodontium is the result of a successful interplay among the resident cell populations. Any imbalance in the anabolic-catabolic axis that exists within the healthy periodontium leads to pathological alteration.[9] There are several features which characterize periodontally involved cementum. These include, the presence of calculus and plaque on the surface of cementum or within surface resorption lacunae. The possibility of toxic substances from plaque penetrating into cementum and thus accounting for cemental cytotoxicity is at present uncertain. So, this review aims to discuss the alterations that may be induced in the cementum, due to periodontal diseases and other factors.

Alterations in the Physical Characteristics

Hardness: Hardness refers to the resistance to wear.[2]Periodontally involved cementum has been suggested to be softer than uninvolved cementum. This may be due to the result of proteolysis of the embedded remnants of Sharpey's fibres by bacterial products.[4]

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Cementum exposed to a periodontal pocket may become decalcified after exposure and would then be soft and porous and may result in an unsatisfactory environment for reattachment of periodontal fibers.[1]

Permeability: The permeability of human teeth in vivo has been investigated using radioactive iodine mouth rinses.[10] Following their extraction and sectioning, the teeth revealed some penetration of the iodine into the exposed cervical portions of the roots.[4] It has been reported that endotoxin derived from cell wall of Gram -negative bacteria which is a lipopolysaccharide (LPS) present in the periodontal pockets gets adsorbed into the relatively permeable cementum. This cementum bound endotoxin exerts a biologically inhibitory effect on gingival fibroblasts.[11]

Thickness*:* Cementum, like bone tissue, has the potential to be dynamically responsive and its growth may be stimulated by tensional forces. It has been suggested that cementum is thicker in areas exposed to tensional forces of the root surface of teeth undergoing orthodontic tooth movement.[7]

Surface Characteristics: surface of a periodontally involved cementum is rough and of an irregular nature. This facilitates both the accumulation and retention of plaque and calculus.[4]

Electron-microscopic examination demonstrates an appearance of 'pebbled' surfaces of periodontally involved cementum, which is covered with layers of bacteria and bacterial remnants.[12] ***Cracks*** in cementum of chronic and aggressive periodontitis can be due to chemical changes from inflammation and bacterial toxin.[13] Cracks can cause spaces between cementum layer which will lead to the exposure of the

dentinal tubules to the environment which consequently causes sensitivity.[14]

Cemental Junctions

The attachment of the cementum to the root dentin occurs via a fibrous junction, called the cemento-dentinal junction (CDJ). Early studies of root permeability indicated that the cemento-dentinal junction represents a barrier against the permeation of substances applied experimentally to the root surfaces.[7] However, Erasquin & Muruzabal have observed necrosis of cells in the deep layers of CDJ after root canal treatment in the molar teeth.[15]

Junction between the root cementum and enamel is referred to as the cemento-enamel junction (CEJ). CEJ serves as a significant reference point in the diagnosis and treatment of dental surface defects. It is a significant landmark for probing the level of the attachment of fibers to the tooth root in the presence of periodontal disease. CEJ also marks the landmark to diagnose cervical enamel projections (CEPs).[16]

Histopathology

Cementoblastoma*:* Cementoblastoma is a rare benign neoplasm of cementoblasts. The microscopic study of decalcified sections shows the presence of trabeculae and sheets of cementum like material and cellular fibro vascular stroma surrounding a few bony trabeculae, adjacent to the apical portion of the root. At places few osteoclastic multinucleated giant cells can also be seen.[17]

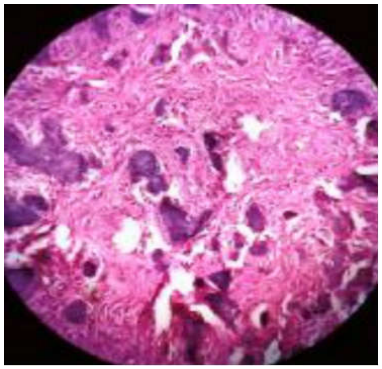

Fig 1 Benign Cementoblastoma. (17)

Gigantiform Cementoma: The lesion predominantly composes of calcified bony or cementum-like masses which are lobulated with rounded margins. The stroma appears scanty, fibrous in nature including relatively few cells. Sparse, irregularly placed lacunae are seen in the hard tissue and most of them are empty. Incremental lines are also seen surrounding cementicle-like structures. The peripheries of the masses show prominent, deeply staining basophilic rest lines. In some areas, the periphery of the lesion is lined by a layer of cells.[18] Plain and polarized light microscopic examination shows three basic collagen bundle arrangements in the hard tissue, the parallel fibre, the quilt-like and the coalescing globule type, as described by **Giansanti** (1970).[19]

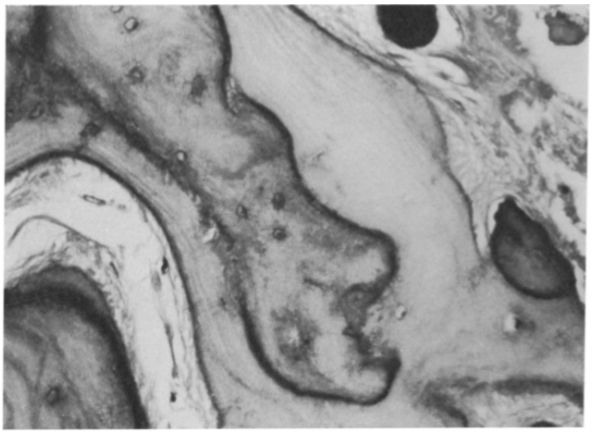

Fig 2 Photomicrograph of Gigantiform Cementoma Showing Sclerotic Hard Tissue With Scanty Stroma And Sparsely Placed Lacunae. (18)

Cementifying Fibroma: Histopathology presents as haphazardly arranged collagen fibres, although a whorled pattern may also be seen. Calcified material are noted throughout the fibrous stoma in the form of small, ovoid/globular, basophilic deposits. Irregular trabeculae of lamellar bone are also evident.[20]

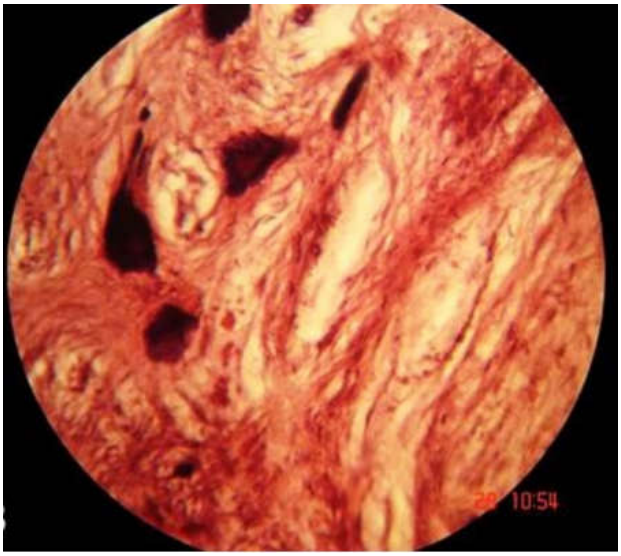

Fig 3 Cementifying Fibroma. (20)

Cemento-Osseous Fibroma (Cof): COF is a rare slow benign fibro-osseous tumour arising from the periodontal ligament. It is formed by a layer of fibrous connective tissue that encircles the roots of the teeth. Histologically, numerous clusters of cementum embedded in the fibrous tissue with areas of bone fragments are observed. In general, COF is more prevalent in the mandible than in the maxilla. The origin of COF is considered unknown. ***Wenig*** et al. suggested that past trauma or local irritants are possible causes for the development of a COF.[21]

Periapical Cemento-Osseous Dysplasia (PCOD): PCOD lesions are non-neoplastic fibro-osseous lesions which can exhibit similar histopathologic and radiologic features with other bone lesions. Histopathologic findings include cementicles/psammoma body-like calcifications.[22] Clinically it presents as a round swelling with smooth margin.[23]
Histologically, it can appear as three stages

Osteolytic stage*:* the tissue consists primarily of cellular connective tissue replacing normal trabecular bone with

calcified structures of insufficient size to be evident radiographically.

Cementoblastic stage: shows a mixture of spherical calcifications and irregularly shaped osteoid and mineralized bone deposits.

Mature stage: the tissue in the mature stage is composed mainly of coalesced spherical calcifications and sclerotic mineralized bone with scant connective tissue.[24]

Pathologic Granules: it was first described by Bass (1951) during a light microscopic study of decalcified sections.[4] Bass et al (1951) described pathologic granules to be highly refractile granules colored varying shades of brown. They may appear to be separate or in clumps, made up of a number of particles and of varying sizes. They are distributed in exposed cementum and the underlying dentine. Therefore, it is found only from the cementoenamel junction apex-ward to the root. In other specimens the granules in the deeper part of the cementum tend to project outward from the cemento-dentinal junction into the cementum for some distance.[25]

Fig 4 Very Thick Cementum with Scattered Large Clumps of Pathologic Granules. (25)

Developmental and Aqcuired Anomalies of Cementum

Hypophosphatasia: results from mutations in the tissue non-specific alkaline phosphatase gene, which codes for the enzyme alkaline phosphatase, which is essential for normal mineralization and development of tissues including cementum. Hypophosphatasia is characterized by bone abnormal development of cementum, which results in the early loss of primary teeth. This condition is now treatable with a fusion protein that replaces the lost alkaline phosphatase protein function.[26]

Fusion: it is characterized by the union of two normally separated tooth germs. It may be resulted from a physical force of pressure, which produces the contact of developing teeth and their subsequent fusion. If this contact occurs before the beginning of calcification, a complete union may occur to form a single large tooth. If the contact occurs after the completion of crown formation, union of the roots alone can be observed. The dentine, however is always confluent in cases of true fusions. The condition is more common in deciduous than in permanent dentition. In some cases, condition has been reported to show a hereditary tendency.

Concrescence: Here the teeth are united by cementum only. It occurs after root formation has been completed, which is thought to arise as a result of traumatic injury or crowding of teeth, with the resorption of interdental bone. This results in the approximation of two roots and fusion by the deposition of cementum between them. It may occur before or after the eruption of teeth.[27]

Hypercementosis/ Cemental Hyperplasia: Hypercementosis is the increase in cementum deposition on a root surface resulting in an abnormal root shape, frequently a bulbous tip at the root apex. It can result from a reactive response to peri-apical inflammation, trauma to the periodontal ligament, or an abnormal developmental process. Several systemic conditions have been related to hypercementosis including atherosclerosis, acromegaly, and Paget disease.[26]

When individual teeth or small groups of teeth are placed in heavy function, the surface of the cementum becomes irregular with spurs extending into the areas of principal fiber attachment. This undulating pattern increases the area of periodontal attachment and strengthens the supporting mechanism. In heavy function the cementum is considerably thickened in the apical portion of the root. These changes indicate that cementum deposition is influenced by function as well as by movement of the teeth.[1]

Cemental /Cementodentinal Tears: Cemental tear is a specific type of root surface fracture and has been explained as the detachment of a fragment of cementum from the root surface. It is rarely detected by non-invasive procedures, owing to its clinical appearance simulating a root fracture or a periodontal or endodontic lesion. As a kind of unusual root fracture, cemental tears can cause rapid periodontal attachment loss. The classical symptom is localized deep periodontal pockets, accompanied by other symptoms, such as a sinus tract or pain. The mechanism by which cemental tears develop is currently not fully known. However, common etiological factors been reported are, aging, gender, trauma, increased cementum fragility, history of periodontitis and occlusal overloading.[28]

Alterations in Cementum Resulting From Periodontal Pathology

Effect of gingival inflammation Subsurface alterations: Cementum may undergo alterations in structure as well as in the composition of its organic and inorganic components consequential to pathological changes in the immediate environment. The longstanding presence of an inflammatory process in the gingival connective tissue results in a net loss of collagen and in breakdown of dento-gingival fibers.

Exposure to the oral environment- Bacterial contamination: More obvious alterations may occur following exposure of the cementum surface to the environment of the periodontal pocket or the oral cavity. Bacterial invasion into cementum and root dentin is a common sequela to chronic periodontal disease.[7] *Adriaens* et al. were able to grow anaerobic bacteria from samples of root dentin of periodontally diseased teeth, which suggested that cementum and root dentin may serve as reservoirs from which recolonization of mechanically debrided root surfaces can occur, as well as infection of the dental pulp.[29]

Root Surface Wall of periodontal pockets: As the periodontal pocket deepens, collagen fibers embedded in the cementum are destroyed and there is exposure of cementum to the oral environment. Penetration of bacteria into the cementum can occur as deep as the CDJ and may also enter the dentinal tubules, thereby resulting in increased sensitivity.

Demineralization: Areas of demineralization are often associated with root caries. Exposure to the oral fluids and plaque bacteria results in the proteolysis of the embedded Sharpey's fiber remnants. Thus, cementum may become softened and may undergo fragmentation and cavitation.[30]

Hyper mineralization*:* The development of a hyper mineralized zone apparently depends on the ionic concentration of inorganic elements in the local environment. *Furseth* found that healthy cementum exposed to the oral environment by a gingivectomy procedure acquired a hyper mineralized surface zone within 21days. Similarly, if the original surface layer of cementum is removed by root planing, a hyper mineralized zone may be re-established within 4 to 8 weeks.[7]

Cementum Resorption

Surface/transient resorption*:* if there is injury or irritation to the cementum, resorbing cells will be attracted to the affected areas of the root surface, and resorption will occur as part of the normal scavenging function of the cells. It has been demonstrated that when there are only minor inflammatory changes, new PDL and cementum begin to form on the external root surface as early as 1 week after the injury.

Inflammatory Resorption: in cases of severe trauma and intense inflammatory response, the damage to the cementum progresses to involve resorption of the intermediate cementum layer that caps the ends of the dentinal tubules. The dentinal tubules are then open and communicate with inflammatory resorptive cells within the PDL and alveolar bone. If allowed to progress, the resorptive process may destroy the root and adjacent alveolar bone.[5]

External Resorption: May be divided into 3 categories:

1. progressive inflammatory resorption
2. cervical resorption; and
3. replacement resorption.

Progressive external inflammatory resorption: this is usually encountered in luxated teeth, where the root resorption is initiated by mechanical trauma, resulting in the removal of cementum on the root surface. The resorptive process is then maintained by microbial stimuli from the infected root canal, which provide the necessary continuous stimulation of the resorbing cells. After a few weeks the condition can be recognized radiographically as peri radicular radiolucent areas, usually encompassing areas of the root and the adjacent alveolar bone.[6]

Cervical Resorption: it is a resorption that appears to follow injury to the cervical attachment apparatus. The injury may be immediately below the attachment of the epithelial or more apically on the root surface where the damage to the cementum has occurred. Cervical resorption may result from orthodontic movement, as a late complication of traumatic injuries, or after internal bleaching, periodontal treatment, or other orthognathic and dentoalveolar surgery. In many instances, the history is obscure and thus is referred to as "idiopathic".[5]

Dento-alveolar ankylosis and replacement resorption**:** Dento-alveolar ankylosis occurs after periodontal ligament necrosis with formation of bone onto a denuded root surface area. It is usually seen as a complication to luxation injuries, especially in avulsed teeth. If less than 20% of the root surface is involved, reversal of ankylosis may occur. Otherwise, ankylosed teeth become incorporated in the alveolar bone and may form a part of the normal bone remodeling process. Consequently, they will gradually resorb and be replaced by bone, hence the term replacement resorption.[6]

Repair

Morphological studies have shown that two different repair matrices become attached to the resorbed root surface. Following the detachment of odontoclasts from the root surface, cementogenic cells repopulate the Howship's lacunae and attach the initial repair matrix to a thin decalcified layer of residual and exposed collagen fibrils. These cells and their respective repair tissues reveal remarkable homologies to the initial genesis of the two major cementum, acellular extrinsic fiber cementum and cellular intrinsic fiber cementum. In analogy to the formation of the genuine dentino cemental junction, the interdigitation of the newly formed collagen fibrils with the residual dentinal matrix fibrils occurs before the new attachment site becomes obscured by electron-dense material the globular accumulation of which is indicative of mineralization. Subsequently, deposited repair matrix usually resembles cellular intrinsic fiber cementum formed on non-resorbed roots. The strong resemblance of the initial formation of the two repair matrices with the initiation of acellular extrinsic fiber cementum and cellular intrinsic fiber cementum on the forming root indicates that repair cementogenesis recapitulates the events occurring during root development.[7]

Effects of Nutritional Deficiency on Cementum

Deficiency of vitamin D during tooth formation may result in the delayed eruption of tooth and cementum loss that leads to tooth loss. Protein collagen is intimately involved in the formation of cementum, at the biochemical level. As the building blocks of protein, amino acids are required for maintenance and repair of the oral tissues as well as for the formation of antibodies necessary to resist infection. Protein deficiency results in poor structural integrity of the dentition, degeneration of the structures supporting the dentition, delayed wound healing, and poor resistance to oral pathogens.[31]

CONCLUSION

Cementum provides a distinctive micro-environment for the ideal healing of periodontal tissues. It also provides tooth attachment and helps maintain occlusal relationships. Cellular intrinsic fiber cementum is mainly involved in repair and adaptive functions. In the absence of disease, the thickness of cementum increases throughout the life of the individual.[3]

Generally, minor resorption defects on the root surface are reversible and heal by reparative cementum formation. Irreversible damage occurs when the cementum surface becomes exposed to the external environment, such as a periodontal pocket. Surgical procedures to restore lost periodontal attachment have been only partially successful. In this context, the discovery of a variety of non-collagenous proteins in cementum has opened a new research area of great therapeutic potential. Conceivably, the future application of cementum-derived growth and/or attachment factors may result in accelerated wound healing and in controlled neo-cementogenesis following periodontal regenerative surgery.[7] Further studies are needed to determine the therapeutic implication of the cementum with root planing or chemical treatment in order to achieve an available root surface for healthy attachment.[8]

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