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Histology of enamel and dentin and its clinical consideration

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Abstract

Teeth constitute approximately 20% of the surfaces of the mouth. Teeth serve several functions. Mastication is the function most commonly associated with the human dentitions, but teeth also are essential for proper speech and, in modern times, for esthetics.the histological factors of the tooth structure and better knowledge about the same can help us to make our clinical practice more easy and accurate. This article focuses on the histology of enamel and dentin and its clinical considerations.

Keywords: Histology; enamel; dentine; clinical consideration; histological features

Introduction

The oral cavity contains a variety of hard tissues and soft tissues. The hard tissues are the bones of the jaws and the tooth. The soft tissues include the lining mucosa of the mouth and the salivary glands. The tooth consists of crown and root. The anatomical crown is covered by enamel and the root by cementum.^[1]

Enamel has evolved as an epithelially derived protective covering for the anatomic crown of the teeth. The enamel is the most highly mineralized tissue in the body, consisting more than 96% inorganic material in the form of apatite crystals and traces of organic material. Although enamel is a dead tissue in a strict biologic sense, it is permeable; ionic exchange can occur between the enamel and the environment of the oral cavity, in particular the saliva.^[2,3]

Dentin is a mineralized, elastic, yellow-white, avascular tissue enclosing the central pulp chamber. The mineral is also apatite, and the organic component is mainly the fibrillar protein collagen. A characteristic feature of the dentin is its permeation by closely packed tubules traversing its entire thickness and containing the cytoplasmic extensions of the cells that once formed it and later maintain it. These cells are called odontoblasts; their cell bodies are aligned along the inner edge of the dentin, where they form the peripheral boundary of the dental pulp. The very existence of odontoblasts makes dentin a vastly different tissue from enamel. Dentin is a sensitive tissue, and more importantly, it is capable of repair, because odontoblasts or cells in the pulp can be stimulated to deposit more dentin as the occasion demands.^[1,4]

Physical Characteristics

Enamel is the most highly mineralized tissue of the body. It forms a protective covering of variable thickness over the entire surface of the crown. The specific gravity of enamel is 2.8. The hardness and density decreases from the surface to the deeper regions.^[2]

The enamel attains a maximum thickness of 2.5 mm on the cusp of crown and a minimum of about 100 microns at the neck of the tooth and bottom of the pits and fissures. The nature of its structure and the hardness renders it very brittle having a low tensile strength.^[5] The color of enamel covered crown ranges from yellowish white to grayish white. It has been suggested that the color is determined by differences in the translucency of enamel which may be attributable to variations in the degree of calcification and homogeneity of the enamel.^[5]

The color of dentin varies from light yellow in deciduous teeth to pale yellow in permanent dentition, becoming darker with age. Dentin supports the brittle enamel under masticatory forces. It has a density of 22.1 gram/ millimeter and its Knoop hardness number is 68. ^[6,7]

The lower content of mineral salts in dentin renders it more radiolucent than enamel. Dentin hardness varies between crown and root dentin in a tooth and between different types of teeth. Dentin hardness is more in the central part than near the pulp or at its periphery. Dentin is harder in permanent teeth than in deciduous teeth.^[7]

Chemical Properties

Owing to its high mineral contentenamel is a brittle tissue, so brittle that it cannot withstand the forces of mastification without fracture unless it has the support of a more resilient tissue, such as dentin. Dentin forms the bulk of the tooth, supports the enamel, and compensates for its brittleness.^[8]

Dentin consists of 70 % inorganic matter, 20% organic material and 10% water by weight. By volume, mature dentin contains approximately 45% inorganic material; 33% organic material and 22% water. On weight basis, dentin is less mineralized than enamel (96% in weight),

but more than bone or cementum (about 65% in weight). this distribution However. global provides an oversimplified view, because dentin is a puzzle of different types of dentin, reflecting different functions and bearing their own specificities.^[8] Unlike enamel, the high organic content of dentin enables it to deform slightly under compression. Another factor contributing to the resiliency of the dentin is the fluid within the dentinal tubules. The fluid-filled dentinal tubules may function as 'hydraulic shock absorbers' dissipating the forces of mastication. Dentin therefore provides a cushion for the overlying brittle enamel.^[9]

Although trace amounts of calcium carbonate, fluoride, magnesium, zinc and other minerals like metal phosphates and sulfates are found in dentin, hydroxyapatite $[Ca_{10}(PO_4)_6(OH)_2]$ is the principal inorganic component of the dentinal matrix. The hydroxyapatite crystals are in the form of flattened plates with the approximate dimensions of 60 to 70 nm in length, 20 to 30 nm in width and 3 to 4 nm in thickness. The high mineral content of dentin makes it harder than cementum or bone, although softer than enamel. ^[2, 6]

The bulk of the organic matrix of dentin (85-90%) consists of collagenous fibrils embedded in the ground substance of mucopolysaccharides (proteoglycans and glycoaminoglycans). The principal type of collagen found in dentin is type I collagen with minor amounts of type V and VI.

The organic and inorganic substances can be separated by either decalcification or incineration. In the process of decalcification the organic constituents can be retained to maintain the shape of the dentin. That is why decalcified teeth and bone can be sectioned to provide clear histological visualization. The enamel, which is made up of 90% mineral content, is lost after decalcification.^[10]

Dr. Saarika Suresh, et al. International Journal of Dental Science and Innovative Research (IJDSIR)

Structure

Enamel is composed of the following:

- 1. Enamel rods (prisms)
- 2. Rod sheaths
- 3. Inter rod substance (cement)

The other structures of enamel includes: Hunter-Schreger bands, Incremental lines of Retzius[,] Structureless outer enamel layer, Perikymata, Enamel rod ends, Cracks[,] Neonatal line or ring[,] Nasmyth's membrane (Primary Enamel Cuticle), Enamel lamellae[,] Enamel tufts[,] Enamel spindles[,] Gnarled enamel, Enamel droplets or enamel pearls[,]

The dentin is composed of the following:

- 1. Odontoblastic processes
- 2. Dentinal tubules
- 3. Peritubular dentin
- 4. Intertubular dentin
- 5. Predentin

Age Changes

Enamel does not contain nerves and is incapable of repair and replacement. Gradually, with age the enamel is worn away in the regions where masticatory forces are applied, which is called 'attrition'. The rate at which structure is lost depends on the location of the surface of the tooth and on the location of the tooth in the mouth. Facial and lingual surfaces lose their structure much more rapidly than proximal surfaces, and anterior teeth lose their structure more rapidly than posterior teeth.

As a result of age changes in the organic portion of enamel, presumably near the surface, the teeth may become darker, and their resistance to decay may be increased. There is insufficient evidence to show that enamel becomes harder with age.^[11]

With advancing age and for functional requirements number of changes are seen in dentin.

Vitality of dentin: Pathologic effects of dental caries, abrasion and attrition cause changes in dentin. These are described as the development of dead tracts, sclerosis, and the addition of reparative dentin^[12]

Reparative dentin: If by extensive abrasion, erosion, caries or operative procedures the odontoblasts processes are exposed or cut, the odontoblasts die or survive, depending on the intensity of the injury. If they survive the dentin that is produced is known as reactionary or regenerated dentin. ^[12]

Dead tracts: Loss of odontoblasts process may also occur in teeth containing vital pulp as a result of caries, attrition, abrasion, cavity preparation or erosion. Dentinal areas characterized by degenerated odontoblasts processes give rise to dead tracts. Dead tracts are probably the initial step in the formation of sclerotic dentin. ^[13]

Sclerotic dentin: Though the sclerotic dentin was harder than normal dentin, its elastic properties were not altered, but its fracture toughness was reduced. The crystals present in the sclerotic dentin were smaller than those present in the normal dentin.^[13]

Clinical Considerations Of Enamel

The course of the enamel rods is of importance in cavity preparations. The choice of instrument depends on the location of the cavity in the tooth. Generally the rods run at a right angle to the underlying dentin or tooth surface. Close to the cementoenamel junction the rods run in a more horizontal direction. In preparing cavities, it is important that unsupported enamel rods are not left at the cavity margins because they would soon break and produce leakage. Bacteria would lodge in these spaces, inducing secondary dental caries.^[1]

Deep enamel fissures predispose teeth to caries. Although these deep clefts between adjoining cusps cannot be regarded as pathologic, they afford areas for retention of caries-producing agents. Caries penetrate the floor of

Dr. Saarika Suresh, et al. International Journal of Dental Science and Innovative Research (IJDSIR)

fissures rapidly because the enamel in these areas is very thin.^[6] As the destructive process reaches the dentin, it spreads along the dentinoenamel junction, undermining the enamel. An extensive area of dentin becomes carious without giving any warning to the patient because the entrance to the cavity is minute. Careful examination is necessary to discover such cavities because most enamel fissures are minute than a single toothbrush bristle and cannot be detected with the dental probe.

Dental lamellae may also be predisposing locations for caries because they contain much organic material. Primarily from the standpoint of protection against caries, the structure and reactions of the outer enamel surface are subject to much current research. In vitro tests have shown that the acid solubility of enamel can be greatly reduced by treatment with fluoride compounds. Clinical trials based on these studies have demonstrated reductions of 40% or more in the incidence of caries in children after topical applications of sodium or stannous fluoride. Incorporation of fluorides in dentifrices is now a wellaccepted means of caries prevention. Fluoride containing mixtures such as stannous fluorides pastes, sodium fluorides rinses, and acidulated phosphate fluoride are also used by the dentist to alter the outer surface of the enamel in such a manner that it becomes more resistant to decay. [14]

The most effective means for mass control of dental caries to date has been adjustment of the fluoride level in communal water supplies to 1 part per million. Epidemiological studies in areas in which the drinking water contained natural fluoride revealed that the caries prevalence in both children and adults was about 65% lower than in nonfluoride areas, and long-term studies have demonstrated that the same order of protection is afforded through water fluoridation programs.^[2] The mechanism of action are believed to be primarily a combination of changes in enamel resistance, brought about by incorporation of fluoride during calcification, and alterations in the environment of the teeth, particularly with respect to the oral bacterial flora.

The surface of the enamel in the cervical region should be kept smooth and well polished by proper home care and by regular cleansing by the dentist. If the surface of the cervical enamel becomes decalcified or otherwise roughened, food debris, bacterial plaques, and so on accumulate on this surface. The gingiva in contact with the roughened, debris covered enamel surface undergoes inflammatory changes. This ensuing gingivitis, unless promptly treated, may lead to more serious periodontal disease.

One of the more developed techniques used in operative dentistry consists of the use of composite resins. These materials can be mechanically "bonded" directly to the enamel surface. In this procedure the enamel surface is first etched with an acid (phosphoric acid 37%) to remove the smear layer on the enamel that was created during cavity preparation. Smear layers are about 1um thick and are made up of burnished cutting debris. Because the particles that constitute the smear layer are very small, the layer is very acid labile. Acid etching of enamel removes this smear layer. This produces an uneven dissolution of the enamel rods and their "sheaths" or enamel "heads" and their "tails" so that a relatively smooth enamel surface becomes pitted and irregular, this achieve mechanical bonding of composite with the enamel.^[1] The same principle is used in coating the susceptible areas of the enamel with the so-called pit fissure sealants.[15]

Depending on the crystal orientation to the surface three types of etching patterns are produced. Crystals dissolve more readily at their ends than on their sides. Hence, those crystals which are perpendicular to the surface are removed preferentially. This results in crystal removal from the rods and is called type I pattern. In the type II pattern the interrod crystals are preferentially removed. The type III pattern is irregular. Prismless enamel found on the surface does not provide enough mechanical retention so etching should go beyond the prismless enamel to the prismatic enamel below it.^[16]

AMELOGENESIS IMPERFECTA (AI): It is a group of inherited defects of enamel that show clinical and genetic heterogeneity. It affects all or only some of the teeth in the primary or permanent dentition. In its mildest form, AI causes discoloration while in its most severe presentation; the enamel is hypo mineralized causing it to be abraded from the teeth shortly after their emergence into the oral cavity.

The treatment of AI aims to relieve pain or tooth sensitivity, to preserve as much tooth structure as possible while preventing further tooth loss, to maintain mastication and to improve the appearance because this has great psychological impact on patient's confidence. It affects the psychology of the patient negatively due to the aesthetic concerns. It should be promptly identified and treated, and existing dentition should be protected so that teeth can be conserved as much as possible. Oral rehabilitation provides a good prognosis and they also have less clinical complications. The Patient should be counseled and motivated and should be taught to maintain good oral hygiene, which helps in maintaining the dentition.[17]

Clinical Considerations of Dentin

The cells of the exposed dentin should not be insulted by bacterial toxins, strong drugs, undue operative trauma, unnecessary thermal changes, or irritating restorative materials. One should bear in mind that when 1mm² of dentin is exposed, about 30,000 living cells are damaged. It is advisable to seal the exposed dentin surface with a nonirritating, insulating substance.

The rapid penetration and spread of caries in the dentin is the result of the tubule system in the dentin. The enamel may be undermined at the dentinoenamel junction, even when caries in the enamel is confined to a small surface area. This is due in part to the spaces created at the dentinoenamel junction by enamel tufts, spindles, and open and branched dentinal tubules. The dentinal tubules provide a passage for invading bacteria and their products through either a thin or thick dentinal layer.^[1]

Electron micrographs of carious dentin show regions of massive bacterial invasion of dentinal tubules. The tubules are enlarged by the destructive action of the microorganisms. Dentin sensitivity of pain, unfortunately, may not be a symptom of caries until the pulp is infected and responds by the process of inflammation, leading to toothache. Thus patients are surprised at the extent of damage to their teeth with little or no warning from pain. Undue trauma from operative instruments may also damage the pulp. Air-driven cutting instruments cause dislodgement of the odontoblasts from the periphery of the pulp and their "aspiration" within the dentinal tubule. This could be an important factor in survival of the pulp if the pulp is already inflamed. Repair requires the mobilization of the macrophage system as healing takes place; as this progress there is the contribution of deeper pulpal cells, through cytodifferentiation into odontoblasts, which will be achieve in formation of reparative dentin.[18]

The sensitivity of the dentin has been explained by the hydrodynamic theory, alteration of the fluid and cellular contents of the dentinal tubules causes stimulation of the nerve endings in contact with these cells. This theory explains pain throughout dentin since fluid movement will occur at the dentinoenamel junction as well as near the pulp.^[1] Erosion of peritubular dentin and smear plug removal accounts for dentin hypersensitivity caused by agents like acidic soft drinks. Brushing after acidic drink

consumption, induced smear layer formation, thus reducing sensitivity.

The basic principles of treatment of hypersensitivity are to block the patent tubules or to modify or block pulpal nerve response. The most inexpensive and the first line of treatment is to block the patent tubules with dentifrice containing potassium nitrate and/ or stannous fluoride. Lasers have been used in the treatment of hypersensitivity with varying success, ranging from 5.2 to 100%.

The permeability of radicular dentin near the pulp is only about 20% that of coronal dentin, and the permeability of outer radicular dentin is about 2% of coronal dentin. This suggests that the outer dentin of the root acts as a barrier to fluid movement across dentin in normal circumstances and recalls the correlation between root planning and hypersensitivity.

Smear layer consists of the cut dentin surface along with the embedded bacteria and the debris. Though the smear layer occludes the tubules and reduces the permeability, it also prevents the adhesion of restorative materials to dentin. Therefore this layer has to be removed by etching and a rough porous surface should be created for bonding agent to penetrate.^[52]

Dentin formation, unlike bone, is not affected by vitamin D deficiency states. Fluoride incorporated during active dentinogenesis, as occurring in dental fluorosis, increases the hardness of dentin.

Dentinogenesis Imperfecta (**Di**): It is a localized mesodermal dysplasia affecting both the primary and permanent dentitions. The disease is inherited in an autosomal dominant fashion with high penetrance and a low mutation rate. It is the most common dental genetic disease, affecting approximately 1 in 8000 births. Clinically, both dentitions are affected. The color of the teeth varies from brown to blue, and is sometimes described as amber or gray. The enamel may show

hypoplastic or hypocalcified defects in about one-third of the patients and in an affected person tends to crack away from the defective dentin. The exposed dentin may then undergo severe and rapid attrition. [19]

The early diagnosis and treatment of DI is very important as it prevents the deterioration of the teeth and occlusion and helps in improving esthetics. In severe cases of DI, two treatment stages of the primary dentition under general anesthesia are recommended. In moderate cases, a one-stage treatment of the primary dentition at age 30 months is done. Composite restoration in severe cases of DI provides durability. Long term follow-up is important to intercept complications and adjust the treatment according to the changing dentition and occlusion.

Conclusion

The tooth is formed from the ectoderm and ectomesenchyme. The enamel is derived from the enamel organ which is differentiated from the primitive oral epithelium lining the stomodeum. Epithelial mesenchymal interactions take place to determine, the shape of the tooth and the differentiation of the formative cells of the tooth and the timing of their secretion. Dentin and pulp are derivatives of dental papilla and the ectomesenchymal cells closer to the outer margins of the enamel organ become dental follicle. The ameloblast produces enamel, cementoblast produces cementum, odontoblast produces dentin and osteoblast produces bone

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Dr. Saarika Suresh, et al. International Journal of Dental Science and Innovative Research (IJDSIR)

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