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Regressive Alterations Of Teeth – A Review Literature

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ABSTRACT

Regressive alteration is a complicated condition that causes enamel and dentine to deteriorate. Erosion, abrasion, abfraction, attrition, resorption, or a combination of these lesions are typically the cause. With intervals of activity and inactivity, regressive alterations are shown to progress slowly. Prevention is still the best way to keep a tooth healthy for as long as possible, even though restorations are sometimes necessary. For the diagnosis and treatment of these lesions, a comprehensive understanding of their origin, symptoms, and signs is essential. This page helps readers diagnose the type of lesion and determine the best course of treatment by outlining the etiological causes.

Keywords: Erosion, Attrition, Abrasion, treatment.

INTRODUCTION

A number of changes in the dental tissues are examples of regressive changes in teeth; both pathological and physiological factors can cause mechanical tooth wear and tear[1]. Regressive alterations, which result in tooth structural wear and tear and functional degradation, are a subset of retrogressive alterations that occur in teeth due to non-bacterial sources.

Occlusal loading forces result in tooth flexures, which in turn produce mechanical microfractures and tooth structure loss in the cervical regions, per research on the process of stress concentration in these regions[2].

Few investigators, have concluded that the cause of non-carious cervical lesions (NCCLs) could be acid in areas of stress concentration which results in either static or cyclic stress corrosion. While some degree of tooth wear is acknowledged as a natural aspect of aging, issues related to tooth wear are concerning[3,4,5].

HISTORY

Miller's 1907 article on observations and studies on tooth wear provided insight into the genesis of non-carious cervical lesions (NCCLs). referred to as denudation, abrasion, erosion, and chemical abrasion, among others[6]. In the late 1970s, the idea that occlusal loading might result in cervical stress and the subsequent loss of cervical tooth structure started to take shape. NCCLs have historically been categorized based on their appearance into flattened, irregular, wedge-shaped, disk-shaped, and figured sections[7].

The research by McCoy[8], Lee and Eakle[9], and Grippo[10] have led to the evolution of the word "abfraction." It is explained as a theoretical mechanism wherein occlusal forces induce enamel stresses and dentin along the cervical region, making it vulnerable to abrasion and erosion. Early in the 1980s, McCoy questioned whether toothbrush abrasion contributed to the development of what was once known as "cervical erosion."

Therefore, it was suggested by McCoy and Grippo in the early 1990s that bruxism might be the main reason for angled notches at the CEJ.

Grippo came to the conclusion that the enamel rods at the CEJ were damaged by the flexure, which caused them to loosen and the tooth structure to flake away as a result. He instilled the name this kind of damage, or "abfraction," in his 1991 article[11]. While Lee and Eakle [9] posited a complex etiology that included occlusal tension, abrasion, and erosion, he maintained that AF is the fundamental cause of all NCCLs. Spranger backed up the multiple etiology of the cervical lesions and proposed that the wear was connected to the development of caries, occlusion and function, the distribution of forces determined from the elastic deformation tests, and anatomy.

CLASSIFICATION

1. ENAMEL
a. Attrition
b. Abrasion
c. Erosion
d. Abfraction
2. DENTIN
a. Dentinal sclerosis
b. Dead tracts
3. PULP
a. Reticular atrophy of pulp
b. Pulp calcification
4. RESORPTION OF TEETH
a. External
b. Internal
5. HYPERCEMENTOSIS
6. CEMENTICLES

ATTRITION

The term attrition was derived from the Latin verb attritium which describes the action of rubbing against something. Attrition is the loss of tooth material brought on by tooth-to-tooth contact; hence, while it is most commonly observed occlusally, it can also happen interproximally when the teeth shift laterally, creating wider interproximal contacts over time.

On the teeth's functional surfaces, tooth wear results in distinct wear facets[12].The following are some of the causes that may contribute to attrition.

1. Developmental defects nutrition
2. opposing restoration
3. improper chewing habits
4. abnormal

Given that dentine is less resistant to wear and erosion than enamel, it was hypothesized that cupping of the incisal borders could result from either acid assault or abrasion. The dental arch becomes shorter as a result of attrition across the teeth's proximal surfaces

SYMPTOMS

- Tooth grinding at night
- Jaw pain, fatigue and limited opening on waking
- Teeth feel loose (localised or generalised)
- Sore teeth or sore gums
- Headaches in the temporal region
- Grinding or clenching of the teeth while awake.

CLINICAL SIGNS

- Tooth wear and marked wear facets, particularly in protrusion or lateral excursion
- Tooth fractures – natural teeth or restorations
- Tooth mobility
- Pulp necrosis – as loads cause limitation of blood supply
- Traumatic ulcers
- Linear alba

Radiographic view reveals shortened crown image, sclerosis of the pulp chambers and the canals due to deposition of secondary dentine, widening of the periodontal ligament space, hypercementosis and loss of alveolar bone might also occur[13].

ABRASION

Abrasion is the pathological loss of tooth structure brought on by an irregular mechanical process[13].

The causes of abrasion are [14]

1. improper brushing technique abrasive dentifrices
2. improper use of toothpicks and dental floss
3. biting on hard objects like pins or finger nails

V-shaped or wedge-shaped notches at the cervical borders are how the lesion appears.

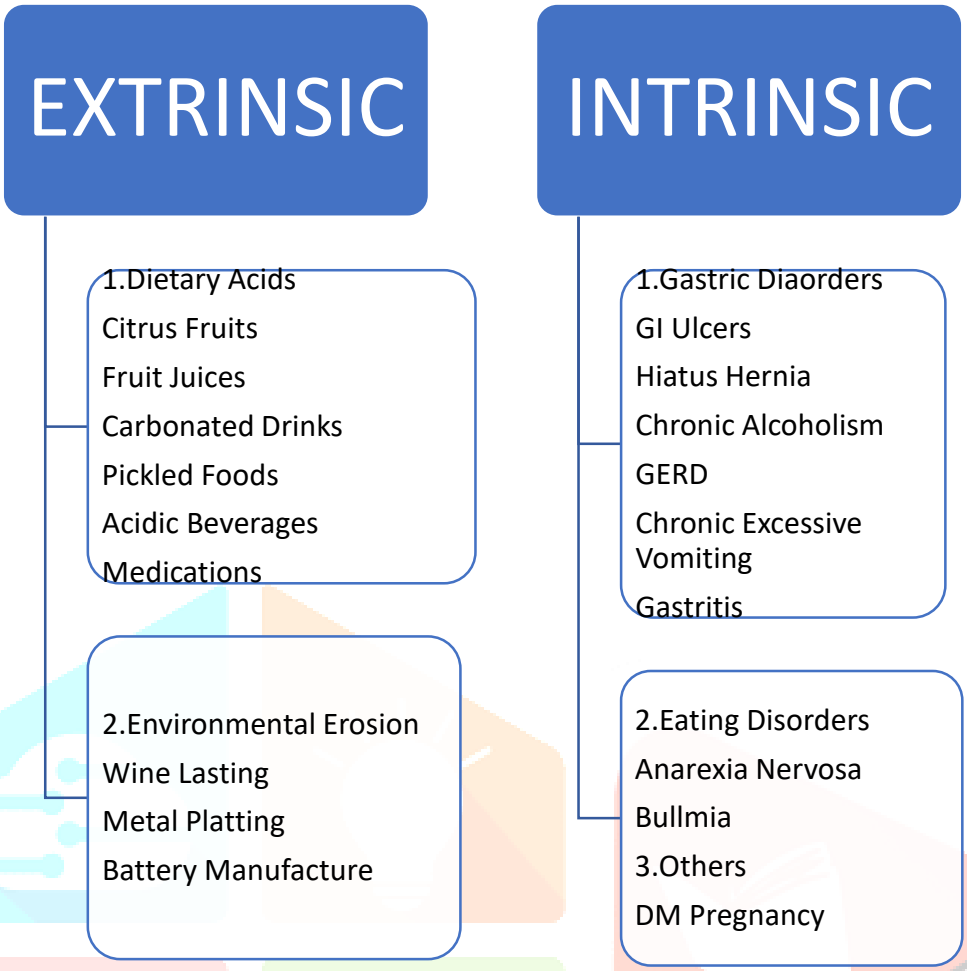
According to studies, wear is influenced by the toothbrush's hand and tooth alignment; a right-handed person's left side experiences more wear, and vice versa[12]. Conversely. The term for tooth wear caused by friction from food boluses is masticatory abrasion.

When the cuspal enamel is removed from teeth of persons who eat abrasive diets, the dentine wears down more quickly than the enamel, giving the teeth a "cupped" or "scooped" appearance. Since a smear layer covers the abraded dentine surface, sensitivity issues are not linked to it.

When a toothbrush injury occurs, the radiographic view reveals a radiolucent defect at the cervical level, partial or complete pulp chamber sclerosis, and narrow semilunar grooves in the teeth's interproximal surfaces close to the cervical area when a dental floss injury occurs. [13]

EROSION

Erosion is the loss of tooth structure by a chemical process that does not involve a known bacterial action.



The lesion is smooth and free of chalkiness, affecting both the buccal and labial surfaces of the teeth. Erosion of palatal surfaces is known as perimolysis. caused by the forcefully directed movement of vomitus, which has a mean pH of 3.8, in the maxillary anterior teeth[15].

Bodecker claims that when gingival crevicular fluid comes into contact with a tooth, it is similarly acidic and may corrode the cervical regions. Drinking caused different levels of dental deterioration.

A more noticeable pH drops results from techniques like keeping the beverage in the mouth for longer. Foods having a pH below 5.5 have been shown to demineralize teeth and function as corroders[16,17]. The corrosive effect of carbonated drinks, which are linked to tooth decalcification, has been linked to the addition of phosphoric and citric acids. Erosion depth could be increased by drinking with a higher flow rate and a smaller exit diameter. Additionally, the impact is amplified as the acid temperature rises[18].

ABFRACTION

Also called as stress lesion

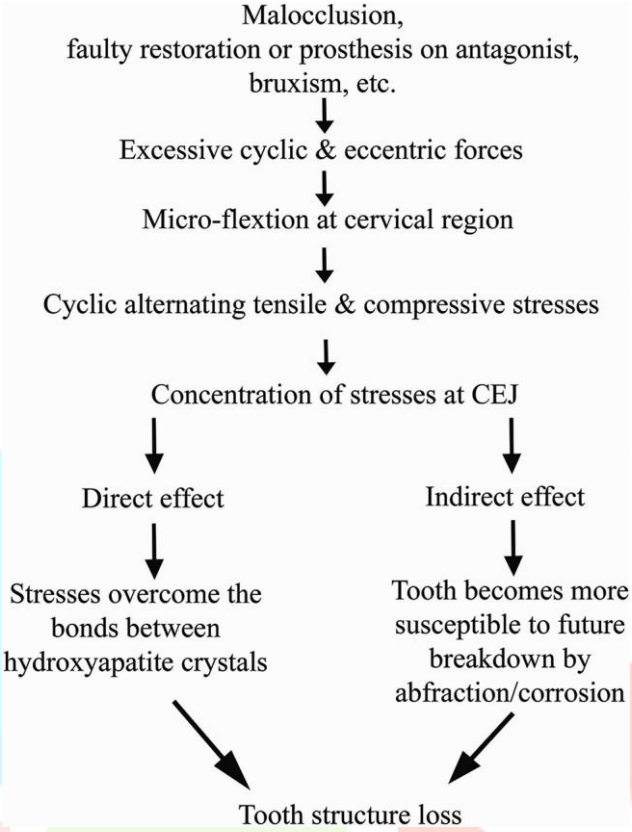
It is a result of the natural dentition's eccentric forces.

The Latin terms ab, which means away, and fraction, which means cracking, were used to create the phrase "abfraction."

Abfraction denotes the pathologic loss of tooth structure caused by mechanical loading, which leaves the cervical regions with wedge-shaped flaws. This results from the separation of thin coating of dentin, cementum, and enamel rods[19]. Occlusal loading forces often transmit stresses that concentrate to cause abfractions.

ETIOPATHOGENESIS

According to McCoy, tooth flexure from tensile pressures caused cervical tooth disintegration, and bruxism may be the main cause of abfraction [20]. Later, Lee and Eakle postulated that the influence of tensile tension from mastication and malocclusion was the main causative element in cervical lesions.



Combining barrelling and bending deformations results in the lesion. This causes the tensile and compressive stresses to alternate, which weakens the dentin and enamel. The tooth fractures or splits when the forces exceed a fatigue limit. Concurrently, compressive stress is applied to the opposite region. The tooth bends in the opposite direction as the force direction changes, and the tensions at this cervical region also reverse. The most flexible zone of the tooth becomes fatigued and fractured as a result of side-to-side bending. These interocclusal stresses result in physical microfractures or abfractions at the cervical region.

RESORPTION

Resorption occurs when cells known as odontoclasts cause long-term, gradual deterioration or loss of tooth structure.
Resorption in deciduous teeth can be either physiological , or in the permanent dentition, it may be pathologically observed.

Both internal and exterior pathological resorption are possible.

The affected tooth is typically asymptomatic when external resorption occurs. The most commonly affected are the maxillary incisors, maxillary and mandibular bicuspids. The tooth may become mobile as the root resorbs, but it will become immobile if ankylosis occurs after resorption.

A disorder known as internal resorption begins in the pulp chamber and spreads via resorption of the dentin around it.A pink patch in the pulp of teeth indicates hyperplastic pulp tissue that fits the resorbed area and peeks through the tooth substance on top. The lesion appears radiolucent, circular, oval, and extended inside the root on radiography, with the pulp chamber and canals spreading. Internally, there are no radiopaque foci or bone trabeculation; the radiolucency is uniform.

DENTINAL SCLEROSIS

A regressive change in tooth composition, sclerosis of primary dentin is typified by calcification of the dentinal tubules. In addition to being caused by caries or abrasions that penetrate the dentin, it also happens as a natural part of aging. It has long been known that a transparent zone in the dentin beneath the cavity can be detected when a ground section of a tooth with a very superficial carious lesion of the dentin is studied using transmitted light. This was easily identified as the result of a discrepancy between the refractive indices of the nearby normal tubules and the calcified or sclerotic dentinal tubules. These dentinal tubules are impervious to dyes because the tubules are occluded by calcification

DEAD TRACTS

In ground sections of teeth, dead tracts in the dentin are visible as a black zone when illuminated by transmitted light and as a white zone when illuminated by reflected light . The refractive indices of the impacted tubules and normal tubules differ, which causes this optical phenomena. When odontoblasts die or the odontoblastic process retracts or degenerates, the dentinal tubules become empty or air filled. Abrasion, erosion, cavity preparation, or dental caries attrition could be the cause of this. Dead tract formation is thought to be the first stage in the formation of sclerotic dentin.

PULP CALCIFICATION

Pulp calcification refers to the accumulation of calcium salts within the dental pulp, resulting in the development of pulp stones or diffuse calcifications. It may arise due to factors such as aging, trauma, inflammation, or certain pathological conditions. While often asymptomatic, these calcifications can pose challenges during endodontic procedures. Radiographic imaging plays a crucial role in identifying and managing pulp calcifications[21].

True denticles are made up of localized masses of calcified tissue that resemble dentin because of their tubular structure. Since the tubules are uneven and limited in number, these nodules really resemble secondary dentin more than primary dentin. Compared to the root canal, they are far more prevalent in the pulp chamber.

False denticles are localized masses of calcified material that do not have dentinal tubules like true denticles. They consist of concentric layers or lamellae formed around a central core, known as a nidus. The exact nature of this nidus is unclear, but it is thought to contain unidentified cells surrounded by reticular fibers that later calcify.

Most frequently observed in tooth root canals, diffuse calcification is similar to the calcification that occurs in other bodily tissues after deterioration. "Calcific degeneration" is a common phrase used to describe this kind of calcification. It typically occurs in columns that run parallel to the pulp's blood vessels and nerves or in amorphous, disorganized linear threads.

HYPERCEMTOSIS

Hypercementosis is a non-neoplastic condition in which excessive cementum is deposited in continuation with the normal radicular cementum[22]. Hypercementosis is linked to a number of local and systemic conditions, including supra-eruption of a tooth, inflammation at the apex of a tooth, traumatic occlusion, Paget's disease, etc., even some cases are idiopathic. Hypercementosis can manifest as a single tooth, several teeth, or a widespread condition. More often, posterior teeth are affected. Hypercementosis appears on radiographs as a changed root form with the shadows of the lamina dura and periodontal membrane maintaining their regular connection. The cementum that forms in teeth with hypercementosis is typically osteocementum (acellular cementum), according to histologic analysis.

Rushton and Cooke (1959) stated that mild traumatic occlusion may cause hypercementosis. In rare cases, excessive occlusal trauma may lead to the formation of serrated hypercementosis (cemental spikes) which follows the course of Sharpey's fibres[23].

CEMENTICLES

The periodontal ligament of the lateral and apical root areas contains cementicles, which are tiny foci of calcified tissue that are not necessarily real cementum. Although the precise mechanism of their development is uncertain, it is generally accepted that they typically indicate areas of dystrophic calcification and are hence an illustration of a degenerative or regressive transformation.

Cementicles may arise from focal calcification of connective tissue between Sharpey's bundles with no apparent central nidus. This calcification occurs as small round or ovoid globules of calcium salts.

CONCLUSION

In summary, regressive alterations of teeth are natural or pathological changes that affect tooth structure over time. There is no way to reverse these regressive tooth alterations. They can be controlled, though, and further development can be avoided. Therefore, accurately identifying the symptoms of a worn dentition should always be the first priority. The treatment regimen should then be planned utilizing an organized evaluation procedure that is founded on a complete understanding of the etiology. A dental surgeon can then precisely restore the patient's function and appearance. Continued research and advancements in dental care will help improve preventive and treatment approaches for these conditions.

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