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REVIEW ARTICLE

Current concepts of trauma from occlusion - A review

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Abstract

It has been a topic of debate among dental care professionals, if trauma from occlusion is linked with periodontal disease or not. There are several schools of thought related to if trauma from occlusion is an etiological factor or cofactor for the occurrence of periodontal diseases. The present review article discusses historical background, etiological factors, classification, relevant terminologies, tissue response, signs and symptoms, advanced diagnostic methods, and treatment.

Introduction

The role of occlusal trauma in the initiation and progression of periodontitis remains a controversial subject in periodontology. Occlusal trauma can only be confirmed histologically; its clinical diagnosis completely depends on clinical and radiographic surrogate indicators which make clinical trials difficult. The present review paper discusses the historical background, etiological factors, classification, tissue response, signs and symptoms, advanced diagnostic methods, and treatment.

Historical Aspects

Ever since Karolyi (1901) postulated that an interaction may exist between "trauma from occlusion" (TFO) and "alveolar pyorrhea," different opinions have been presented in the literature regarding the validity of this claim.[1]

In the 1930s, Box (1935) and Stones (1938) reported experiments in sheep and monkeys, the results seemed to indicate that "TFO is an etiologic factor in the production of periodontal disease in which there is vertical pocket formation associated with one or a varying number of teeth" (Stones 1938).[1]

Glickman and Smulow[2] proposed the theory in the early 1960s that a traumatogenic occlusion may act as a cofactor in the progression of periodontitis. This theory is known as the "co destructive theory." Goldman[3] proved that occlusal trauma was not the cause of soft tissue lesions such as Stillman's clefts and McCall's festoons. Waerhaug[4] proved the involvement of TFO in the pathogenesis of infrabony pockets.

Definition and Terminology

Excessive occlusal force is defined as occlusal force that exceeds the reparative capacity of the periodontal attachment apparatus, leading to occlusal trauma, and/or causes excessive tooth wear (loss).[5,6]

Occlusal trauma

The injury resulting in tissue changes within the attachment apparatus, including cementum, supporting alveolar bone, and periodontal ligament, as a result of occlusal force(s). A reduced periodontium or an intact periodontium caused by periodontal disease may be affected by occlusal trauma.[7]

TFO was defined by Stillman as "a condition where injury results to the supporting structures of the teeth by the act of bringing the jaws into a closed position."[1]

The World Health Organization in 1978 defined trauma from occlusion as "damage in the periodontium caused by stress on the teeth produced directly or indirectly by teeth of the opposing jaw."[1]

In "glossary of periodontic terms" (American Academy of Periodontology 1986), occlusal trauma was defined as "an injury

to the attachment apparatus as a result of excessive occlusal force."[1]

Primary occlusal trauma

The injury resulting in tissue changes from excessive occlusal forces applied to a tooth or teeth with normal periodontal support.[8] In the presence of normal clinical attachment levels, normal bone levels, and excessive occlusal force(s), primary occlusal trauma occurs.

Secondary occlusal trauma is injury resulting in tissue changes from normal or excessive occlusal forces applied to a tooth or teeth with reduced periodontal support.[8] In the presence of attachment loss, bone loss, and normal/excessive occlusal force(s), secondary occlusal trauma occurs.

Fremitus

When a tooth is subjected to occlusal forces, a palpable or visible movement on the tooth is appreciable, called as fremitus.[7]

Bruxism or tooth grinding

A habit of grinding or clenching of the teeth.[7] Both tooth and attachment apparatus may get damaged by the forces generated.

Forces During Jaw Movements

During the process of chewing, swallowing, or any parafunctional habits, such as bruxism and clenching, the teeth and their periodontium are predisposed to functional dynamic loading.

Frequently, in normal healthy adults, tooth-to-tooth contact or near contact occurs during mastication. The lateral guiding cusps (commonly of cuspids and bicuspids) come in contact, and during closure, the jaw, follows a wide lateral path. Relatively, a low magnitude of forces (averaging 81 N) and short-acting, with a duration of about 20–50 ms, are generated by these contacts.

Forces at final closure in the intercuspal position are much greater (averaging 262 N) and are also longer acting with an average duration of about 115 ms. An average force of 296 N and a duration of about 700 ms occur in the intercuspal position, during chewing and swallowing.

Classification OF TFO

Glickman's classification (1953)

According to the duration of cause:[8]

1. Acute TFO.
2. Chronic TFO.

Acute TFO

An abrupt occlusal impact, caused by chewing on a hard object, restorations or prosthetic appliances that may alter the occlusal forces.

Acute trauma may lead to pain, sensitivity, and increased mobility of the tooth.

Chronic TFO

It is more frequent and has more clinical significance. The gradual changes in occlusion caused by tooth wear, drifting movement, and extrusion of the teeth along with parafunctional habits lead to chronic TFO.

According to the nature of cause, chronic TFO is classified into: [8]

1. Primary TFO.
2. Secondary TFO.

- Primary TFO: TFO occurs as the result of alterations in occlusal forces.
- Secondary TFO: It occurs as a result of reduced ability of the tissues to resist the occlusal forces.

Box's classification[9]

Physiologic occlusion

A condition, in which the systems of forces acting on the tooth during the occlusion are in a state of equilibrium, and they do not and cannot change the normal relationship existing between the tooth and its supporting structures, defined by box.

Traumatic occlusion

The damage produced in the periodontium is due to the overstress produced by the occlusion.

Hamp, Nyman, and Lindhe's classification (1975)[9]

This classification is based on a horizontal component of tissue destruction that has occurred in the interradicular area.

- Degree I: Horizontal loss of periodontal tissue support not exceeding one-third of the width of the tooth.
- Degree II: Horizontal loss of periodontal support exceeding one-third of the width of the tooth.
- Degree III: Horizontal through-and-through destruction of the periodontal tissue in the furcation area.

Etiological Factors[10]

Precipitating factors

The irritants and the devasting occlusal forces that further destroy the tissues are weakened by the predisposing factors.

Predisposing factors

Factors which take the place of those contributing to the histopathologic lesion are listed as developmental factors, functional mechanisms, and the systemic component. They can be divided into:

1. Intrinsic factors: Consist of the morphology of the roots, alveolar process, and the orientation of the occlusal surfaces and roots to the forces, in which the tooth gets exposed to.
2. Extrinsic factors: Consist of plaque, parafunctional activities, bone lossor loss of teeth, and malocclusion created iatrogenically.

Etiology For Primary TFO[8]

1. A "high filling;"
2. Prosthetic replacement which creates undesired forces on abutment and opposite teeth;
3. Drifting or extrusion of teeth into unreplaced missing teeth space;
4. Movement of teeth into functionally unacceptable positions by orthodontic procedures.

Etiology For Secondary TFO[8]

Reduced height of bone with:
1. Normal periodontium,
2. Marginal periodontitis.

TFO And Plaque-Associated Periodontal Disease

Analysis of human autopsy material[1]

The assessments made from cadaver specimens have a limited to questionable value when "cause–effect" relationships between occlusion, plaque, and periodontal lesions are to be described. The conclusions drawn from this type of research can be controversial. This can best be illustrated if "Glickman's concept" is compared with "Waerhaug's concept" of what autopsy studies have revealed regarding TFO and periodontal disease.

Glickman's concept[11,12]

Glickman (1965, 1967) claimed that, if forces of an abnormal magnitude are acting on teeth harboring subgingival plaque, then the alley of the spread of a plaque-associated gingival lesion can be altered.

The periodontal structures can be divided into:
1. The zone of irritation.
2. The zone of codestruction.

The zone of irritation consists of the marginal and interdental gingiva. The soft tissue zone is surrounded by the hard tissue (the tooth) on one side and has no impact by occlusal forces. This means that gingival inflammation cannot be initiated by TFO but rather due to irritation from plaque.

The zone of codestruction consists of the periodontal ligament, cementum, and alveolar bone and is coronally delineated by the transseptal and the dentoalveolar collagen fiber bundles.

In contrast to the co-destructive theory, Waerhaug and Glickman, both had examined autopsy specimens, but Waerhaug also measured the distance from the subgingival plaque to the periphery of the associated inflammatory cell infiltrate in the gingiva and the adjacent alveolar bone surface. He came to the conclusion that angular bony defects and also infrabony pockets occur equally often at periodontal sites which are unaffected by TFO like in traumatized teeth.[4]

Various animal studies using the Squirrel Monkey[13-15] and Beagle Dog[16-20] evaluated the excessive jiggling force's effects in the experimentally induced periodontitis.

The conclusions of these studies are as follows:
1. Occlusal trauma does not induct gingival inflammation.
2. A traumatogenic occlusion will lead in increased mobility, widening of PDL, and crestal bone loss along with bone volume but no attachment loss in the absence of inflammation.
3. When gingival inflammation was present, excess jiggling forces did not cause increased attachment loss in squirrel monkeys, but accelerating occlusal forces may increase attachment loss in beagle dogs.
4. Although there will be no difference in attachment level, after treating the inflammation in the existence of mobility, it will at least decrease the mobility of teeth and help in gaining bone density.

Tissue Response To TFO[8]

There are three stages of tissue response: Injury, repair, and adaptive remodeling of the periodontium.

Stage I Injury[8]

Immoderate pressure initiates resorption of the alveolar bone, along with widening of the periodontal ligament space. Immoderate tension leads to elongation of the periodontal ligament fibers and alveolar bone apposition. In areas of excessive pressure, there are numerous blood vessels which are reduced in size; in areas of excessive tension, they are expanded. Greater pressure leads to changes in the PDL, such as compression of the fibers, producing hyalinization.

Stage II Repair[8]

In the normal periodontium, repair occurs persistently, and increased reparative activity is stimulated due to TFO. When due to excessive occlusal forces, bone is resorbed, the body reinforces the bony trabeculae which is thinned, with new bone. This process of compensating for the lost bone is termed as "buttressing bone formation."

Buttressing bone formation occurring within the jaw is called central buttressing and on the bony surface is called "peripheral buttressing."

A shelflike thickening of the alveolar margin or an evident bulge in the facial and lingual bone may be produced by peripheral buttressing which is called as "lipping."

Stage III Adaptive remodeling of the periodontium[8]

If the destruction due to the occlusion surpasses the repair process, the periodontium is remodeled so that it can maintain a structural relationship. This leads to thickening of the PDL, which will be funnel shaped at the crest and angular defects in the junctional epithelium without any pocket formation. The involved teeth become mobile. There will also be an increase in vascularization.

Signs[21,22]

Clinical signs
1. Mobility and periodontal ligament widening Positive fremitus test
2. in the apical region and in bifurcationareas, lamina dura thickening can be seen.
3. Occlusal prematurities.

Radiographic signs[8,22,23]
1. Periodontal ligament widening, mostly with lamina dura thickening along the root's lateral aspect, apical region, and bifurcation.
2. Vertical destruction of the interdental septum.
3. Radiolucency and alveolar bone condensation.
4. Resorption of root.
5. Migration of tooth.
6. Tooth fracture.
7. Thermal sensitivity.

Diagnosis of TFO[21,22]

For the correct diagnosis of TFO, proper clinical history taking and clinical examination of the patients are vital.
1. Cardinal manifestation of primary TFO is increased tooth mobility. The mobility can be assessed by mechanical and electronic instrument. Subjective assessments of mobility are done as in Miller classification assigned from 0 to 3 score.
2. Tilting and migration of individual teeth or of complete segments. The percussion of teeth on tapping with a blunt instrument changes from a resonant note with a healthy supporting structure to a dull note if there is primary TFO in attachment apparatus.
3. Careful palpation of the muscles of mastication to ascertain whether there is hypertrophy or sign of hypertonicity with possible spasm of one group of muscle.
4. Palpation of TMJ and observation of any deviation of the mandible in various paths of closure.
5. Fremitus test: [23,24] Measures the vibratory patterns of the teeth during contact positions and during movements.

The following classification system is used:
- Class 1: Mild vibration or movement detected.
- Class II: Easily palpable vibration but no visible movement.
- Class III: Movement visible with the naked eye.

Goals And Treatment Considerations[25]

TXTreatment considerations that must be considered include one or more of the following: [25]
1. Occlusal adjustment of the tooth.
2. Correction of parafunctional habits.
3. Temporary, provisional, or long-term stabilization of mobile teeth with appliances.
4. Orthodontic corrections.
5. Occlusal readjustments.
6. Extraction of indicated teeth.

Occlusal adjustment

Occlusal adjustment means, setting up of functional relationships appropriate to the periodontium by: Coronoplasty, dental restorations, tooth removal or by orthognathic surgery.[26]

Indications and contraindications for occlusal adjustment

Given by World Workshop in Periodontics (1989)[27]

Indications
1. To fortify repair within the periodontal attachment apparatus by reducing traumatic forces to teeth that exhibit increased mobility or fremitus.
2. To obtain functional association and masticatory effectiveness along with restorative treatment, orthodontic, orthognathic surgery, or jaw trauma.
3. As an adjunct, reducing the damage from parafunctional habits.
4. To recontour teeth, responsible for causing soft tissue injury.

Contraindications
1. Prophylactic adjustment.
2. As a treatment of primary, microbial-induced inflammatory periodontal disease.
3. Based on a patient history, correction of bruxism, without exact evidence of damage, pathosis, or pain.
4. When the patient's emotional state impedes a satisfactory result.
5. When severe extrusion, mobility or malpositioning of teeth is present where occlusal adjustment would not be sufficient.

Management of parafunctional habits

The perfect diagnosis of bruxism can be acheived by taking proper history and by assessing the clinical features of the patient. Methods by which the patient with bruxism can betreated: Electromyographic biofeedback[28,29] a physical therapy course, medications aimed at altering sleep arousal or anxiety level and the appliances for maxillary stabilization.

Splint

A splint is an appliance used for immobilization or stabilization.[30] Splinting is stabilization, achieved by joining two or more teeth to increase resistance to the forces applied. The types being the short-term splint, the provisional or long-term splint.

Indications and Contraindications for Splinting

Given by World Workshop in Periodontics (1989)[27]

Indications
1. To stabilize the teeth with increased mobility that have not been corrected by occlusal adjustment and periodontal treatment.

2. When there is an interference with the normal functioning and patient discomfort.
3. Splinting extremely mobile teeth, before periodontal instrumentation and occlusal adjustment procedures, facilitating the treatment.
4. To prevent tipping or drifting and extrusion of teeth.
5. Following orthodontic movement to stabilize the teeth.
6. To create enough occlusal stability while replacing the missing teeth.
7. Following acute trauma.

Contraindications

1. If the treatment of inflammatory periodontal disease has not been done.
2. If occlusal adjustment for the reduction of trauma and/or interferences has not been addressed previously.

Occlusal reconstruction

In cases where no other treatment could achieve occlusal equilibration, occlusal reconstruction has to be done. It consists of recontouring the occlusal contacts by providing crowns, bridges, or implant-supported prosthesis.

Extraction

A tooth which has a poor prognosis and by the extraction of which, the prognosis of the remaining teeth improves, then the tooth in question should be extracted.

Conclusion

Inconclusive evidence based on well-controlled prospective human studies has led to the unsure treatment of periodontium affected with TFO. Removal of the anomalous occlusal forces and stabilization of the affected tooth/teeth is the most relevant therapy for teeth affected by TFO.

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