IJFANS INTERNATIONAL JOURNAL OF FOOD AND NUTRITIONAL SCIENCES
ISSN PRINT 2319 1775 Online 2320 7876
Research Paper , UGC CARE Listed (Group -I) Journal Volume 11, Iss 12, 2022

Trauma from Occlusion and Periodontium: A Review

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Abstract: Trauma from occlusion (TFO) is the term for any occlusal force that damages periodontal structures beyond our periodontium's adaptive capacity. There are differing opinions on whether TFO is a cofactor or an etiological factor in the development of periodontal diseases. This review study aims to elucidate the true meaning of TFO, including its etiological aspects, historical context, pertinent terminology, signs and symptoms, and improved diagnostic techniques.

Introduction: A term used to describe pathologic alterations or adaptive changes which develop in the periodontium as a result of undue forces produced by the masticatory muscles. Stillman (1917) “a condition where injury results to the supporting structures of the teeth by the act of bringing the jaws into a closed position”. WHO 1978 “damage in the periodontium caused by stress on the teeth produced directly or indirectly by teeth of the opposing jaw”. In “Glossary of Periodontic Terms” (AAP 1986), “an injury to the attachment apparatus as a result of excessive occlusal force”. Other terms used are: traumatizing occlusion, occlusal trauma, traumatogenic occlusion, periodontal traumatism, overload, etc.While the role of occlusion in the progression of periodontal disease has been discussed and studied for over 100 years it has been and remains a controversial subject[1]. It is well understood that trauma from occlusion does not initiate or accelerate attachment loss due to inflammatory periodontal disease. The relationship between trauma from occlusion associated with progressively increasing tooth mobility causing an accelerated attachment loss in patients with inflammatory periodontal disease. For these reasons when treating periodontal patients with occlusal issues the first aim of therapy should be directed at alleviating plaque-induced inflammation. Once this has been accomplished efforts can then be directed at adjusting the occlusion. This may result in a decrease in mobility, decrease in the width of the periodontal ligament space, increase in overall bone volume. Finally, in cases planned for regenerative therapy, consideration to stabilizing mobile teeth should also be given prior to surgical intervention.[2]

Historical background:

Karolyi (1901) proposed the possibility of a relationship between alveolar pyorrhea and trauma resulting from occlusion.

Research on sheep and monkeys reported by Box (1935) and Stones (1938) appeared to suggest that trauma from occlusion is an etiologic factor in the development of that type of periodontal disease where there is vertical pocket formation connected to one or more teeth.

Early in the 1960s, Glickman and Smulow[3-5] put out the hypothesis that a traumatogenic occlusion would contribute to the development of periodontitis. The "co destructive theory" is the name given to this idea. Goldman[6] shown that soft tissue lesions like McCall's festoons and Stillman's clefts were not caused by occlusal stress. Waerhaug's[7,8] research demonstrated TFO's role in the aetiology of Infra-bony pockets.

Various Classifications of Trauma from Occlusion

Glickman's classification (1953)[1]

According to duration of cause:

i. Acute TFO

ii. Chronic TFO.

According to nature of cause:

i. Primary TFO

ii. Secondary TFO.

Box's classification[9]

Physiologic occlusion

Box defined it as a condition, in which the systems of forces acting upon 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.

Traumatic occlusion

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

Hamp, Nyman, and Lindhe's classification (1975)[2]

This classification is based on a horizontal component of tissue destruction that has occurred in the interradicular area, i.e., degree of horizontal root exposure or attachment loss.

• **Degree I:** Horizontal loss of periodontal tissue support notexceeding one-third of the width of the tooth

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• **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

Acute trauma from occlusion

Causes: An abrupt occlusal impact/ by biting on a hard object, Restorations/prosthetic appliances.

Manifestations: Tooth pain,

Sensitivity to percussion

Increased tooth mobility

If the force is dissipated by a shift in the position or by wearing away or correction of the restoration, injury heals and the symptoms subside.Otherwise, periodontal injury may worsen and develop into necrosis accompanied by periodontal abscess formation or persist as a symptom-free chronic condition.[10]

Chronic trauma from occlusion: More common & significant

Gradual changes by: Tooth wears, Drifting movement & extrusion

This condition is usually associated with parafunctional habits such as bruxism and clenching.with duration of time pathological changes are seen in the tooth supporting structures.[11]

Primary Trauma from occlusion: Primary TFOResult of alterations in occlusal forces, it is called primary trauma from occlusion. Occurs if

T.F.O. Is considered the primary etiologic factor in periodontal destruction examples

1) The insertion of a "high filling,"

2) Insertion of prosthetic

3) Drifting movement or extrusion of teeth

4) Orthodontic movement of teeth into functionally unacceptable positions.

Changes produced by primary trauma do not alter the level of connective tissue attachment and do not initiate pocket formation because the supracrestal gingival fibers are not affected and therefore prevent apical migration of the functional epithelium."[10]

SecondaryTrauma from occlusion: It Results from reduced ability of the tissues to resist the occlusal forces; it is known as secondary trauma from occlusion.Secondary TFO Occurs when the adaptive capacity of the tissues to withstand occlusal forces is impaired by bone loss resulting from marginal inflammation which Reduces the periodontal attachment area and alters the leverage on the remaining tissues. The periodontium becomes more vulnerable to injury, and previously well-tolerated occlusal forces become traumatic.

TFO And Plaque-Associated Periodontal Disease Analysis of human autopsy material[2]

When describing "cause–effect" correlations between periodontal, plaque, and occlusion diseases, the assessments derived from cadaver specimens are of limited to dubious utility. This kind of study might provide contentious results. The easiest way to convey this is to contrast "Glickman's concept" with "Waerhaug's concept" of what postmortem findings of TFO and periodontal disease have shown.

Glickman's concept[3]

- Glickman (1965, 1967) claimed that the pathway of the spread of a plaque-associated gingival lesion can be changed if forces of an abnormal magnitude are acting on teeth harboring subgingival plaque.[14]
- The character of the progressive tissue destruction of the periodontium at a "traumatized tooth" will be different from a "non-traumatized" tooth.
- Instead of an even destruction of the periodontium and alveolar bone (suprabony pockets and horizontal bone loss) according to Glickman, the sites with uncomplicated plaque-associated lesions, sites which are also exposed to abnormal occlusal force will develop angular bony defects and infrabony pockets.[8]
- The periodontal structures can be divided into two zones

1. The zone of irritation

2. The zone of co-destruction

The zone of irritation includes the marginal and interdental gingiva. The soft tissue of this zone is bordered by hard tissue (the tooth) only on one side and is not affected by forces of occlusion. This means that gingival inflammation cannot be induced by trauma from occlusion but is the result of irritation from microbial plaque. The plaque-associated lesion at a "non-traumatized" tooth propagates in the apical direction by first involving the alveolar bone and only later the periodontal ligament area.

The zone of co-destruction includes the periodontal ligament, the root cementum, and the alveolar bone, and is coronally demarcated by the trans-septal (interdental) and the dento-alveolar collagen fiber bundles.[15]

The fiber bundles which separate the zone of co-destruction from the zone of irritation can be affected from two different directions:

1. From the inflammatory lesion maintained by plaque in the zone of irritation
2. From trauma-induced changes in the zone of co-destruction.

The spread of an inflammatory lesion from the zone of irritation directly down into the periodontal ligament may hereby be facilitated. This alteration of the "Normal" pathway of spread of the plaque-associated inflammatory lesion results in the development of angular bony defect.

Waerhaug's concept

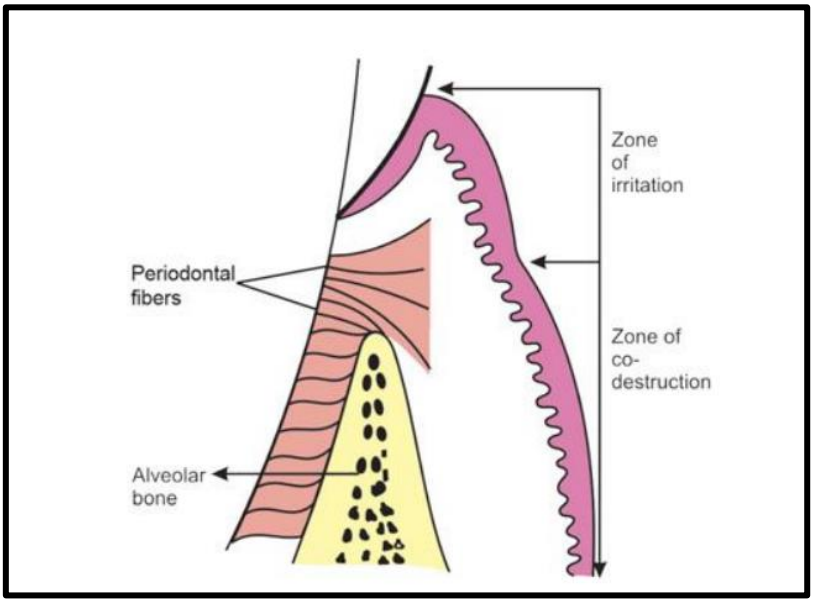
Waerhaug (1979) examined autopsy specimens similar to Glickman but in addition measured the distance between the subgingival plaque:

(1) The periphery of the associated inflammatory cell infiltrate in the gingiva

(2) The surface of the adjacent alveolar bone.

- He concluded from his analysis that angular bony defects and infra-bony pockets occur equally often at periodontal sites of teeth which are not affected by trauma from occlusion as in traumatized teeth.[1]
- The loss of connective tissue attachment and the resorption of bone around teeth exclusively the result of inflammatory lesions associated with subgingival plaque.

- Angular bony defects and infra-bony pockets occur when the subgingival plaque of one tooth has reached more apical level than the microbiota on the neighboring tooth and when the volume of the alveolar bone surrounding the roots is comparatively large.
- Waerhaug's observations support findings presented by Prichard (1965) and Manson (1976) which imply that the pattern of loss of supporting structures is the result of an interplay between the form and volume of the alveolar bone.
- Authors tend to accept Glickman's conclusions that trauma from occlusion is an aggravating factor in periodontal disease e.g. Macapanpan& Weinmann 1954; Posselt & Emslie 1959; Glickman &Smulow 1962, 1965[14].
- While others accept Waerhaug's concept e.g. Lovdahl et al. 1959; Belting & Gupta 1961; Baer et al. 1963; Waerhaug 1979.



Schematic diagram of Zone of Irritation & Zone of Destruction

Pathogenesis of trauma from occlusion (stages of tissue response)

Stage I: Injury

- Tissue injury is produced by excessive occlusal forces. The body then attempts to repair the injury and restore the periodontium. This can occur if the forces are diminished or if the tooth drifts away from them.

- if the offending force is chronic, the periodontium is remodeled to cushion its impact. The ligament is widened at the expense of the bone, resulting in angular bone defects without periodontal pockets and the tooth becomes loose.
- Slight excessive pressure stimulates the resorption of alveolar bone that result in widening of pdl ligament space and narrowing of blood vessels.
- Slight excessive tension cause elongation of pdl ligament & apposition of alveolar bone and blood vessel become enlarged.
- Greater pressure causes the compression of pdl ligament which produce area of hyalinization that result in subsequent injury to the fibroblast & other connective tissue cells leads to necrosis of area of ligament.
- Vascular changes results in retardation and stasis of blood flow within 30 min. and at 2-3 hrs blood vessel to be packed with erythrocyte and between 1–7day disintegration of blood vessel.
- Severe tension cause widening of pdl, thrombosis, hemorrhage, tearing of pdl ligament and resorption of alveolar bone.
- Severe pressure enough to force the root against bone result in necrosis of pdl ligament & bone. The bone is resorbed from viable pdl ligament adjacent to necrotic areas and from marrow spaces, called undermining resorption.
- Injury to the periodontium produces a temporary depression in mitotic activity and the rate of proliferation and differentiation of fibroblasts in collagen formation and in bone formation. These return to normal levels after dissipation of the forces.

Stage II: Repair

- Repair is constantly occurring in the normal periodontium and trauma from occlusion stimulates increased reparative activity.
- The damaged tissues are removed and new connective tissue cells and fibers, bone, and cementum are formed in an attempt to restore the injured periodontium.
- When bone is resorbed by excessive occlusal forces, the body attempts to reinforce the thinned bony trabeculae with new bone. This attempt to compensate for lost bone is called **BUTTRESSING BONE FORMATION**.[10]
- When buttressing bone formation occur within jaw, it's called **CENTRAL BUTTRESSING**. In central buttressing, the endosteal cell deposit new bone which restores the bony trabeculae and reduce size of marrow spaces.

- When buttressing bone formation occurs on bone surface, called **PERIPHERAL BUTTRESSING** – occurs on facial and lingual surface of alveolar plate. It may produce a shelf-like thickening of alveolar margin, referred as **LIPPING**.[12]

Stage III: Adaptive Remodelling of the Periodontium

- If the repair process cannot keep pace with the destruction caused by the occlusion, the periodontium is remodeled in an effort to create a structural to the tissues.
- This results in a thickened periodontal ligament, which is funnel shaped at the crest, and angular defects in the bone, with no pocket formation. The involved teeth become loose. Increased vascularization has also been reported.

Effects Of Insufficient Occlusal Force

- Insufficient occlusal force may also be injurious to the supporting periodontal tissues.
- Insufficient stimulation causes thinning of the periodontal ligament, atrophy of the fibers, osteoporosis of the alveolar bone, and reduction in bone height.
- Hypofunction can result from an open-bite relationship, an absence of functional antagonists, or unilateral chewing habits that neglect one side of the mouth.

Reversibility of traumatic lesions

- Trauma from occlusion is reversible. When trauma is artificially induced in experimental animals, the teeth move away or intrude into the jaw. When the impact of the artificially created force is relieved, the tissues undergo repair.
- Although trauma from occlusion is reversible under such conditions, it does not always correct itself nor it is always temporary and of limited clinical significance. The injurious force must be relieved for repair to occur.
- If conditions in humans do not permit the teeth to escape from or adapt to excessive occlusal force, periodontal damage persists and worsens.
- The presence of inflammation in the periodontium as a result of plaque accumulation may impair the reversibility of traumatic lesions.

Clinical Signs of Trauma from Occlusion[15.16]

- Increasing tooth mobility and migration or drifting.

- Persistent discomfort on eating.
- Occlusal prematurities.
- Wear facets in presence of other clinical indicators.
- Tooth migration.
- Fractured tooth (teeth).
- Thermal sensitivity.
- **Fremitus test:**is the movement of a tooth or teeth subjected to functional occlusal forces. this can be assessed by palpating the buccal aspect of several teeth as the patient taps up and down.
- **Periodontometers:** It was a research tool used in the 1950s and 1960s to standardise the measurement of even minor tooth displacement.
- **Periotest**: This device was produced in Germany in the late 1980s to provide a more reliable method for determining tooth mobility. It is designed to measure the reaction of the periodontium to a defined percussion, delivered by a tapping instrument.[13]

Radiographic Signs of Trauma from Occlusion[16,17]

Injury phase: Loss of the laminadura in apices, furcations and/or marginal areas - results in widening of the periodontal ligament space.

Repair phase: It results in an attempt to strengthen the periodontal structures to better support the increased load - manifested by a widening of the periodontal ligament space, which may be generalized or localized.When variations in width between the marginal area and midroot or between the mid-root and apex are detected, it means that the tooth is being subjected to increased forces.

More advanced traumatic lesions

- It may result in deep angular bone loss and when itcombined with marginal inflammation may lead to intra-bony pocket formation.
- In terminal stages these lesions extend around the root apex, producing a wide radiolucent periapical image i.e. cavernous lesion.
- Root resorption may also occur as a result of excessive forces on the periodontium, particularly those caused by orthodontic appliances.[14]

Management of TFO: A goal of periodontal therapy in the treatment of occlusal traumatism should be to maintain the periodontium in comfort and function.

In order to achieve this goal a number of treatment considerations must be considered including one or more of the following:

1) Occlusal adjustment

2) Management of parafunctional habits

3) Temporary, provisional or long-term stabilization of mobile teeth with removable or fixed appliances

Occlusal adjustment:[18,19]

it defined as reshaping the occluding surfaces of teeth by grinding to create harmonious contact relationships between the upper and lower teeth

Indications for Occlusal Adjustment (By the World Workshop in 1989)[20]

1) To reduce traumatic forces to teeth that exhibit:

- Mobility or fremitus to encourage repair within the periodontal attachment apparatus.
- Discomfort during occlusal contact or function.

2) To achieve functional relationships and masticatory efficiency in conjunction with restorative treatment, orthodontic, orthognathic surgery or jaw trauma when indicated.

3) As adjunctive therapy that may reduce the damage from parafunctional habits.

4) To reshape teeth contributing to soft tissue injury.

5) To adjust marginal ridge relationships and cusps that are contributing to food impaction

Splinting

Indications for Splinting (By the World Workshop in 1989)[20]

1) Stabilize teeth with increasing mobility that have not responded to occlusal adjustment and periodontal treatment.

2) Stabilize teeth with advanced mobility that have not responded to occlusal adjustment and treatment when there is interference with normal function and patient comfort.

3) Facilitate treatment of extremely mobile teeth by splinting them prior to periodontal instrumentation and occlusal adjustment procedures.

4) Prevent tipping or drifting of teeth and extrusion of unopposed teeth.

5) Stabilize teeth when indicated following orthodontic movement.

6) Create adequate occlusal stability when replacing missing teeth.

7) Splint teeth so that a root can be removed and the crown retained in its place.

8) Stabilize teeth following acute trauma.

Conclusion:

In the treatment of periodontal disorders, knowledge of the impact of trauma from occlusion on the periodontium is helpful. Under some circumstances, occlusion-related stress to teeth with plaque-associated periodontal disease may accelerate the disease's course and contribute to its destructive process. Clinically, this knowledge reinforces the need for appropriate management of periodontal disease linked to plaque and give more strength to treatment plan. This treatment will also arrest the destruction of the periodontal tissues even if the occlusal trauma persists.

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