Periodontal pocket

The periodontal pocket :- defined as a pathologically deepened gingival sulcus, is one of the most important clinical features of periodontal disease. All types of periodontitis, share histopathologic features, such as tissue changes in the periodontal pocket, mechanisms of tissue destruction, and healing mechanisms.

Classification

Deepening of the gingival sulcus may occur as a result of **coronal** movement of the gingival margin, **apical** displacement of the gingival attachment, or a **combination** of the two processes. Pockets can be classified as follows:

1- **Gingival pocket** (also called False or "pseudo-pocket") is formed by gingival enlargement without destruction of the underlying periodontal tissues. The sulcus is deepened because of the increased bulk of the gingiva.

2- **Periodontal pocket** produces destruction of the supporting periodontal tissues, leading to the loosening and exfoliation of the teeth. Based on the location of the base of the pocket in relation to the underlying bone, periodontal pockets can be classified into the following types:

   A- Suprabony (supra-crestal or supra-alveolar) occurs when the bottom of the pocket is coronal to the underlying alveolar bone. The pattern of destruction of the underlying bone is **horizontal**.

   B- Intrabony (infrabony, subcrestal, or intra-alveolar) occurs when the bottom of the pocket is apical to the level of the adjacent alveolar bone. The lateral pocket wall lies between the tooth surface and the alveolar bone. The pattern of bone destruction is **vertical (angular).**

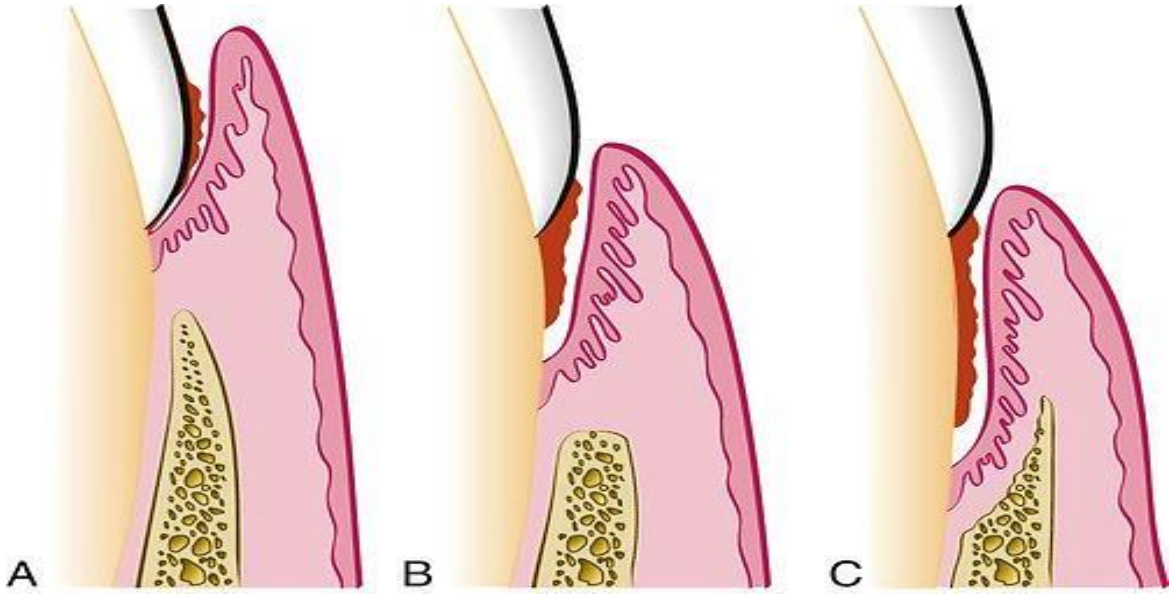

Fig. :- Pockets can be classified into :- A. Gingival pocket, B. Suprabony pocket, C. Infrabony pocket.

Pockets can involve one, two, or more tooth surfaces, and they can be of different depths and types on different surfaces of the same tooth and on proximal surfaces of the same interdental space.

Pockets can also be spiral (i.e., originating on one tooth surface and twisting around the tooth to involve one or more additional surfaces) ,these types of pockets are **most common** in furcation areas.

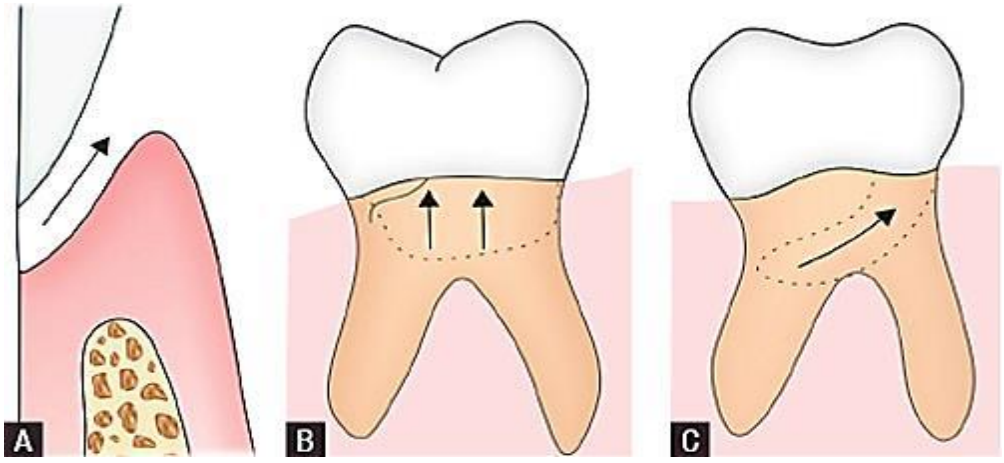

Fig.:- A. Simple pocket, B. Compound pocket, C. Spiral (complex) pocket.

Clinical Features

Clinical signs & symptoms that suggest the presence of periodontal pockets **include:-**

- ☒ Bluish red thickened marginal gingiva
- ☒ Bluish red vertical zone from the gingival margin to the alveolar mucosa
- ☒ Gingival bleeding and suppuration
- ☒ Tooth mobility
- ☒ Diastema formation
- ☒ Localized pain or pain "deep in the bone.

The only reliable method of locating periodontal pockets and determining their extent is careful probing of the gingival margin along each tooth surface.

Table :- Correlation of Clinical and Histopathologic Features of the Periodontal Pocket

Clinical Features	Histopathologic Features
1. The gingival wall of the pocket presents as bluish red discoloration; flaccidity; a smooth, shiny surface; and pitting on pressure.	1. The discoloration is caused by circulatory stagnation; the flaccidity by the destruction of gingival fibers and surrounding tissues; the smooth, shiny surface by atrophy of the epithelium and edema; and the pitting on pressure by edema and degeneration.
2. Less frequently, the gingival wall may be pink and firm.	2. In such cases, fibrotic changes predominate over exudation and degeneration.
3. Bleeding is elicited by gently probing the soft tissue wall of the pocket.	3. Ease of bleeding results from increased vascularity, the thinning and degeneration of the epithelium, and the proximity of engorged vessels to the inner surface.
4. When explored with a probe, the inner	4. Pain on tactile stimulation is caused by the ulceration of

aspect of the pocket is generally painful.	the inner aspect of the pocket wall.
5. In many cases, pus may be expressed with the application of digital pressure.	5. Pus occurs in pockets with suppurative inflammation of the inner wall.

Pathogenesis of the periodontal pocket

The initial lesion in the development of periodontitis is the inflammation of the gingiva in response to a bacterial challenge. Changes involved in the transition from the normal gingival sulcus to the pathologic periodontal pocket are associated with different proportions of bacterial cells in dental plaque. Healthy gingiva is associated with few microorganisms, mostly coccoid cells and straight rods. Diseased gingiva is associated with increased numbers of spirochetes and motile rods.

Early concepts assumed that after the initial bacterial attack, periodontal tissue destruction continued to be linked to bacterial action. **More recently** it was established that the host's immune-inflammatory response to the initial and persistent bacterial attack lead to collagen and bone destruction. These mechanisms are related to various cytokines, some of which are produced normally by cells in non-inflamed tissue and others by cells that are involved in the inflammatory process, such as polymorphonuclear leukocytes (PMNs), monocytes, and other cells, leading to collagen and bone destruction.

Pocket formation **starts** as an inflammatory change in the connective tissue wall of the gingival sulcus. The cellular and fluid inflammatory exudate causes degeneration of the surrounding connective tissue, including the gingival fibers. Just apical to the junctional epithelium, collagen fibers are destroyed and the area is occupied by inflammatory cells and edema.

The two mechanisms associated with collagen loss are:-

1- Collagenases and other enzymes secreted by various cells in healthy and inflamed tissue, such as fibroblasts, polymorphnuclear leukocyte (**PMNs**), and macrophages, become extracellular and destroy collagen, these enzymes that degrade collagen into small peptides are called matrix metalloproteinases (**MMP**)

2- Fibroblasts phagocytize collagen fibers by extending cytoplasmic processes to the ligament-cementum interface and degrade the collagen fibrils.

As a consequence of the loss of collagen, the apical cells of the junctional epithelium proliferate along the root, extending fingerlike projections two or three cells in thickness . As a result of inflammation, PMNs invade the coronal end of the junctional epithelium in increasing numbers.

When the relative volume of **PMNs** reaches approximately **60%** or more of the junctional epithelium, the tissue loses cohesiveness and detaches from the tooth surface. Thus the coronal portion of the junctional epithelium detaches from the root as the apical portion migrates, resulting in its apical shift, and the oral sulcular epithelium gradually occupies an increasing portion of the sulcus.

Extension of the junctional epithelium along the root requires the presence of healthy epithelial cells. Marked degeneration or necrosis of the junctional epithelium impairs rather than accelerates pocket formation. (This occurs in necrotizing gingivitis, which results in an ulcer rather than pocket formation.) Degenerative changes seen in the junctional epithelium at the **base** of periodontal pockets are usually **less severe** than those in the epithelium of the **lateral** pocket wall. Because the migration of the junctional epithelium requires healthy, viable cells, it is reasonable to assume that the degenerative changes seen in this area

occur after the junctional epithelium reaches its position on the cementum. With continued inflammation, the gingiva increases in bulk and the crest of the gingival margin extends coronally. The apical cells of the junctional epithelium continue to migrate along the root (apically) and its coronal cells continue to separate from it. The epithelium of the lateral wall of the pocket proliferates to form bulbous, cordlike extensions into the inflamed connective tissue.

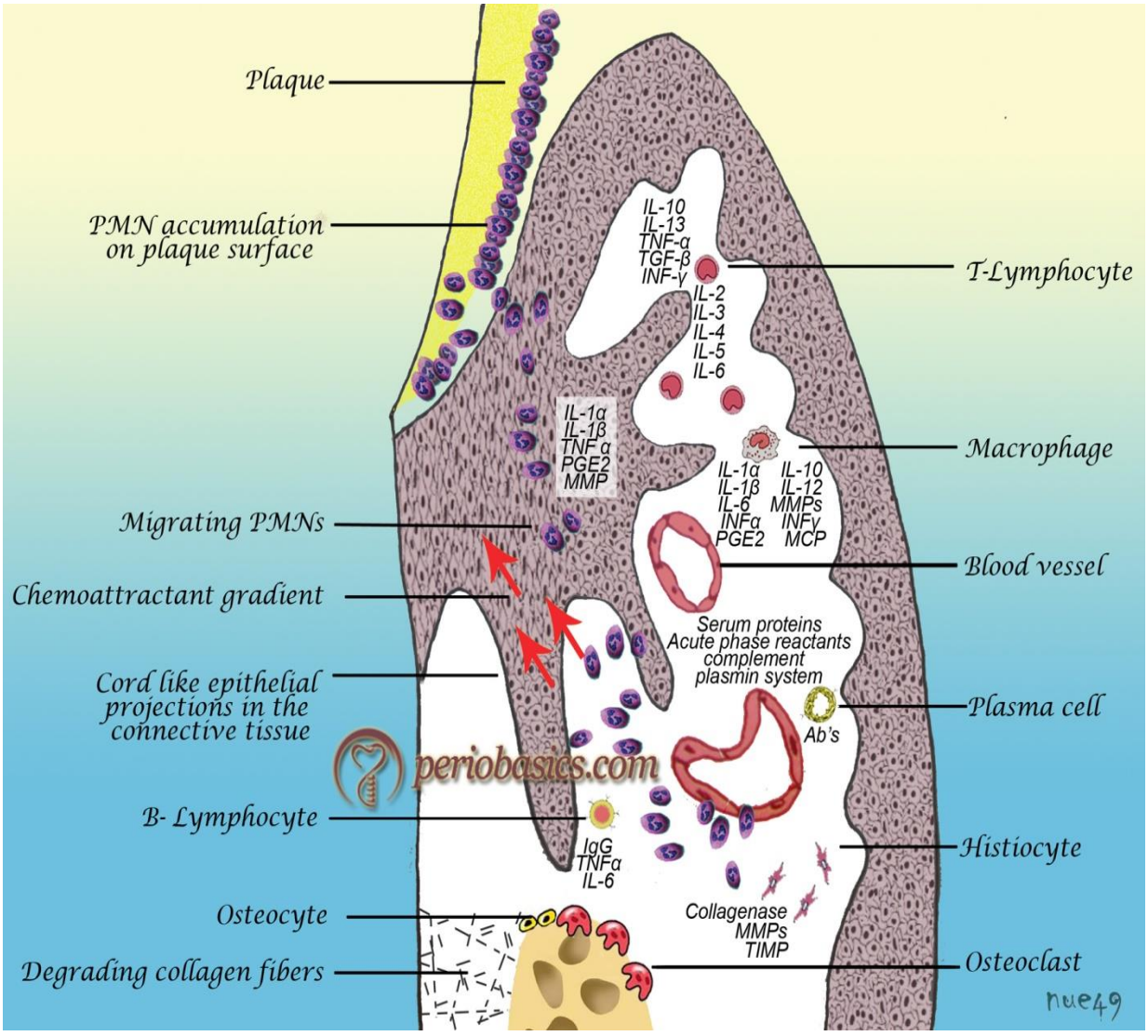

Fig. :- Pathogenesis of the periodontal pocket.

Histopathology

Once the pocket is formed, several microscopic features are present.

Soft Tissue Wall

The connective tissue is **edematous** and **densely infiltrated** with **plasma cells** (**approximately 80%**), **lymphocytes**, and **PMNs**. The blood vessels are increased in number, dilated, and engorged, particularly in the sub-epithelial connective tissue layer.

The connective tissue exhibits varying degrees of degeneration. Single or multiple necrotic foci are occasionally present. In addition to exudative and degenerative changes, the connective tissue shows proliferation of the endothelial cells, with newly formed capillaries, fibroblasts, and collagen fibers.

The junctional epithelium at the base of the pocket is usually much **shorter** than that of a normal sulcus. The coronoapical length of the junctional epithelium is reduced to only 50 to 100 μm.

The **most severe degenerative changes** in the periodontal pocket occur along the **lateral wall**. The epithelium of the lateral wall of the pocket presents striking proliferative and degenerative changes. Epithelial buds or interlacing cords of epithelial cells project from the lateral wall into the adjacent inflamed connective tissue and may extend farther apically than the junctional epithelium. These epithelial projections, as well as the remainder of the lateral epithelium, are densely infiltrated by leukocytes and edema from the inflamed connective tissue. Progressive degeneration and necrosis of the epithelium lead to ulceration of the lateral wall, exposure of the underlying inflamed connective tissue, and suppuration.

The severity of the degenerative changes is **not necessarily related** to pocket depth. Ulceration of the lateral wall may occur in shallow pockets, and deep pockets are occasionally observed in which the lateral epithelium is relatively intact or shows only slight degeneration.

Bacterial Invasion

Bacterial invasion of the apical and lateral areas of the pocket wall has been described in human chronic periodontitis . Bacteria may invade the intercellular space under exfoliating epithelial cells, but they are also found between deeper epithelial cells and accumulating on the basement lamina. Some bacteria traverse the basement lamina and invade the subepithelial connective tissue.

Micro-topography of gingival wall

Scanning electron microscopy has permitted the description of several areas in the soft tissue (gingival) wall of the periodontal pocket in which different types of activity take place. These findings suggest that the pocket wall is constantly changing as a result of the interaction between the host and the bacteria. The following areas have been noted:

1. Areas of relative quiescence showing a relatively flat surface with minor depressions and elevations and occasional shedding of cells .

2. Areas of bacterial accumulation, which appear as depressions on the epithelial surface, with abundant debris and bacterial clumps penetrating into the enlarged intercellular spaces.

3. Areas of emergence of leukocytes, in which leukocytes appear in the pocket wall through holes located in the intercellular spaces .

4. Areas of leukocyte-bacteria interaction, in which numerous leukocytes are present and covered with bacteria in an apparent process of phagocytosis.

5. Areas of intense epithelial desquamation, which consist of semi attached and folded epithelial cells, sometimes partially covered with bacteria .

6. Areas of ulceration, with exposed connective tissue .

7. Areas of hemorrhage, with numerous erythrocytes.

The transition from one area to another could result from bacteria accumulating in previously quiescent areas and triggering the emergence of leukocytes and the leukocyte-bacteria interaction. This would lead to intense epithelial desquamation and finally to ulceration and hemorrhage.

Periodontal Pockets as Healing Lesions

Periodontal pockets are chronic inflammatory lesions and thus are constantly undergoing repair. Complete healing does not occur because of the persistence of the bacterial attack, which continues to stimulate an inflammatory response, causing degeneration of the new tissue elements formed in the continuous effort at repair.

The condition of the soft tissue wall of the periodontal pocket results from the interplay of the destructive and constructive tissue changes. If the inflammatory fluid and cellular exudate predominate, the pocket wall is bluish red, soft, spongy, and friable, with a smooth, shiny surface; at the clinical level, this is generally referred to as an **edematous pocket wall**. If there is a relative predominance of newly formed connective tissue cells and fibers, the pocket wall is more firm and pink, clinically referred to as a **fibrotic pocket wall** .

Edematous and fibrotic pockets represent opposite extremes of the same pathologic process not different disease entities. **Fibrotic pocket walls may be misleading because they do not necessarily reflect what is taking place throughout the pocket wall.** In some cases, inflammation and ulceration on the inside of the pocket are walled off by fibrous tissue on the outer aspect . Externally the pocket appears pink and fibrotic, despite the inflammatory changes occurring internally.

Pocket Contents

Periodontal pockets contain debris **consisting principally of** microorganisms and their products (enzymes, endotoxins, and other metabolic products), gingival fluid, food remnants, salivary mucin, desquamated epithelial cells, and leukocytes. Plaque-covered calculus usually projects from the tooth surface . Purulent exudate, if present, consists of living, degenerated, and necrotic leukocytes; living and dead bacteria, serum ,and a scant amount of fibrin.

Pus is a common feature of periodontal disease, but it is only a **secondary sign**. The presence of pus or the ease with which it can be expressed from the pocket merely reflects the nature of the inflammatory changes in the pocket wall. **It is not an indication of the depth of the pocket or the severity of the destruction of the supporting tissues**. Extensive pus formation may occur in shallow pockets, whereas deep pockets may exhibit little or no pus.

Surface morphology of tooth wall

The following zones can be found in the bottom of a periodontal pocket :

1. Cementum covered by calculus.

2. Attached plaque, which covers calculus and extends apically from it to a variable degree .

3. The zone of unattached plaque that surrounds attached plaque and extends apically to it.

4. The zone of attachment of the junctional epithelium to the tooth. The extension of this zone, which in normal sulci is more than 500 µm, is usually reduced in periodontal pockets to less than 100 µm.

5. A zone of semi destroyed connective tissue fibers may be apical to the junctional epithelium.

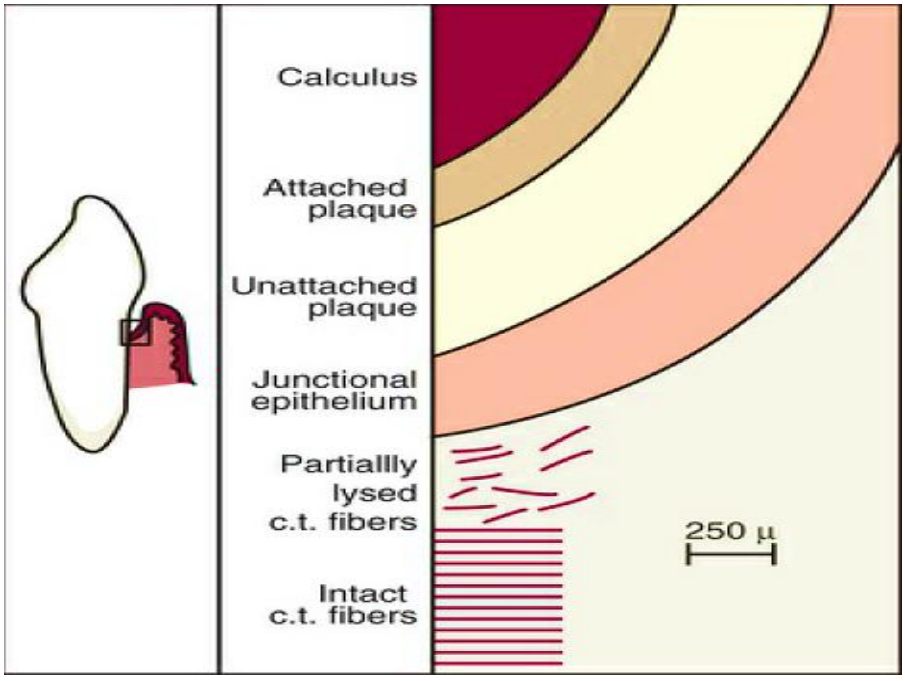

Fig. :- Tooth wall zones of the periodontal pocket.

Periodontal disease activity

Periodontal pockets go through periods of exacerbation and quiescence, resulting from episodic bursts of activity followed by periods of remission. **Periods of quiescence** are characterized by a reduced inflammatory response and little or no loss of bone and connective tissue attachment. A buildup of unattached plaque, with its gram-negative, motile, and anaerobic bacteria , starts a **period of exacerbation** in which bone and connective tissue attachment are lost and the pocket deepens. This period may last for days, weeks, or months and is eventually followed by a period of remission or quiescence in which gram-positive bacteria proliferate and a more stable condition is established.

These periods of quiescence and exacerbation are also known as **periods of inactivity and periods of activity**. Clinically, active periods show bleeding, either spontaneously or with probing, and greater amounts of gingival exudate.

Relationship of attachment loss and bone loss to pocket depth

Clinical attachment loss is the distance between the base of the pocket and cemento-enamel junction ,the severity of the attachment loss is generally but not always correlated with the depth of the pocket. This is because the degree of attachment loss depends on the location of the base of the pocket on the root surface, whereas pocket depth is the distance between the base of the pocket and the crest of the gingival margin. Pockets of the same depth may be associated with different degrees of attachment loss , and pockets of different depths may be associated with the same amount of attachment loss .

Extensive attachment and bone loss may be associated with shallow pockets if the attachment loss is accompanied by recession of the gingival margin, while slight

bone loss can occur with deep pockets (gingival pocket) . **So clinical attachment loss better than pocket depth** in reflection of the degree periodontal destruction.

Area Between Base of Pocket and Alveolar Bone

Normally, the distance between the apical end of the junctional epithelium and the alveolar bone is relatively **constant**. The distance between the apical extent of calculus and the alveolar crest in human periodontal pockets is most constant, having a mean length of **1.97 mm** (±33.16%). The distance from attached plaque to bone is never less than 0.5 mm and never more than 2.7 mm.

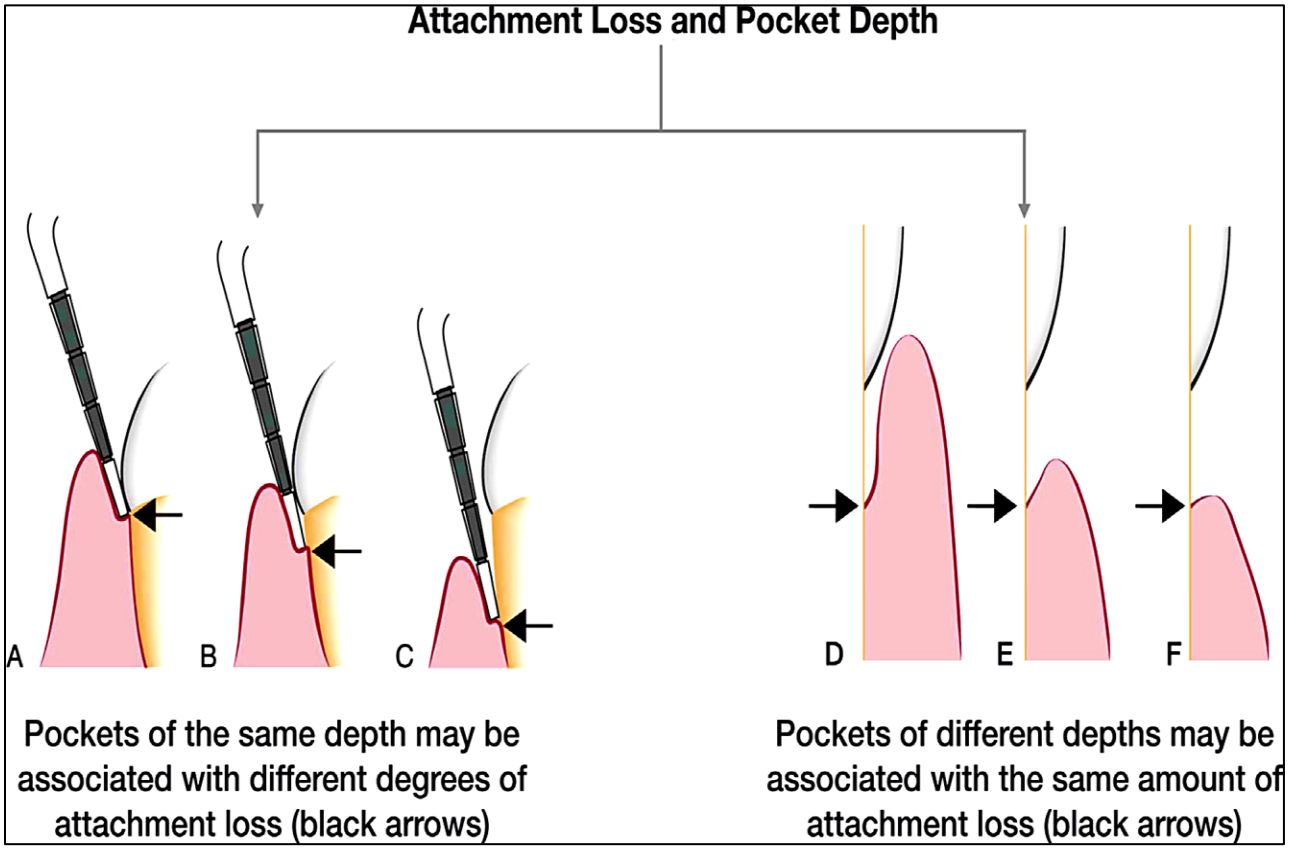

Fig. :- Relationship of attachment loss and bone loss to pocket depth.