ETIOLOGY, PATHOGENESIS, MODERN CONCEPTION IN THE ONSET AND EVOLUTION OF PERIODONTAL DISEASE. CLASSIFICATION OF PERIODONTAL DISEASE.

LECTURE N2

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Etiopathogenesis

1. local factors:

- **causal factors**: bacterial plaque;

- **contributing factors**: calculus, occlusal trauma, dental caries, edentation, dento-maxillary anomalies, parafunctions, bad habits, iatrogenic factors.

Iatrogenic factors: gingiva trauma during caries cavities preparation (neck and proximal surfaces), high occlusal obturations, cervical and proximal cavities obturations with overocclusion, traumatic action of matrix and pins in proximal caries restoration, absence of gingival wall margin beveling in proximal cavities, arsenic bandages incorrect applied.

Bad habits: oral breathing, infantile deglutition, onicophagia, lips biting, toothpicks trauma, traumatic toothbrush, finger sucking, abnormal pressure on dental arches, shoemaker.

Smoking, alcohol consumption, chemical irritations, radiation action.

Other local factors: smoking, alcohol consumption, chemical irritations, radiation action.

Rough surface and retentive for bacterial plaque, interradicular perforations, false ways in desmodontium, repeated trauma in excessive subgingival grinding, stamp crowns not adapted right to the neck, incorrect prosthetic devices, orthodontic devices with excessive forces, teeth trauma neighbor to the surgical treatment (extraction of teeth, tumor extirpation).

Occlusal trauma: acute and chronic, primary and secondary.

Anomalies dento-maxillary: crowded teeth, open occlusion, deep occlusion.

Parafunctions: bruxism (daily, night), psychologic factor, neurologic factor, occlusal factor, professional factor.

Role of diet in periodontal disease release: deficiency of vitaminB6, PP favor the calculus accumulation, food reach in calcium, phosphorus, bicarbonate, proteins, glucydes. The amount of ascorbic acid in food decrease the calculus amount.

2. general factors, systemic (hereditary, nervous system disturbance, endocrine disturbances, immune system disfunctions, cardiovascular system, hematologic system diseases, hepatic diseases, nutrition deficiencies).

Nervous system disturbances: psychological stress, constant and intense stress, anxious neuroses, obsessive, depressive, oligophrenia, major psychoses.

Endocrine disturbances: hypofunction of the thyroid gland, hypofunction of pituitary, hypofunction of parathyroids, hypofunction of gonads, hypofunction of corticosuprarenal.

Immune system disfunctions: AIDS (lymphocytes T, subclass TH4 helper), agranulocytosis, agammaglobulinemia.

Cardiovascular system dysfunction: atherosclerosis.

Hepatic diseases: cirrhosis.

Hematologic system diseases: gingival hyperplasia, gingival bleeding, ulcerations of mucosa, alveolar bone demineralization.

Nutrition deficiencies: hypovitaminosis A, hypovitaminosis D, B1, B2, PP, C

Dental plaque – a soft deposit that forms the biofilm adhering to the tooth surface or other hard surfaces in the oral cavity, including removable and fixed restorations.

Classification:

1. dental plaque (supra, subgingival);
2. material alba;
3. rest of food;
4. calculus.

Materia alba – is a soft accumulation of bacteria and tissue cell that lack the organized structure of dental plaque and are easily displaced with a water spray.

Calculus – is a hard deposit that is formed by the mineralization of dental plaque and it is generally covered by a layer of unmineralized plaque.

1. supragingival plaque – situated above the gingival margin and is in direct contact with the gingival margin (marginal plaque).

2. subgingival plaque – is found below the gingival margin, between the tooth and the gingival sulcular tissue.

Marginal plaque is of prime importance in the development of gingivitis.

Supra and subgingival plaque lead to calculus formation and root caries.

Subgingival plaque in soft tissue destruction characteristic of different forms of periodontitis.

Formation

Dental plaque is visible after 1-2 days with no oral hygiene measures plaque is white, grayish, yellow and has a globular appearance.

Plaque deposit prefere to form in cracks, pits, fissures in the tooth structure: under overhanging restorations and around malaligned teeth.

Phases:

1. the formation of dental pellicle;

2. initial colonization by bacteria;

3. secondary colonization and plaque maturation.

1. the formation of dental pellicle. The surface of teeth, fixed and removable restorations are coated with a glycoprotein pellicle. Pellicle derived from components of saliva and crevicular fluid, bacteria, debris, host tissue cell products.

Enamel pellicle with aminoacid composition differs from that of saliva, means the pellicle is formed by selective adsorbtion of the environmental molecules.

Enamel pellicle formation include electrostatic, Van der wals, hydrophobic forces. Hydroxyapatite surface has a predominance of negatively charged OH- (phosphate groups) that directly-indirectly interact with positively charged of components of salivary and crevicular fluid macromolecules.

Pellicle with a protective barrier function providing lubrication for the surfaces and preventing tissue dessication.

2. The initial bacteria colonizing the pellicle – coated tooth surface is predominantly gram+ facultative microorganisms – Actinomyces viscosus, Streptococcus sanguis.

Gram+ facultative microorganisms adhere to the pellicle through molecules adhesions (on bacterial surface) that interact with receptors in the dental pellicle. Ex.Actynomyces viscosus (protein structure-fimbrial) adhere to proline – rich proteins.

3. Secondary colonizers are microorganisms that do not initially colonize clean tooth surface (Prevotella intermedia, Prevotella loescheii, Caprocytophaga, Fusofacterium nucleatum, Porphyromonas gingivalis).

Coaggregation – the adherence of microorganisms to one another (highly specific stereochemical interaction of protein and carbohydrate molecules located on the bacterial cell surfaces).

Microorganisms: Fusofacterium nucleatum and strptoccocus sanguis, Prevotella loescheii and Actinomyces viscosus, Capnocytophaga ochracea and Actinomyces viscousus.

Coaggregation predominates at different gram- species (Fusofacterium nucleatum and Porphyromonas gingivalis). The early colonizers (Streptococci, Actinomyces) utilize O2 and lower reduction – oxidation potential of the environment which favors the growth of anaerobic species.

Gram+ utilize sugar as an energy source and saliva as a carbon source. Bacteria (in mature plaque) use aminoacids and small peptides as energy source.

Dental calculus

Calculus

Soft dental deposits which haven’t been removed by a professional tooth brushing and in presence of poor oral hygiene and due to a permanent cover by bacteria and a sufficient salivary flux cause the mineralization and calcification of deposit. Calculus play a major role in maintaining and accentuating periodontal disease by keeping plaque in close contact with the gingival tissue and creating areas where plaque removal is impossible.

Supra- and subgingival calculus

Calculus is an adherent calcified or calcifying mass that forms on the surface of natural teeth, dental prosthesis, restorations, implants.

Ordinarily calculus consists of mineralized bacterial plaque.

Supragingival calculus – located coronal to the gingival margin and is visible in the oral cavity. Has white, whitish-yellow, hard and clay-like consistency and is easy detached from the tooth surface.

After removal it may recurs rapidly with prevalence oral surface of mandibular incisors. The color of calculus depends by using tabacco or food pigments. It may be localized on a single tooth or group of teeth, or generalized. It also may be localized on the buccal surfaces of the molars opposite Stenon's duct and as it was mentioned above – on the lingual surface of the mandibular anterior teeth (central incisors) opposite Wharton's duct. Calculus has a bridge-like structure if occupies the interdental papilla of adjacent teeth or cover the occlusal surface of teeth with functional antagonists.

Subgingival calculus – is located below the crest of marginal gingiva and therefore is not visible on routine clinical examination. It requires careful examination with an explorer. It is usually dense, dark, brown or greenish black and hard or flint-like in consistency, it is firmly attached to the tooth surface.

Supra- and subgingival calculus generally occur together but one may be present without the other and usually extend near but do not reach the base of periodontal pockets in chronic periodontal disease.

A hypothesis existed about the origin of supra- and subgingival calculus.

Supra- has been reffered to as salivary calculus and subgingival as a serumal calculus based on first derived from the saliva and the second from the blood serum. That means saliva supplies the minerals for the formation of supragingival calculus, whereas the gingival fluid which resembles serum is the main mineral source for subgingival calculus. These terms are no longer use.

Prevalence

Studies based on patients who had no oral hygiene or dental care, supragingival calculus formation started early in life, soon after tooth eruption, localization was as previous, and continued to accumulate with age reaching a maximum at about 25-30 years – facial surfaces with less accumulation, appeared to be symmetric, premolars were without calculus. By age 30 all teeth had subgingival calculus without any pattern of predilection.

Norwegians had good oral hygiene and frequent visits for dental care through their lives. But supragingival calculus still formed on facial surfaces of upper molars and lingual surfaces of lower incisors in 80% of teenagers, it didn't extend to other teeth and didn't increase with age.

X-ray examination (radiographs) permits to see supra- and subgingival calculus forming irregular contours, irregularly sharp projections into the interdental space.

Composition

Supragingival calculus consists from: 70-90% inorganic, 10-30% organic components.

Inorganic content:

- 75,9% calcium phosphate $Ca_3(PO_4)_2$
- 3,1 calcium carbonate $CaCO_3$
- Magnesium phosphate $Mg(PO_4)_2$
- Calcium 39%
- Phosphorus 19%
- Carbon dioxide 1,9%

- Magnesium 0,8%
- Amounts od sodium, zinc, strontium, bromine, iron, copper, manganese, tungsten, gold, aluminum, silicon, fluorine
- 2/3 of inorganic component is crystalline in structure
- Hydroxyapatite 58%
- Magnesium white lockite 21%
- Octacalcium phosphate 21%
- Brushite 9%

Organic content:

- Consists of a mixture of protein-polysaccharide complexes, desquamated epithelial cells, leukocytes;
- 1,9-9,1% carbohydrate: galactose, glucose, rhamnose, mannose, glucuronic acis, galactosamine, glucosamine, galacturonic acid, arabinose acid;
- Salivary proteins 5,9-8,2% aminoacids;
- Lipids 0,2% in form of neutral fats, free fatty acids, cholesterol, cholesterol esters, phospholipids;
- Ca/P ratio is higher in subgingival region;
- Na content increases in depth of periodontal pockets.

Attachment to the tooth surface

1. attachment by means of an organic pellicle;

2. penetration of calculus bacteria into cementum;

3. mechanical locking into surface irregularities – such as resorbtion lacunae and caries;

4. close adaptation of calculus undersurface depressions to the gently sloping mounds of the unaltered cementum surface;

Formation

Calculus is attached to dental plaque that has undergone mineralization. The soft plaque is harden by precipitation of mineral salts which usually starts between 1^{st}-14^{th} day of plaque formation but calcification occurs since 4-8 hours. Calcifying plaques may become 50% mineralized in 2 days and 60-90% mineralized in 12 days.

Early plaque contains a small amount of inorganic material which increases as the plaque develops into calculus. Early plaque of heavy calculus formers contains more calcium, three times more phosphorus, less potassium than that of non calculus formers, suggesting than phosphorus may be more critical than calcium in plaque mineralization.

Rate of formation and accumulation

The starting time and rates of calcification and accumulation of calculus vary from person to person, in different teeth. So persons may be classified as: heavy, moderate, slight calculus formers, non-calculus formers. Calculus formation continues until it reachs a maximum, after which it may be reduced in amount, so the maximal accumulation (reversal phenomenon) may be explained by the vulnerability of bulky calculus to mechanical wear from food and from the checks, lips, tongue.

Theories regarding the calculus mineralization

1. **mineral precipitations results** from local rise in the degree if saturation of calcium and phosphate ions, there are several ways:

- a rise in the PH of saliva causes precipitation of calcium phosphate salts by lowering the precipitation constant;

- colloidal proteins in saliva bind calcium and phosphate ions and maintain a super saturated solutions with respect to calcium phosphate salts;

- phosphatase liberated from dental plaque, desquamated epithelial cells, or bacteria precipitate calcium phosphate by hydrolyzing organic phosphates in saliva, thus increases the concentration of free phosphate ions;

2. **seeding agents** include small foci of calcification that enlarge and coalesce to form a calcified mass. It is suspected that the intercellular matrix of plaque plays an active role.

Role of microorganisms in calculus mineralization

Mineralization of plaque starts extracellularly around both gram+ and gram- organisms, it also may starts intralellularly. Filamentous organisms, diphtheroids, and Bacterionema and Veillonella species have the ability to form intracellular apatite crystals. Calculus formation spreads until the matrix and bacteria are calcified.

WHO Classification in periodontal diseases

K.05. gingivitis and periodontal diseases included the diseases of edentulous interalveolar crest

K.05.0. acute gingivitis

K.05.00. acute streptoccocic gingivostomatitits

K.05.08. other acute form of gingivitis with precision

K.05.1. chronic gingivitis

K.05.10. marginal simple

K.05.11. hyperplastic

K.05.12. ulcerative

K.05.13. descuamative

K.05.18. other chronic gingivitis with precision

K.05.2. acute periodontitis

K.05.20. periodontal abscess with gingival origin without fistula

K.05.21. periodontal abscess with gingival origin withfistula

K.05.22. acute pericoronarities

K.05.28 other acute periodontitis with precision

K.05.3 chronic periodontitis

K.05.30. simple

K.05.31. complicated (accompanied)

K.05.32 chronic periodontitis

K.05.33 chronic hyperplastic folliculitis

K.05.38 other form of chronic periodontitis with precision

K.05.4. chronic periodontitis. Juvenile periodontitis

K.06.0. gingival recession (including postinfections, after intervention)

K.06.00. localized

K.06.01. generalized

K.06.09. gingival recession without precision

K.06.1. gingival hyperplasy (overgrowth)

K.06.10. gingival fibromatosis

K.06.18. other gingival hyperplasy with precision

K.06.2. gingival lesions and lesions of edentulous alveolar crest in association with trauma

K.06.20. due to traumatic occlusion

K.06.21.due to tooth brushing

K.06.22. functional keratosis

K.06.23. hyperplasy due to prosthesis irritation

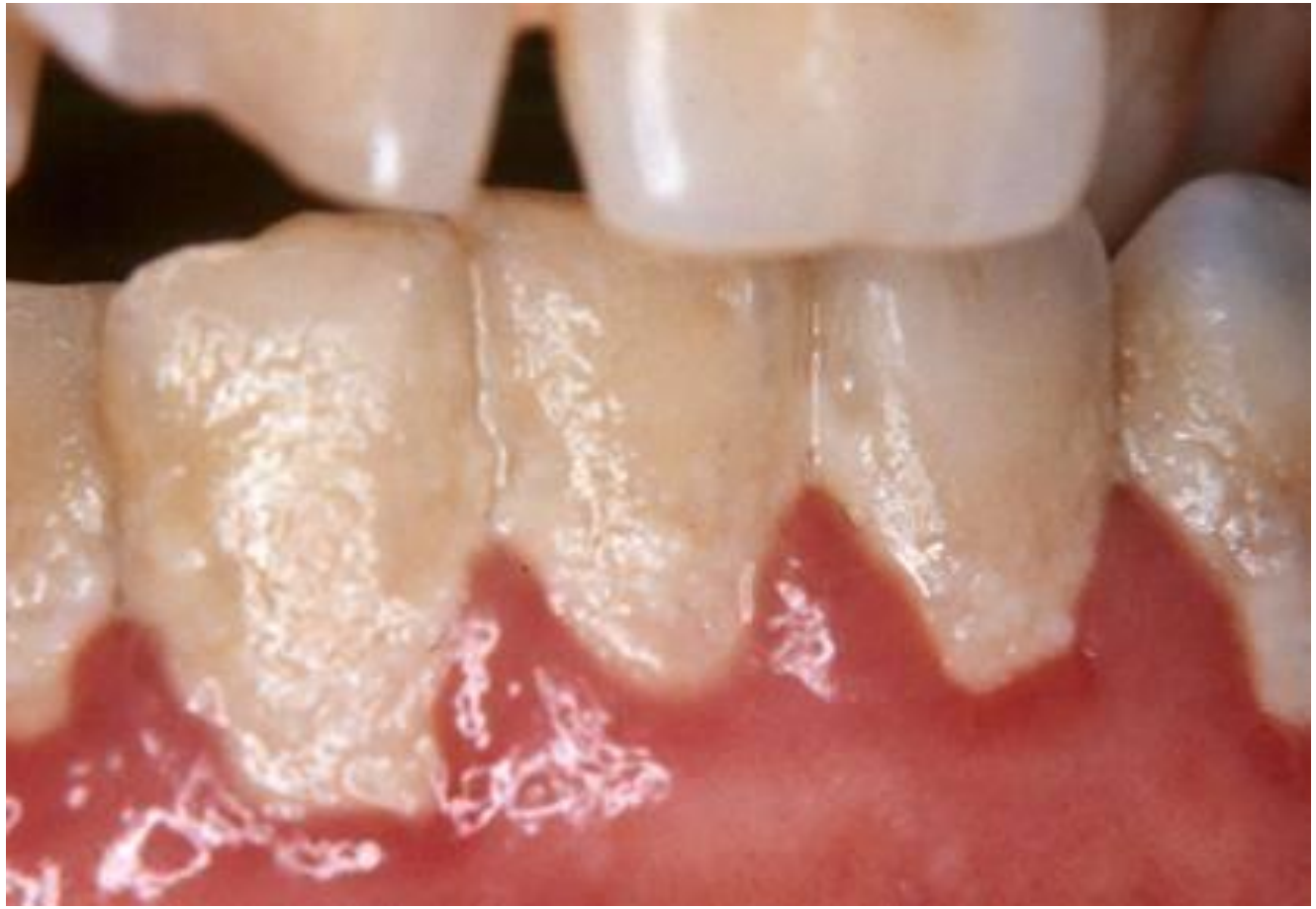
K.06.80. gingival adult cyst

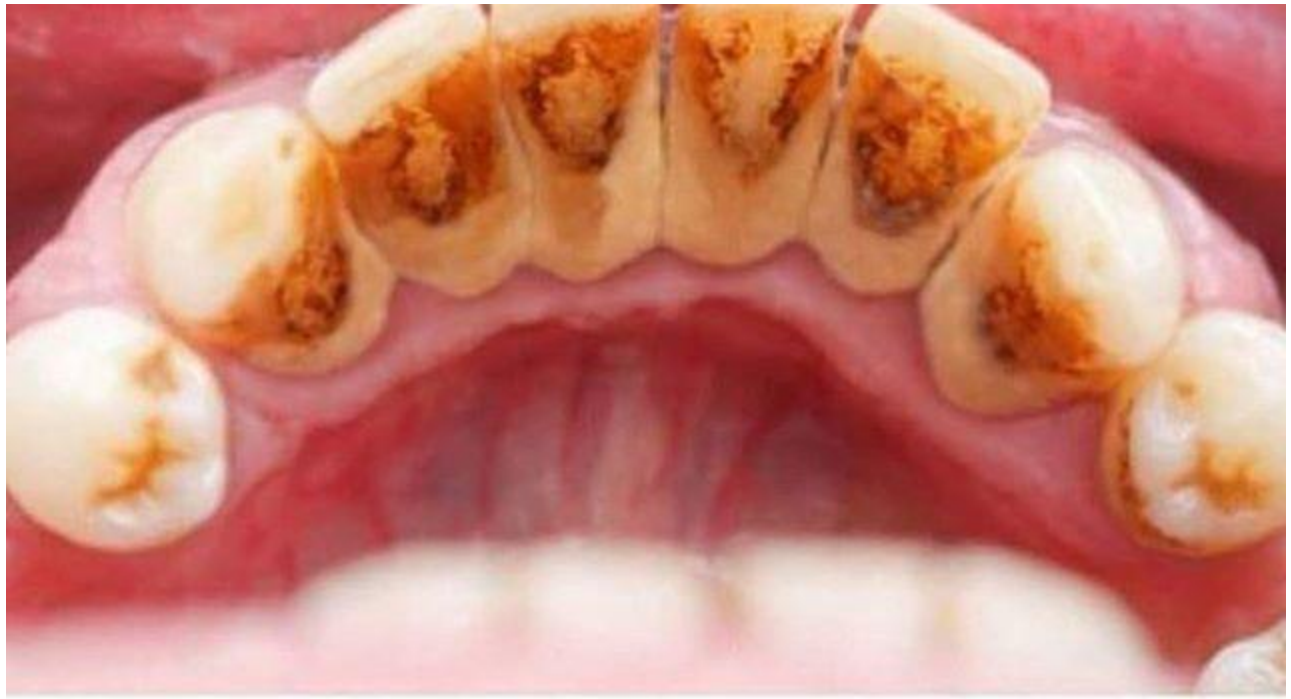
K.06.81. giant cell epulide

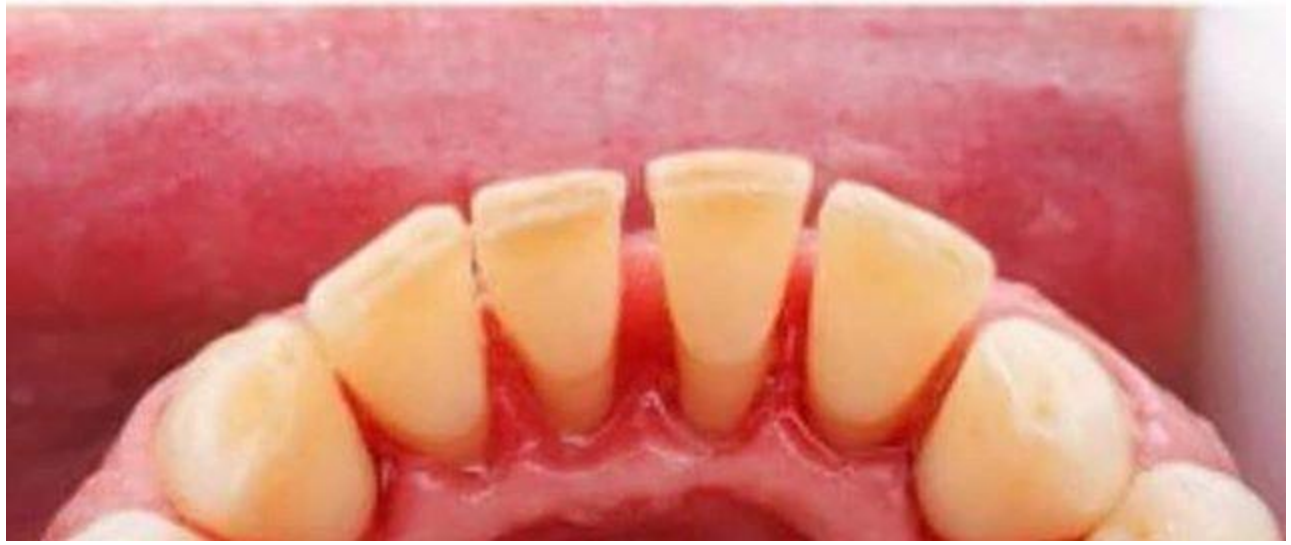
K.06.82. fibrous epulis

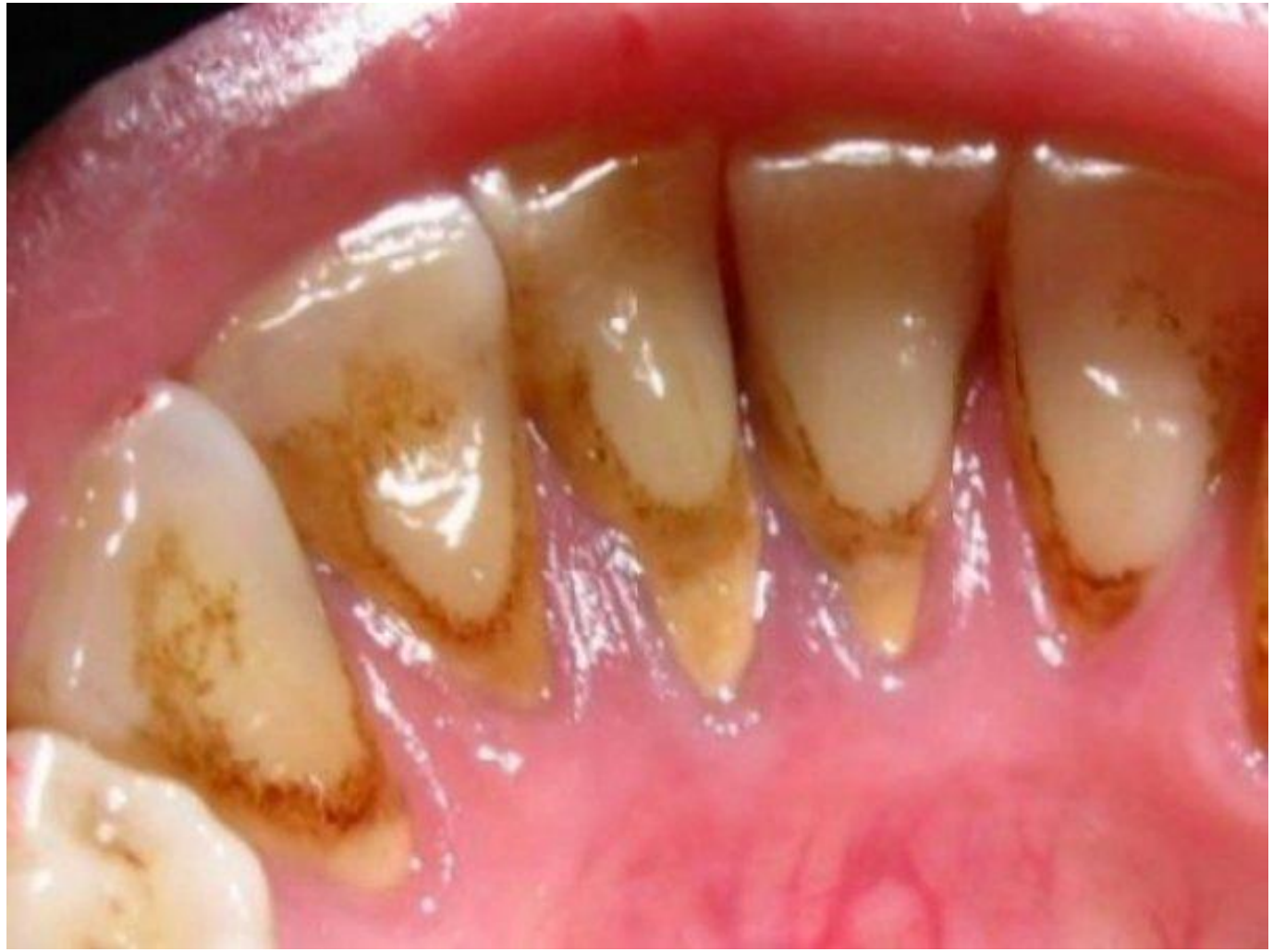
K.06.83. piogenic gingival granuloma

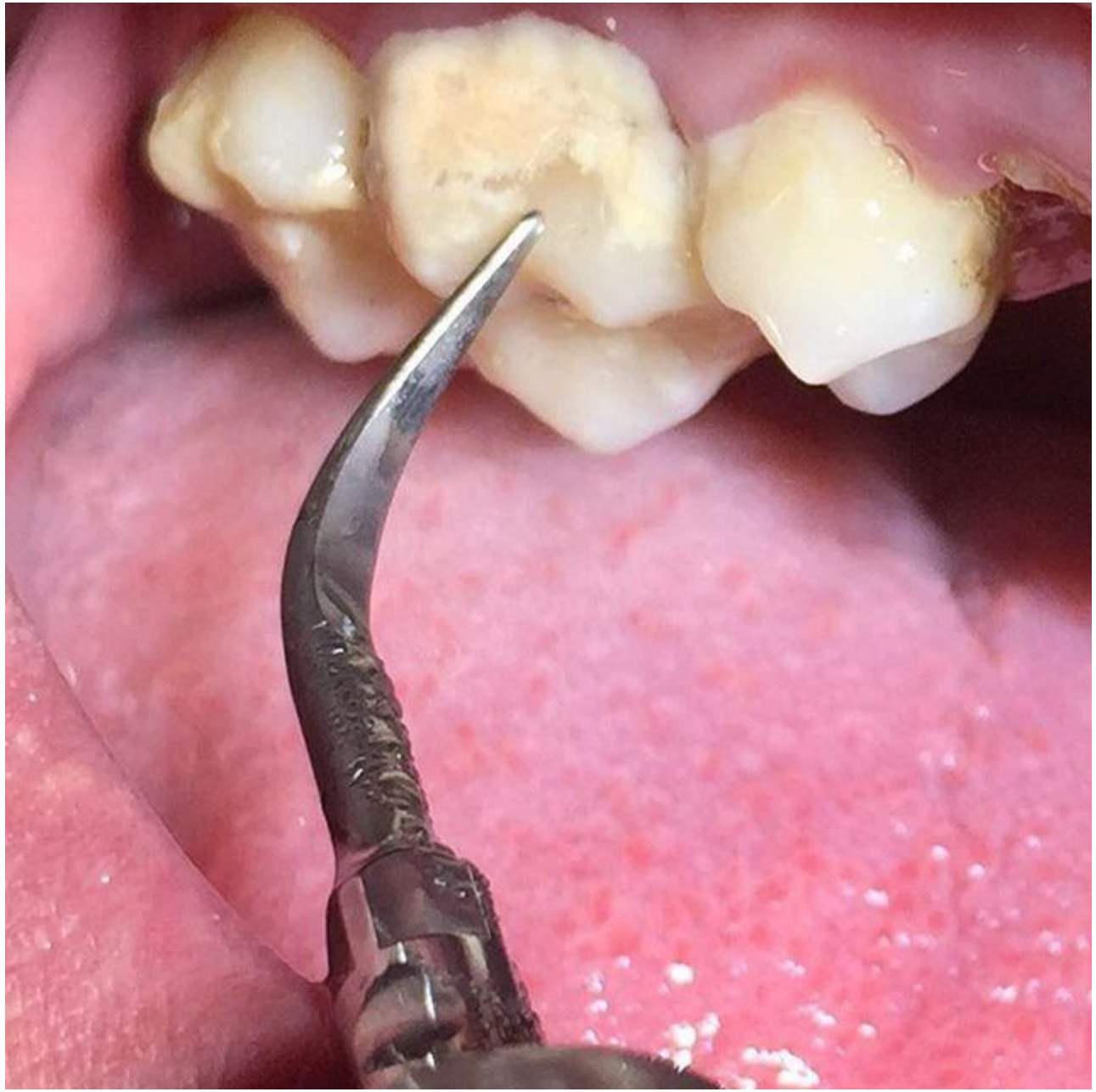
K.06.84. floating gingival crest












Dental Premier