**Risk factors for inflammatory periodontal disease**

The main etiological factor for the development of inflammation in periodontal tissues is plaque microorganisms.

The growth rate of plaque is directly related to oral hygiene, without which the signs of gingival inflammation appear after 10-15 days.

Plaque that is present above and below the gums for a long time will mineralize and form tartar. The rough surface of the plaque creates favorable conditions for the attachment of bacteria, and causes mechanical trauma to the gums.

Anaerobic microflora capable of causing inflammation of the periodontal tissues are called periodontopathogens.

The main periodontopathogenic species are:

Aggregatibacter actinomycetemcommitans;

Tannerella forsythensis;

Prevotella intermedia;

Porphyromonas gingivalis;

Treponema denticola.

Streptococcus intermedius, fusobacteria, actinomycetes, yeast-like fungi of the genus Candida also have periodontopathogenic properties.

A combination of the following factors is necessary for the development of the disease:

- the presence of sufficient numbers of periodontopathogens in the biofilm;

- favourable conditions for their growth and reproduction, lack of antagonists;

- sensitivity of human organism to microorganisms and products of their activity.

Mechanisms of the damaging effects of periodontopathogens

Expressed susceptibility to invasion

Invasiveness is the ability of pathogenic microorganisms to spread beyond the ecological niche, bypassing the resistance of the normally functioning non-specific resistance system of the organism.

Active invasion of epithelial structures by microorganisms is considered

as a transition of gingivitis to periodontitis. In this process, along with pathogenic microflora, indicators of the organism's resistance play an important role. The process of periodontopathogen invasion is promoted by the anatomical features of the gingival sulcus, numerous gingival microtraumas, reduced concentration of secretory immunoglobulin A in the oral fluid, nutritional features.

Secretion of toxins and enzymes

Periodontopathogens produce histolytic enzymes (collagenase, protease, keratinase, neuramidase, etc.), which contribute to the destruction of periodontal connective tissue structures.

Infective agents release bacterial toxins (lipopolysaccharides) and chemotaxins. Polymorphonuclear leukocytes, platelets, monocytes and macrophages formed from them release prostaglandins, which directly or indirectly (acting on lymphocytes) activate osteoclasts that cause bone resorption of interalveolar septa.

**Local risk factors** contribute to increased plaque formation, difficulty with oral hygiene and chronic trauma to periodontal tissues.

These include:

- Poor oral hygiene;

- Poor brushing techniques, flossing and toothpicking;

- Prevalence of soft foods, easily fermentable carbohydrates in the diet;

- Reduced salivary flow, increased viscosity of saliva;

- abnormal oral mucous membrane development (short and incorrectly attached frenulum of the lips and tongue, shallow vestibule);

- abnormal dental position (crowding, dystopia) and occlusion (deep incisal overlap);

- functional overloading of periodontal tissues due to primary or secondary adentia, occlusal anomalies);

- the presence of fixed orthodontic appliances;

- overhanging edges of fillings and lack of contact points between teeth;

- wrongly made orthopaedic constructions;

- Oral piercings;

**Systemic factors** leading to morphological, biochemical and immunological changes in the oral cavity and the body as a whole are the background for the development of inflammation in periodontal tissues.

These include:

- General somatic pathology: endocrine disorders (diabetes mellitus,

pituitary-adrenal system pathology, hypofunction of reproductive

glands);

- gastro-intestinal diseases, systemic connective tissue diseases, allergic pathology;

- dietary disorders: hypovitaminosis A, C, D, calcium and protein deficiencies;

- Pregnancy and puberty;

- stress and neurosis;

- smoking;

- Environmental pollution.