

## A study of the microbiological status in patients using implant supported fixed dentures.

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**Introduction:** Today, dental science and practice have accumulated considerable experience in the prosthetic rehabilitation of patients through the use of dental implants. Along with the positive results of implantation, various complications are observed when performing this method. Such complications include peri-implantitis, a pathological process (traumatic, resorptive, inflammatory, ulcerative) in the area of the implant location [3]. This problem can occur both in the early and late periods after implantation and prosthetics. The most likely reason for the development of peri-implantitis may be the penetration of an infection of the oral cavity into the area of contact between the implant and the bone [2,4,5].

Timely detection of the composition of the mixed microflora of the oral cavity, which causes the development of inflammatory processes in the area of intraosseous implants, is important for the development of treatment methods for such patients.

**The purpose of this study** was to study the qualitative and quantitative composition of the microflora of the oral cavity, which determine the occurrence of peri-implantitis.

The microbial landscape in peri-implantitis is currently known and represents a wide variety of aerobes and anaerobes [2,9]. The inflammatory process of tissues in the peri-implant zone is the main cause of destruction and resorption of bone tissue in the implant area [2].

**Materials and methods:** When studying the microbiological status of the examined, a certain relationship was found between changes in the microflora of the oral cavity and the clinical form of periimplantitis. Thus, the microflora of the oral cavity in mild forms of periimplantitis included all taxonomic groups of microorganisms determined in healthy individuals. The frequency of their detection almost did not differ from the norm, minor changes were not of a regular nature (aerobic and anaerobic gram-positive and gram-negative microbes). Similar results were obtained in a quantitative study of the contents of the oral cavity, although for certain groups of bacteria a difference was revealed, manifested in a decrease in CFU / ml for streptococci "salivarius" (from 7.4 to 5.4), peptococci with (7, 0 to 5.5) and, on the contrary, an increase in the concentration of fusobacteria from 2.8 to 5.7 and "odontogenic" bacteroids from 3 to 4.8 ( $p < 0.05$ ).

With moderate severity of periimplantitis, the number of most species and groups that make up the normal flora of the oral cavity was reduced: green streptococci from 100.0 to 68.5% ( $p < 0.01$ ), staphylococci from 90.0 to 45.7% ( $p < 0.01$ ), diphtheroids from 55.0 to 5.7% ( $p < 0.01$ ), Neisseria from 90.0 to 37.1% ( $p < 0.01$ ). Less significantly ( $p > 0.05$ ) decreased the number of pneumococci (25.0 to 14.3%), lactobacilli (from 90.0 to 68.5%). With a slight decrease in the total number of bacteroids to 83.3%, their species composition noticeably changed: if in healthy people and in patients with mild peri-implantitis, Pr. melaninogenicus and other bacteroids, then with moderate severity, the frequency of their isolation sharply decreased, and the number of odontogenic anaerobes increased from 10.0 to 83.3% ( $p < 0.05$ ). An increase in the frequency of inoculation of fungi of the genus Candida from 25.0 to 50.0% ( $p > 0.05$ ) was also noted.

Protozoa, as well as spirochetes, were found in native preparations in isolated cases (2.8% each).

In patients with severe forms of peri-implantitis, there were no hemophils, pneumococci, and other cocci; many times lower was the inoculation of non-greening streptococci, staphylococci, diphtheroids, neisseria, veillonella ( $p < 0.05$  and  $p < 0.01$ ). At the same time, the frequency of detection of fungi of the genus Candida increased (from 15 to 50.0%) and odontogenic bacteroids (from 10.0 to 83.3%). The number of greening streptococci was 2.3 times less than in healthy ones, staphylococci - 4.3 times, Neisseria - 3.6 times, lactobacilli - 2.6 times, veillonella - 2 times. At the same time, the concentration of fusobacteria increased by 2 times, and by 3.2 times - actinomycetes. The total number of bacteroids increased by 1.6 times, of which odontogenic - by 1.7 times. In 5 (16.6%) patients, the study revealed trichomonas, spirochetes and spirilla (the latter - only in 6.6%).

**Results:** According to some researchers [1,3,6], the main symptom of periimplantitis and the site of the onset of the inflammatory process is the implant-gingival pocket, the study of the biocenosis of which is of particular interest. With a pocket depth of not more than 4 mm, in most cases, there was a slightly lower frequency of inoculation of aerobic-microaerophilic flora than in healthy individuals ( $p>0.05$ ). Species such as pneumococci, diphtheroids were not detected. In the anaerobic part of the microflora, significant changes affected only the group of streptococci "mutaus" (90 and 64%,  $p<0.05$ ) and peptostreptococci (95 and 60%,  $p<0.05$ ). The concentration of detected microorganisms also remained almost unchanged, although cfu/ml of peptostreptococci in patients of this group decreased from 6.2 in the norm to 4.4. The change in the spectrum of microorganisms isolated from periodontal pockets with a depth of more than 6 mm was even more pronounced and had a significant character (90.0 and 40.0%,  $p<0.01$  and 95 and 28.5%,  $p<0.01$ ). With severe gum bleeding, most representatives of the normal microflora were sown less often: streptococci "Salivarius" in 80.0 and 22.8% ( $p<0.01$ ), staphylococci - 50.0 and 17.0% ( $p<0.05$ ), lactobacilli - in 25.0 and 5.7% ( $p<0.05$ ). The number of these microorganisms decreased by 1.-1.8 times, the difference in CFU/ml for peptostreptococci was especially significant - 6.2 in normal and 3.4 in patients. The frequency of detection of bacteroids decreased slightly (from 90.0 to 82.8%), however, the proportion of "odontogenic" bacteroids increased sharply from 2.8 to 61.8% ( $p<0.05$ ). A twofold increase in the inoculation of fungi of the genus *Candida* (from 15.0 to 31.0%,  $p>0.05$ ) is noteworthy. The frequency of detection of actinomycetes increased in the presence of serous-purulent exudate (from 10.0 to 14.3%), the quantitative indicator of this microorganism increased by 3.2 times - from 1.7 to 5.5 cfu/ml with suppuration.

The results of our studies indicate that as the inflammatory-destructive process becomes more severe and the intensity of its clinical manifestations increases, significant changes occur in the microbiological status of patients with peri-implantitis. They relate to the biocenosis of the periodontal pocket: some groups of bacteria characteristic of healthy individuals disappear (*Neisseria*, pneumococci, other cocci), the detection of most obligate and facultative anaerobes characteristic of this biotope (*Veillonella*, peptostreptococci, lactobacilli, green and non-green streptococci) is significantly reduced. The leading role begins to play "odontogenic" bacteroids, fungi of the genus *Candida*, to a lesser extent (due to a higher concentration) - actinomycetes and fusobacteria. In severe forms of peri-implantitis, rarely identified *A. actinomycetemcomitans*, opportunistic aerobic species - *P. aeruginosa* and *S. aureus* also appear in periodontal pockets.

Protozoa also seem to play a significant role in the development of a more severe course of the disease, since their detection in native smears becomes more and more frequent as the disease progresses from mild to more severe forms.

When **analyzing** the total number of associates in the oral cavity (both representatives of the "normoflora" and opportunistic microorganisms), it turned out that 10-9 species and groups of microorganisms were mainly sown in the control group and in patients with mild periimplantitis. In the control group, such results were obtained in 90% of the examined, with mild severity of peri-implantitis - in 96%. In the moderate and severe course of the disease, there was a shift towards a decrease in the number of associates. In the first case, 8-6 species were seeded from the oral cavity in 82.7% of patients; ) and 5 (46.6%) species.

Carrying out such an analysis, where the content of the implant-gingival sulcus served as the material for the study, showed that the total number of associates here is significantly less. In the microbiocenosis of this biotope, the number of associates was: in healthy people - 7-6 (80.0%); in patients with mild peri-implantitis - 6-3 (80.0%); moderate and severe - respectively 5-3 (74.3%) and 4-2 (93.4%).

In addition, in the implant-gingival grooves with a depth of more than 6 mm and a severe course of the disease, a clear pattern was traced: the leading position was occupied by "odontogenic" bacteria found in 80.0% of patients of the second group and 86.0% of patients of the third group. It should be emphasized that *Acinobacillus actinomycetemcomitans* were detected only with abscess formation and the presence of purulent exudate were detected in 13.3% of cases; they

were the only representatives of the periodontopathogenic flora in high titers and in 2 more cases were isolated in association with *Por. gingivalis*.

The study of samples of the oral cavity and implant-gingival grooves showed that in the oral cavity of healthy individuals and in patients of the first group, the microflora is quite abundant and consists of 9-10 or more associates. With the development of periimplantitis, the number of participants in the biocenosis decreases to 8-6 with an average degree and up to 7-8 with a severe degree of periimplantitis. An even greater "depletion" occurs in the implant-gingival grooves: if 7-6 species (groups) of bacteria are sown in healthy people, then in patients with an average degree - 5-3, and with a severe course - no more than 4-2 species. In the oral cavity and in the implant-gingival grooves of patients with moderate and severe peri-implantitis, one or two representatives of this group dominated. This trend is especially pronounced in the implant-gingival grooves.

According to some researchers [4,6], a violation of the ratio of the number and composition of microorganisms can eventually acquire nosological independence and serve as an additional factor in the pathogenesis of chronic inflammatory diseases of the oral cavity. As the results of our studies have shown, in patients with peri-implantitis, the severity of the identified violations of the biocenosis may be different. The distribution of patients according to the severity of dysbiotic disorders was carried out by us on the basis of the calculation of the dysbiosis index (DI). Numerical values were obtained by dividing the quantitative indicators of microorganisms according to the following formula:

$$DI I = \frac{\text{Streptococcus salivarius} + \text{mutans} + \text{other obligate microorganisms}}{\text{total number of microorganisms}}$$

$$DI II = \frac{\text{facultative organisms} + \text{obligate microorganisms}}{\text{total number of microorganisms}}$$

So, in the control group, the normal state of the microflora was noted (DI 0.8-0.7). Dysbiotic shift (DI 0.7-0.6) was observed in patients with mild CGP. Dysbiosis I degree (DI 0.6-0.4), diagnosed with CGP moderate. Dysbiosis II degree (DI 0.06 or less) corresponded to a severe degree of periimplantitis. Such systematization is conditional and does not take into account all possible quantitative fluctuations. However, it allows a more differentiated approach to the diagnosis and complex treatment of the disease.

**Discussion:** Thus, microbiological studies have shown that in patients with peri-implantitis, the biocenosis of the oral environment is significantly disturbed. The most pronounced violations of the microflora, which are in the nature of dysbiosis, are observed in patients with a severe degree of the disease. The results of clinical and microbiological studies of patients indicate that the degree of violations of the biocenosis of the oral cavity is interconnected with the clinical form of periimplantitis. At the same time, it was found that dysbiotic changes aggravate the clinical picture of the disease.

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