SUMMARY

Periodontitis is an infectious disease concerning supporting tissues of the teeth. The primary etiological agent for disease development and progression is the subgingival biofilm, but recently it is known that host factors may modify the pathological process or may affect the severity and/or extent. The increasing levels of some specific pathogenic subgingival bacteria such as Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, Treponema denticola, Tannerella forsythia, Fusobacterium nucleatum, Prevotella intermedia and others can result in periodontal destruction and possibly correlate with disease severity (3, 4, 5). Data from controlled studies show high prevalence of P. gingivalis, T. forsythia and Tr. denticola which represent the red complex (coexistence of these three species) in patients with moderate and severe chronic periodontitis (3, 5, 9). Parallel investigation of probing depth (PD) and clinical attachment level (CAL) with the microbiological testing may give a confirmation of relation between subgingival pathogenic bacteria and severity of periodontitis (1, 2, 7).

Key words: periodontal pathogens, chronic periodontitis, clinical periodontal examination.

INTRODUCTION:

Chronic periodontitis is one of the most significant diseases that cause teeth loss. The key role of several anaerobic bacteria in subgingival microbiota has been discussed in the literature (pathogenic potential of the red and the orange complexes) (4, 5, 9, 10). There is a need for further studies on the correlation of clinical parameters PD and CAL and the levels of key periodontal pathogens subgingivally.

AIM:

To evaluate the presence and the levels of main periodontal pathogens in the periodontal pockets in patients with chronic periodontitis in relation with clinical measurements.

MATERIALS AND METHODS:

In this study 20 patients with chronic periodontal disease were evaluated based on their clinical parameters (gingival bleeding, pocket depth, clinical attachment level, recession, furcation involvement, mobility, hygiene status) and microbiological analysis. Microbial evaluation included the detection of three periodontopathogenic bacteria: Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, Treponema denticola, by means of real-time polymerase chain reaction (MIP Pharma GmbH – PET test).

RESULTS:

In our study T. denticola and P. gingivalis were established in great percent of subgingival simples (P. gingivalis - in 95,8%; T. denticola – in 98,2%). These pathogens were closely associated with moderate and deep periodontal sites but they also present in lower levels in shallow periodontal pockets (table 1 and diagram 1.). No one of investigated periodontal sites demonstrates the presence of A. actinomycetemcomitans.

Diagram 2 presents the results of the evaluation of gingival bleeding on probing. The data showed high PBI average values in all investigated patients except one who demonstrates average value of 0.5.

Table 1. Levels of periodontal pathogens in different periodontal pockets.

<table>
<thead>
<tr>
<th>MO</th>
<th>Periodontal pockets</th>
<th>P. gingivalis</th>
<th>T. denticola</th>
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<tbody>
<tr>
<td>shallow</td>
<td>7,4x10⁴</td>
<td>5,9x10⁴</td>
<td></td>
</tr>
<tr>
<td>moderate</td>
<td>3,4x10⁵</td>
<td>1,6x10⁵</td>
<td></td>
</tr>
<tr>
<td>deep</td>
<td>5,3x10⁵</td>
<td>7,4x10⁵</td>
<td></td>
</tr>
</tbody>
</table>
Diagram 1. Levels of periodontal pathogens in different periodontal pockets in graphic.

Diagram 2. Evaluation of gingival bleeding.

Diagram 3. Clinical parameters.
Diagram 3 allows direct comparison of three clinical measurements: periodontal pocket depth, gingival bleeding and loss of attachment. There may be simultaneously viewed evaluated clinical parameters in each patient. It is evident the correlation between the loss of attachment and the pocket depth and gingival bleeding in the investigated patients.

Diagram 4 and Diagram 5 present available amounts of $P. \text{gingivalis}$ and respectively $T. \text{denticola}$ depending to the measured PPD. In this investigation periodontal pockets were differentiated as shallow (<4mm), medium (4-6mm) and deep (>6mm). Periodontitis we observed were moderate and severe according to received average values of CAL. In regard with this we found a clear distinction in the levels of the detected pathogens, that correlate to the increasing depth of the periodontal pockets (average values) as well as to changes in the CAL (average values) and the disease’s severity.

**DISCUSSION:**

The similar analyses in specialized literature are focused on the detection of the presence of $A. \text{actinomycetemcomitita}$, $T. \text{denticola}$ and $P. \text{gingivalis}$ as significant pathogens implicated in periodontal lesions (1, 3, 5, 8, 10, and 11). Recently various authors suggest that these main periodontal pathogens present in supragingival plaque as well as subgingivally in periodontitis subjects (6).

In current investigations $T. \text{denticola}$ and $P. \text{gingivalis}$ were detected in subgingival plaque samples from active periodontal sites in high levels. Our microbiological data included expression of these bacteria as counts $x10^n$ levels and % sites colonized – prevalence. In our study great proportions of $T. \text{denticola}$ and $P. \text{gingivalis}$ were observed in samples of periodontal sites in diseased individuals. The amount of the investigated bacteria is low in the shallow pockets and they levels increase with the increasing pocket depth. Possibly these pathogens could exist in small proportion in shallow periodontal sites and proliferate due to inflammatory process, and multiplicate in favorable for them environment with enough nutrients and impaired relations between the epithelial cells. This may to some extent explain higher levels of detected periodontal pathogens in deeper sampling sites.

Our study was limited in evaluation of only three pathogen bacteria. Our results show any presence of $A. \text{actinomycetemcomitita}$ in all plaque samples in investigated patients. Based on literature data for microbial etiology of periodontal diseases this microorganism seems to be related with aggressive and refractory periodontitis more than the chronic periodontitis. Like other investigators we attempt to trace levels of detected periodontal pathogens with severity of periodontal destruction, measured with the loss of both - attachment and bone.
Conclusion:
The results of this investigation confirm the strong relation between the levels of *T. denticola* and *P. gingivalis* and their presence together in progressive periodontal lesions, evaluated with clinical measurements. Diminution and/or elimination the proportion of pathogen species belong to red and orange complexes as well as the total bacterial number subgingivally could be main goal of infection control in periodontal treatment.

The results obtained in the present study are preliminary. The severest limitation of this study being the low number of subjects included. Further studies are planned to increase the number of patients and to obtain statistically reliable results.

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REFERENCES:

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