Gingival inflammatory infiltrate analysis in patients with chronic periodontitis and diabetes mellitus

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Abstract
Diabetes mellitus and periodontal disease are two pathological entities that destructively emphasize each other. The aim of our study was the histological and immunohistochemical analyze of the inflammatory infiltrate in the gingival tissue at patients with diabetes mellitus (DM) and periodontal disease.

Materials and Methods: The study was achieved on gingival tissue from 40 patients with DM and specific symptoms of periodontal disease. We used Hematoxylin–Eosin and trichromic Goldner–Szekely staining and CD20cy and CD45RO antibodies.

Results: In patients with DM under 10 years, we found an intense periodontal lymphocyte inflammatory infiltrate and in patients with a DM evolution more than 10 years, the lymphocyte inflammatory infiltrate had a less intensity. The pattern was mostly diffuse in lamina propria. Many patients had a very abundant plasmocyte infiltrate. During immunohistochemical exam, 25 cases presented CD20 positive immunostaining. The intensity of the inflammatory infiltrate with B-lymphocytes was very low (score 1). All immunohistochemical analyzed cases presented CD45RO positive immunostaining, with a mixed pattern of the T-cell lymphocyte infiltrate. Conclusions: The inflammatory infiltrate in diabetic periodontal disease was polymorph, mostly with a diffuse pattern in gingival chorion. The intensity of the lymphocyte infiltrate was higher in patients with chronic periodontitis and DM less than 10 years. Positive CD45RO T-lymphocytes were more numerous compared to positive CD20 B-lymphocytes and they were present intra and under epithelial in the gingival of all the patients, no matter of the DM time evolution.

Keywords: diabetes mellitus, inflammation, periodontal, lymphocytes, plasmocytes.

Introduction
Diabetes mellitus is recognized as an important risk factor for more severe and progressive periodontitis, infection or lesions resulting in the destruction of tissues and supporting bone that form the attachment around the tooth [1, 2].

Both diabetes and chronic periodontitis seem to share a common pathogenesis that involves an enhanced inflammatory response that can be observed at local and systemic level. Severe chronic forms of periodontitis can result in systemic response to the bacteria and bacterial products that are disseminated due to breakdown of the periodontal apparatus. Chronic hyperglycemia has been closely associated with an inflammatory response that has been linked to complications observed in diabetes [3, 4].

The purpose of this study was to go through a histological and immunohistochemical analyze of the inflammatory infiltrate from gingival tissue in patients with diabetes mellitus and periodontal disease.

Materials and Methods
The studied material was represented by gingival tissue fragments gathered after the extractions of irretrievable teeth from 40 patients diagnosed with DM in the Diabetes and Nutrition Clinic of the Emergency County Hospital in Craiova. Our study took place during 2006 and 2008. All the patients selected for the present study accused specific clinical symptoms of the periodontal disease by the moment of our investigation: gingival inflammation, gingival bleeding and recession, dental mobility or loss.

In addition to the specific symptoms, another including criteria for our study was the lesion macroscopic aspect. Therefore, patients with macroscopic aspects suggestive for different types of periodontal disease associated to the DM were accepted for our research (gingival sessile and pediculated overgrowth, gingival bleeding, great loss of periodontal attachment with deep periodontal pockets).

The mucosal fragments gathered after the extractions of irretrievable teeth were immediately fixed...
in 10% neutral formalin solution and then processed through the usual technique of paraffin inclusion. This technique is indicated for both histological and immunohistochemical exams in order to preserve the antigenicity of the different structures. There have been used the Hematoxylin–Eosin and trichromic Goldner–Szekely stainings for the histological study and concentrated antigens of DAKO Cytomation for the immunohistochemical study. The antigens dilutions and the performed pre-treatments are presented in Table 1.

Table 1 – The antibodies used for the immunohistochemical study

<table>
<thead>
<tr>
<th>Antibody</th>
<th>Clone</th>
<th>Dilution</th>
<th>Antigen retrieval</th>
<th>Code</th>
</tr>
</thead>
<tbody>
<tr>
<td>CD20cy</td>
<td>L26</td>
<td>1:50</td>
<td>5 MW cycles in citrate buffer</td>
<td>M0755</td>
</tr>
<tr>
<td>CD45RO</td>
<td>UCHL1</td>
<td>1:50</td>
<td>5 MW cycles in citrate buffer</td>
<td>M0742</td>
</tr>
</tbody>
</table>

The immunostaining for lymphocyte markers (CD20 and CD45RO) was considered positive when it presented a membranar staining pattern. We considered positive cells only those with undoubtedly positive cytoplasm or membranar staining, whereas the cells with an ambiguous staining were considered negative.

The lymphocyte infiltrate intensity (for both B- and T-lymphocytes) was appreciated through a qualitative gradation system in four degrees:

- *score 0* – absence of positive inflammatory cells;
- *score 1* – reduced lymphocyte infiltrate represented by a small number of positive or isolated cells, identified in 100× magnification microscopic area;
- *score 2* – lymphocyte infiltrate with a moderated number of positive cells;
- *score 3* – abundant lymphocyte infiltrate with a large number of positive cells.

Results

The histological analyze of inflammatory infiltrate in lamina propria was essentially based on the study of lymphocyte-type infiltrate which was furthermore analyzed through immunohistochemical methods.

Table 2 – Lymphocyte inflammatory infiltrate intensity and pattern in patients with DM of our study

<table>
<thead>
<tr>
<th>DM evolution</th>
<th>Less than 10 years (cases)</th>
<th>More than 10 years (cases)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lymphocyte infiltrate intensity</strong></td>
<td><strong>Absent</strong></td>
<td><strong>Discrete</strong></td>
</tr>
<tr>
<td>No. of cases</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td><strong>Lymphocyte infiltrate pattern</strong></td>
<td>Diffuse</td>
<td>4 diffuse</td>
</tr>
</tbody>
</table>

Patients with DM of more than 10 years evolution had a predominant lymphocyte inflammatory infiltrate of a low intensity (ten cases), followed by moderate intensity (five cases). Although few patients with DM of more than 10 years evolution had an absent lymphocyte inflammatory infiltrate (only isolated cells) on the biopsy fragments (four cases).

Whatever the DM time evolution and inflammatory infiltrate intensity, the lymphocyte inflammatory infiltrate pattern was predominant diffuse in lamina propria. Only two patients with DM of a less than 10 years evolution and moderate lymphocyte inflammatory infiltrate had a nodular under-epithelial lymphocyte infiltrate pattern.

Each case was examined in optical microscopy on usual and special trichromic GS stainings, with great interest only for the chorion inflammatory infiltrate. The intraepithelial inflammatory infiltrate analyze was made through immunohistochemical method, due to the interpretation difficulties through histological method.

There has been made a qualitative determination of the inflammatory infiltrate and it was appreciated the inflammatory infiltrate intensity (the inflammatory infiltrate was absent when only isolated cells were identified, or it was discrete, moderate or intense), and also its pattern in the chorion (diffuse or nodular). It is valuable to note that the inflammatory infiltrate had a non-homogeneous aspect, with variable intensities from a case to another and from an area to another on the same preparation. Therefore, it was necessary a strict examination of the entirely each preparation for a correct appreciation of the inflammatory process intensity.

The 40 analyzed cases were divided as follows:

- 20 patients with DM of a less than 10 years evolution (nine patients with intense inflammatory infiltrate, six patients with moderate inflammatory infiltrate and three patients with discrete inflammatory infiltrate);
- 20 patients with DM of more than 10 years evolution (10 patients with discrete inflammatory infiltrate, five patients with moderate inflammatory infiltrate and one patient with intense inflammatory infiltrate).

In Table 2, we presented the appreciation of the lymphocyte inflammatory infiltrate of all the 40 patients included in our histological and immunohistochemical study after the made biopsies, regarding the inflammatory cells intensity and pattern. From this table it is suitable to mark that patients with DM of a less than 10 years evolution had a predominant lymphocyte inflammatory infiltrate of maximum intensity (nine cases), followed by moderate intensity (six cases) and discrete intensity (three cases). The lymphocyte inflammatory infiltrate was absent in two cases where we identified only rare isolated lymphocytes.

Some patients in our study with DM of a less than 10 years’ evolution and incipient periodontal disease presented mononuclear and also PMN leukocytes into the chorion and the epithelium. This leukocyte infiltrate was discrete and the PMNs were numerous only in patients with identified clinical periodontal pockets (Figure 1). Another category of patients with DM of a less than 10 years evolution were those who presented lymphocytes and also plasmocytes, macrophages, fibroblasts and more important, numerous neoforination capillaries (Figure 2) with a granulation tissue pattern. These patients had a rigorous metabolic control and generally a properly oral hygiene.
However, many patients presented a very abundant plasmocyte infiltrate, where plasmocytes were the predominant inflammatory cells (Figure 3). The hyperplastic aspect of the gingival epithelium and the collagen lysis and fragmentation phenomena into the chorion predominated at these cases.

The studied patients with DM of more than 10 years evolution had especially an inflammatory infiltrate made only by inflammatory cells involved in chronic inflammation, associated with narrow and sclerotic sanguine vessels, atrophic phenomena of suprajacent epithelium and collagen sclerosis with thick bands into the chorion (Figure 4). However, patients with precarious oral hygiene and bad metabolic control also associated abcedated areas with numerous PMNs. Some of these patients also presented predominant plasmocyte inflammatory infiltrate with a reduced number of lymphocytes.

It is valuable to know that the high density of plasmocytes compared to the other inflammatory cells was associated with a bad controlled DM and a diabetic periodontitis of highly aggressiveness and advanced lesions.

We encountered CD20 positive immunostaining with B-lymphocytes in 33 cases of all 40 immunohistochemical analyzed ones (19 cases with DM of less than 10 years evolution and 10 cases of more than 10 years evolution) presented and CD20 negative immunostaining in seven cases.

The seven CD20 negative immunostaining cases (without B-lymphocytes into the inflammatory infiltrate) were framed with 0 score and represented patients with DM of more than 10 years evolution.

All cases with B-lymphocyte infiltrate revealed subepithelial pattern. The B-lymphocyte infiltrate with intraepithelial pattern was found in no cases.

The B-lymphocyte inflammatory infiltrate intensity was very low (score 1) in patients with DM of more and also less than 10 years evolution (Figure 5). However, we noticed B-lymphocyte small and nodular pattern aggregate points associated with reduced B-lymphocyte infiltrate (Figure 6) in four patients with DM of less than 10 years evolution (Table 3).

All immunohistochemical analyzed cases showed positive CD45RO immunostaining, in other words they presented T-lymphocytes inflammatory infiltrate.
Figure 5 – CD20 immunostaining in a patient with DM of more than 10 years evolution, reduced B-lymphocyte subepithelial infiltrate (ob. 20×).

Figure 6 – CD20 immunostaining, patient with DM of less than 10 years evolution, B-lymphocyte aggregates with nodular pattern (ob. 10×).

Table 3 – Intensity and distribution for both T- and B-lymphocyte inflammatory infiltrate

<table>
<thead>
<tr>
<th>DM evolution</th>
<th>Less than 10 years evolution</th>
<th>More than 10 years evolution</th>
</tr>
</thead>
<tbody>
<tr>
<td>T-lymphocyte infiltrate intensity</td>
<td>Score 2</td>
<td>Score 3</td>
</tr>
<tr>
<td></td>
<td>8 cases</td>
<td>12 cases</td>
</tr>
<tr>
<td>T-lymphocyte infiltrate pattern</td>
<td>5 diffuse intra- and subepithelial</td>
<td>12 diffuse intra- and subepithelial</td>
</tr>
<tr>
<td></td>
<td>3 nodular subepithelial</td>
<td></td>
</tr>
<tr>
<td>B-lymphocyte infiltrate intensity</td>
<td>Score 1</td>
<td>Score 1</td>
</tr>
<tr>
<td></td>
<td>20 cases</td>
<td>15 cases</td>
</tr>
<tr>
<td>B-lymphocyte infiltrate pattern</td>
<td>16 cases diffuse subepithelial</td>
<td>15 cases diffuse subepithelial</td>
</tr>
<tr>
<td></td>
<td>4 cases nodular subepithelial</td>
<td>–</td>
</tr>
</tbody>
</table>

The analysis of T-lymphocyte inflammatory infiltrate pattern highlighted the subepithelial and intraepithelial presence of these cells. All the study cases had a mixed pattern of the T-lymphocyte infiltrate (subepithelial associated with intraepithelial pattern).

As with B-lymphocytes, T-cell inflammatory infiltrate intensity was appreciated by means of a qualitative grading system with three scores.

In order to appreciate the T-lymphocyte inflammatory infiltrate intensity, we encountered a moderate inflammatory infiltrate (score 2) in eight patients with DM of less than 10 years evolution and an intense infiltrate (score 3) in 12 similar patients. Five patients with moderate T-cell inflammation intensity had a diffuse intra- and subepithelial pattern for T-lymphocyte and the other three similar patients had a diffuse intra-epithelial and a nodular subepithelial infiltrate pattern (Figure 7). All the 12 cases with an intense T-lymphocyte infiltrate intensity (score 3) showed a diffuse intra- and subepithelial T-cell pattern (Figure 8).

All the 20 patients with DM of more than 10 years evolution presented a reduced T-lymphocyte infiltrate intensity (score 1) with diffuse intra- and subepithelial pattern.

Figure 7 – CD45RO immunostaining, patient with DZ of less than 10 years evolution, moderate intensity for intra- and subepithelial T-lymphocyte infiltrate, subepithelial nodular pattern (ob. 10×).

Figure 8 – CD45RO immunostaining, patient with DZ of less than 10 years evolution, abundant intra- and subepithelial T-lymphocyte infiltrate (ob. 10×).
Discussion

In most cases, DM is associated with generalized chronic periodontitis with frequent exacerbations and short-term remissions; in addition, the periodontium is frequently affected by varied unfavorable factors [5]. The most important manifestation of DM is the hyperglycemia, because of vascular changes that lead to severe ischemia of many types of organs [6].

The histological study of gingival fragments from patients with DM highlighted in most of the cases significant changes in gingival epithelium and lamina propria.

Patients of the actual study presented at intraepithelial level PMN and rare lymphocytes that have been associated with epithelial cells swelling and intercellular bridges disintegration. The intraepithelial presence of inflammatory cells suggested an accelerated vascular permeability and also the growth of gingival epithelium permeability for this type of cells, with degenerative consequences over the epitheliocytes that have been evolved to ulcerations.

The present histological study of inflammatory infiltrate focused on the study of lymphocyte infiltrate in patients with DM of less than 10 years evolution compared to those with DM or more than 10 years evolution. However, the qualitative analyze of the inflammatory infiltrate on HE staining preparations allowed the revealing of a polymorph inflammatory infiltrate with PMNs, plasmocytes, macrophages. So, there has been detected in some cases, no matter of the evolution period of the DM, a big number of plasmocytes that predominated into the inflammatory infiltrate. These cases with plasmocyte predominance of the gingival inflammation were represented by the patients with low metabolic control of DM and aggressive periodontitis.

We also detected PMNs in both studied groups; these cells sometimes formed micro-abscessed areas. The micro-abscesses correlated with a bad metabolic control and a precarious oral hygiene and have been found in patients with clinical periodontal abscesses.

In general, the aspect of the inflammatory infiltrate was characteristic to the chronic inflammation that seems to be, as suggested by other authors, the result of the organism inability to stop the inflammatory response [7]. However, there were some situations when the polymorph inflammatory infiltrate made by lymphocytes, plasmocytes, macrophages and fibroblasts was associated with a large number of neoformation capillaries resulting into zones with granulation tissue aspect. These cases were represented by patients with rigorously controlled DM and organism with good response through granulation tissue as a preceding healing process phase.

Many studies sustain the predominance of inflammatory infiltrate plasmocyte cells of the diabetic periodontitis that secret immunoglobulins. Thus, Seppälä B et al. demonstrated that patients with long-term DM type 1 and a poor metabolic control presented high levels of plasmocytes into the gingival biopsies compared to the controls [8]. Also Kawamura JY et al. most recent study [9] showed that there was an intense, mostly plasmocyte inflammatory infiltrate with a diffuse pattern into the whole dense conjunctive tissue with abundant vascularization of lamina propria onto the gingival biopsies. The same study shows that the neutrophil infiltrate was generally poor and only in patients with clinical signs of periodontal abscesses it was rich, corresponding to the out study. The most numerous inflammatory cells were the IgG secreting cells, followed by the lymphocytes. The same study remarks that the plasmocyte inflammatory infiltrate intensity grew according to the periodontal disease aggressiveness.

In our study the lymphocyte inflammatory infiltrate intensity was qualitatively appreciated on histological preparates using a four degrees system (absent, discrete, moderate and intense) and on the immunohistochemical ones using a 0 to 3 score (0 – absent, 1 – discrete, 2 – moderate and 3 – intense). We could notice just from the beginning that the inflammatory infiltrate intensity was variable from a case to another, and also on the same preparate, making the correct appreciation more difficult. A similar aspect was noticed by Sohoel PD et al. [10] in their study when using the indirect fluorescence method.

Regarding the lymphocyte inflammatory infiltrate pattern, we noticed a diffuse pattern into the gingival conjunctive tissue (perivascular predominance) and also a nodular pattern (grouped in small focuses) of the analyzed inflammatory lymphocyte cells. The nodular pattern of the inflammatory infiltrate was met in patients with DM of less than 10 years evolution and it was rarely met compared to the diffuse pattern. The same pattern aspects of the inflammatory infiltrate were described by Lins RD et al. [11]. They similarly found a most frequent diffuse pattern of the inflammatory infiltrate into the chorion compared to the nodular pattern.

Referring to the lymphocyte inflammatory infiltrate intensity, our study showed that patients with less than 10 years DM had lymphocyte inflammatory infiltrate with maximum intensity (nine cases), followed by moderate intensity (six cases) and then discrete intensity (three cases). The lymphocyte inflammatory infiltrate was formed by isolated rare lymphocytes in two cases. In patients with less than 10 years DM the lymphocyte inflammatory infiltrate had a predominant small intensity (10 cases), followed by moderate intensity (five cases) and intense intensity (only one case). However, the lymphocyte inflammatory infiltrate was absent (isolated cells) on biopsies of four patients with more that 10 years DM.

We did not find any study in the literature that could quantify the inflammatory infiltrate intensity degree on usually stained preparates in patients with DM and periodontitis, and only one study made a comparison study between chronic gingivitis and chronic periodontitis without specifying the periodontitis cause. Lins RD et al. [11] study established, similarly to us, that the inflammatory infiltrate intensity in patients with chronic periodontitis was usually intense (88% of total cases) and the moderate inflammatory infiltrate was
found in 12% of the cases. The same study shows that de lymphocytes were predominant cells in 32% of the cases, plasmocytes in 32% and the lympho-plasmocyte inflammatory infiltrate in 36%. Karakuş A et al. [12] correlated the micro-vascular density to the DM gingival inflammation degree and demonstrated that there are no significant differences regarding the number of blood vessels in patients with diabetic periodontitis, no matter of the inflammatory infiltrate degree.

In our immunohistochemical study, we analyzed the aspects of the lymphocyte inflammatory infiltrate using CD20 as marker for B-lymphocytes and CD45RO as marker for T-lymphocytes. Anti-CD20cy monoclonal antibody, L26 clone marks B-lymphocytes line and is very useful for identification of neoplasm with B-lymphocytes origin if used together with a panel of immunohistochemical markers. The antibody reacts with an antigen (CD20) very useful for normal and neoplastic B-cells identification. The epitope of CD20 antigen has an intra-cytoplasmic localization and the antibody is utile for Hodgkin and Sternberg-Reed cells identification from Hodgkin lymphomas.

CD20 is a non-glycosylated trans-membranar of 33kD, expressed by B-cells precursors and mature B-cells, but it is lost during transforming into plasmocytes. The N- and C-terminal portions of the protein are localized on cytoplasm part of cellular membrane and only a small part of the protein expresses on the cellular surface [14].

In normal lymphoid tissues, the anti-CD20 antibody marks the cells of germinal centre, the lymphocytes of manta area and some inter-follicular lymphocytes, but not the T-lymphocytes, the histiocytes or the plasmocytes [16, 17]. There was no sign of marking at this antibody into the epidermis, sebaceous glands, pneumocytes or many other tested non-lymphoid normal tissues [16].

Positive reaction at this antibody was present in the most B-tested lymphomas. The mark for this antibody showed that during B-cells differentiation, the antibody is not expressed by very mature lymphoid cells, but begins to express during incipient maturation stages; it is then very intensively expressed in mature B-lymphocytes and after that, the CD20 antigen disappears into the plasmocytes [13]. Other studies gathered similar results, demonstrating a positive staining for this antibody in all B-lymphomas with big immunoblastic cells [16] and in all B-lymphomas without acute lymphoblastic leukemia and malign plasmocyte lymphomas [17].

The anti-CD45RO antibody reacts with the unique epitope for CD45RO and marks the most thyocytes, the inactive T-lymphocyte subpopulation, including CD4 and CD8, also the activated mature T-lymphocytes. This marker is excellent for formalin-fixed and paraffin-embedded prepares and for reactive T-lymphocytes and T-cells neoplasms on routine processed biopsy materials. CD45RO monoclonal mouse anti-human, UCHL1 clone, is marking the CD45RO epitope in normal and tumor T-cells, being useful for T-cells lymphoma detection [17].

The CD45 antigen is a trans-membranar protein expresses by the most nucleate hematopoietic cells and is coded by a single gene onto the chromosome 1. This antigen presents many isoformes, in the human leucocytes being identified five different isoformes: ABC, AB, BC, B and O. These isoformes are recognized by the CD45RA, CD45RB, CD45RC and CD45RO. All the CD45 isoformes present the same intracellular segment that has thyro-phosphatase activity. Variable leucocytes expresses characteristic CD45 isoformes, thus T-lymphocytes express CD45 corresponding to their maturation and activity. B-lymphocytes generally express ABC isoform and monocytes and dendrite cells generally express B and O isoformes. The granulocytes express only B and O isoformes [18].

The marked cells by anti-CD45RO antibody generally present a cellular membrane-limited mark. In normal tissues, the antibody marks the most thyocytes, inactive CD4 and CD8 T-lymphocytes subsets and activated mature T-lymphocytes. Most of all, granulocytes and monocytes are marked, while normal B-lymphocytes and NK cells are negative [19].

We found in a study using a large panel of normal tissues that the antibody had marked the lymphocytes into the T-cells area from amygdale, spleen and reactive lymph nodes [17]. Into the thymus, there have been marked 90% of the cortical and 50% of the medullar thyocytes, while the thymic blast cells have been negative. We constantly noticed a membranar mark in mature myeloid cells and in 20% of the macrophages. A diffuse, but weak cytoplasmic mark was noticed in glandular, squamous, transition epithelium, hepatocytes, sincitiotrophoblasts and smooth muscle [17].

With few exceptions, this antibody marks tumor cells from cutaneous T-cells malign lymphomas and the most reactive T-lymphocytes were positive on 85 cutaneous biopsies with inflammatory lesions [20].

There are few studies about the inflammatory infiltrate intensity and pattern depending on the lymphocyte type (T or B) determined by immunohistochemical methods on gingival biopsies in patients with diabetic periodontitis.

In our study, we remarked a higher intensity of T-lymphocytes (positive CD45RO) compared to B-lymphocytes (positive CD20cy) in both analyzed patients groups. Regarding lymphocyte pattern, we noticed that while T-lymphocytes were present at gingival intra- and also subepithelial level, B-lymphocytes were identified only subepithelial. The intra-epithelial T-lymphocytes presence suggests that the gingival epithelial permeability is raised in DM patients, allowing the lymphocytes and also the PMNs to penetrate, as indicated before, contributing to the epithelial integrity alteration. Both lymphocyte types had a diffuse and nodular pattern in patients with less than 10 years evolution DM, while the pattern for patients with more than 10 years DM was exclusively diffuse.

After Kawamura JY et al. [9], the positive CD45RO T-lymphocytes were the second more predominant type of cells in patients with DM type 1 and periodontitis after plasmocytes, their number being statistically superior to the non-diabetic patients. As a result, the intensity of positive CD20cy B-lymphocytes infiltrate
was lower than the intensity of positive CD45RO T-lymphocytes infiltrate, as shown by the same study.

Without specifying the DM implication in their etiology, studies of inflammatory infiltrate with B- and T-lymphocytes are more numerous and they identify these types of cells with markers and using diverse methods. Lins RD et al. [11] study on patients with chronic gingivitis and periodontitis showed that 88% of the chronic periodontitis cases were positive for CD20 and the intensity score was 2 in 13 cases and 1 in nine cases. The same study identifies only the positive CD4 from the T-lymphocytes (T-helper lymphocytes) in 88% of the patients with chronic periodontitis, with intensity score 1 and an isolated infiltrate pattern of these cells.

Conclusions

Patients with DM and periodontitis presented major histological alterations of the gingival epithelium and also an inflammatory infiltrate with a variable morphology depending on the DM evolution, metabolic control and oral hygiene.

The periodontitis diabetic inflammatory infiltrate was polymorph, plasmocyte predominant in patients with aggressive periodontitis and a poor metabolic control of the DM, and PMN predominant in patients with clinical periodontal abscesses, the lymphocyte always being constant.

The lymphocyte inflammatory infiltrate was mostly diffuse into the gingival chorion of the diabetic patients no matter of the DM evolution. The nodular pattern was characteristic for the patient with a less that 10 years evolution DM. The lymphocyte inflammatory infiltrate intensity was higher in patients with periodontitis and less than 10 years evolution DM compared to the patients with more than 10 years evolution DM.

CD45RO positive T-lymphocytes were more numerous than CD20 positive B-lymphocytes and they were present gingival intra and subepithelial in all diabetic patients, regardless of the DM evolution.

References


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