DENTAL MANAGEMENT OF THE CARDIAC PATIENTS
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ABSTRACT
Recent epidemiological studies reveal that oral pathogens are co-factors in the formation of atheroma plaques and the advanced periodontitis represents a risk factor for cardiovascular diseases. The pathogenic bacterial burden represents the essential in the association of coronary artery disease with periodontitis, which is extremely relevant to the periodontal lesions that develop in the oral cavity, a space crossed by the most unexpected bacterial combinations from alimentation and air. The main objective of the study is to determine the prevalence of bacterial burden in cardiac patients with periodontal disease and to follow up the effects of dental procedures associated with low-doses of antibiotics in relation with coronary atherosclerotic disease. The study implies a lot of 92 patients undergoing the following investigations: 1. Panoramic radiography of the oral cavity, 2. Clinical examination of the oral cavity, 3. Bacterial examination on the dental plaque’s level 4. identification of the presence of serum inflammatory markers, 5. Bio-humoral examinations, 6. Cardiology specialty examination. The control of periodontal infection could lead to the improvement in the levels of systemic inflammation markers and in those of endothelial dysfunction’s markers. As far as the control of periodontal infection decreases the incidence of coronary atherosclerotic cardiovascular events can be assumed that the dental management of cardiovascular disease is in progress.

Keywords: periodontal disease, atherosclerosis, periodontal pathogens

INTRODUCTION
Recently, the researchers have redefined the relationship between the oral health wellness and the patients’ area, stressing on the importance which functional integrity of the dental surface has in the context of organic equilibrium [1,2]. Various epidemiologic studies have proved that there were associations between different degree types of parodontopathies and systemic affection such as atherosclerosis, heart failure or stroke. Oral pathogens turned out to be co-factors in the atheroma plaque formation and the evolutive parodontopathies are a risk factor for cardiovascular diseases.

Periodontal infectious-inflammatory processes generates physiopathological sublayer for the atherosclerosis, and the previous triggers bio-histopathological modifications in the structure of the arterial aortic walls, of the carotid, and of coronary arteries, with the known major implications and consequences [3,4].

HYPOTHESIS. AIMS.
Both chronic marginal parodontopathy and ischemic heart disease, under their distinct clinical forms, take the lead in special pathology, so that in the daily practice we often encounter associations of these, in
various degrees of difficulty, to which we must find a solution. The relation between infection-inflammation-atherosclerosis and the ischemic heart disease and coronary atherosclerosis is well known, but currently the researchers’ opinions differ widely concerning the opportunity of associating the dental procedures and the antibiotics in the therapy of chronic marginal periodontitis, as well as the efficiency of this association measured by the limitation of the atheroslerotic effect of the aortic wall inflammation (conditioned by periodontal pathogens or their toxines), respectively of the coronary clinical events [5].

In literature, papers revealing encouraging results came into sight, pointing out the number of cases involving patients treated by combined dental procedures and antibiotics in comparison with the ones treated only by simple dental procedures, where less atherosclerotic determinations, and milder forms of ischemic heart disease occured.

The activity in a dental medicine cabinet is marked by a large number of patients affected by different levels of parodontopathy, and also with its association with ischemic coronary disease, thereby the idea of some more favourable and quicker results obtained through the conformed methods motivated us.

The main objective of the study is to determine the prevalence of the bacterial burden in the case of cardiac patients suffering from periodontal disease and to observe the evolution of the dental procedure’s effects in association with low-dose of antibiotics.

**MATERIAL AND METHODS**

I made a prospective study, from January 2008 through March 2011, on a lot of 92 subjects (Table 1) suffering from chronic marginal paradontopathy and cardiac disease. The examinations took place in the individual cabinet in Cluj-Napoca.

<table>
<thead>
<tr>
<th>Characteristics of the group</th>
<th>n=92</th>
</tr>
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<tbody>
<tr>
<td>Gender (Female/Male)</td>
<td>65.2%/34.8%</td>
</tr>
<tr>
<td>Age (18-65 years old)</td>
<td>41.2/57.8</td>
</tr>
<tr>
<td>Origin (Urban/Rural)</td>
<td>70.7%/29.3%</td>
</tr>
</tbody>
</table>

The patients were asked to give their consent for their introduction in the study, and thus issues linked to ethics and deontological nature being eliminated.

5 days ahead of the dental procedures’ debut, the patients were administrated 650 mg Augmentin (amoxiciline and clavulcanic acid) twice a day in a span of 12 hours and 2x250 mg Metronidazol, respectively post-procedural 2 days, same amount.

The whole research lasted for 36 months; the examination was performed after each 12 months, with the exception of the dental procedures maintenance made every 4 months, followed by bacteriologic examination and the measurement of the serum antibodies.

Having in view the fact that the initial examination were identical for all the patients, later investigations were reported to their initial ones in order to perform a comparative analysis of the study in a consistent manner.

The atherosclerotic lesions were evaluated: intima media thickness he level of internal carotid, ecographic appreciation of the atherosclerotic plaques from the carotid and coronaries, the evaluation of the factors which lead to the inflammation (C-reactive protein, white blood cells, globulele albe, fibrinogen) and the clinical manifestations resulted through coronary events, unstable angina pectoris, heart failure etc.

The investigations were made according to following plan: 1. Panoramic radiograph of the oral cavity; 2. The clinical examination of

The Russell Periodontal Index (PI) was used, built upon the data recorded after the examination of the marginal periodontium of each tooth.

The statistical analysis was made with the help of an application named EPIINFO, version number 6.0, a program belonging to the Center of Disease Control and Prevention - CDC (Center of Disease Control and Prevention) from Atlanta and WHO (World Health Organisation), suited for medical statistics processing. Averages of the parameters were calculated, S-au calculat medii ale parametrilor, intervals for frequencies, standard deviations, statistical significance test by Student method (t-Test) and $\chi^2$.

RESULTS

The first examination performed by us was the panoramic radiograph of the oral cavity. It is known that the Radiologic examination in parodontopathy is important for the diagnosis in order to appreciate the presence and the extension of the erosion of the alveolar bone, and in some special cases even highlights some etiological factors [6].

The oral clinical examination marked the reduction of some lesions of the soft-tissue, of the epithelial insertion and of other inflammatory processes corresponding to points 1 and 2 from Russell IP for the patients fitting in the two degrees 1 and 2 of the IP, meaning from 35,8% to 33,7%, after 1 year, to 25,0% after 2 years and to 9,8% after 3 years.

Sampling for the bacteriologic examination was taken from the parodontal bags and from the gingival channels, respecting the asepsy and atisepsy rules. Three successive bacteriological examinations were performed for the initial examination on a time span of 7 days in order to identify the perennial bacteria. Those pathogenic bacteria which were identified in all the three examinations entered the case study of bacterial burden. Viridans Streptococci was the most frequent, being present in the case of 60 patients (65,2%), followed by Prevotella intermedia (31,5%), Treponema denticola (28,3%) and Porphyromonas gingivalis (27,2%). Actinobacillus actinomyctemcomitans with only 5 presences (5,4%) came the last one during our study (fig.1).

In 3 years time, Prevotella intermedia and Tonerella forsythesis cannot be spotted in any patient, Streptocucus viridans and Treponema denticola are decreasing significantly, from an initial 65,2% , to 27,2%, respectively from 28,3% to 6,5% (fig.1).

Actinobacillus actinomyctemcomitans maintains the same prevalence during the first three evaluations (5,4%), decreasing only at the final evaluation (3,3%).

In the initial evaluation, after the three series of bacteriologic examinations, we identified the number of constant pathogen agents in the samples taken from the parodontal bags as being the following:

- 3 pathogen agents: Streptococcus viridans+ Treponema denticola+Porphyromonas gingivalis in a number of 22 patients (23,9%)
- 2 pathogen agents: Streptococcus viridans+Prevotella intermedia – identified at 27 patients (29,3%)
- 1 pathogen agent was identified at 43 patients (46,8%).

The association of 3 pathogen agents, initially encountered at 23,9% of the patients, has a decreasing trend reaching 4,3% on the 3 years evaluation.
The association of the 2 pathogen agents, such as Prevotella intermedia and T. forsythensis disappears.

A significant increase of the percentage of the patients without pathogen agents across the 3 years from initially 0,0% to 66,3% on the last evaluation after 3 years was observed (fig.2).

If initially, with the exception of Actinobacillus actinomycetemcomitans, increased levels of antibodies in all pathogen agents were determined, during the 3 years are significantly reduced. In three years, only one case of increased level of Viridans Streptococcus was recorded. The significant reduction in the serum levels was recorded at all pathogen agents (p<0,001) (fig.3).

Increased values of C-reactive protein were initially recorded in the case of 27,2% of the patients, percentage which significantly decrease at 10,9% in 3 years.

Initially increased values of Fibrinogen were determined in almost 60% of the patients (58,7%); this percentage decreases significantly after 3 years (22,8%).

In both cases of C-reactive protein and of fibrinogen the values decrease significantly (p=0.013, respectively p<0.001).
The values of arterial pressure >140/90 mmHg were initially recorded at 35.9% of the patients, this percentage revealing a decreasing trend, reaching 17.4% (p<0.001) at the 3 years evaluation.

A normal ECG was recorded at 64.1% of the patients at the initial evaluation, this percentage increasing to 71.2% in a year, to 77.2% in 2 years and to 79.3% in 3 years.

Regarding the intimate/media thickness report of the carotid (IMT), ecocardiographic examination initially revealed a percentage of 17.4% with the IInd degree and 17.4% with the Ist, and 65.2% with 0 degree. Significant modifications in the IMT at the IInd degree patients (p>0.05) were not recorded, but only in the case of the ones with the Ist and 0
degrees (p=0,033) (table 2).

Carotid artery stenosis was initially present in 30,4% of the patients, this percentage being maintained until the final evaluation, when it reached 22,8% (p>0,05). During the 3 years, only a single case passed from moderate to severe stenosis and other 7 cases passed from severe to normal stenosis.

Regarding the coronary lesions, we recorded: left common carotid >1 = initially 53,3%, and in the end 67,4% of the patients, initial left-sided intraventricular portion > 1 at 64,1% patients initially and 70,7% in the end and initial right coronary portion > 1 at 70,7% patients initially and 64,1% in the end (table 1).

Table 2.

<table>
<thead>
<tr>
<th>Examined coronary</th>
<th>Female (F)</th>
<th>Before</th>
<th>Male (M)</th>
<th>Before</th>
<th>After 3 years</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N.</td>
<td>%</td>
<td>N.</td>
<td>%</td>
<td>N.</td>
</tr>
<tr>
<td>Left common coronary</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;1</td>
<td>49</td>
<td>53,3</td>
<td>62</td>
<td>67,4</td>
<td></td>
</tr>
<tr>
<td>≥1</td>
<td>43</td>
<td>46,7</td>
<td>29</td>
<td>31,5</td>
<td></td>
</tr>
<tr>
<td>&lt;1</td>
<td>0</td>
<td>0,0</td>
<td>1</td>
<td>1,1</td>
<td></td>
</tr>
<tr>
<td>Initial intraventricular portion</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;1</td>
<td>59</td>
<td>64,1</td>
<td>65</td>
<td>70,7</td>
<td></td>
</tr>
<tr>
<td>≥1</td>
<td>22</td>
<td>23,9</td>
<td>22</td>
<td>23,9</td>
<td></td>
</tr>
<tr>
<td>&lt;1</td>
<td>11</td>
<td>12,0</td>
<td>5</td>
<td>5,4</td>
<td></td>
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<tr>
<td>Right common coronary</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;1</td>
<td>65</td>
<td>70,7</td>
<td>59</td>
<td>64,1</td>
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<tr>
<td>≥1</td>
<td>22</td>
<td>23,9</td>
<td>28</td>
<td>30,4</td>
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<tr>
<td>&lt;1</td>
<td>5</td>
<td>5,4</td>
<td>5</td>
<td>5,4</td>
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</table>

DISCUSSIONS

The relation infection-inflammation-atherosclerosis-periodontal disease is described and sustained in broad studies in medical literature. Periodontitis reflects a variety of oral pathology ranging from gingivitis to severe periodontitis with the loss of alveolar bone and tooth. It is believed that periodontal pathology is due to the accumulation of dental plaque (subgingival biofilms) with the infection and later inflammation of the mucus [7].

It is possible for atherosclerosis to exacerbate the periodontal disease by determining an systemic inflammatory reaction or even by the agency of subclinical ischemia. Some of the recent studies revealed an increased risk of 1,5-2 for the patients suffering from periodontal disease in developing some atherosclerotic complications [8]. The presence of an inflammatory outbreak in the oral cavity which might potentiate the atherosclerotic process by stimulating the umoral ways and those mediated by the cell. The level of inflammation of the periodontal disease is enough in order to create a systemic inflammatory response as it is highlighted by the increase of C-reactive protein’s level of serum. In addition, the presence of the periodontal infection can lead to bacteriemi episodes with the inoculation of the atherosclerotic arterial plaques with periodontal pathogens such as Porphyromonas gingivalis, Actinobacillus actinomycetemcomitans, Bacteroides Forsythus etc., which are able to boost the inflammatory process by developing and multiplying, condition that generates the instability of atherosclerotic plaque [9].
From a study called „Kuopio Ischaemic Heart Disease Risk Factor“ [10], upon a group of 1023 male patients (46-64 years old), during 1991-1993 it was concluded that the subjects without a debut of coronary heart disease, that the IMT significantly increased in the same rhythm with the levels of IgA titre for P. gingivalis, Actinobacillus actinomycetemcomitans.

In another study, [11] on 657 „etiologic bacterial burden“ was defined as the accumulated colony on subject by A. actinomycetemcomitans, P. gingivalis, T. Forsythensis and T. Denticola. The number of white blood cells and IMT shows a significative increase along with the etiologic periodontal bacterial burden. The intensive periodontal therapy [12] for the patients suffering from severe marginal parodontopathy, meaning the classic procedures associated with local antibiotics determined on a short-term the visible reduction of C-reactive protein’s serum level, therefore the decrease of the inflammatory process in the periodontal and general vascular substrate.

CONCLUSIONS

1. If parodontal disease is one of the major leading reason for tooth loss, by the quality of periodontal treatments this phenomena could be prevented.

2. The clinical oral cavity examination at the end of the research study conducted in the spirit of Russell Periodontal Index powerfully illustrates the effect of associating antibiotherapy with the dental procedures, by the restoration of the inflammatoy infected gingival substrate and of the gingivodental ligaments proved by the increase of the number of patients with 0 periodontal index.

3. The bacterial factors (pathogen agents) highlighted on the occasion of the initial evaluation identified the existence of six pathogens, and the associations between them in the determination of bacterial burden were mentioned in the previous pages. Nonetheless, in spite of all the periodontal treatment associated with antibiotherapy, both total elimination of the pathogens, or in the cases of all patients were not possible.

4. The inflammatory factors (markers) especially C-reactive protein behaved as exact with the response of the inflammation, in parallel with the elimination of the lesional substrate through dental procedure combined with antibiotics.

Along with C-reactive protein, in total synergism, the serum levels of the white blood cells and fibrinogen decreased, the latter being more constant to the intensity of the infectious process without having a specific character.

5. Blood pressure values were amended as the aggressive inflammatory processes were restored, with the rehabilitation of gromerulary filtration, the decrease of creatine as a consequence of bacterial elimination treatment.

6. The electrocardiographic examination at the end of medical monitoring highlighted some improvement in the electric signals and the disparition of the ischemic lesionage in all the 6 cases (6,5%).

Atherosclerotic manifestation was validated by acocardiographic measurements, 2 D and colour Doppler, IMT and carotid and coronary stenosis-like aspects.

The ischemic process was validated by static EKG image and 6 EKG force demonstrations, as well as the clinical angina pectoris effort manifestations.

No intolerance reaction to the associated treatment was remarked.

To the extent that the control of periodontal infections decreases the prevalence of cardiac-coronary atherosclerotic events, we may say that the dental management of cardiovascular diseases is in progress.
REFERENCES
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