Destructive periodontitis, its prevalence among chronic periodontitis patients, with cohort incentive conditions

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Abstract:

Sixty-six patients have been diagnosed and distinguished as having destructive periodontitis out of one thousand slowly growing chronic periodontitis (S.G.Ch.P.), constituted 6.6%, females was 68.18%, male was 31.81%, a significant difference was found between male and female.

Possible activating and inverting factors was studied, restraint stress and depression appear having a significant relationship, nutritional factor as well showed a significant influence on inversion of S.G.Ch.P. into DP.

High gingival index, sever bone destruction; deep packet and an eventual tooth mobility with generalized distribution pattern were the characteristic clinical feature of the disease.

In conclusion local bacterial and systemic conditions either psychic or somatic may interfere in the activation and inversion of the adult periodontitis into destructive periodontitis under the basis of systemic and psychological factors.

Key word: Adult periodontitis, destructive periodontitis, stress, nutrition atherosclerosis.

Introduction

According to the clinical criteria of chronic periodontitis, the presence of periodontal pocket with radiological bone loss is essential in diagnosis and differentiation. Earlier histometric studies suggested that chronic periodontitis is a slowly progressing disease as it stayed inactive (1). It could be activated to get harmful progression when take sever clinical gingival signs with unexpected bone loss followed with deep packet formation (2), this activation associated with gingival invasion by pocket bacteria (3,4), and invading the alveolar bone as well (5,6,7). The bacterial invasion encourage the immunological reactions on gingival level to liberate a huge of destructive enzymes and proteins (8,9) which seemed to be already systemically stimulated by the antigens of periodontal pathogens (10), this reaction may exposed clinically as an exacerbation of active inflammatory reactions (11). A significant positive correlation have been found between the histomatic index, bacterial activation, especially that of black pigmented bacteria (12). The clinical indicators had explained that the bacterial activation ends in active tissue destruction (13). It has been demonstrated that the bacterial invasion into the connective tissue in advanced periodontitis were mostly cocci, mobile rods, filaments and spirokettts (4,7). The bacterial flora of active sites of chronic periodontitis pockets have been demonstrated as predominated with B. forsythus, P. gingivalis, P. micrus, Actinobacillus actinomycetes temcomitance, W. rectus and B. intermedius (14, 15, 16).

Many earlier studies showed that
nutrition is an essential systemic factor in periodontal health and disease. However, shortage in calcium intake is negatively influencing alveolar bone building, calcium has long been a candidate to modulate periodontal disease in influencing the mineral density of alveolar bone, low calcium intake is a risk factor for periodontal disease and could result in more severe periodontal breakdown

Vitamin B. complex positively influencing wound healing and could result in statistically significant superior clinical attachment level gain, its shortage can give a contrary worse result

The effect of menopausal period on periodontitis and osteoporosis are characterized by the loss of bone mass. Osteocalcin level have been postulated as a marker of inhibition of bone formation. It has been found that osteocalcin level in gingival cervical fluid is correlated positively with periodontitis

Periodontal disease, especially measured by alveolar bone loss is a strong and independent predictor tooth loss in postmenopausal women

It seems important to estimate the prevalence of the destructive periodontitis and the possible cohort incentive systemic and/or psychological conditions which help in converting the slowly growing chronic periodontitis into a destructive type.

Materials and Methods

One thousand patients were diagnosed as having adult type periodontitis. Referred to the department of periodontology, college of dentistry, Mosul University to get periodontal therapy. The patient were submitted under a special regimen of diagnosis included a profound personal interrogation about their condition, precise notes about their social, economic, psychological and general health were taken, psychological and systemic events and landmarks were recorded. Profound history of oral illness complains of a past and present illness, previous treatments, and drugs intakes. All patients assessed for systemic check up, routine blood analyses, blood sugar and cholesterol as well. Female hormone analyses were recommended, as needed, especially the menopausal hormone imbalance, cholesterol analyses included VLDL, LDL, HDL total lipids, Triglyceride and finely the uric acid.

Gingival index (GI) \(^{(21)}\), clinical pocket depth (CPD), was measured with WHO, CPITN probe \(^{(22)}\). Full mouth x-ray were taken (ortho-pantograph) and periapical films with long cone machine. Tooth mobility was also recorded according to modified miller Index \(^{(23)}\). The criteria used to identifying destructive type were the age, sex, pattern of lesion, mode of distribution, evidence and design of bone loss, variable degree of tooth mobility, gingival bleeding upon probing, gingival distortion, presence of variable amount of plaque and calculus. Pocket depth exceeded 4mm on any side of the tooth was considered as having chronic periodontitis.

Pregnant women and diabetic patients have been excluded from this study.

Bacteriologic culture of random pocket contents smears have been performed on blood agar and chocolate agar, gram stain to study the morphology and pattern of hymolysis, for identification; IMVC, catalase, oxidase and coagulase biochemical's were used [MAST DIAGNOSTICA, U.K].

Results

Table 1: Out of one thousand adult
periodontitis, 6.6% were diagnosed as having destructive evolution (66 patients). The average age of female was 31.8 ± 2.8, whereas that of male was 35.4 ± 3.3. Female constituted 68.1% (45 out of 66), male constituted 31.9% (21 out of 66). A significant difference was obtained according to t test in the distribution of the disease on sex (P<0.01), but insignificant on age distribution (Table 4.B).

The Gingival index scores of DP patients were 2.501, that of female were 2.301, and that of male was 2.707. Insignificant variables were obtained by their mean according to T test between the female and male gingival index scores.

The total number of teeth present in the oral cavity of all the 1000 patients was 20462 that of DP patient were 1782, which equal to 8.7088% of total teeth number. Female had 899 teeth equal to 4.3935% of total number and 50.4489% of DP teeth number. Male had 883 teeth, equal to 4.3153% of total teeth number and equal to 49.561% of DP teeth number.

Teeth showing loss of surrounding bone exceeded 20% of root length in x-ray were 1041 (58.4175%) of DP teeth, and 5.078% of total teeth number. Female had 609 teeth, equal to 2.97% of the total and 58.5% of DP teeth. Male had 432 teeth, equal to 2.112% of total and 41.498% of DP teeth.

Mobile teeth with grade 1, 2 or 3 according to modified Miller index were 774 in both sex, equal to 43.433% of DP teeth number, and equal to 3.782% of total teeth number, represented 74.3515% of the teeth involved with bone loss 20% or more of root length. Female showed 433 mobile teeth, equal to 55.943% of DP teeth, and 2.116% of total teeth number, and equal to 71.1% of teeth involved with 20% of bone loss. Male showed 341 mobile tooth, equal to 44.056% of DP teeth, and 1.66% of total teeth number which equal to 78.935% of the teeth involved with 20% of bone loss. Insignificant differences were obtained in the intra oral distribution of the disease between male and female (T. test).

Table 2: Slowly growing chronic periodontitis (S.G.Ch.P.) represented with the other 934 patient, constituted 93.4% of total patient number, female were 435, their age average was 38.1 ± 2.8, constituted 43.5% of total patients, equal to 46.5738% of S.G.Ch.P. patients, male was 499, their age, average was 42.7 ± 3.1, constituted 49.9% of total patients, equal to 53.426% of S.G.Ch.P. patients. Insignificant differences were obtained in patient’s age on sex distribution.

The means GI scores showed that total S.G.Ch.P. scored 1.090.11, female-scored 1.170.12, male scored was 1.220.31. The difference was insignificant according to t test on sex distribution. The number of present teeth in S.G.Ch.P. patient was 18680, constituted 91.2911% of total present teeth, female had 8765 tooth, equal to 42.835% of total, and equal to 46.4556% of total present teeth and equal to 51.879% of S.G.Ch.P. teeth number, and 47.3609% of total teeth number. Female had 5314 tooth, constituted 54.834% of S.G.Ch.P. present teeth, equal to 25.97% of total. Male had 4377 tooth, constituted 45.1656% of S.G.Ch.P. teeth and 21.3908% of total patient teeth.

Insignificant intra oral distribution of disease was obtained between the two sexes. Significant difference was obtained in number of teeth involved with periodontal pocket in privilege to female over male (Table 4B).

The mobile teeth were 331, constituted 1.37% of S.G.Ch.P. teeth,
equal to 1.6176% of total teeth number, and 3.4155% of teeth involved with periodontal pocket 4mm in depth. Female had 167 mobile tooth, constituted 1.905% of S.G.Ch.P. present teeth, equal to 0.216% of total teeth, and equal to 3.142% of teeth involved with 4mm pocket depth. Male had 164 mobile teeth, constituted 1.654% of S.G.Ch.P. teeth, equal to 0.8014% of total teeth number, and 3.7408% of teeth involved with 4mm periodontal pocket, insignificant differences were obtained in intra oral distribution of S.G.Ch.P. disease when comparing the two sex data.

High significant differences were obtained in GI, clinical pocket depth alveolar bone loss as seen in x-ray and tooth mobility data when comparing the DP data with that S.G.Ch.P. in exception of patients age (P<0.01) (Table 4A).

The Complementary test:

Table 3 : Direct personal interview revealed that 26 patient out of 66 showed diseases activation accompanied with the restraint stress, constituted 39.39%, female was 18, equal to 27.272% male was 8, constituted 12.12%, this result means that 40% of DP female and 38% of DP male had strain stress and depression. Eighteen patient had nutritional problems, constituted 27.27%, female was 11, constituted 16.67%, and male was 7, equal to 10.606% of total DP patients. Female constituted 61.11% of total nutritional factor number, while male constituted 38.89% of the nutritional factor number. Both, psychological and nutritional conditions appeared significant when compared to the total DP number, and also when compare female to male data (P<0.05) the females appeared more susceptible than male.

The menopausal factor included nine women, constituted 20% of DP female number, equal to 13.636% of total DP patient. It appeared significant according to t test when compare to total DP female. High total cholesterol level over 320 mg/dl was appeared in 8 patient, showed high risk level of HDL and LDL, constituted 12.12% of total DP patient, 4 female and 4 male, each constituted 6.06% of total DP patients. Female constituted 8.89% of female DP patient, male constituted 19.05% of male DP patients, thus male showed significant difference over female in cholesterol factor (P<0.05), table 3.

Other five patients, 2 male and 3 female showed that the disease activation could associated with a traumatic hand scaling, they constitute 7.576%, female constitute 6.67% of female DP patients, while male constitute 9.523% of DP male.

Bacteriologic test:

Random smear of pocket contents, purified, cultured, stained then identified biochemically, revealed that the bacterial flora was rich with black pigmented bacteroids, while the S.G.Ch.P. pockets showed increased number of S. sangings. The microorganisms existed in both types were almost identical in species. Bacteroids, fusobacterium, spirochetes and streptococcus were clearly identified in both groups.

Discussion

High gingival index scores, spontaneous bleeding, distortion of gingival pattern and contours, generalized advanced destruction of alveolar bone, deep pockets, tooth migration with eventual tooth mobility were the common characteristic features of destructive periodontitis, this profile could be suggested as a specific entity differentiable from the slowly growing chronic periodontitis.
The major bacteriological components demonstrated in this study related to DP pockets was the bacteroids, while S.G.Ch.P. pockets showed increased number of streptococcus bacteria. This bacteriological picture could suggest that there is inversion of bacterial flora from the predominated streptococcus into the predominated Bacteriod, which could be exposed as a progression of adult periodontitis into the destructive periodontitis.

A significant differences were obtained in comparing the data of DP to that of S.G.Ch.P., while the inter group variables were generally insignificant in both groups. In exception, and according to sex, females showed significant variable in their number over male in DP. On other hand, the disease history seemed to be sudden; the activation occurs on previously existent adult periodontitis, which could suggest that there is inversion of S.G.Ch.P. into DP. This result could support the concept of intense disease activity (5, 6), the inversion could be induced by systemic condition, either somatic or psychic (25). A hypothesis of an increased risk for destructive periodontal disease due to psychic stress has long been promoted, however the research on the influence of stress on periodontal diseases is still in its infancy (26). Recent study showed that restraint stress modulates the progression of periodontal inflammation with positive correlation (27, 28). Positive correlations have been demonstrated between the severity of periodontal disease and the presence of antigen of periodontal pathogens in circulating blood (29). A familial distribution of HLA-A and HLA-B antigens demonstrated that there is a family high risk of DB (30,31).

It has been suggested that stress have a negative influence on healing process (27) and positively influencing the periodontal breakdown (28).

In the present study, the psychological and nutritional factors appeared significantly influencing the inversion of S.G.Ch.P. into DP in both sex. The epidemiologic studies have shown that periodontitis may be associated with presence of atherosclerosis, DNA from periodontal pathogens has been detected in atherosclerotic lesions, the data confirm that DNA of periodontal pathogen can be detected in atherosclerotic plaque, especially that of porpheromous entermidius(32).

The behavioral factor in this study appeared insignificant, previous studies have suggested that risk for adult periodontitis has genetic (heritable) component, it has been confirmed that approximately half of the variance in disease in the population is attributed to genetic variance, the basis for the heritability of periodontitis to be biological and not behavioral in nature (33). In conclusion, there are a local bacterial and systemic either psychic or somatic factors could interfere in the activation of bacterial flora of S.G.Ch.P., and able to invert the adult periodontitis into destructive lesion under the basis of genetic and immunologic behavior. The clinical characteristic of DP could suggest a distinguishable clinical entity of the disease.

References :

4- Sagli R , Newmann M G, Carranza Jr F A, and Pattison G L: Bacterial invasion of
gingival connective tissue in advanced periodontitis in man, J Periodontol, 1982 ;
53(4) : 217-222.
5- Duran B M, Perdix G, Delaland J : La
parodontite aggressive; une entite clinic, J
6- Socransky S S, Haffajee A D, Goodson J
M, and Lindhe J: Jew concept of
destructive periodontal diseases, J Clin.
7- Dzink J L, Socransky S S, Haffajee A D:
The predominant cultivable flora of active
and inactive sites of destructive periodontal
316-323.
8- Suzuki J B, Martin S A, Vincent J W, and
Falker J R: Local and systemic production
of immunoglobulin to periodontal
pathogens in periodontal disease, J
9- Genco R J, Slats J, Mouton C, and Murray
P: Systemic immune response to oral
organisms in: anaerobic bacteria, Plenum
publishing co., New York, U.S.A., 19,
10- Martin S , Falker JR W, Suzuki J, Hawley
C, and Mackler B : Local and systemic
immunoglobulin reactive to bacteroid
gingivalis in rapidly progression and adult
periodontitis, J periodontol Res., 1986 ;
(21)2 : 351-354.
11-Baumann H, Gualic J: The acute phase
response, Immunology today, 1994; 15(1):
74-80.
12- Harper D S, Robinson P T: Correlation
between microbial, histometric and
clinical indicators of periodontal disease
before and after root planning, J Clin.
13- Haffajee A, Socransky S S, Ebensol J, and
Smith D: Clinical microbiological and
immunological factors associated with the
treatment of active periodontitis lesion, J
14- Mark Ide, Daljit J, Paula Y C, Marthin C,
Barclay G R, and Wilsin R F: The short –
term effect of treatment of chronic
periodontitis on circulating level of
endotoxin, C-reactive protein, tumor
Necrosis factor– a, and inter leukin– 6, J
periodontol, 2004 ; 75 (3) : 420428.
15-Hui-Wen Y, Yu-Feng H, and Ming-Young
C: Occurrence of porphyromonas
gingivalis and Tannerella forsythenisia in
periodontaly diseases and healthy subject, J
periodontol, 2004; 75(9) : 1077-1083.
16-Becker T, Prior K, Ehmke B, and
Flemming T F : Specific antibiotics in
the treatment of periodontitis , A proposed
strategy, J periodontol, 2004 ,75(1) : 169-
175.
17-Nishide M, Grossi S G, Dunford R G, Alex
WHO, Trevisan M, Genco R J: Calcium
and the risk for periodontal disease, J
periodontol, 2000 ; 71 (7) : 1057-1066.
18-Neiva RF, Nociti Jr. FH, Soehlen S, Wong
H L: Effect of Vitamin B-complex
supplementation on periodontal wound
healing, J periodontol, 2005 ; 76(7) :1084-
1091.
19-Bullon P, Goberna B, Guerrero J M, Perez-
cano R, Sahauquil M A : Serum , Saliva,
and gingival cervical fluid osteocolcin :
their relation to periodontal status and bone
mineral density in postmenopausal women,
J periodontol, 2005 ; 76(4) :513-519.
20-Tezal M, Wactawski W J, Grossi S G,
Domochowski J, Genco R J : Periodontal
disease and incidence of tooth loss in post
menopausal women, J periodontol, 2005;
76(7) : 1123-1128.
21- Amamo J, Bay I: Problems and proposal
for recording gingivitis and plaque,
International Dental Journal; 25(2) : 229-
235.
22- Amamo J: The new WHO method
(C.P.I.T.N.) for assessment of periodontal
treatment need, J periodontol, 1983 ; 2 (3)
: 343 -350.
23- Millers S C D: Textbook of
periodontology, Blakiston, Philadelphia,
24- Giovannoli J L, Heins P: Classification of
the radiographic aspect of infrabony lesions
related to root proximities, J periodontol,
1988; 2(1) : 31- 40.
25- Buatongsri V, Songpaison Y, Hongpuason
N, Phantom V P, Clarke N : The
distribution of sever periodontitis in urban
and rural high risk to stress group of
population, Current Rent. Journal, 2002,
25(1) : 1-7.
26- Tetsuo T, Nobuyo S, Shin S, Hitoshi K,
Takashi Y, and Toshihide N: Effect of
restraint stress on the progression of
experimental periodontitis in rats J
periodontol, 75: 200.
27- Coimmer G, Janda M, Wies everlasting P K,
Jakse N, Polansky R, Petri C: Coping with
stress ; its influence on periodontal disease,
J periodontol, 2002 ; 73(11) : 1343-1351.
J D: Relationship of clinical depression to
periodontal treatment outcome ,J
periodontol, 2002 ; 73(4): 441-449.
29- Gmur R, Hrodek K, Sozer V, and
Gygenheim B: Double blind analysis of
the relation between adult periodontitis and
systemic host response to suspected
Figure (1): Different cases of Destructive periodontitis representing the clinical and radiological features.
### Table 1: distribution of destructive periodontitis.

<table>
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<tr>
<th></th>
<th>Number of patients according to sex</th>
<th>Number of patients out of 1000</th>
<th>Percent of the destructive periodontitis patients number according to the sex out of 66</th>
<th>Percent of mobile teeth of S.G.Ch.P. patient</th>
<th>Tooth mobility</th>
<th>Number of involved teeth with perio. Pockets and bone loss &gt;20% of root length</th>
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<tr>
<td>Total</td>
<td>66</td>
<td>6.6%</td>
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<td>33.2±5.1%</td>
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<tr>
<td>Female</td>
<td>45</td>
<td>4.5%</td>
<td>68.18%</td>
<td>31.8±2%</td>
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<tr>
<td>Male</td>
<td>21</td>
<td>2.1%</td>
<td>31.81%</td>
<td>35.4±3%</td>
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### Table 2: distribution of S.G.Ch.P. periodontitis.

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<th>Number of patients according to sex</th>
<th>Number of patients out of 1000</th>
<th>Percent of the destructive periodontitis patients number according to the sex out of 943</th>
<th>Percent of mobile teeth of S.G.Ch.P. patient</th>
<th>Tooth mobility</th>
<th>Number of involved teeth with perio. Pockets &gt;4mm depth</th>
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<tr>
<td>Total</td>
<td>93</td>
<td>93.4%</td>
<td>-</td>
<td>40.9±5%</td>
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<tr>
<td>Female</td>
<td>43</td>
<td>43.5%</td>
<td>46.5738%</td>
<td>38.1±2%</td>
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<tr>
<td>Male</td>
<td>49</td>
<td>49.9%</td>
<td>53.426%</td>
<td>42.7±3%</td>
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9
Table 3: Possible factor associated with disease activation and inversion of S.G.Ch.P. into DP

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<tr>
<th>Sex</th>
<th>Number of DP patients according to sex</th>
<th>No. of patient per sex</th>
<th>Total factor number</th>
<th>% of patient number out of total factor No. according to sex</th>
<th>% of factor No. out of female DP (45 female)</th>
<th>% of factor No. out of male DP (21 male)</th>
<th>% of factor No./sex out of DP total No.</th>
<th>% of total factor No. out of DP total patient</th>
<th>t-test</th>
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<tr>
<td>Psycho logic factor</td>
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<tr>
<td>M</td>
<td>21</td>
<td>8</td>
<td>26</td>
<td>30.769%</td>
<td>40%</td>
<td>12.12%</td>
<td>39.39%</td>
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<td>69.23%</td>
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<tr>
<td>M</td>
<td>21</td>
<td>7</td>
<td>18</td>
<td>38.89%</td>
<td>24.44%</td>
<td>1.606%</td>
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<td>9</td>
<td>100%</td>
<td>20%</td>
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<td>13.636%</td>
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<td>Atherosclerotic factor</td>
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<tr>
<td>M</td>
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<td>8</td>
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<td>8.89%</td>
<td>19.05%</td>
<td>6.06%</td>
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<tr>
<td>Behavioral factor</td>
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<tr>
<td>M</td>
<td>21</td>
<td>2</td>
<td>5</td>
<td>40.0%</td>
<td>6.67%</td>
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<td>3.03%</td>
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Psychological factor: Restraint stress and depression.
Atherosclerotic factor: High total cholesterol, HDL, LDL.