

Original

Aggressive periodontitis presents a higher degree of bilateral symmetry in comparison with chronic periodontitis

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Abstract: The purpose of this cross-sectional study was to evaluate the occurrence of bilateral symmetry in the distribution of clinical parameters in subjects with generalized aggressive periodontitis (GAP) and severe chronic periodontitis (SCP). The sample comprised 53 subjects with GAP and 33 with SCP. Probing depth (PD) and clinical attachment loss (CAL) were recorded from both buccal and lingual interproximal sites of incisors and molars. The symmetry of periodontal destruction was analyzed in terms of intraclass coefficient correlations (ICC) for pairs of contralateral sites at which PD and/or CAL was ≥ 5 mm at one of the sites of the subjects in each group. GAP patients had a higher proportion of both PD and CAL ≥ 5 mm and also a higher mean proportion of subjects having PD and/or CAL ≥ 5 mm at one or both sites. The GAP group had 20 pairs of contralateral sites with PD (ICC = 0.22-0.63) and 26 pairs with CAL (ICC = 0.20-0.63), the correlation being statistically significant, while the SCP group had only 2 pairs (ICC = 0.36-0.48) with PD and 5 pairs with CAL (ICC = 0.33-0.58) showing a significant correlation. It can be concluded that GAP shows more symmetric periodontal destruction than SCP.

Keywords: aggressive periodontitis; chronic periodontitis; diagnosis; epidemiology; periodontal attachment loss.

Introduction

Periodontitis is a chronic inflammatory disease that affects the tooth-supporting structures, leading to loss of bone support and ultimately tooth loss. It can be either chronic or aggressive, and the diagnosis is determined using a combination of anamnesis, and clinical and radiographic findings (1). Aggressive periodontitis is characterized by rapid disease progression and severe periodontal destruction in fairly young individuals (2), whereas chronic periodontitis has a much slower progression rate. Despite these differences, both diseases can present in more severe forms and lead to tooth loss and edentulism (3). According to Albandar (1), aggressive periodontitis is characterized by early onset (before 25 years of age), involvement of several teeth with a distinct pattern of bone and attachment loss, radiographic vertical bone loss on the interproximal surfaces of posterior teeth or horizontal bone loss in advanced cases, with bilateral symmetry. The disease initiates on the interproximal surfaces of permanent first molars and incisors, which explains the advanced loss of periodontal support in these areas. Other criteria include rapid progression, lack of systemic involvement compromising the immune response, strong familial aggregation, suggesting a genetic role in onset of the disease (4), and involvement of specific bacterial species (5,6).

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Periodontal disease is highly prevalent and one of the main causes of tooth loss in the United States and other countries. According to the National Health and Nutrition Examination Survey (NHANES) 2009-2012, an estimated 46% of adults (approximately 140 million) aged 30 years or older in the United States have periodontitis, 8.9% having more severe disease and 37.1% mild to moderate disease (7). A systematic review on the epidemiology of aggressive periodontitis found a disease prevalence of between 1% and 5% in African populations, whereas the prevalence in Caucasians from Europe or North America was between 0.1% and 0.5%. Among Hispanics (0.5-1%) and African Americans (2.6%) the prevalence was higher in North America and from 0.2% to 1% in Asia. In South America, disease prevalence varied from 0.3% to 2% and was dependent on ethnicity (8).

Symmetry is a property whereby an object shows invariant transformation. Different concepts are available when referring to symmetry, and it is found in mathematics, art, architecture and other fields. Symmetric behavior is frequently observed in some health conditions, such as pneumonia (9), caries (10), tooth loss (11,12). A few studies have evaluated the similarities between clinical parameters of contralateral sites in individuals with aggressive and chronic periodontitis (13-18). In a study of juvenile periodontitis, which is now referred to as aggressive periodontitis (19), Horman and Frandsen (13) found a "mirror-like" pattern in the distribution of bone loss between the right and left sides of the arch. Burmeister et al. also reported contralateral symmetry of interdental periodontal attachment loss in subjects with this disease (14). A bilateral symmetric pattern among patients with chronic periodontitis has also been demonstrated using clinical and radiographic approaches (15-18).

It is important to determine whether chronic periodontitis and aggressive periodontitis show a specific and symmetric site distribution pattern, which would have the potential to facilitate diagnosis and implementation of epidemiological studies. The aim of this cross-sectional study was to evaluate the occurrence of bilateral symmetry in the distribution pattern of clinical parameters in individuals with generalized aggressive and severe chronic periodontitis.

Materials and Methods

Participants

Fifty-three individuals with generalized aggressive periodontitis and 33 with severe chronic periodontitis were selected from a pool of patients seeking treatment

at the Periodontics Clinics of the School of Pharmacy, Dentistry and Nursing, and the Dental School in Sobral, Federal University of Ceara, Brazil. These patients had been enrolled previously in a study conducted to evaluate the presence of *Aggregatibacter actinomycetemcomitans* and its leucotoxicity among family members (5). Generalized aggressive periodontitis (GAP) was diagnosed following the criteria recommended by the American Academy of Periodontology (AAP) (19): familial aggregation, rapid attachment loss and bone destruction, age at onset and clinical presentation as generalized interproximal attachment loss affecting three permanent teeth besides incisors and first molars. Individuals with severe chronic periodontitis (SCP) were diagnosed following the Centers for Disease Control and Prevention (CDC) and AAP case definitions (20). It was defined as having ≥ 2 interproximal sites with a clinical attachment loss (CAL) of ≥ 6 mm (not on the same tooth) and 1 or more interproximal site(s) with a probing depth (PD) of ≥ 5 mm.

The research protocol was approved by the Ethics Committee, Federal University of Ceara, Brazil (#20/08, 03/14/2008). All participants were informed about the purpose of the study and provided written consent.

Inclusion criteria

Inclusion criteria included a diagnosis of generalized aggressive periodontitis or severe chronic periodontitis based on the established criteria. Other inclusion criteria comprised the presence of at least 20 teeth, no periodontal treatment in the previous 6 months, no antibiotic therapy in the previous 3 months, absence of systemic alterations that could affect periodontal status, no medications associated with gingival enlargement, or pregnant or lactating women.

Variables and data source

All individuals were examined and PD (measured from the gingival margin to the bottom of the pocket), and CAL (measured from the cemento-enamel junction to the bottom of the pocket) were recorded. One calibrated examiner performed all measurements with a manual periodontal probe (UNC-15, Trinity Commerce and Industry Ltd., São Paulo, Brazil). For reproducibility analysis, duplicate measurements were performed on two occasions, one prior to initiating the study, and a second during the study. Twelve individuals diagnosed with chronic and aggressive periodontitis were evaluated twice, with a three-day interval. Data were analyzed and the Intraclass correlation coefficient (ICC) was 0.88 and 0.83 for PD and CAL measurements, respectively.

Table 1 Description and comparison of the clinical characteristics of the subjects

	Generalized aggressive periodontitis (<i>n</i> = 53)	Severe chronic periodontitis (<i>n</i> = 33)
Age (\pm SD) Years	29.6 \pm 4.8 [†]	39.6 \pm 8.1
Female (%)	33 (62.3)	19 (57.6)
Tooth count	25.7 \pm 2.0	25.6 \pm 1.3
Mean (\pm SD) PD [§] mm	4.2 \pm 2.1 [†]	3.3 \pm 1.7
Mean (\pm SD) CAL [§] mm	4.9 \pm 2.6 [†]	3.8 \pm 2.1
% Sites [§] Mean (\pm SD) - PD \geq 5 mm	37.2 \pm 18.4 [†]	20.8 \pm 12.5
% Sites [§] Mean (\pm SD) - CAL \geq 5 mm	43.3 \pm 19.2 [†]	26.6 \pm 16.3
% of subjects with PD \geq 5 mm [§] in at least one contralateral site Mean (\pm SD)	45.1 \pm 16.1 [†]	25.9 \pm 16.6
95% Confidence Interval	38.8-51.4	19.5-32.4
% of subjects with CAL \geq 5 mm [§] in at least one contralateral site Mean (\pm SD)	52.6 \pm 13.8 [†]	31.9 \pm 15.2
95% Confidence interval	47.2-58.0	25.9-37.8
% of subjects with PD \geq 5 mm [§] in both contralateral sites Mean (\pm SD)	13.9 \pm 8.5 [†]	7.7 \pm 8.0
95% Confidence interval	10.6-17.3	4.7-10.9
% of subjects with CAL \geq 5 mm [§] in both contralateral sites Mean (\pm SD)	32.3 \pm 11.1 [†]	12.5 \pm 9.8
95% Confidence interval	28.0-36.6	8.7-16.3

[†]Student's *t*-test, *P* < 0.05; [§]interproximal sites of incisors and molars; SD: standard deviation; PD: probing depth; CAL: clinical attachment loss

Clinical parameters were evaluated at six sites per tooth (mesiobuccal, buccal, distobuccal, mesiolingual, lingual and distolingual) for all teeth, except third molars. Only interproximal sites were considered for evaluation of bilateral symmetry. Both mesial and distal sites at incisors and first molars were considered. For second molars, only the mesial surfaces were evaluated in an effort to avoid gingival excesses at the distal sites due to the presence of third molars. The Fédération Dentaire Internationale (FDI) tooth numbering system was used to present the data.

Statistical analysis

Data regarding age, tooth count, mean PD and CAL, mean proportions of sites with PD \geq 5 mm and CAL \geq 5 mm and the mean proportions of subjects with at least one site showing PD \geq 5 mm and/or CAL \geq 5 mm were compared by independent's *t*-test. In order to determine whether periodontal destruction was present at both contralateral sites, the mean proportions of subjects with both sites showing PD \geq 5 mm and/or CAL \geq 5 mm were also analyzed using the same statistical test.

Bilateral symmetry of disease was evaluated by comparing PD and CAL at the site level. In order to compare sites at which the disease was present, analyses were performed only when at least one contralateral site showed PD and/or CAL \geq 5 mm. The intraclass correlation coefficient (ICC) was applied to evaluate the correlation between contralateral sites within the same group. Statistical significance was set at *P* < 0.05 and the analysis was performed with SPSS for Windows, version 17 (IBM SPSS, Armonk, NY, USA). The ICC was only applied when at least 16 individuals in each group had

at least one contralateral site that met the predetermined clinical criteria (PD and/or CAL \geq 5 mm). This sample size calculation (MedCalc, MedCalc Software, Ostend, WF, Belgium) was performed to provide 80% power with α = 0.05 and substantial ICC (0.65) (21).

Results

Data on the demographics and clinical characteristics of the studied population are presented in Table 1. Generalized aggressive periodontitis showed a higher degree of bylateral periodontal destruction than severe chronic periodontitis. The comparison of mean PD and CAL \geq 5 mm at interproximal sites and the mean proportion of such sites between groups showed statistically significant differences between them. When the proportion of subjects with at least one of the evaluated pairs of sites with PD \geq 5 mm and CAL \geq 5 mm, as well as the proportion of subjects with both contralateral sites with PD \geq 5 mm and CAL \geq 5 mm were compared, statistically significant differences were also shown.

Table 2 presents the number of individuals with paired contralateral sites showing PD and CAL \geq 5 mm in at least one of the two sites. ICC for PD and CAL along with *P* values are also presented. This evaluation was only performed when sites with PD and/or CAL \geq 5 mm could be paired with contralateral sites in at least 16 of the individuals in each group.

In the aggressive periodontitis group, 24 pairs of PD values were analyzed; 20 pairs showed a statistically significant correlation of between 0.22 and 0.63. When CAL was evaluated, the aggressive periodontitis group included 27 pairs of sites eligible for correlation analysis, and 26 of these pairs presented a statistically significant

Table 2 Pairs of contralateral interproximal sites and correlation values between PD and CAL parameters in the generalized aggressive periodontitis group

Sites	Generalized aggressive periodontitis				Severe chronic periodontitis			
	PD		CAL		PD		CAL	
	Pairs	ICC	Pairs	ICC	Pairs	ICC	Pairs	ICC
17-27 MB	34	0.22	41	0.24	20	NS	21	NS
16-26 DB	37	0.36	39	0.49	20	NS	20	0.35
16-26 MB	38	0.28	39	0.29	17	NS	19	0.33
12-22 DB	24	0.28	28	0.36	12	NE	12	NE
12-22 MB	31	0.36	35	0.35	9	NE	13	NE
11-21 DB	30	0.24	37	0.27	6	NE	11	NE
11-21 MB	25	NS	29	0.39	7	NE	9	NE
17-27 ML	37	0.38	39	0.30	17	0.37	18	0.48
16-26 DL	38	0.27	39	0.41	17	0.48	17	0.58
16-26 ML	34	0.42	34	0.52	17	NS	18	0.38
12-22 DL	16	0.36	20	0.42	5	NE	7	NE
12-22 ML	23	0.42	26	0.28	8	NE	9	NE
11-21 DL	24	0.39	30	0.24	4	NE	8	NE
11-21 ML	25	0.22	26	0.51	7	NE	9	NE
47-37 ML	24	0.47	26	0.42	12	NE	14	NE
46-36 DL	25	NS	26	0.23	5	NE	7	NE
46-36 ML	25	0.63	27	0.63	4	NE	4	NE
42-32 DL	13	NE	15	NE	4	NE	5	NE
42-32 ML	10	NE	19	NS	4	NE	6	NE
41-31 DL	12	NE	17	0.20	4	NE	9	NE
41-31 ML	8	NE	20	0.51	4	NE	5	NE
47-37 MB	19	0.33	22	0.40	7	NE	8	NE
46-36 DB	24	NS	24	0.29	20	NE	5	NE
46-36 MB	21	0.48	22	0.51	20	NE	5	NE
42-32 DB	22	0.22	26	0.24	17	NE	9	NE
42-32 MB	17	0.36	25	0.49	12	NE	8	NE
41-31 DB	18	0.28	24	0.29	9	NE	9	NE
41-31 MB	16	0.28	26	0.36	6	NE	10	NE

ICC: intraclass correlation coefficient; Values in bold type are significant; NE: not evaluated; NS: not statistically significant; MB: mesiobuccal; DB: distobuccal; ML: mesiolingual; DL: distolingual

correlation of between 0.20 and 0.63. The highest correlations were obtained from mesiolingual sites of teeth 36 and 46 for both PD and CAL (i.e. 0.63). The mean PD and CAL for contralateral sites of aggressive periodontitis patients, evaluated by ICC, are presented in Fig. 1.

In the severe chronic periodontitis group, only 6 pairs of sites were included for both PD and CAL analyses. Significant correlations for PD were found in 2 pairs (ICC = 0.36 and 0.48), and for CAL in 5 pairs (ICC between 0.33 and 0.58).

Discussion

The present study compared the symmetry of periodontal parameters in chronic and aggressive periodontitis. Cross-sectional analysis was performed because this approach allows an understanding of disease distribution in a determined time frame, which is of interest for both screening, and also epidemiological and clinical approaches for clarifying the occurrence of disease. Interproximal sites were evaluated with the aim of identi-

fying symmetry in the distribution of clinical parameters. These sites were chosen because they have also been proposed as clinical criteria for classifying both aggressive and chronic periodontitis and their severity (19,20). Interproximal sites are more reliable for detection of periodontal disease since measurements performed at buccal/lingual sites may be influenced by abrasion from brushing and gingival recession, which can lead to over-estimation of disease (20).

Site-specific analysis was performed using an ICC approach, and only applied when at least 16 of the subjects in each group had PD and/or CAL ≥ 5 mm at one of the contralateral sites. The use of the ICC makes it possible to determine the degree to which two measurements tend to be similar in a clinically relevant manner. Thus, to verify symmetry among continuous data, this is one of the recommended approaches (22). Among a total of 28 pairs of sites, 24 presented this criterion for PD and 27 for CAL in subjects with aggressive periodontitis, and a statistically significant difference was observed at 20 and

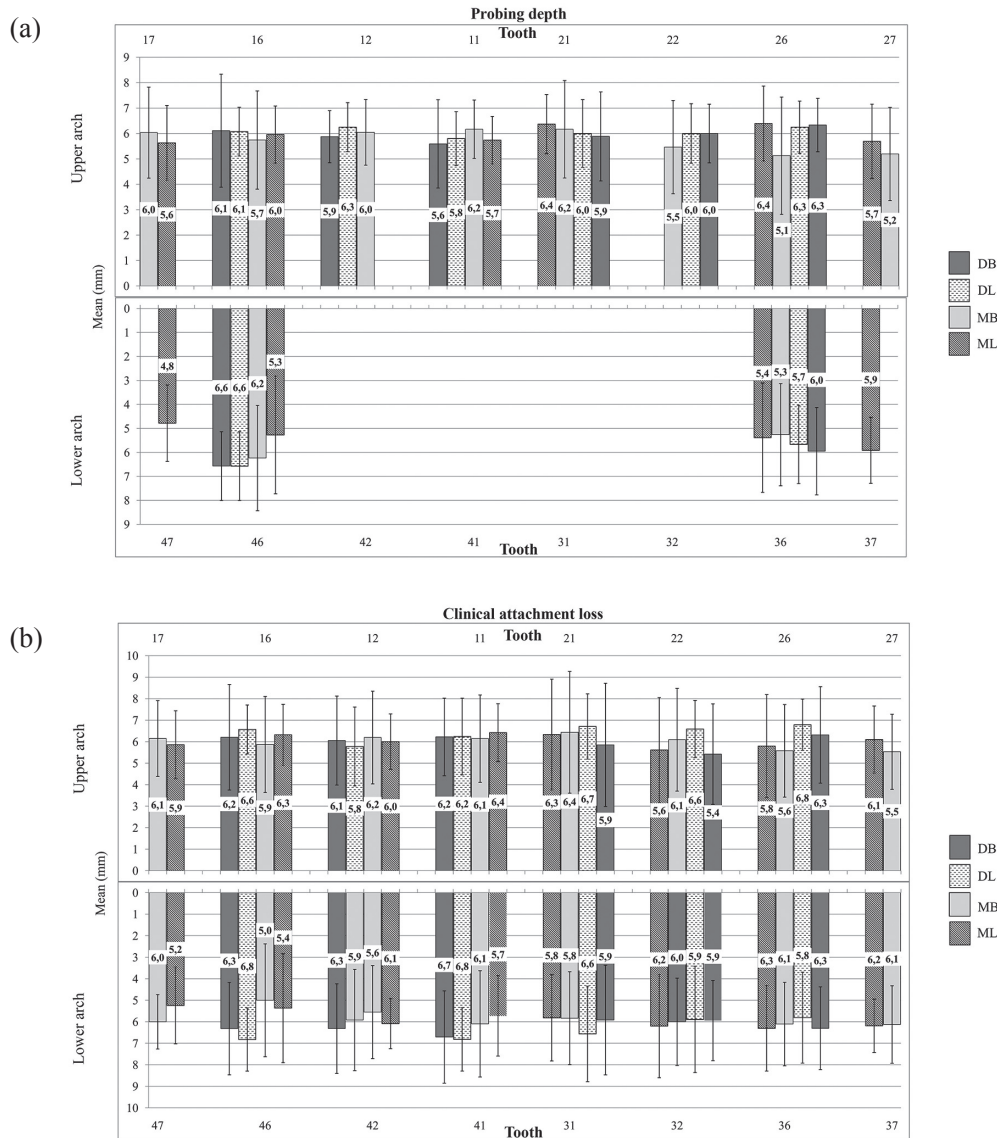


Fig. 1 Mean probing depth (a) and clinical attachment loss (b) for interproximal surfaces with at least one site ≥ 5 mm in patients with generalized aggressive periodontitis.

26 sites, respectively. Contralateral sites in the aggressive periodontitis group presented ICC values of between 0.22 and 0.63 for PD. Clinical attachment loss ICC values ranged from 0.20 to 0.63. The highest correlations for both PD and CAL were observed at mesiolingual sites of mandibular first molars. Overall, among the 46 pairs of sites that showed significant correlations, 28 presented a fair correlation (0.2 to 0.4), 16 a moderate correlation (0.4 to 0.6), and two of them showing the highest ICC for both PD and CAL were considered to have a substantial correlation (0.6 to 0.8), as described by Landis and Koch (21). In the severe chronic periodontitis group, only 7 pairs of sites presented a significant correlation. Fair and moderate correlations were found for four and three of the analyzed sites, respectively (21). The mean propor-

tion of sites where PD and/or CAL ≥ 5 mm was evident for at least one of them was also greater for aggressive than for chronic periodontitis. These results show that aggressive periodontitis is characterized by more severe periodontal destruction as well as a more symmetric pattern than chronic periodontitis. However, in patients with chronic periodontitis, the mean proportions of sites showing PD and CAL ≥ 5 mm were 20.8% and 26.6%, respectively. Thus, although the disease is present, the periodontal destruction is distributed more randomly.

Around 40% of the interproximal sites evaluated presented moderate to strong correlations with the respective contralateral site in patients with generalized aggressive periodontitis, compared with only 11% (three sites) in patients with severe chronic periodontitis. These

results can be attributed to the main differences between aggressive and chronic periodontitis; onset at an early age along with rapid destruction at specific sites such as the first molars and incisors (19), could have created a more symmetric destruction pattern in comparison with the more diverse pattern seen in chronic periodontitis. The symmetric distribution pattern of aggressive periodontitis strengthens the concept that its etiology is associated with not only local factors but also genetic or environmental systemic factors, which together may contribute to the bilateral symmetry of the disease (23,24). It has been suggested that the destruction can be due to the eruption pattern, in which the first molars are the first permanent teeth to erupt (23). In accordance with this, the greatest correlations ($ICC \geq 0.5$) found in one-fourth of the patients with aggressive periodontitis were for the first molars and incisors. One of the theories used to explain bilateral symmetry in aggressive periodontitis is that genetic or environmental systemic disorders occur along with the extensive systemic and metabolic changes produced during puberty, and that these may disappear with time. The teeth affected for the longest period of time would exhibit the most severe periodontal lesions (24). Furthermore, the prevalence of periodontal pockets was associated with the dates of tooth eruption, and the longer the teeth are in the mouth, the greater the chance of pocketing (25).

The results of this study suggest that, especially in aggressive periodontitis, the presence of a diseased site can be an indication that a contralateral site may also be involved. Similar results have been reported by Horman and Frandsen (13) and Burmeister et al. (14), in which the distribution of clinical parameters in subjects with aggressive periodontitis, reported as juvenile periodontitis, was evaluated. Analyzing 156 patients Horman and Frandsen (13) observed a pattern of bone loss, referred to as a “mirror effect”, between the teeth of the right and left jaws. Burmeister et al. found symmetry between attachment loss at contralateral sites in patients with aggressive and also severe periodontitis. However, the authors’ criteria used to classify severe periodontitis (generalized pattern of severe destruction with attachment loss of at least 5 mm at 8 or more teeth, at least 3 of which were not first molars or incisors) and the patients’ mean age (25.4 years) were compatible with what is now known as generalized aggressive periodontitis (19).

On the other hand, unlike the results of the present study, bilateral symmetry has also been found in chronic periodontitis (15-18). Rise et al. reported a high PD correlation (97%) between the left and right sides of periodontal sites (15). Based on clinical findings,

Mombelli and Meier (16) reported symmetry between clinical parameters at contralateral sites at the tooth and quadrant levels. Darby et al. (18) also found a significant right-left distribution in all clinical parameters evaluated in individuals with severe chronic periodontitis. Radiographic bone loss from panoramic radiographs between contralateral sites was analyzed and ICC values between 0.53 and 0.79 were found, the highest values being observed at the mesial sites of teeth 17 and 27 (17). Nevertheless, the criteria used to perform symmetry analysis by these authors (15-18) allowed some different interpretations, such as symmetry between sites with no periodontal destruction. In the present study, the correlation was made when the disease was present, PD and/or $CAL \geq 5$ mm, in at least one of the contralateral sites. This criterion avoided comparison between shallow sites against each other, which can possibly yield high correlations. Despite the high correlation reported, Rise et al. evaluated patients with a mean PD of 2.4 mm, which may not be clinically relevant (15). Darby et al. suggested that bilateral symmetry may exist after they found no differences between the mean PD values for most of the contralateral sites they evaluated, except for the maxillary lateral incisors and mandibular second molars (18). Similar results were obtained in the present study when the mean PD and CAL were evaluated (data not shown). However, when the ICC—a reliable statistic test—was applied, only a few contralateral sites showed a significant correlation. In the study by Persson et al. the patients were receiving supportive periodontal therapy, which suggests that the observed correlations were due to the fact that the patients had already been treated, and their affected sites had become shallower over time, and then became correlated (17). Mombelli and Meier detected PD correlations between both sides, but the regression coefficients of determination were low, which meant that between only 14% and 26% of PD variability on the right side could be explained by the respective value on the left side (16).

Chronic periodontitis has been reported more frequently in older subjects (7,26), whereas aggressive periodontitis shows characteristic occurrence in young subjects aged less than 30 years (19,27,28). These features are in accord with the present results, as older subjects with an average age of 39.6 years presented with chronic periodontitis. The average age of patients with aggressive periodontitis was 29.6 years. The early age at onset characteristic of aggressive periodontitis probably indicates a higher rate of disease progression, leading to severe destruction of periodontal tissues in a shorter period of time relative to chronic periodontitis. It is estimated that progression of

bone loss in aggressive periodontitis is three to four times faster than in chronic periodontitis (1). Even though the subjects with chronic periodontitis were older than those with aggressive periodontitis, the average age was lower (39.6 years) than that in the studies by Persson et al. (17) and Darby et al. (18). Despite the above shortcomings, these two studies suggested a contralateral symmetry in the clinical and radiographic parameters of subjects with chronic periodontitis, and the average subject age was 47.5 and 55.5 years, respectively. However, inclusion of older subjects would have been a study limitation, as tooth loss increases with age, and this specific sample population has a high prevalence of tooth loss (29). One of the inclusion criteria of this study was the presence of at least 20 teeth, and this would have limited the sample size as well as the age of patients in the chronic periodontitis group. Data from the Brazilian National Health Survey has shown that 11% of the Brazilian population (16 million people) are edentulous, and that 23% have lost at least 13 teeth. In the state of Ceara, where this study was conducted, these numbers are higher, 29% of the population over 18 years of age having lost 13 or more teeth. On the other hand, as mentioned before, unlike the former studies, a strict criterion was chosen in order to analyze symmetry, i.e., at least one contralateral site with PD and/or CAL \geq 5 mm. Thus, even if the patients were older and more periodontal destruction had been observed, use of the above criterion would not have confirmed whether the disease distribution was symmetric, as observed in patients with generalized aggressive periodontitis.

The findings of the present study should be viewed in consideration of its limitations and strengths. Generalized aggressive periodontitis was diagnosed on the basis of strict inclusion criteria that had been employed in a previous study for microbial analysis and to examine the familial distribution of the disease (5). The criteria used to analyze data were of clinical relevance; only sites with clinical signs of periodontitis were included. The novelty of this study resides in comparing individuals with chronic and aggressive periodontitis from two urban areas with comparable socioeconomic and cultural conditions who sought dental treatment at a reference dental school in the area. This offered the potential for a good degree of comparability in terms of the demographics of the included individuals. The main limitation was the small sample size of patients with chronic periodontitis. However, was justified due to the strict inclusion criteria regarding tooth count and the inclusion of patients with the most severe chronic periodontitis, accounting for the lowest prevalence (around 9% of the population) in large epidemiological studies (7,26).

As our results showed that patients with aggressive periodontitis had a higher proportion of sites with periodontal destruction on both sides, and that these sites presented significant moderate and strong correlations between their clinical parameters, it can be concluded that generalized aggressive periodontitis was characterized by more symmetric periodontal destruction than severe chronic periodontitis. This finding may contribute to a better understanding of the disease and act as a guide for diagnosis in future epidemiological studies. Furthermore, during diagnosis and treatment planning of aggressive periodontitis, it is important to pay attention to patients who have sites showing periodontal destruction on one side and lacking such destruction on the other, especially for incisors and molars. Along with major signs of aggressive periodontitis such as rapid attachment loss and bone destruction, and familial aggregation, it can be suggested that such patients have an increased risk of developing the disease. Therefore, preventive strategies such as early diagnosis and treatment could be implemented for these patients in order to prevent the breakdown of those sites.

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Conflict of interest

None declared.

References

1. Albandar JM (2014) Aggressive periodontitis: case definition and diagnostic criteria. *Periodontol* 2000 65, 13-26.
2. Meng H, Xu L, Li Q, Han J, Zhao Y (2007) Determinants of host susceptibility in aggressive periodontitis. *Periodontol* 2000 43, 133-159.
3. Schatzle M, Faddy MJ, Cullinan MP, Seymour GJ, Lang NP, Burgin W et al. (2009) The clinical course of chronic periodontitis: V. Predictive factors in periodontal disease. *J Clin Periodontol* 36, 365-371.
4. Kinane DF, Hart TC (2003) Genes and gene polymorphisms associated with periodontal disease. *Crit Rev Oral Biol Med* 14, 430-449.
5. Silveira VR, Nogueira MV, Nogueira NA, Lima V, Furlaneto FA, Rego RO (2013) Leukotoxicity of *Aggregatibacter actinomycetemcomitans* in generalized aggressive periodontitis in Brazilians and their family members. *J Appl Oral Sci* 21, 430-436.
6. Haubek D, Johansson A (2014) Pathogenicity of the highly leukotoxic JP2 clone of *Aggregatibacter actinomycetemcomitans* and its geographic dissemination and role in aggressive periodontitis. *J Oral Microbiol* 6, doi: 10.3402/jom.v6.23980.
7. Eke PI, Dye BA, Wei L, Slade GD, Thornton-Evans GO,

- Borgnakke WS et al. (2015) Update on prevalence of periodontitis in adults in the United States: NHANES 2009 to 2012. *J Periodontol* 86, 611-622.
8. Susin C, Haas AN, Albandar JM (2014) Epidemiology and demographics of aggressive periodontitis. *Periodontol* 2000 65, 27-45.
9. Kang EY, Staples CA, McGuinness G, Primack SL, Muller NL (1996) Detection and differential diagnosis of pulmonary infections and tumors in patients with AIDS: value of chest radiography versus CT. *AJR Am J Roentgenol* 166, 15-19.
10. Jackson D, Fairpo CG, Burch PR (1973) Distribution of symmetric and asymmetric patterns of caries attack in human permanent maxillary incisor teeth: genetic implications. *Arch Oral Biol* 18, 189-195.
11. Hirschfeld L, Wasserman B (1978) A long-term survey of tooth loss in 600 treated periodontal patients. *J Periodontol* 49, 225-237.
12. McFall WT Jr (1982) Tooth loss in 100 treated patients with periodontal disease. A long-term study. *J Periodontol* 53, 539-549.
13. Horman J, Frandsen A (1979) Juvenile periodontitis. Localization of bone loss in relation to age, sex, and teeth. *J Clin Periodontol* 6, 407-416.
14. Burmeister JA, Best AM, Palcanis KG, Caine FA, Ranney RR (1984) Localized juvenile periodontitis and generalized severe periodontitis: clinical findings. *J Clin Periodontol* 11, 181-192.
15. Rise J, Tollefsen T (1984) Reliability of plaque and periodontal measurements estimated by the internal consistency method. *Acta Odontol Scand* 42, 293-296.
16. Mombelli A, Meier C (2001) On the symmetry of periodontal disease. *J Clin Periodontol* 28, 741-745.
17. Persson RE, Tzannetou S, Feloutzis AG, Bragger U, Persson GR, Lang NP (2003) Comparison between panoramic and intra-oral radiographs for the assessment of alveolar bone levels in a periodontal maintenance population. *J Clin Periodontol* 30, 833-839.
18. Darby IB, Polster A, Gan JS, Guo Q, Henein N, Heredia A et al. (2012) Left-to-right distribution of periodontal disease. *Int J Dent Hyg* 10, 74-79.
19. Tonetti MS, Mombelli A (1999) Early-onset periodontitis. *Ann Periodontol* 4, 39-53.
20. Eke PI, Page RC, Wei L, Thornton-Evans G, Genco RJ (2012) Update of the case definitions for population-based surveillance of periodontitis. *J Periodontol* 83, 1449-1454.
21. Landis JR, Koch GG (1977) The measurement of observer agreement for categorical data. *Biometrics* 33, 159-174.
22. Koo TK, Li MY (2016) A guideline of selecting and reporting intraclass correlation coefficients for reliability research. *J Chiropr Med* 15, 155-163.
23. Kaslick RS, Chasens AI (1968) Periodontosis with periodontitis: a study involving young adult males. I. Review of the literature and incidence in a military population. *Oral Surg Oral Med Oral Pathol* 25, 305-326.
24. Kaslick RS, Chasens AI, Bressman E, Lazzara R, Egito J (1971) Investigation of periodontosis with periodontitis: ultramicroanalysis of gingival fluid, gross examination of the periodontal ligament and approach to treatment. *J Periodontol* 42, 428-434.
25. MacGregor ID, Sheiham A (1974) Patterns of periodontal pocketing in Western Nigerian populations. *J Periodontol* 45, 402-409.
26. Eke PI, Dye BA, Wei L, Thornton-Evans GO, Genco RJ (2012) Prevalence of periodontitis in adults in the United States: 2009 and 2010. *J Dent Res* 91, 914-920.
27. Albandar JM, Muranga MB, Rams TE (2002) Prevalence of aggressive periodontitis in school attendees in Uganda. *J Clin Periodontol* 29, 823-831.
28. Elamin AM, Skaug N, Ali RW, Bakken V, Albandar JM (2010) Ethnic disparities in the prevalence of periodontitis among high school students in Sudan. *J Periodontol* 81, 891-896.
29. Cardoso M, Balducci I, Telles DM, Lourenço EJ, Nogueira Júnior L (2016) Edentulism in Brazil: trends, projections and expectations until 2040. *Cien Saude Colet* 21, 1239-1246.