Original Article

Clinical and Microbiological Profiles of Aggressive and Chronic Periodontitis in Congolese Patients: A Cross-sectional Study

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ABSTRACT

Background: Chronic and aggressive periodontitis were the main forms of periodontitis according to the 1999 classification of periodontal diseases and conditions. Their profile in Congolese patients is still undescribed. **Aim:** The aim of this study was to compare the profile of chronic periodontitis (ChP) with that of aggressive periodontitis (AgP) in Congolese patients. **Materials and Methods:** Thirty-two patients with ChP and 20 with AgP who consulted the dental services at any of the four medical centers in Kinshasa, from April 2017 to April 2018, were enrolled in the cross-sectional study. All patients underwent a full mouth examination, including assessment of the probing pocket depth and clinical attachment level at six sites per tooth. Microbial samples were collected in the deepest pocket in the maxilla and the deepest pocket in the mandible. A deoxyribonucleic acid (DNA) analysis was performed using DNA strip technology. Fisher exact test, the chi-square test, the *t* test, and the Mann–Whitney test were used for the statistical analysis. **Results:** Patients with AgP were significantly younger than those with ChP ($P < 0.001$). There was no significant difference in the prevalence of *Porphyromonas gingivalis*, *Tannerella forsythia*, *Treponema denticola*, or *Prevotella intermedia* between the AgP and ChP groups ($P > 0.05$). *Aggregatibacter actinomycetemcomitans* was detected in 10% of cases in the AgP group and in none of those in the ChP group ($P = 0.143$). **Conclusion:** This study shows that the clinical profiles of ChP and AgP are similar in Congolese patients. There were no microbiological differences between these two forms of periodontitis.

Keywords: *Aggressive periodontitis, bacteria, chronic periodontitis, microbiological profile, periodontitis*

Introduction

Periodontitis is an inflammatory/biofilm-induced disease characterized by loss of the tissues supporting the teeth. Microbes within the dental biofilm initiate inflammation that can lead to breakdown of tissues in a susceptible host.[1]

According to the 1999 classification of periodontal diseases and conditions,[2] three primary forms of periodontitis are distinguished, of which aggressive periodontitis (AgP) and chronic periodontitis (ChP) are the most common. The difference between these two forms of periodontitis has been a matter of intense debate for the past two decades,[3] and studies distinguishing these forms are being published nowadays.[4-10] According to a recent workshop on the

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classification of periodontal and peri-implant diseases and conditions, there is currently insufficient evidence to consider AgP and ChP as two pathobiologically distinct diseases.[11] This has led to patients with the chronic and aggressive forms being grouped under a single category of "periodontitis,"[12] implying that research should continue to provide scientific evidence that can support or refute this framework. Fine *et al.*[13] have recently concluded that localized AgP differs in many ways from the far more common ChP and supported classifying it as a distinct form of periodontitis. Furthermore, comparing these two forms of periodontitis in Africa, where this disease is reported to be very common, may be of some interest.

The microbiota associated with periodontitis seems to vary markedly according to geographic location[14] and between different ethnic or racial groups.[15-17] According to Mayorga-Fayad *et al.*,[18] the evidence suggests that each country must establish its own oral microbiological profile to formulate adequate measures and more specific treatments.

Various studies have attempted to determine biological, clinical, and microbiological differences between AgP and ChP to facilitate the diagnosis and treatment of periodontal disease. A systematic review by Mombelli *et al.*[19] concluded that the presence or absence of the periopathogens assessed in their study could not discriminate between subjects with AgP and those with ChP. Another study analyzed the prevalence and levels of 51 species by checkerboard DNA-DNA hybridization and found few differences in microbial species between AgP and ChP but that *Porphyromonas gingivalis* and *Treponema denticola* were associated with ChP.[20]

Acquisition of additional microbial data at the population level would contribute to better clinical diagnosis and a better treatment plan.[20-22]

The objective of this study was to compare the clinical and microbiological profiles of AgP and ChP in Congolese patients.

Materials and Methods

Study design

The subjects of this cross-sectional study were selected from patients who attended for dental services at any of the four medical centers in Kinshasa, that is, the Biamba Marie Mutombo Hospital, University Clinic, Ngaliema Clinic, or Boyambi Clinic, between April 2017 and April 2018. The study protocol was approved by the ethics committee at the School of Public Health, University of Kinshasa (approval number ESP/015/2017) and conducted according to the tenets outlined in the Declaration of Helsinki and to the STROBE guidelines.

Selection of study population and clinical examination

The study included 52 patients (18 men and 34 women) of mean age 44 ± 18.5 (range, 14–72) years. Twenty patients had AgP and 32 had ChP. AgP and ChP were defined according to the criteria published by the American Academy of Periodontology.[2] The mean age was 25.2 ± 11.9 years in the AgP group and 55.7 ± 10.5 years in the ChP group. The sample size was determined according to the method described in Figure 1.

The study inclusion criteria were as follows: at least 20 teeth present, age 12 years or older, a probing pocket depth (PPD) of at least 5 mm and a clinical attachment level (CAL) of 3 mm for at least three teeth (at least one in the maxilla and another in the mandible), and a diagnosis of AgP or ChP. For AgP, at least two of the teeth involved were required be a first molar or incisor. Patients who had taken antibiotics during the previous 6 months and those were receiving or had received periodontal treatment during the previous 6 months were excluded, as were patients who were pregnant or lactating.

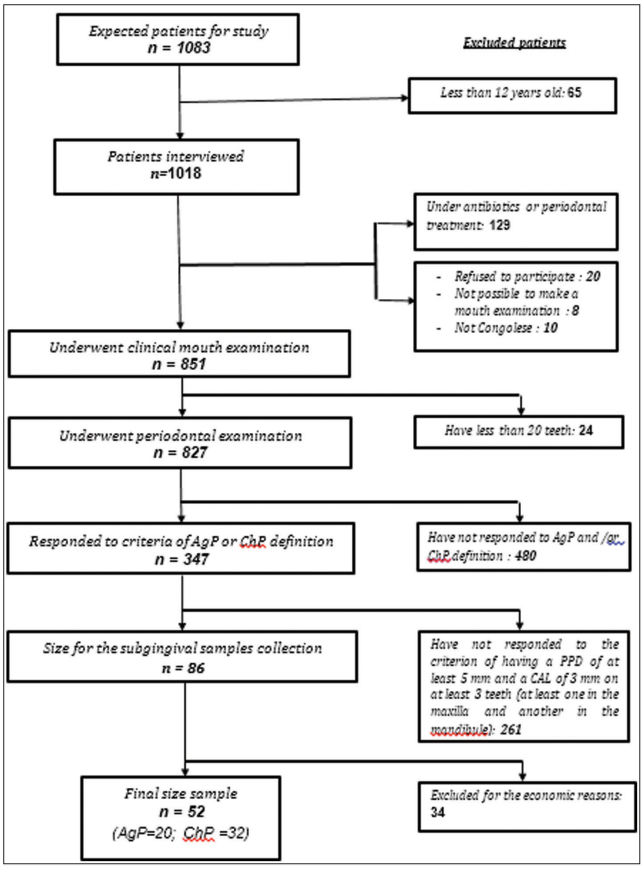

Figure 1: Flowchart of study sample. AgP = aggressive periodontitis, CAL = clinical attachment level, ChP = chronic periodontitis, PPD = probing pocket depth

A full mouth examination was performed using a PCP 10 periodontal probe (Hu-Friedy, Chicago, Illinois). All teeth, except for the third molars, were examined. Teeth that were erupting, supernumerary, or partially impacted were also excluded. The PPD and CAL were assessed at six sites (mesiobuccal, buccal, distobuccal, mesiolingual, lingual, and distolingual) per tooth. The plaque index and bleeding on probing (BoP) were also recorded. All measurements were performed by the same trained examiner (EKK); 20 patients were examined and reexamined 1 h later. The κ coefficients calculated for PPD and CAL were 0.82 and 0.84, respectively.

Microbial sampling

Subgingival biofilm samples were collected using a previously described method.[16] They were obtained from the two deepest pockets per patient (one in the upper arch and another in the lower arch) determined during the clinical examination.

The sampled site was isolated using sterile cotton rolls. The supragingival plaque was first removed with sterile gauze and then a sterile Gracey curette. A sterile paper point was introduced into the pocket and left in place for 10 s. The paper point was then removed and placed immediately in an Eppendorf plastic tube. The tube was then closed and placed in the micro-IDent kit provided by Biocentric Laboratory (Hain Lifescience, Nehren, Germany).

DNA analysis

The plaque samples were sent to France where polymerase chain reaction (PCR) analysis was performed by the Biocentric Laboratory (Bandol, France). The technique used in this laboratory is based on semiquantitative PCR using DNA-strip technology (Hain Lifescience). The threshold for detection of bacteria is 10^3 for *Aggregatibacter actinomycetemcomitans* and 10^4 for *P. gingivalis*, *Tannerella forsythia*, *T. denticola*, and *Prevotella intermedia*. DNA was extracted from the specimen by thermolysis at 95°C. The DNA was selectively amplified by subsequent PCR. The amplified DNA was denaturized and then hybridized with specific probes on strip.[23]

Statistical analysis

The statistical analysis was performed using Statistical Package for Social Sciences software, version 20.0 (IBM, Armonk, New York). Comparisons between AgP and ChP were performed using Fisher exact test, the *t* test, the chi-square test, and the Mann–Whitney test.

Results

The mean patient age was significantly lower in the AgP group than that in the ChP group (25.2 ± 11.9 years vs. 55.7 ± 10.5 years; $P < 0.001$). Furthermore, there was no significant difference in patient sex or educational level between the AgP and ChP groups ($P = 0.065$ and $P = 0.375$, respectively). All current smokers had ChP but there was no difference in smoking status

Table 1: Demographic/lifestyle characteristics of patients with aggressive and chronic periodontitis

Characteristic	AgP (*n* = 20)	ChP (*n* = 32)	*df*	*P* value	95% CI	Power (%)
Age, years	25.2 ± 11.9	55.7 ± 10.5	50	<0.001[b]	(-30.804; -24.171)	100
Sex						
Male	50.0	25.0	1	0.065[c]	(0.916–9.830)	45.5
Female	50.0	75.5				
Smoking status						
Smoker	0.0	12.5	1	0.151[d]	-	37.2
Nonsmoker	100	87.5				
Previous dental care						
No	80.0	43.8	1	0.020[d]	(1.403–18.858)	73
Yes	20.0	56.2				
Study level[a]						
Low	50.0	62.5	1	0.375[c]	(0.194–1.860)	14.5
High	50.0	37.5				

AgP = aggressive periodontitis, ChP = chronic periodontitis, df = degree of freedom, CI = confidence interval

The data are presented as the mean and standard deviation or as the percentage

[a]Educational level (low: primary and secondary school, high: undergraduate or higher)

[b]*t* test

[c]Pearson chi-square test

[d]Fisher exact test

between the two study groups (P = 0.151) [Table 1]. There was also no difference in the plaque index or number of missing teeth between the two clinical forms of periodontitis. BoP was greater in the AgP group than that in the ChP group (P = 0.025) [Table 2].

Table 3 compares the AgP and ChP groups according to the presence or absence of periopathogens, that is, *P. gingivalis, T. forsythia*, *T. denticola*, and *P. intermedia*, and found no significant difference in their distribution in the plaque samples (P > 0.05). *A. actinomycetemcomitans* was detected in 10% of

Table 2: Clinical characteristics of aggressive and chronic periodontitis

Characteristic	AgP (n = 20)	ChP (n = 32)	P value[a]	95% CI	Power (%)
Plaque index[b]	0.90 ± 0.15	0.92 ± 0.78	0.694	0.000 (0.000–0.000)	94.9
Bleeding on probing[b]	46.78 ± 25.26	30.44 ± 18.62	0.025	19.000 (0.000–34.000)	31.7
Missing teeth	0.44 ± 0.97	0.69 ± 155	0.646	0.000 (0.000–0.000)	9
Teeth with PPD 3 mm, n[b]	5.11 ± 2.23	1.75 ± 1.37	<0.001	4 (2.000–5.000)	100
Sites with PPD 3 mm, n[b]	6.00 ± 2.54	2.13 ± 1.72	<0.001	4 (3.000–6.000)	100
Teeth with PPD ≥4 and <6 mm, n[b]	5.56 ± 2.40	7.19 ± 272	0.034	-2 (-3.000–0.000)	51.4
Sites with PPD ≥4 and <6 mm, n[b]	8.40 ± 3.59	9.56 ± 3.75	0.370	1 (-4.000–1.000)	17.1
Teeth with PPD ≥6 mm, n[b]	1.15 ± 2.25	1.44 ± 124	0.027	-1 (-1.000–0.000)	8.5
Sites with PPD ≥6 mm, n[b]	2.30 ± 4.69	1.81 ± 1.96	0.024	-1 (-2.000–0.000)	7.6
Teeth with CAL 2 mm, n[b]	4.50 ± 2.65	1.75 ± 1.46	<0.001	3 (1.000–5.000)	99.2
Sites with CAL 2 mm, n[b]	5.33 ± 3.08	2.65 ± 3.15	0.004	3.5 (1.000–6.000)	78
Teeth with CAL ≥3 and <5 mm, n[b]	4.80 ± 2.28	6.44 ± 2.90	0.037	-2 (-3.000–0.000)	49.6
Sites with CAL ≥3 and <5 mm, n[b]	6.53 ± 3.73	8.19 ± 3.99	0.189	-1.5 (-4.000–0.000)	27.5
Teeth with CAL ≥5 mm, n	2.20 ± 4.02	2.25 ± 2.31	0.112	-1 (-1.000–0.000)	5
Sites with CAL ≥5 mm, n	4.56 ± 9.23	3.25 ± 3.65	0.177	0 (2.000–0.000)	10

AgP = aggressive periodontitis, CAL = clinical attachment level, ChP = chronic periodontitis, PPD = probing pocket depth, CI = confidence interval

The data are shown as the mean and standard deviation

[a]Mann–Whitney U test

[b]Missing data for two patients

Table 3: Comparison between aggressive periodontitis and chronic periodontitis according to the absence or presence of periopathogens

Bacterium	AgP (n = 20)	ChP (n = 32)	df	P value	CI	Power (%)
P. gingivalis						
Absent	10.0	0	1	0.143[a]	-	44.6
Present	90.0	100				
T. forsythia						
Absent	10.0	0	1	0.143[a]	-	46.6
Present	90.0	100				
T. denticola						
Absent	0	0		-	-	-
Present	100	100		-	-	-
A. actinomycetemcomitans						
Absent	90	100	1	0.143[a]	-	0.446
Present	10	0				
P. intermedia						
Absent	30.0	25	1	0.693[b]	(0.369–4.474)	0.069
Present	70.0	75				

AgP = aggressive periodontitis, *A. actinomycetemcomitans* = *Aggregatibacter actinomycetemcomitans*, ChP = chronic periodontitis, *P. intermedia* = *Prevotella intermedia*, *P. gingivalis* = *Porphyromonas gingivalis*, *T. forsythia* = *Tannerella forsythia*, *T. denticola* = *Treponema denticola*, *df* = degree of freedom, CI = confidence interval

The data are shown as the percentage

[a]Fisher exact test

[b]Chi-square test

cases in the AgP and in none of those in the ChP group ($P = 0.143$).

Discussion

The objective of this study was to compare the clinical and microbiological profiles of AgP and ChP in Congolese patients. Its findings indicated a marked difference in patient age between the AgP group and the ChP group but that none of the five types of bacteria assessed could differentiate the two forms of periodontitis by its presence or absence.

Age was the main demographic characteristic that distinguished ChP from AgP. The 1989 classification used age-dependent terms, such as "adult" and "juvenile," to categorize periodontitis. The 1999 classification estimated that age was not an appropriate parameter for distinguishing the different forms of the disease and recommended its deletion.[2,24] However, in daily practice and in epidemiological studies, age has continued to be an essential criterion for distinguishing between AgP and ChP.[25,26] A pilot study that tested the consistency of diagnosis of AgP and ChP by certified periodontists found that awareness of patient age influenced the clinical diagnosis when a distinction was made between AgP and ChP.[27]

Our results suggest that patients who have consulted a dentist previously are less likely to develop AgP than ChP. Given that AgP is considered to be a severe type of periodontitis, it is likely that Congolese patients who have sought dental care would be protected from a severe form of the disease.

The distribution of clinical features between the two types of periodontitis was slightly different; BoP in particular was greater in the AgP group than that in the ChP group. BoP has been reported to be a sign of gingival inflammation and a possible sign of progression of the disease, whereas its absence is a reliable predictor of periodontal health.[28,29] The higher BoP in the AgP group may reflect the rapid progression of this severe form of periodontitis. The differences in PPD and CAL found in this study seem to be nonconclusive.

A. actinomycetemcomitans, reported to be a key pathogen in AgP,[30,31] was detected at low prevalence and only in the AgP group in this study. This finding is in agreement with other studies in which this organism was detected at low prevalence.[18,32] Other researchers have also reported an absence of *A. actinomycetemcomitans* in patients with AgP.[33] Variations according to geographic location and ethnic or racial group could explain the low prevalence of *A. actinomycetemcomitans* in Congolese patients diagnosed clinically with AgP. The other bacteria assessed in this study, that is, *P. gingivalis*, *T. forsythia*, and *T. denticola*, were all detected at high prevalence. This is in agreement with our previous pilot study in which red complex bacteria were present at high prevalence.[17]

No statistically significant difference in periopathogen status was found between our ChP and AgP groups. The presence of *P. gingivalis*, *T. forsythia*, *T. denticola*, *P. intermedia*, and *A. actinomycetemcomitans* could not differentiate between the two forms of periodontal disease. A systematic review by Mombelli *et al.*[19] concluded that the presence or absence of *P. gingivalis*, *A. actinomycetemcomitans*, *P. intermedia*, *T. forsythia*, and *Campylobacter rectus* could not discriminate between subjects with AgP and those with ChP. Other studies have not found a microbiological difference between these two forms of periodontal disease.[34,35] Another study found no differences in the immunological and microbiological profiles of ChP and AgP and concluded that these two clinically distinct forms of periodontitis might result from common pathogenic mechanisms.[35]

Within the limitations of this study, ChP and AgP seem quite similar in Congolese patients except for clinical periodontal inflammation that was more visible in AgP. No microbiological difference was found between these two forms of periodontitis. However, given the low power of the tests used to compare microbiological profile, studies with great samples are needed in Congolese patients. Age remained an essential factor for differentiation between AgP and ChP.

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Conflicts of interest

There are no conflicts of interest.

Author contributions

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Data acquisition: Em Kalala-Kazadi.

Data analysis: Em Kalala-Kazadi, Selena Toma, Jérôme F. Lasserre, Michel C. Brecx.

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Guarantor: Michel C. Brecx.

Ethical policy and institutional review board statement

The study protocol was approved by the ethics committee at the School of Public Health, University of Kinshasa (approval number, ESP/015/2017) and conducted according to the tenets outlined in the Declaration of Helsinki.

Patient declaration of consent

Not applicable.

Data availability statement

The data set used in this study is available on request from Dr. Em Kalala-Kazadi (kalalaem@gmail.com).

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