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ORIGINAL RESEARCH

Microbiological Profile of Patients with Chronic Periodontitis

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***ABSTRACT*:**
Background: Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, and Tannerella forsythia have been described as the major periodontal pathogens. The present study was conducted to assess microbial profile of patients with periodontitis. **Materials & Methods:** The present study was conducted on 40 subjects of chronic periodontitis. 40 healthy subjects were taken as control. Patients were divided into 2 groups. Group I were patients of chronic periodontitis and group II were healthy subjects (controls). Samples of the subgingival plaque were taken and analyzed by real-time polymerase chain reaction. **Results:** The mean PD in group I was 3.68 mm and in group II was 1.32 mm, AL was 4.29 mm in group I and 1.02 in group II, gingival index was 0.89 in group I and 0.07 in group II, BOP was 45.4 in group I and 3.5 in group II. The difference was significant (P< 0.05). AA was seen in 45% in group I and 12% in group II, P gingivalis was 32% in group I and 5% in group II, T. forsythia was 11% in group I and 2% in group II, P oralis was 10% in group I and 1% in group II, A israelii was 47% in group I and 20% in group II, clostridium spp was 34% in group I and 11% in group II, P gingivalis was 56% in group I and 22% in group II. The difference was significant (P< 0.05). **Conclusion:** There was increased level of AA, P gingivalis, T. forsythia, P oralis, A israelii and clostridium spp in periodontitis patients as compared to healthy subjects.
Key words: Microbiological, P gingivalis, Periodontitis.

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INTRODUCTION

The human microbiome is important in the establishment and maintenance of human health. Due to many ecological determinants, the oral cavity comprises a very complex and diverse microbiota in the human body.[1] For most part, the oral microbiota presents a symbiotic relationship with the host; however, major disturbances in this microbial community (dysbiosis) may lead to the development of several oral diseases, such as gingivitis, different forms of periodontitis, caries, and endodontic infections.[2] Periodontal diseases are among the most common infectious oral diseases associated with a pathogenic biofilm. The resultant inflammatory process adds to further destruction of the periodontal apparatus and eventual tooth loss.[3]

Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, and Tannerella forsythia have been described as the major periodontal pathogens. Many in vitro studies have focused on the detection and quantification of these three species, even though it is recognized that other species may also contribute to the pathogenesis of periodontal disease. The prevalence of periodontal pathogens varies between individuals from the same environment and from different countries.[4]

A cause–effect relationship between the aggregation of bacterial deposits in the area of the gingival crevice and gingival inflammation.[5] The individual and local expression of signs of gingival inflammation also depends on host-derived factors modulating the inflammatory response to microbial colonization. This is reflected in an inconstant

local and systemic expression of inflammatory mediators.[6] The present study was conducted to assess microbial profile of patients with periodontitis.

MATERIALS & METHODS

The present study was conducted in the department of Periodontics. It comprised of 40 subjects of chronic periodontitis. 40 healthy subjects were taken as control. Chronic periodontitis was defined as having >5 teeth with periodontal sites with probing depths (PD) >5 mm. All were informed regarding the study and written consent was obtained. Ethical clearance was obtained prior to the study.

General data such as name, age, gender etc. was recorded. Patients were divided into 2 groups. Group I were patients of chronic periodontitis and group II were healthy subjects (controls).

Plaque index, gingival index, probing depth and bleeding on probing were assessed for all the patients. Samples of the subgingival plaque were taken with paper points from four teeth of each individual. The samples were divided into two parts. One part was immediately cultivated, while the other one was stored at –20°C until analyzed by real-time polymerase chain reaction. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant (P< 0.05).

RESULTS

Table I Distribution of patients

Groups	Group I	Group II
Status	Chronic Periodontitis	Healthy (Control)
Number	40	40

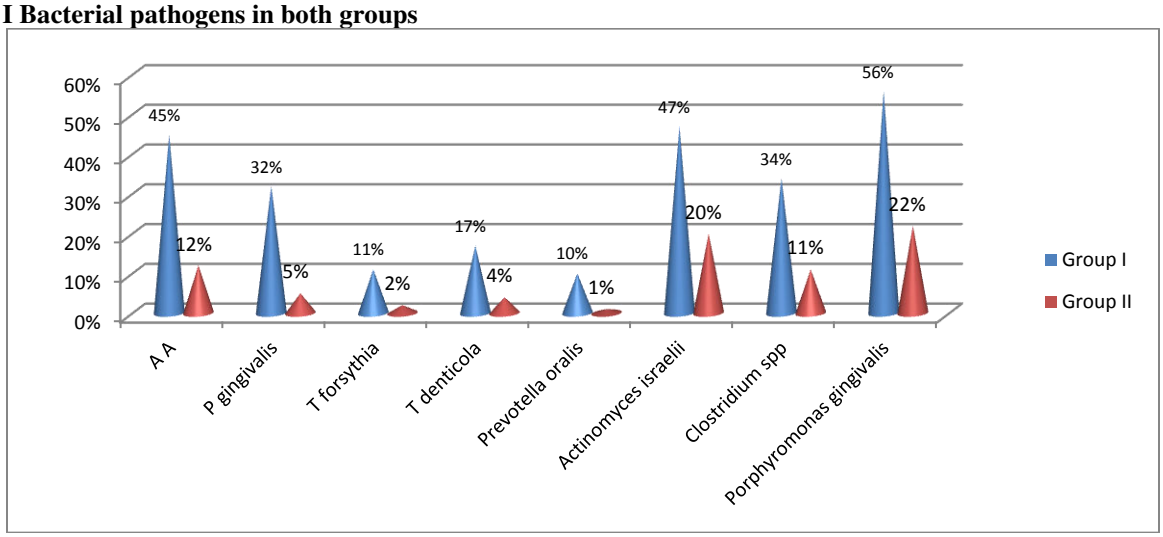
Table I shows that group I was study group i.e chronic periodontitis and group II was healthy ie control. Each group had 40 subjects.

Table II Comparison of parameters

Parameters (Mean)	Group I	Group II	P value
Probing depth (mm)	3.68	1.32	0.02
Attachment level (mm)	4.29	1.02	0.01
Gingival index (mm)	0.89	0.07	0.01
BOP (mm)	45.4	3.5	0.04

Table II shows that mean PD in group I was 3.68 mm and in group II was 1.32 mm, AL was 4.29 mm in group I and 1.02 in group II, gingival index was 0.89 in group I and 0.07 in group II, BOP was 45.4 in group I and 3.5 in group II. The difference was significant (P< 0.05).

Graph I Bacterial pathogens in both groups



Graph I shows that AA was seen in 45% in group I and 12% in group II, P gingivalis was 32% in group I and 5% in group II, T. forsythia was 11% in group I and 2% in group II, P oralis was 10% in group I and 1% in group II, A israelii was 47% in group I and 20% in group II, clostridium spp was 34% in group I and 11% in group II, P gingivalis was 56% in group I and 22% in group II. The difference was significant (P< 0.05).

DISCUSSION

The overall diversity of the periodontitis-associated microbiota is very broad, with potential involvement of several hundred different species and subspecies. These organisms may aggregate in various configurations, some of which have been associated with distinctive patterns of cytokine expression, as measured in gingival crevicular fluid.[7] Subjects with aggressive periodontitis were characterized by a higher interleukin-1beta/interleukin-10 ratio than were periodontally healthy subjects, suggesting an imbalance between pro- and anti-inflammatory cytokines in aggressive periodontitis. Only a few individual species show a unique association with disease.[8] A. actinomycetemcomitans and Porphyromonas gingivalis have been suspected to be of particular importance in the disease process as a result of their pathogenic potential demonstrated in animal models and an association with disease progression and clinical response to therapy, as found in prospective and retrospective clinical trials.[9] A. actinomycetemcomitans displays a broad genetic and phenotypic diversity and is heterogeneously distributed in various populations and cohorts worldwide.[10]The present study was conducted to assess microbial profile of patients with periodontitis.

In present study, group I was study group ie chronic periodontitis and group II was healthy ie control. Each group had 40 subjects.Haffajee et al[11] in their study included thirty-three patients with clinical and radiologic proof of aggressive and advanced chronic periodontitis and 20 healthy subjects. Clinical indices were recorded as six-point measurements on each tooth. A total of 284 anaerobic isolates (224 isolates from patients and 60 isolates from healthy controls) were identified. Forty different anaerobic species were isolated, with a mean of 6.78 species per patient and 3 species per healthy control (after adjusting for multiple comparisons, $P < .001$) were found for Prevotella intermedia/ nigrescens, Fusobacterium nucleatum, T forsythia, Treponema denticola, and Veillonella parvula. The first four species were associated with the aggressive periodontitis group and V parvula with healthy subjects.

We found that mean PD in group I was 3.68 mm and in group II was 1.32 mm, AL was 4.29 mm in group I and 1.02 in group II, gingival index was 0.89 in group I and 0.07 in group II, BOP was 45.4 in group I and 3.5 in group II. AA was seen in 45% in group I and 12% in group II, P gingivalis was 32% in group I and 5% in group II, T. forsythia was 11% in group I and 2% in group II, P oralis was 10% in group I and 1% in group II, A israelii was 47% in group I and 20% in group II, clostridium spp was 34% in group I and 11% in group II, P gingivalis was 56% in group I and 22% in group II. The difference was significant ($P< 0.05$).

Researchers have been struggling for years to develop reliable diagnostic tests capable of defining and identifying etiological and risk factors for periodontal diseases, particularly at the earliest phases of periodontal infection. In this context important progress in the understanding of the complex interactions between periodontal microbiota and host in health and disease has been made. In polymicrobial periodontal infections, determination of the microbial taxa is the first step to comprehend the dynamic interactions among microorganisms, host and environment.[12]

CONCLUSION

Authors found that there was increased level of AA, P gingivalis, T. forsythia, P oralis, A israelii and clostridium spp in periodontitis patients as compared to healthy subjects.

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