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Research article

EVALUATION OF AGGREGATIBACTER ACTINOMYCETEMCOMITANS LEVELS IN LOCALIZED AGGRESSIVE PERIODONTITIS BEFORE AND AFTER PERIODONTAL SURGERY

*****Saranyan Ravi, Priya Kesavan, Manovijay Balagangadharan, Raja Arasapan, Nisha N, Ann Joseph Anthraper

Department of Periodontics, Vinayaka Missions Dental College and Hospital, Salem, Tamilnadu, India

*Corresponding author: ravisaranyan@gmail.com

ABSTRACT

Background The role of microorganisms in the etiology of periodontal disease is well understood. The association of specific organisms in the pathogenesis of periodontal disease was established by the specific plaque hypothesis. This study examined the effects of periodontal surgery on Aggregatibacteractinomycetemcomitans (Aa) levels in localized aggressive periodontitis before and after periodontal surgery. **Method**: A clinical study was done on 24 male and 16 female patients who underwent surgical periodontal therapy. Bacterial counts were assessed from the plaque samples and gingival specimens. **Results:** Mean reduction of pre and post operative bacterial counts were statistically significant at 1%.COnclusion: A reduction of bacterial count was observed in plaque and gingival tissue samples after surgery.

Keywords: Aggregatibacteractinomycetemcomitans, Aggressive periodontitis, Periodontal surgery**,** Bacterial count, Biopsy

INTRODUCTION

Aggressive periodontitis is a disease of the periodontium caused by specific microorganism that differs from other forms of periodontitis in clinical, microbiological and histo-pathological features[1]. Aggressive periodontitis is a disease of the periodontium occurring in an otherwise healthy adolescent which is characterized by a rapid loss of alveolar bone about more than one tooth of the permanent dentition[2].

Localized aggressive periodontitis (LAP) have genetic factors that play an important role in the pathogenesis of the disease[3].However it is uncertain how the genetic factors are expressed. The increased incidence of localized aggressive periodontitis would suggest an x- linked inheritance with reduced penetrability[4,5]. While the precise etiology of LAP is uncertain, microbial sampling of the affected sites point towards the relationship with aggregate bacteractinomycetemcomitans (Aa). Aa is a facultative, gram negative, capnophilicnon motile coccobacilli. Slots 1976, Newman and Socransky 1976[6,7] identified Aa from the sub gingival flora of patients with LAP. Over 90% of patients with LAP have elevated levels of serum antibodies to Aa in both gingival crevicular fluid and gingival tissues[8,9].These antibodies may modulate the disease process by influencing the colonization and proliferation of the bacterium in the periodontal pocket.

Moreover Aa produces a number of virulent factors that can penetrate the gingival tissues and plays a role in the pathogenesis of the disease (Zambon 1983)[9].Factors that promote Aa colonization in the oral cavity include adhesions, bacteriocins, and invasions. Aa can interact and adhere to all components of the oral cavity, tooth surface, epithelial cells and intercellular matrix. It is also able to elicit its own uptake into epithelial cells and its

spread to adjacent cells usurping normal epithelial function. It also enhances its own survival by elaborating factors that interfere with the host's defense system and pass through the epithelial cell barrier causing periodontal destruction thus migrating to deeper tissues[10].

MATERIALS AND METHODS

The study was conducted according to the university norms of Annamalai University. Written informed consent of the patients was obtained. This study was conducted at division of periodontia Rajah Muthiah Dental College and Hospital, Chidambaram Annamalai University Tamilnadu India. The period of study was three months.

Inclusion criteria: Both male and female patients were included for the study. 24 male and 16 female patients who attended the dental op, diagnosed as aggressive periodontitis in the age group of 15-25 years were selected for the study.

Patients chosen were free from any systemic diseases, have not undergone oral prophylaxis or any other periodontal treatment and not taken antibiotics 6 months prior to the study.

Exclusion criteria:

Pregnant and lactating women, smokers, patients with immune-modulatory therapy, systemic diseases were excluded from the study.

Grouping:

Patients were divided into 2 groups randomly. Group 1 received surgical treatment and systemic tetracycline. Group 2 received nonsurgical treatment and systemic tetracycline.

Sample collection: A baseline microbial sampling was done from each subject .The sample sites chosen were first molar and central incisor of the same side. The selected teeth were isolated with sterile cotton rolls and the loosely adherent plaque in direct proximity to the sample site was carefully removed using sterile cotton gauze.

Methodology: A sterile paper point was introduced into the mesiobuccal interproximal pocket until resistance was met[11]. It was kept in place for 10 seconds and after removal the collected sample was then transferred into a test tube containing sterile saline. The bacterial deposits were dispersed in a vortex mixture for 60 seconds.

From the above solution one loopful of plaque suspension was placed over a glass slide and smeared. It was then air dried, heat fixed and stained by gram staining to confirm that the organism was Aggregatibacteractinomycetemcomitans It was then sent for culture to estimate the culture count [11]. Inflammed granulation tissue and the diseased soft tissue wall of the pocket was taken for biopsy and sent for histopathological examination, swell as to look for Aa in the gingival tissues(fig:1, fig:2,fig:3). It was expressed as scanty-25-50CFU, moderate-50-100 CFU and abundant-100-150CFU, where CFU stands for colony forming units. The subjects were examined after a period of 3 months.

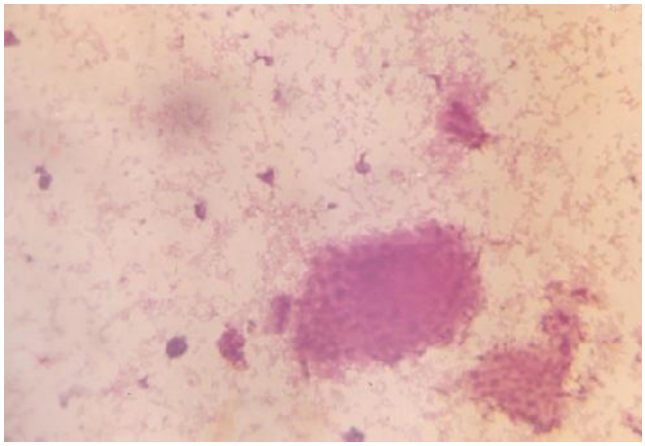

Fig 1: Gram stained smear of Aa revealing cocobacillary& pleomorphic forms x120

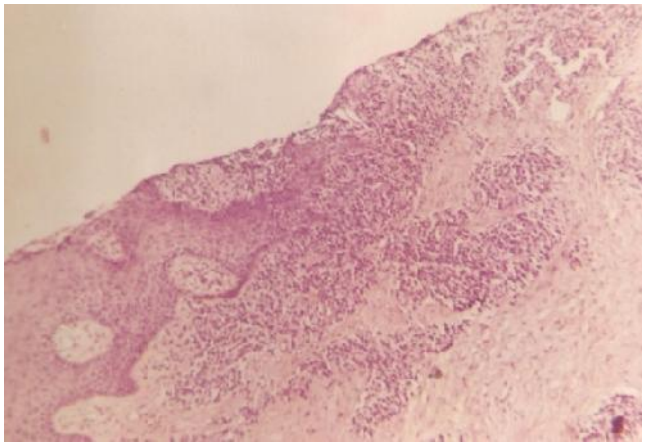

Fig 2: ulcerated membrane showing inflammatory infiltration of cells in the submucosax100

RESULTS

Gingival biopsy of group 1 patients showed a complete elimination of microorganisms where as in group 2 there was a reduction but not complete elimination of the organisms. As shown in table 1 colony forming units of Aa were measured following pre and post operative treatment.

Table 1: colony forming units of Aa were measured following pre and post operative treatment

Patient		Bacterial count	
	Pre treatment CFU		Post treatment CFU
4		25-50	ND
4	Group -1	50-100	
12		100-150	
8	Group - II	50-100	25-50
12		100-150	50-100

Group I p<o.o1 highly significant. Group 2 p<0.001 not significant, ND:

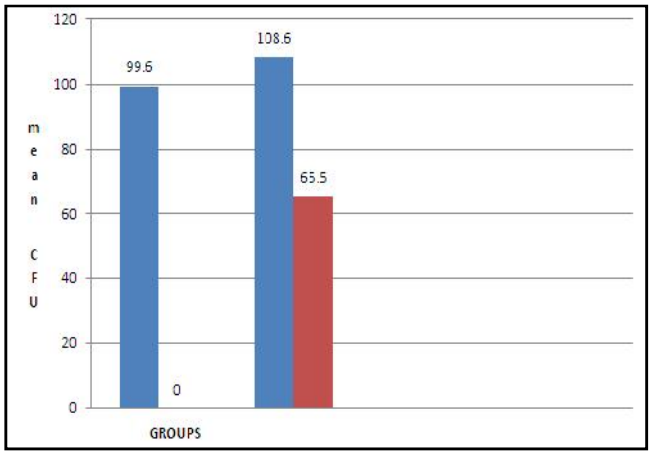

Fig 3: Mean CFU of A.a in group 1 and group 2 pre and post treatment

DISCUSSION

Most forms of periodontal disease are apparently caused by specific bacterial species, and the treatment should be directed towards the elimination of periodontal pathogens. Various clinical data are available indicating that periodontal surgery alone or in combination with systemic antimicrobial treatment can promote healing in LAP.

Suppression of Aa cannot be satisfactorily obtained using only nonsurgical clinical regimens where as it can be obtained with surgical therapy and systemic tetracycline therapy[12] as it has been well established that Aa invades the gingival tissue[13]. The findings of the present study is in concurrence with the above study as shown in Table 1

Furthermore surgical procedures suppress Aa only from the clinically treated sites, where as systemic tetracycline therapy can remove the organism both from the infected sites and buccal mucosa. Studies of Lindhe (1984)[14] demonstrated that treatment of LAP involving removal of subgingival plaque and inflammed periodontal tissue resulted in resolution of gingival inflammation and gain of clinical attachment.

Slots and Rosling (1983)[12] showed systemic administration of tetracycline suppressed Aa infection and its effects were observed in deep pockets and total number of Aa recovery per site were markedly reduced following antibiotic therapy. The results of the present study coincides with the above study

Christersson et al 1985[15] reported that surgical elimination of granulation tissue either by curettage or in conjunction with modified widman flap resulted in suppression of Aa often to undetectable levels and concluded that substantial supression of Aa can be accomplished by the surgical removal of periodontal tissues. The result of the present study is in concurrence with the above study.

Kornman and Robertson [16] and kunihara 1985[17] showed that periodontal surgery combined with systemic tetracycline has been shown to reliably suppress subgingival Aa which in accordance with the present study.

Though various antibiotic regimens are available antiobiotic sensitive test was done which showed that Aa was highly sensitive to tetracycline. Though Aa strains showed resistance to tetracycline, modified forms of tetracycline derivatives (doxycycline) are widely continued to be used in the management of LAP.

Inspite of various treatment modalities available for the management of LAP it has been established that surgical therapy in conjunction with systemic antibiotic therapy prove to be the most effective method in obtaining periodontal health along with suppression of Aggregatebacteractinomycetemco-mitans.

CONCLUSION

Within the limitations of the present study, it can be concluded that there was a reduction in Aggregatibacteractinomycetemcomitans in both the groups. However the reduction in the bacterial count was more evident in the surgical group than the non-surgical group.

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Conflict of Interest: Nil

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