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Original Research Article

The effect of smoking in the polymorph nuclear neutrophils' functions in patients with gingivitis

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ABSTRACT

Introduction: Smoking is a habit that is widespread in today's world as its primary constituent called Nicotine makes it very addictive for the users. Smoking is an important cause of diseases related to gums. If not quitted, it may lead to progressive adverse conditions in the oral cavity like gingivitis, periodontitis and so on. This is because the functions of neutrophils are significantly affected by smoking. As a result of the suppression of the activity of the neutrophils, the overall oral health of the individuals also become compromised.

Objective: This article aims to describe the pattern and clearly delineate the difference in the condition of gingiva between smokers and nonsmokers by explaining the changes brought about by smoking on the polymorphonuclear neutrophils.

Materials and Methods: Three groups of people were selected for this study. The groups were matched by age and gingival condition. Two groups consisted of people with the habit of smoking, and they were further categorized into mild smokers and heavy smokers depending on the number of cigarettes smoked per day. The third group consisted of people with no habit of smoking. This group was the control group in the study. Blood samples were taken and Polymorphonuclear neutrophils were isolated. The functions of PMNs like chemotaxis and phagocytosis were studies under different methods.

Results: The mean value for the all the three groups under clinical parameters like plaque index, gingival index, and the functions of neutrophils like chemotaxis and phagocytosis are obtained and after elucidating the results, it is found that smoking impairs the overall oral hygiene and the functions of neutrophils against gingival infections.

Conclusions and Recommendations: It is proven that there is debasing alteration in the functions of neutrophils which could have led to the localized destruction. Hence it suggests that smoking impairs the functions of neutrophils against gingival infections. And there may be a higher possibility of this condition to progress into the next stage called periodontitis. Hence it is advisable to quit the habit as and when it is possible, and it is the role of dentists to identify such conditions and educate the patients on the adverse effects of smoking.

1. Introduction

Periodontal diseases owe its origin to the lack of balance between the host and the parasite. The extent and intensity of the disease depend on the interaction between the host immune system and the microorganisms. The etiological roles of microorganisms have already been established well in literature. It is now known that the usage of tobacco, cigarettes in specific is the major risk factors for the periodontal diseases. Neutrophils form the first line of defense for the human body, and it is the same for the oral cavity.[1,2] Their functions include phagocytosis,

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chemotaxis, oxidative burst, superoxide and hydrogen peroxide production, protease inhibition etc., They are predominantly phagocytic and chemotactic in response to bacterial functions. Smoking is found to impair the functions of neutrophils.[3] Such changes in the numbers and functions of neutrophils can make the individual more prone to infections. An underlying mechanism which explains this is the change in the host response and neutrophil function by the tobacco products. More accurately it is found to impair chemotaxis and phagocytosis. This inability to neutralize the periodontal infections and increased stimulation of potentially destructive processes thereby causes the periodontal tissue breakdown. The probability of the occurrence of these periodontal diseases in smokers is 2.5 times greater than non smokers.[4,5] Epidemiological evidence prove that smoking is a strong risk factor for the origin of periodontitis when compared with other periopathogens viz., *Actinobacillus actinomycetemcomitans*, *Porphyromonas gingivalis*, etc.,[6] Tobacco interferes with the normal immunological surveillance of neutrophils and macrophages. The negative role is also on the reparative and regenerative properties of cells of the periodontium, adult stem cells etc., Distinct destruction have been attributed to the stimulation of osteoclastic activity of the smoke of tobacco. On an average, 1 million plus polymorphonuclear neutrophils come into the oral cavity through the sulcular fluid. They shield the gingiva from the invading microbes by secreting hydrolytic enzymes and producing oxygen radicals. This is evident from the periodontitis patients with underlying neutrophils deficiency. This study is attempted to explicate the consequence of smoking on chemotaxis and phagocytosis of Polymorphonuclear neutrophils in gingivitis patients.

2. Materials and Methods

30 subjects in the age group of 20-50 YEARS were selected as study population. It was a retrospective study. The subjects were divided into 3 groups each containing 10 patients. Patients with a smoking history for at least 5 years were considered. Patients who smoke less than 5 cigarettes per day were considered as light smokers. Patients who smoke more than 10 cigarettes per day were considered as heavy smokers. Non-smokers were selected as the control group. All subjects were systemically healthy, and they were excluded if they were with any systemic complications, if they were under any medications and if they were with previous history of periodontal therapy.

The investigation was approved by the ethical committee of Vivekanandha Dental College for Women, Thiruchengode, Namakkal district, Tamil Nadu, India. All the participants signed an informed consent before the commencement of the work. Clinical parameters that were recorded are the Plaque index and Gingival Bleeding index. All the measurements were executed by the same examiner. Blood specimens were obtained from the subjects by venipuncture under strict sterile conditions. After centrifugation, the serum was isolated and then the neutrophils were collected for subjecting them into the analyses for chemotaxis and phagocytosis.

2.1. Chemotaxis assay

The chemotaxis assay[7] assembly comprised of a lower compartment filled with the chemo-attractant case (in Hanks balanced salt solution; HiMedia Labs). The upper compartment consisted of a syringe with 5μm pore size calcium acetate filter paper pasted with glue at one end which contained the cell suspension. It was then placed inside the lower compartment and kept undisturbed for about 1 hour at room temperature. Subsequently, the cell contents in the upper compartment were emptied and the compartment was immersed in 70% methanol in such a way that the glue liquefied. The filter paper strip was then removed carefully, and further stained with hematoxylin and was fixed on a glass slide to observe under the microscope.

2.2. Phagocytosis assay

For the phagocytic assay[7,8] the sample was spread on Sabourauds 2% dextrose broth for 48 h at 37°C to obtain organisms in the yeast phase particularly. The cells be assorted with the neutrophil rich cell suspension and set aside uninterrupted for about 30min at 37°C. The complete congregation was centrifuged at 1500 rpm for 5 min. The supernatant was then discarded, and the smears were prepared alongside with the residue, air dried as well as stained with Giemsa stain.

3. Results

Table 1: Mean values of clinical paramters and neutrophil activities

Groups	Numbers	Plaque index	Gingival bleeding index	Chemotaxis	Phagocytosis
I	10	0.56	42.4	115.48	4.26
II	10	0.64	40.8	98.26	3.34
III	10	1.26	40.2	87.77	2.93

1. Non-smokers with chronic gingivitis
2. Light smokers with chronic gingivitis
3. Heavy smokers with chronic gingivitis

4. Discussion

Periodontal diseases are destructive disease of the periodontium that result due to the bacterial activity in the host oral cavity. The immune inflammatory system of

the host plays a major role in the elimination of the harmful effects of microorganisms in the individuals. Smoking is proven to impair the host inflammatory response.[9] Neutrophils are the first line of defense against all the microorganisms that invade the human body. The harmful toxic effects of the cigarettes alter the functions of the neutrophils.[6] Gingivitis is more likely to progress into periodontitis[10] under such conditions where the functions of the neutrophils are compromised. This is proven to be right in the study. 30 subjects with gingivitis in the age group of 20-50 YEARS were selected as study population. The subjects were divided into 3 groups each containing 10 patients. Clinical parameters that were recorded are the Plaque index, Gingival Bleeding index, Probing pocket depth. All the measurements were performed by the same examiner. Blood specimens were obtained from the subjects for subjecting them into the analyses for chemo taxis and phagocytosis.

4.1. Plaque index

On Comparison among the three groups, the mean PI was found to be in the order of Heavy Smokers < mild smokers < Non-smokers (0.56<0.64<1.26). This agrees with the study by Feldman RS et al. (1983)[11] and Preber et al (1980).[12] However, it was also contradicted by Thomson MR et al in 1993.[13]

4.2. Gingival bleeding index

On comparison among the three groups, the BI was in the order of Heavy Smokers < mild smokers < Non-smokers (40.2<40.8<42.4). This result agrees with Linden GJ and BH Mullay (1994).[14]

4.3. Chemotaxis

On comparison, Heavy smokers < mild smokers < non-smokers (87.77<98.26<115.48). Nobel et al. in 1975[15] and Seow et al. in 1994[16] have found that chemotaxis is suppressed in smokers when compared to the counter part.

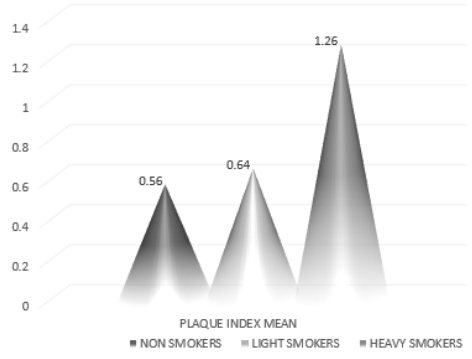

Fig. 1: Plaque index

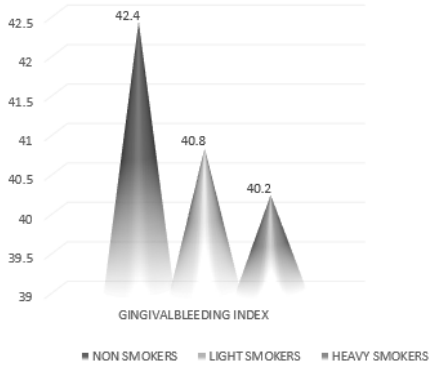

Fig. 2: Gingival bleeding index

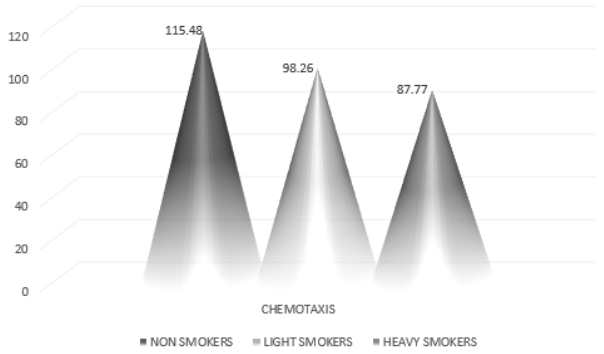

Fig. 3: Chemotaxis

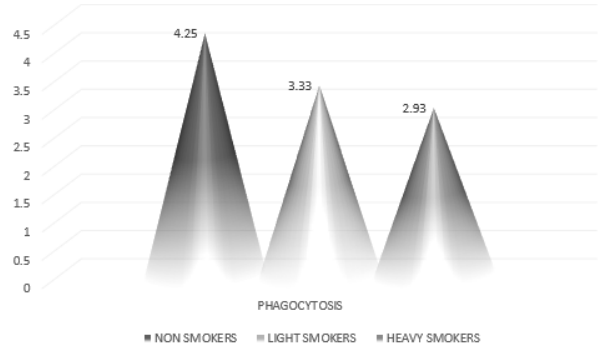

Fig. 4: Phagocytosis

4.4. Phagocytosis

On comparison, Heavy smokers < mild smokers < nonsmokers (2.93<3.33<4.25). Similar results have been found out by Mac Farlane et al in 1992.[17] Gunstch A et al. has also concluded that phagocytosis was decreased in smokers compared to nonsmokers in the year 2006.[2]

5. Conclusion

The downward regulation of the immune systems of smokers is due to the deleterious effects of smoking. In this

study, it is proven that there is alteration in the phagocytosis and chemotaxis which could have led to the localized destruction. Hence it suggests that smoking impairs the functions of neutrophils against gingival infections. And there may be a higher possibility of this condition to progress into the next stage called periodontitis. But the precise changes in the immunological mechanisms included in the rapid tissue destruction are currently not discussed and described. Further studies must be undertaken to describe the same. While it is likely that some smoker may not be affected by the defect in function of neutrophils, these data aim to a continuous need for investigation of the environmental and genetic factors of resistance and susceptibility to periodontitis.

6. Source of Funding

None.

7. Conflict of Interest

None.

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