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Profile of Patients with Periodontal Disease: A Descriptive Study.

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ABSTRACT

Background: The most common form of periodontal disease has been defined as "an inflammatory process affecting one or more of the supporting tissues of teeth – the gingival tissue, the periodontal membrane and the alveolar bone. In this study the index used for assessing the periodontal status of the population was Community Periodontal Index of Treatment Needs (CPITN). **Methods:** Study subjects included patients diagnosed to have periodontal disease and a total of 200 patients were selected for the study. **Results:** In this study, 21% of patients were smokers, 12% of patients consumes smokeless tobacco, 26% of patients were alcoholics and 30% of patients had bad practices of oral cleaning. **Conclusion:** Modifiable risk factors were more commonly associated with periodontal disease.

Keywords: Periodontal disease, smoking, alcohol.

INTRODUCTION

Periodontal disease is an inflammatory disease that affects the soft and hard structures that support the teeth. In its early stage, called gingivitis, the gums become swollen and red due to inflammation, which is the body's natural response to the presence of harmful bacteria. In the more serious form of periodontal disease called periodontitis, the gums pull away from the tooth and supporting gum tissues are destroyed. Bone can be lost, and the teeth may loosen or eventually fall out.

Chronic periodontitis, the most advanced form of the disease, progresses relatively slowly in most people and is typically more evident in adulthood. Although inflammation as a result of a bacterial infection is behind all forms of periodontal disease, a variety of factors can influence the severity of the disease. Important risk factors include inherited or genetic susceptibility, smoking, lack of adequate home care, age, diet, health history, and medications.

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The two most common diseases affecting the oral cavity are dental caries (tooth decay) and periodontal disease.[1] (Periodontal ligament is the connective tissue attaching the tooth to the alveolar bone.) Dental caries are more commonly found in children and young adults because of the consumption of large amount of carbohydrates in the form of sucrose. This becomes the main source of acid for demineralization when microorganisms have acted upon them.

A large body of epidemiological research indicates that tobacco use has deleterious effects producing a wide spectrum of diseases including cancers of the mouth, lung, larynx, pharynx; diseases of the heart, circulatory system and lungs; and if used during pregnancy, produces adverse effects on the fetus.[2] There is a growing body of scientific evidence indicating that nicotine contributes to the progression of periodontal disease, and is detrimental to healing following periodontal therapy. Nicotine has toxic effects on peripheral circulation, which causes gingival vasoconstriction. As a result a decreased number of immune cells are available in the gingival tissue, which translates into a weakened defense-reparative system.[3]

The most common form of periodontal disease has been defined as "an inflammatory process affecting one or more of the supporting tissues of teeth – the gingival tissue, the periodontal membrane and the alveolar bone.[4] More recently the American Academy of Periodontology defined chronic periodontitis as "an infectious disease resulting in inflammation within the supporting tissues of teeth, progressive attachment and bone loss characterized by pocket formation and or recession of gingiva.[5] In early epidemiological studies, gingivitis and periodontitis were combined and considered a

continuum.[6] Studies of experimental gingivitis by Loe and coworkers demonstrated that accumulation of plaque led to gingivitis and that gingivitis was treated by plaque removal.[7] This evidence suggested a plaque-gingivitis periodontitis concept. Currently, the diagnosis of periodontal disease is 5 based on probing depth, clinical attachment level, CAL, the radiographic pattern and extent of alveolar bone loss, gingival inflammation measured as bleeding on probing or a combination of those measures.[8] In addition, consideration might be given to age, gingival recession, tooth mobility, medical and dental history, and signs and symptoms including pain and microbial deposits. Although a standard case definition is a central requirement in epidemiological research, a plethora of case definitions has been used in population based studies without an accepted standard and with a wide range of signs and symptoms employed.[9]

MATERIALS AND METHODS

A Descriptive case series study was carried out at a tertiary care hospital to know the profile of patients with periodontal diseases. Study subjects included patients diagnosed to have periodontal disease and a total of 200 patients were selected for the study. These patients were selected by using non probability purposive random sampling. Patents were explained about the needs of the study and relevant information were gathered by interview technique. A thorough clinical examination and oral cavity examination was made and findings were noted. Data was entered in Microsoft excel and was analyzed using SPSS 21.0.Results were presented as percentages.

In this study the index used for assessing the periodontal status of the population was Community Periodontal Index of Treatment Needs (CPITN). The CPITN is an ideal index for epidemiological studies because it uses accepted clinical criteria, partial mouth scoring and a simple recording procedure, which allows rapid assessment of individuals for periodontal conditions related to treatment needs.

RESULTS

The highest number of patients were in the age group of more than 50 years (32%) followed by 41 – 50 years (20%), less than 20 years (18%), 21 – 30 years (16%) and 31 – 40 years (14%) [Table 1].

Table 1: Age Distribution

Age group	Frequency	Percentage
Less than 20 years	36	18.0
21 – 30 years	32	16.0
31 – 40 years	28	14.0
41 – 50 years	40	20.0
More than 50 years	64	32.0
Total	200	100

Patients comprised of males and females, male constituted 62% and females 38% [Table 2].

Table 2: Gender Distribution

Gender	Frequency	Percentage
Male	124	62.0
Female	076	38.0
Total	200	100

The proportion of illiterate patients was 6%, patients with income less than 5000 per month was 65%, patient with no occupation was 24% and 71% of patients belong to low socio economic status [Table 3].

Table 3: Socio demographic Profile of patients

Variables	Frequency	Percentage
Illiterate	12	6.0
Income less than 5000per month	130	65.0
Not working	48	24.0
Low socio economic status	142	71.0

In this study, 21% of patients were smokers, 12% of patients consumes smokeless tobacco, 26% of patients were alcoholics and 30% of patients had bad practices of oral cleaning [Table 4].

Table 4: Risk factor Profile of patients

Risk factors	Frequency	Percentage
Smoking	42	21.0
Smokeless tobacco	24	12.0
Alcohol	52	26.0
Un hygienic oral cleaning	60	30.0

DISCUSSION

While taking into consideration the periodontal disease status among different age groups, it was found that with advancing age shallow and deep periodontal pockets (grades 3&4) had remarkably increased and were highest in the more than 50 year age group. This gradual worsening of periodontal conditions with increasing age reflects the progressive nature of periodontal disease. The progressive nature of the disease with increasing age was also reported by Anil S et al (1990)[10] in their study conducted to find the periodontal conditions of a selected population in Trivandrum district. In their study, the disease prevalence in the 35-44 age group was very high. The study reports that 77% of the subjects belonging to that particular age group were having the disease and when compared to the younger age group, this was found to be very high. The disease prevalence was very high in the poorest socioeconomic group when compared to other groups. In rest of the group's disease prevalence was almost similar. The reason for decrease in periodontal disease with increase in income may be

due to the paying capacity of the subjects for treatment procedures, which were found unaffordable to those in the lowest socio-economic status.

It should also be noted that in subjects who were consuming both forms of tobacco, the prevalence is high and from the results it's clear that smokers are at high risk. It may be due to the fact that, smokers have poor oral hygiene, which leads to an increase in plaque and calculus formation, and many studies had supported this fact.[8,11] There is a growing body of scientific evidence indicating that nicotine contributes to the progression of periodontal disease. Exposing periodontal tissues to tobacco smoke may effect periodontal break down in two potential pathways: tobacco smoke may function directly as a source of gingival irritation; and indirectly a systemic mechanism may alter vascular tissue and haemodynamics.[12] As a result of gingival vasoconstriction decreased number of immune cells are available in the gingival tissue, which translates into a weakened defence-reparative system. Many studies have shown that smoking is the main factor responsible for periodontitis.

When considering tobacco chewing alone it was found that this factor alone is not responsible for periodontal disease formation. Robertson P.B et al (1990)[13] in their study "Periodontal effects associated with the use of smokeless tobacco" in professional foot ball players reported that 46% had oral mucosal lesions, located primarily in the mandible at sites where the smokeless tobacco quid was placed. They also reported that the use of smokeless tobacco was not necessarily associated with severe forms of periodontal disease though sites adjacent to mucosal lesions in smokeless tobacco users showed significantly greater recession and attachment loss than in sites not adjacent to lesions in users or comparable sites in non-users. Similar findings have been observed by Thomas and Johnson (1987)[14] in their study "Smokeless tobacco and its effect on oral tissues". They reported that tobacco chewers show greater destruction with respect to gingival recession whereas chewing of tobacco did not cause any other destructive changes in the periodonti um. The exact reason for decrease in periodontitis in tobacco chewers is unknown. One of the arguments is that, while chewing the betel quid there will be an increase in salivary flow. The flow causes a physical cleaning action and a biochemical action and due to these reasons the bicarbonate content, pH and buffering capacity are all greatly increased which has its effect on the oral cavity.

CONCLUSION

In this study, Modifiable risk factors were more commonly associated with periodontal disease.

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