Research Article

WBC Count in Patients with Chronic Periodontitis

S. Vaishali*, Sohara Parveen, Vishunupriya V, Gayathri R
Saveetha Dental College, 162, P.H. Road, Chennai, Tamilnadu, India.
***Corresponding author's E-mail:** vaishudevasri1348@gmail.com

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ABSTRACT

The aim is to study the white blood cell count in patients with chronic periodontitis. The objective of study was to determine the changes in WBC count in plasma of the patients with chronic periodontitis compared to healthy controls. Chronic periodontitis is a common disease of the oral cavity consisting of chronic inflammation of the periodontal tissues which is caused by the accumulation of profuse amounts of dental plaque. The purpose of this study is to investigate the relationship between WBC count in patients with chronic periodontitis which is a very important component in the pathogenesis of periodontal diseases. The reason is to analyse the difference in white blood cell count in patients with chronic periodontitis. The changes in WBC count in periodontitis were analysed.

Keywords: Chronic periodontitis, polymorphonuclear leukocyte, bacterial biofilm, Immunoinflammatory.

INTRODUCTION

In recent years, great attention of the world researches has been focused on the possible association between oral health and systemic diseases, so that numerous contradictory results can be found in literature[1]. Since recently, there has been a new supposition that poor oral hygiene can cause an increased systemic response of inflammatory mediators in peripheral blood of patients with periodontitis, elevating the leukocyte count as well[2].

Periodontal disease poses a significant challenge to the patients and the oral healthcare professionals[3]. When periodontal disease affects the bone and supporting tissue, it is termed periodontitis and is characterised by the formation of pockets or spaces between the tooth and gums. This may progress and cause chronic periodontal destruction leading to loosening or loss of teeth. The dynamics of the disease are such that the individual can experience episodes of rapid periodontal disease activity in a relatively short period of time, followed by periods of remission. Chronic periodontitis is a common disease of the oral cavity consisting of chronic inflammation of the periodontal tissues that is caused by accumulation of profuse amounts. In the early stages, chronic periodontitis has few symptoms and in many individuals the disease has progressed significantly before they seek treatment. Symptoms may include the following: Redness or bleeding of gums while brushing teeth, using dental floss or biting into hard food (e.g. apples) (though this may occur even in gingivitis, where there is no attachment loss), gum swelling that recurs, gingival recession, resulting in apparent lengthening of teeth, deep pockets between the teeth and the gums, loose teeth in later stages and so on[4].

The rate of progression of periodontal disease in an individual is dependent on the virulence (or strength of attack) of the bacterial plaque and on the efficiency of the local and systemic immunoinflammatory responses in the person (host). The overall balance between the bacterial plaque challenge and the body's immunoinflammatory responses is critical to periodontal health. Smoking and diabetes are well-established risk factors for periodontal disease. Stress has also been linked to periodontal disease, but it is not clear whether the relationship has a physiological basis or is due simply to the fact that individuals under stress are less likely to perform regular good oral hygiene[5]. Bacteria with varying pathogenicity have been identified and correlated with various forms of periodontitis[6]. The current concepts on the etiology of periodontitis considers three groups of factors that determine whether a disease will occur in a subject. Those include a susceptible host, the presence of pathogenic organism, absence or a small proportion of beneficial bacteria. Under susceptible host, it is partly hereditary but can be influenced by environmental & behavioural factors. The various factors include genetic variations or mutations, smoke, stress, systemic diseases like diabetes and increased interleukin-1 production. Common pathogens include *T.forsynthia, P.intermedia, F.nucleatum, A.actinomycetocomitans*[7]. The formation of plaque on and around the teeth represents a massive accumulation of bacteria that are usually present in the oral cavity. This bacterial colonisation is relatively independent of food intake, degree of salivation, mastication and malocclusion[8].

The mechanisms by which bacterial organisms in the plaque destroy gingival tissues, periodontal fibres, alveolar bone and cause changes in the cementum are not known. However, there are several direct and indirect mechanisms that could cause the initial changes in the superficial tissues and the progressive destruction of connective tissue and bone. Direct mechanisms include

production of histolytic enzymes (hyaluronidase, chondroitinsulfatase, collagenase, and other proteinases) by several of the plaque bacteria. Cytotoxicagents, such as endotoxin, are formed during disintegration of Gram-negative organisms, and toxic metabolities (e.g., ammonia, organic acids, hydrogen sulfide) are produced by the plaque bacteria. All these substances may severely affect normal tissue metabolism or produce inflammatory responses, which may themselves be destructive. Finally, agents produced by the plaque organisms may indirectly stimulate host-mediated responses that lead to tissue destruction. Prominent among these indirect mechanisms are the release of endogenous enzymes and occurrence of immunopathological responses[8]. Periodontal inflammation can deteriorate systemic conditions thought the pathology caused by leukocytes[9]. Leukocytes, before all polymorphonuclear leukocytes are the major systemic cells of phagocytosis and the first cells of host defence mechanism against infective agents[10]. White blood cells (WBCs), also called leukocytes or leucocytes, are the cells of the immune system that are involved in protecting the body against both infectious disease and foreign invaders. All white blood cells are produced and derived from a multipotent cell in the bone marrow known as a hematopoietic stem cell. Leukocytes are found throughout the body, including the blood and lymphatic system. The number of leukocytes in the blood is often an indicator of disease, and thus the WBC count is an important subset of the complete blood count[4]. During periodontitis as a bacterial infection, neutrophils are initially predominant cells of host defence mechanism, and have a significant role in inflammation and pathogenesis[6]. As already reported aggressive periodontitis is strongly associated with impaired neutrophil function, which even more emphasises the importance of neutrophils in the pathogenesis of periodontal disease[11]. It is supposed that there is a difference in the count of these cells in periodontitis of various severities due to accumulation of oral biofilm when compared to healthy subjects[12]. Leukocytes, especially neutrophils, produce a number of specific molecules being directly responsible for the inflammatory response, including, besides local, also systemic response of the host as well, thus bringing them into mutual connection[13]. Changes are total leukocyte and neutrophil counts in the leukocyte formula point to the presence of infection and inflammation, which can be the risk factors for systemic conditions and diseases[14]. The aim of this pilot study was to investigate and compare the total leukocyte count in the peripheral blood of patients suffering from chronic periodontitis and with healthy subjects.

MATERIALS AND METHODS

Patient Selection

The current study included 40 subjects selected from patients presenting to Department of Periodontics, Saveetha Dental College and Hospital. The subjects were divided into two groups. Group I included 20 healthy individuals and group II included 20 chronic periodontitis patients. Subjects suffering from any systemic disease, smokers, pregnant women, subjects being under periodontal treatment, subjects taking antibiotics or having taken antibiotics six months prior to the research were excluded. All these conditions were considered to possibly influence the leukocyte count[10]. Clinical criteria for patient inclusion in the research procedure and for the diagnosis of periodontitis were as follows: depth of periodontal pockets, radiological analysis to reveal bone defects, oral hygiene status, clinical attachment level were measured. None of the subject had received periodontal treatment for 6 months prior to the study[10]. Furthermore, each patient received a detailed explanation regarding the study procedure and gave their oral consent to participate in the study.

Sample Collection

2 ml of venous blood were collected from each subject and were transferred to 5 cm long EDTA-anticoagulated tubes. Then the leukocyte count from the blood samples collected were estimated by using automated cell counters.

Assessment of WBC

WBCs were counted with automated cell counters via standard techniques (Coulter STK-R and Coulter STK-S, Coulter Electronics, and Sysmex SE-9500, TOA Medical Electronics).

The sample is aspirated by the automated cell counter from the collection tube, and after lysis of red blood cells and platelets, WBCs were counted by u84.75 457.56 Tm(an)4(ti)17(5

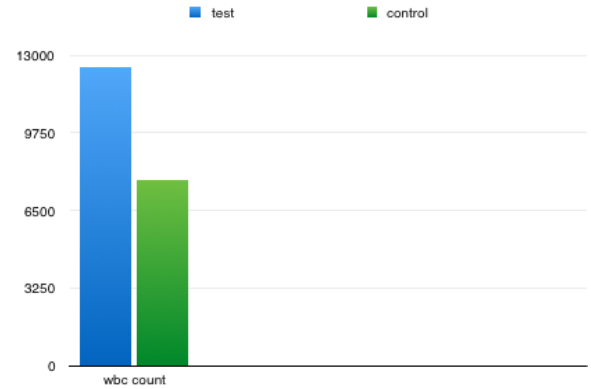
Table 1: Shows the t-test value (T-test) - **Group Statistics**

	Group	N	Mean	Std. Deviation	Std. Error Mean
WBC Count	Test	20	12495.00	953.925	213.304
	Control	20	7795.00	1504.546	336.427

Table 2: Independent Samples Test Value

		Levene's Test for Equality of Variances		t-test for Equality of Means					95% Confidence Interval of the Differences	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
WBC Count	Equal Variances Assumed	8.071	.007	11.799	38	.000	4700.00	398.349	3893.585	5506.415
	Equal Variances not Assumed			11.799	32.151	.000	4700.00	398.349	3888.740	5511.260

RESULTS AND DISCUSSION



Graph 1: shows the leukocyte count in patients with chronic periodontitis and control group.

The research was conducted on 20 patients with chronic periodontitis and 20 healthy subjects. In both groups of subjects, there were evident changes in the values of clinical parameters of periodontitis when compared to group of subjects with healthy periodontium. Table 1 shows the mean value of leukocyte count in patients with chronic periodontitis and control groups. The mean value of leukocyte count in patients with chronic periodontitis was significantly higher than control group. So it is evident that the leukocyte count in patients with chronic periodontitis is higher when compared with control group.

Our study results showed an increase in total leukocyte count in patients with chronic periodontitis compared to healthy subjects.

The findings obtained in the present study confirmed the concept according to which stronger inflammation of the periodontium of subjects with chronic periodontitis could be the result of increased leukocyte count in peripheral blood, which is in conformity with the basic function of leukocytes in infection and inflammation. In addition, the results of this study are in accordance with the results of other authors.

Kweider have shown that patients suffering from periodontitis have significantly higher levels of fibrinogen and leukocytes when compared to control subjects[15]. Inflammatory oral diseases such as periodontitis probably influences the total leukocyte count in the circulation considerably. Total leukocyte counts as indicators of inflammation at the same time show the association between oral disease, especially periodontitis, and systemic diseases in which the infection is an etiologic factor (cardiovascular diseases, especially myocardial infarction)[16]. The starting ground was that periodontitis is a local chronic inflammation occurring in response to a long-term presence of specfic bacteria in the oral biofilm, causing systemic consequences as well[17]. Dorn suggested that the capacity of the oral biofillm bacteria to invade not only periodontal tissues but also the tissues of coronary arteries makes them possible factors connecting periodontitis and coronary artery disease[18]. Changes in leukocyte count can be considered as a qualitative markers of periodontal inflammation. The subgingival biofilm bacteria activate the acute phase hepatic response, which further increases total count of leukocytes and other inflammatory markers, which can predispose the patient to systemic diseases as well[19]. The chronic infections, such as periodontitis, are associated with increased risk for cardiovascular disease[1]. Kowalik suggested that the increase in number of leukocytes in periodontitis is mainly due to an increase in the number of polymorphonuclear leukocytes[20]. In addition, Monteiro earlier demonstrated elevation of total leukocyte count in periodontitis[21] and Christsn said that lowering of leukocyte levels following periodontal therapy[22]. Mattila suggested that such an increased leukocyte count in periodontitis may carry a high risk of coronary heart disease, because there are several plausible mechanisms by which the WBCs can promote atherosclerosis, thrombosis, and myocardial ischemia[23]. Leukocytes have a wide range of biological effects, some of which protect against vascular diseases while some are damaging[24]. Periodontal infection leads to bioflim formation, inflammation and attachment loss. Continued

inflammation results in signalling of fibroblasts and production of pro- inflammatory cytokines in the tissues.

Antibodies specific to oral bacteria circulate in the peripheral blood[3]. Renvert had also stated that recurrent acute coronary syndrome events are predicted by serum WBC counts and is a diagnosis of periodontitis[25]. Periodontal bacteria and their products can directly invade the periodontal tissue and gain access to the systemic circulation[26]. Epidemiological studies suggest that periodontitis is associated with increased risk of systemic diseases like cardiovascular ischemia and atherosclerosis. Any infection in the body leads to increase in W.B.C. count. Activation of the immune system and inflammation may be detected by an increase in a number of markers, including white blood cell count[27].

CONCLUSION

Thus our study concluded that the chronic periodontitis has an association with increased WBC count. It also acts as a quantitative marker for diagnosis of systemic diseases. As increase in W.B.C. count can act as a risk factor for many systemic diseases, so to avoid the risk of systemic diseases, non surgical therapy should he advocated. In case of periodontitis, patients are advised to take necessary treatment to avoid further health risks.

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