The Effects of Cigarette Smoking on Human Gingival Tissues (A Histopathological Study)

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Abstract

Gingival biopsy specimens were obtained from 9 smokers and 7 non- smokers during routine flap surgical procedure to investigate the effects of cigarette smoking on gingival epithelium and connective tissue. Although no remarkable histopathological difference was noted between gingival tissues of smokers and non- smokers, but smokers revealed relatively more epithelial keratosis, inflammatory cells, blood-vessels and collagen fibres in connective tissue. Congestion of the capillaries were also more prominent in the smokei's gingival tissues (JPMA 44:210,1994).

Introduction

Several epidemiological and clinical studies have shown an increased prevalence and more severe periodontal disease in smokers than non-smokers[1-4]. This maybe due to the increased plaque accumulation as well as calculus[4-6]. The effect of cigarette smoking on the immune system has also been reported by some investigators, showing elevated or functionally impaired blood leukocytes[7,8] and lymphocytes[9,10], fall in serum IgG and IgA levels[11,12] and low natural killer cell activity[11,13] . A significant increase in gingival blood flow in cigarette smokers has been reported recently[14]. The alteration in the immune system of smokers may contribute to patients susceptibility to infection and malignant diseases[15,16]. Several studies have shown the effect of cigarette smoking on periodontal health, gingival blood flow and host defense mechanism but no study has investigated its effect on the gingival epithelium and connective tissues. The present study was designed to investigate the long term effects of cigarette smoking on the gingival epithelium and connective tissues in patients with chronic periodontitis.

Patients and Methods

The subjects of this study were 16 physically healthy individuals who attended department of periodontology, Dental Faculty , Dicle University, Diyaibakir, for the treatment of periodontitis. The smokers group included 9 subjects (7 males and 2 females) consuming 20 to 30 cigarettes perday (mean 23 cigarettes) for the past 15 to 25 (mean 19.4) years. Their ages ranged from 35 to 41 years (mean 37.7 years). Seven subjects (4 males and 3 females), age range 29 to 40 years (mean 35 years), abstained from smoking and formed the non-smokers group. The patients had moderate to severe periodontitis as evidencedby penodontal pockets with probing depth of 5mm or more and alveolar bone loss by periapical dental X-rays. They were fully informed of the objectives of the study. Medical history was obtained before taking the biopsy. Subjects with systemic diseases such as tuberculosis, diabetes mellitus, rheumatoid arthritis or cardiac disease were excluded from the study. Gingival biopsy specimens were obtained during routine periodontal surgical procedures and fixed in formalin. Paraffin blocks were prepared and 4-6 mm thick sections were stained with hematoxylin-eosin to study the histological changes. Tissues were also stained with Masson trichrome and Verhoeff Vangieson stains

to examine the connective tissue changes.

Results

Figures 1 to 5 show histopathological analysis of gingival biopsies obtained from cigarette smokers and nonsmokers under light microscope. Epithelium with various degrees of acanthosis and a thick squamous cell layer was noticed (Figures 1 and 2).

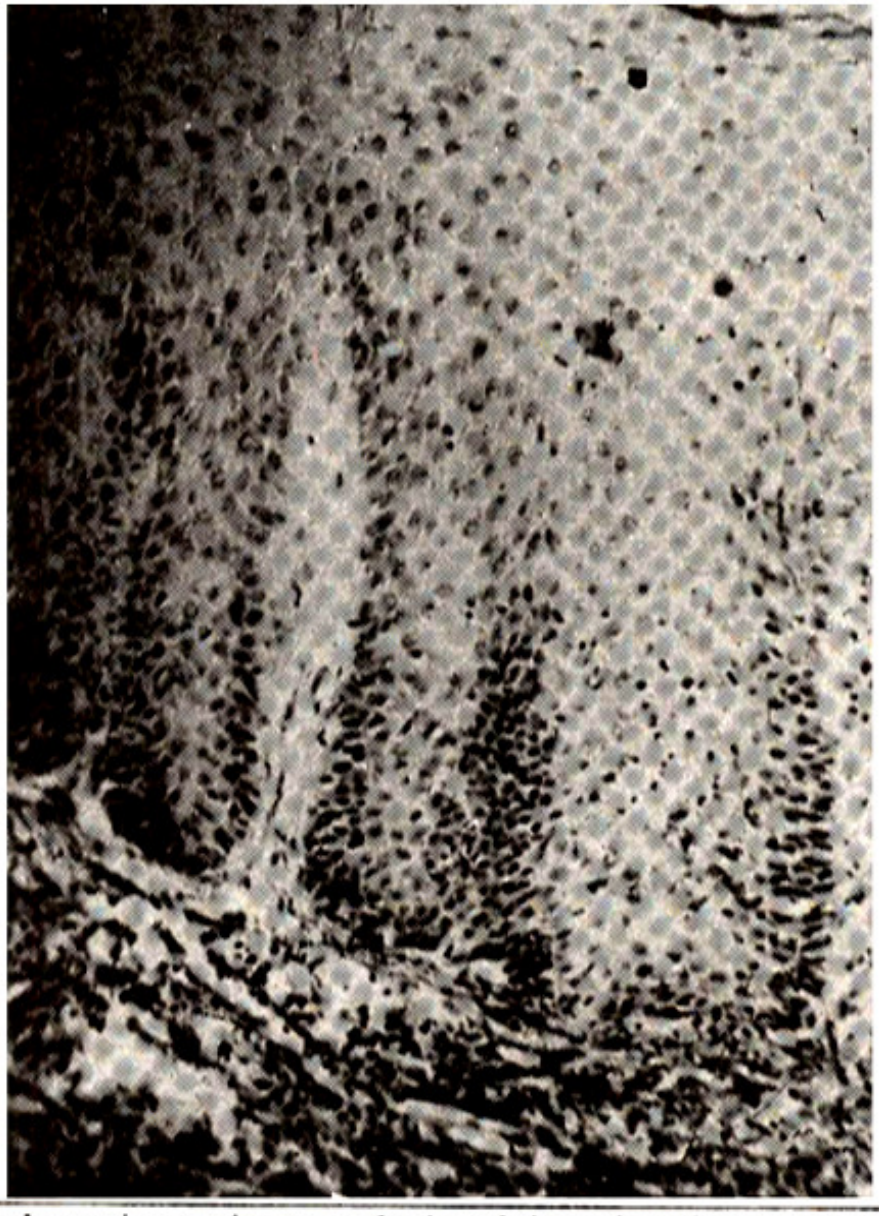

Figure 1. Parakeratosis, prominent acanthosis and elongation of retepegs in the epithelium of one cigarette smoker's gingiva (B- 1416-91)(H+E x 63).

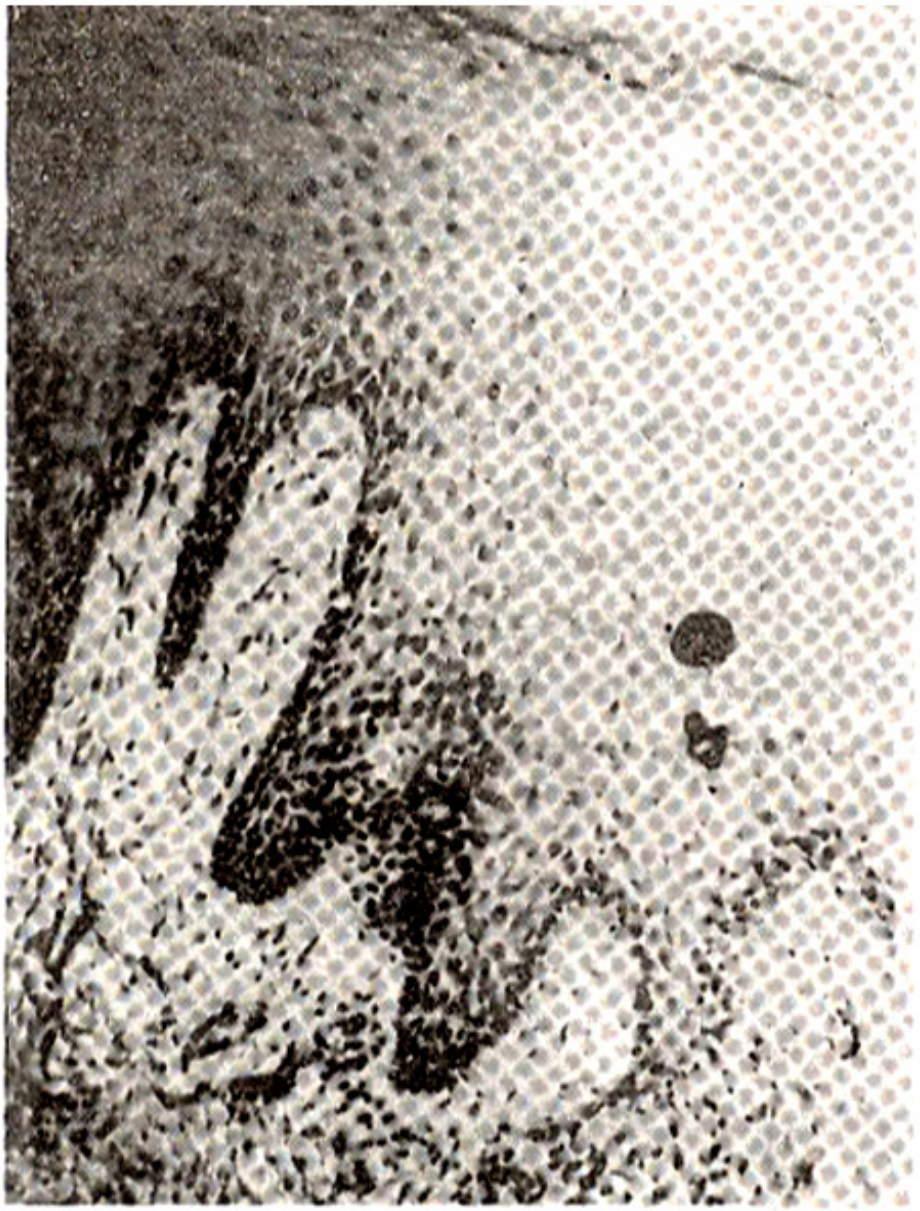

Figure 2. Parakeratosis and prominent acanthosis, elongation of retepegs in the epithelium of one non-smoker's gingiva (B-2079- 91)(H+E x 63).

In some cases the upper layer of epithelium showed vacuolized and enlarged cells along with some structural changes. Various thickness of parakeratosis in the epithelium was also remarkable. Edema and increased number of vessels showing dilatation and congestion appeared in the strome, particularly in the subepithelial region (Figure 3).

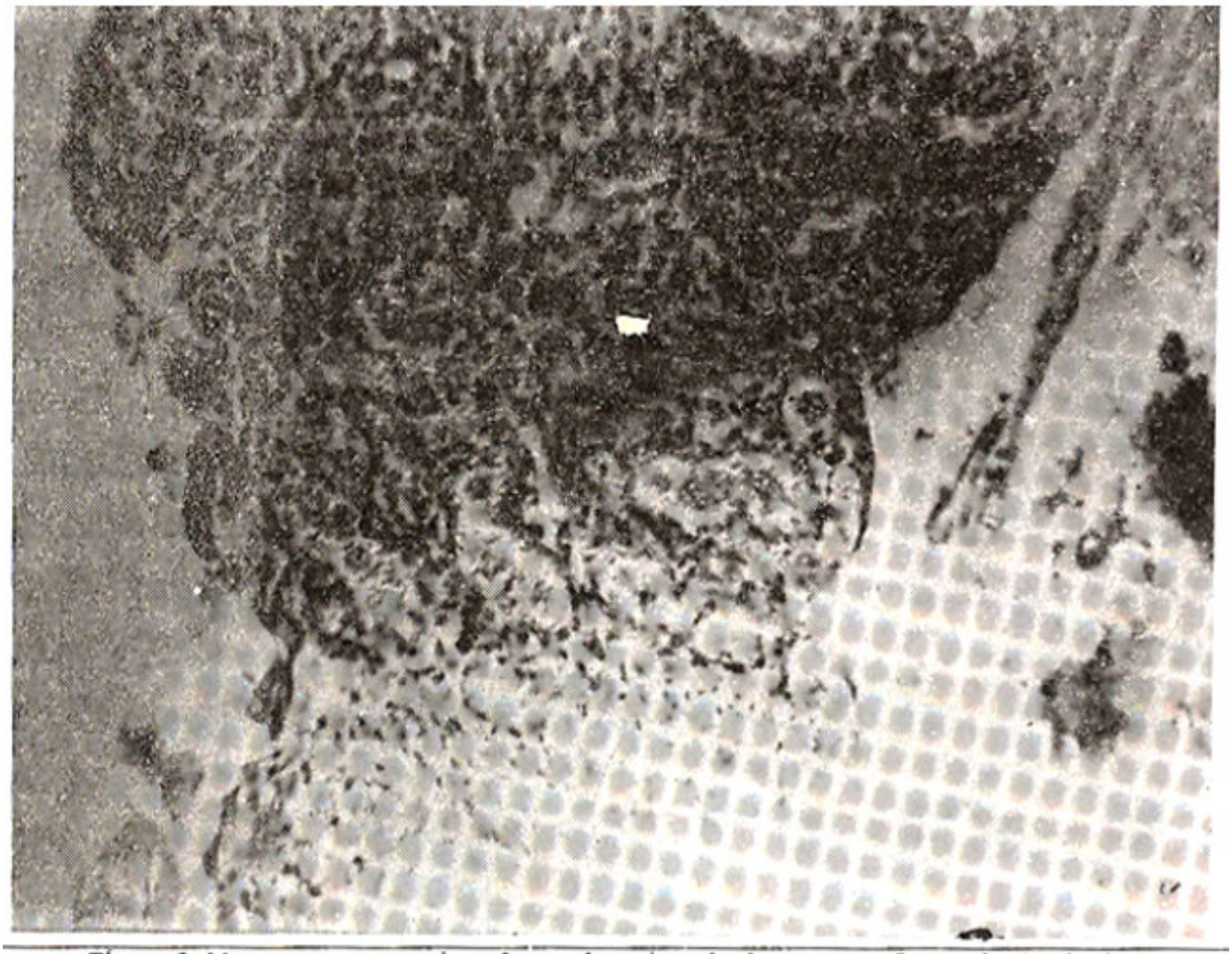

Figure 3. Numerous congestioned vessel sections in the stroma of a smoker's gingiva (B-2076-91)(H+E x 63).

The connective tissue was infiltrated by the inflammatory cells mostly lymphocytes, few plasma cells, polymorphonuclear leukocytes and histiocytes, particularly densely located around the vessels. In some cases these cells appeared diffuse while in the others localized (Figures 4 and 5).

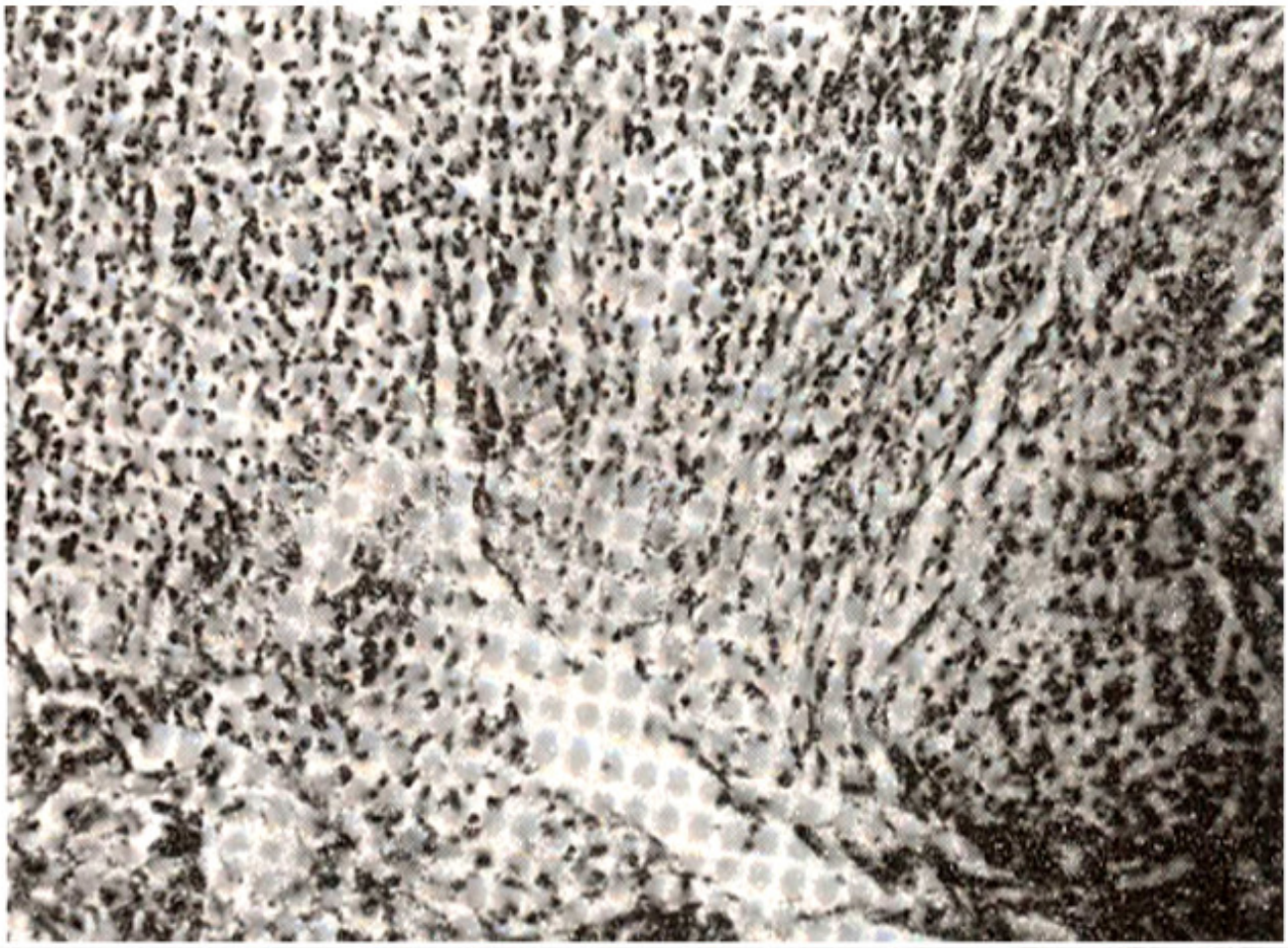

Figure 4. Numerous capillaries and dense mononuclear inflammatory cell infiltrate in the stroma of a smoker's gingiva (B-2076- 91)(H+E x 63).

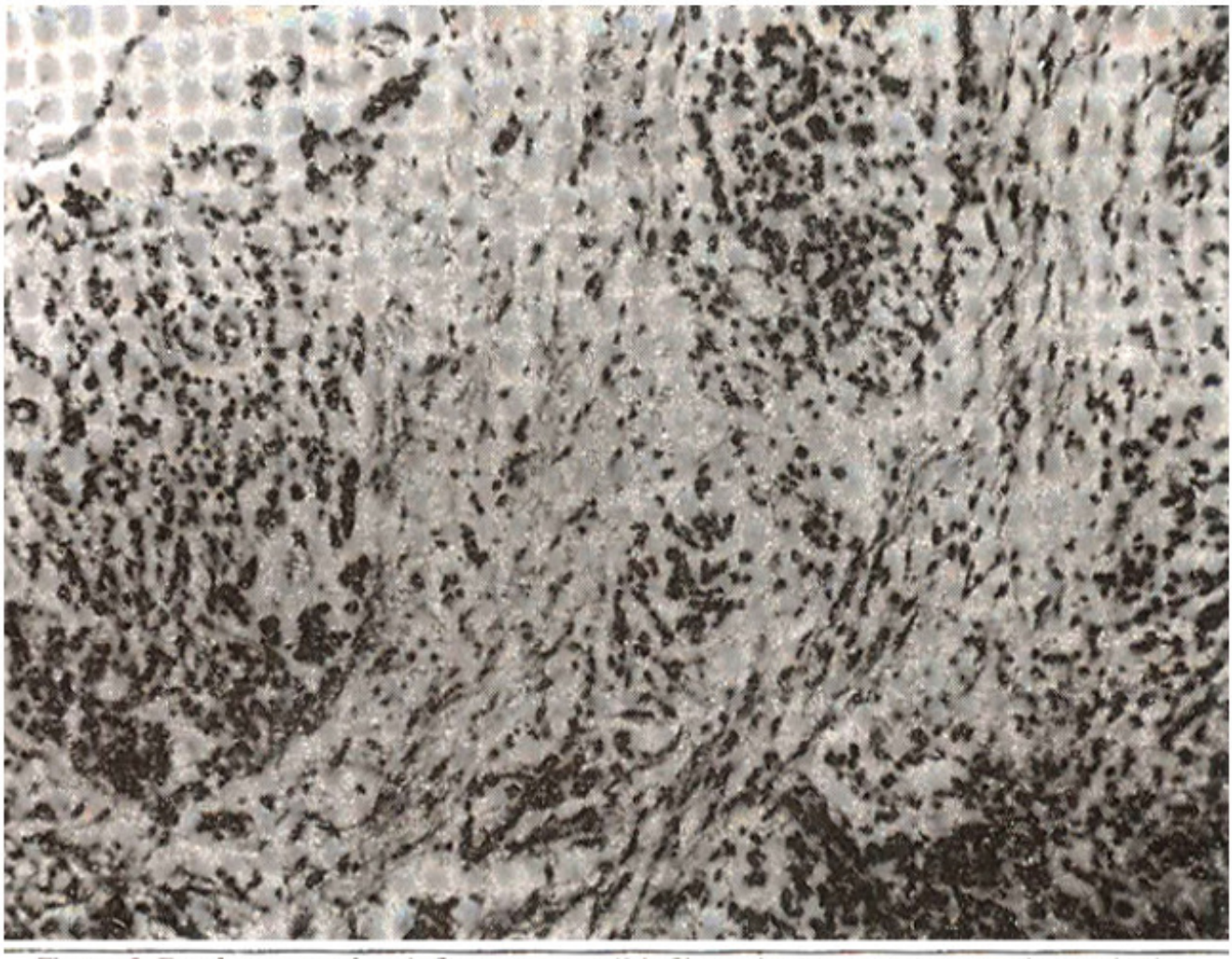

Figure 5. Focal mononuclear inflammatory cell infiltrate in the stroma on smoker's gingiva (B-2079-91)(H+E x 63).

Masson trichrome and. Verhoeff Vangieson staining methods also showed increase in connective tissue. Although no remarkable histopathological difference was noted between the gingival tissues of the cigarette smokers and non smokers, the smokers showed relatively more epithelial keratosis, inflammatory cells and increased number of vessels and collagen fibres in the connective tissue. Congestion of the capillaries were also more prominent in the smokers gingival tissue.

Discussion

There are controversial observations on the effect of smoking on gingival tissue. Some reports indicated that smokers had more gingival inflammation[5], some found no difference2 and others observed less gingival bleeding and inflammation in smokers than non-smokers[17,18]. In this study inflammation was more often seen in smokers. Tobacco smoking can produce oral keratosis which may be associated with malignant transformation[19,20]. In this study also smokers had increased keratosis of the gingival epithelium. Some investigators also found that collagen production was apparently stimulated by nicotine[21]. When collagen fibres were evaluated, we have also found that they increased in connective tissue of smokers gingiva. Nicotine, a major component of the particular phase of tobacco smoke, has been shown to induce vascular changes in gingival tissues[22] similar to the

exudative vasculitis in periodontal inflammation. Some investigators reported that the number of blood vessels found in the marginal gingiva of smokers were less than in non-smokers[23]. Baab and Oberg[14] indicated that cigarette smoking caused a significant increase, rather hana decrease in human gingival circulation. Smoking has divergent effects on blood flow to different parts of the body. It increases blood flow of skeletal muscles[24], intestines and uterus[25] but it decreases flow to fore-ann, skin and hands[26,27].

These different effects of smoking on different parts of the body may be due to the complex responses to the vasoactive hormones released by smoking. In smokers blood flow to the head may be affected in different ways than at the periphery, in order to maintain blood flow to the brain. The increased number and the congestion of the capillaries found in smokers in this study may be a part of that mechanism.

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