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Research Article

Morphological Changes of Gingiva in Str Diabetic Rats

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Abstract

Gingivitis and periodontitis are chronic bacterial diseases of the u mellitus is responsible for tooth deprivation both by decay and diabetes results in a diabetic status in experimental animals simila the study was to investigate the relationship between the ging streptozotocin-induced diabetes mellitus. Forty male Wistar ra experimental). Diabetes mellitus was induced by 45 mg/kg IV s marginal gingival and the relevant gingival papilla showed inflar epithelium as well as marked thickness of the arteriole in the dia control group. The results suggested a probable application of a r patients in order to control the progress of disease complicatio investigation can be used as a routine assay for the control of the

1. Introduction

Gingivitis and periodontitis are chronic bacterial diseases of the u
factor in the development of the periodontal disease is the host
stimuli. Systemic factors modify all forms of gingivitis and perio
immune and inflammatory defense. It has been reported [1] t
inflammation induced by mechanical or chemical irritations in the g
suggested that in diabetes mellitus the unmyelinated small dia
neuropathy. Furthermore, many studies have been focused in th
mellitus. Microangiopathy at the bone tissue was suggested as a
can have an impact on the bone through multiple pathways, s
changes in insulin levels, higher concentrations of advanced g
associated with glycosuria, reduced renal function, lower ins
inflammation [3].

Furthermore, diabetes mellitus is responsible for tooth deprivatio
diseases are associated with a higher experience of caries, a h
gingival and periodontal problems. Patients with high blood pres
have poorer gingival or periodontal conditions, fewer teeth, and hi
found that a larger number of oral *streptococci* adhered to the toc
mice that spontaneously develop insulin-dependent diabetes mellit

The aim of the study was to investigate the relationship betw
presence of microangiopathy in streptozotocin-induced diabetes m

2. Material and Methods

Forty male Wistar rats of average body weight of 200 g were
(control, $n = 10$). They were housed five per cage at a constan
light/12-hour dark (light period 00.8–20.00 hours) cycle. Food a
cared for accordance with the principles of the "Guide for the
animals of group A were injected once IV with streptozotocin 45
experiment was 90 days. The blood glucose levels were estimated
Division, Miles Laboratories, Rexdale, Ontario, Canada) in blood
was also qualitatively assayed in urine with urineteststrips (Glukot
well as their food intake was determined. The animals were s
obtained from the incisor area of the mandible were washed with
for further histological examination with the light microscope. T
stained with haematoxylin-eosin by light microscope. The values
$m \pm SD$). The statistical analysis was performed by Student's *t*-tes
acceptable level of significance.

3. Results

The induction of diabetes mellitus was assessed the day after strep
such as frequent urination, increased appetite, and weight loss. In
animals exerted a hyperphagia accompanied with an increa
streptozotocin animals had increased serum glucose and increas
severity of diabetes was indicated by the statistically significantly
in comparison to controls $P <$,001 (Table 1). The quantity of da
compared to controls. Blood glucose levels were significantly inc
levels of Hb A1c were lower in the control compared to the expe
group was frequent and the shavings of the cages needed to be ch

Table 1: Clinical indices and laboratory finding

The experimental animals had a mortality of 10% during the e remained alive until the end of the experimental procedure.

The histological findings of the experimental group were as follov from the marginal gingivae and the gingival papillae from the incis formation of new vessels with various wall thicknesses, and hyper site of the gingiva, which was in contact with the tooth surface.

The gingival specimens obtained from the molar area of the mandi to high hyperceratosis and mild inflammation. All the experime which was not observed in the control animals (see Table 2, Figure

Table 2: Histological findings.

Figure 1: (□) Focal perivascular and diffus angiogenesis, and (*) hyperplasia of the squan

Figure 2: (*) Hyperplasia of the squamo inflammation of the lamina propria, and (↑) th

Figure 3: (*) Normal gingival mucosa. nor vasculature, and absence of inflammation.

4. Discussion

Recent drastic increase in diabetic population poses serious pro services. The recent drastic increase in the diabetic population p socioeconomic services. The most important issue in the clinica complications that contribute to a high morbidity and mortality [7,

Hyperglycaemia, as a common feature of diabetes mellitus, is a ca endothelial function.

Diabetes mellitus results in the development of large and m independently of the presence of atherosclerosis. The abnormalit

laminin, fibronectin, type IV collagen, and connective tissue with l calcium. It is of particular interest that accumulation of PAS-posit are recognized as the histological markers of diabetic microangiopa

In this study, the administration of streptozotocin in rats induced a to that observed in diabetic patients, as shown by the manifeste reported literature [2, 13, 14].

The changes observed in bed vasculature concerned macro- and r is known to impair the function of various organs and systems and nephropathy, and neuropathy [15]. Periodontal disease is consider has long been observed that diabetic patients have greater tooth l comparable age [16]. The severity of the periodontal disease is ab [17]. The phenomenon may be due to the high tissue glucose cor products of the impaired glucose metabolism and it is recogni narrowing of vessel lumen diameter. This process induces disabiliti vasculature [12, 18]. Our results are in agreement with those o vessel lumen diameter in diabetic subjects as observed by the inc [9].

These changes can be aggravated through inflammatory cell infilt treatment in the gingivomucosal tissue as reported by Fehér et a pathophysiology of diabetic microangiopathy and its pathoge hyperglycemia, early stimuli elicit adaptive reactions of tissues sh and changes of microangiopathy. The impaired glucose metabolism with a narrowing of vessel lumen diameter. This situation in denta extraction sockets, periradical lesions, and periodontal disease [4,

In addition, it has been suggested that the unmyelinated small-dia diabetes mellitus, which indicates that in the streptosotocin-in prerequisite for neurogenic inflammation induced by mechanical o permeability. Furthermore, diabetic changes may be accompanied tissue [21].

This process induces disabilities of vessel wall such as narrowed ve an abnormal vasculature [12, 19]. Since the ability of the dia especially during increased blood flow causing severe disturbances poor nourishment, which, in relation to the high blood glucose l microflora in the oral cavity [22]. The presence of PAS material de index of severe diabetic damage [9]. Therefore, slow flow of r destruction process of the periodondium [23].

An important injury that leads to severe handicap in diabetic patie leads to blindness [24]. Most recently, it has been proven that the in the presence of the periodontal disease [25]. In addition, th diabetes is related to tooth loss and tempomandibular joint disfunc

Therefore, the investigation of the surrounding oral cavity tissue signs that may alert the physician to control or prevent the histological gingival analysis may be routinely utilized for the co considered as a diagnostic method of the severity of the disease.

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