Autoradiographic Analysis of Epithelial Proliferation in Chronic Gingivitis

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Proliferative processes in the gingival mucosa are studied by autoradiography in 55 patients with chronic gingivitis. It is found that the number of DNA-producing epithelial cells does not change. Increased label intensity in chronic catarrhal and hypertrophic gingivitis in comparison with the control testifies to a sufficient compensatory reaction. Regenerative processes are suppressed in chronic atrophic gingivitis. The number of pathological mitoses increases with the development of pathomorphological changes in all studied types of gingivitis.

Key Words: *chronic inflammation; gingival epithelium; proliferation; radioautography*

Proliferative activity of epithelial cells is important for assessing the regenerative potential of the digestive system tissues. The intensity of proliferative processes in the epithelium has been studied in different parts the alimentary canal [4,7,8,12]. It was demonstrated that the rate of gingival epithelium renewal is comparable to that of the lower segments of the alimentary canal, and the content of pathological mitoses in gingival epithelium is consistent with the average values recorded for normal epithelium [4,6,8]. Meanwhile, the data on cell regeneration in the prosthetic bed are scarce and controversial [2,7,9-11,13-15].

We did not find any significant age- or sex-dependent differences in the proliferation of gingival epithelium, although the proliferative processes are more intense in women [6].

In the present study we investigated division of epithelial cells in chronic gingivitis.

MATERIALS AND METHODS

Cellular proliferation was studied in bioptates obtained from 55 patients (24 men and 31 women over 40 years) with chronic diseases of the parodontium. Diagnosis was based on pathomorphological studies. Nineteen patients (9 men and 10 women) had chronic catarrhal gingivitis (CCG), 19 patients (8 men and 11 women) had chronic hypertrophic gingivitis (CHG), and 17 patients (7 men and 10 women) had chronic atrophic gingivitis (CAG). Bioptates obtained from 17 individuals (8 men and 9 women of the same age) who did not wear dentures and had no pathological changes in the parodontium served as the control. The classification of parodontosis [3] was used for diagnosis.

Bioptates (3×3 mm pieces of mucosa) were obtained upon extirpation of teeth or roots, fixed in Carnoy's fluid, processed by standard methods, and sections were stained with hematoxylin and eosin.

The bioptates were incubated in a thermostat at 37°C for 60 min in medium 199 containing 5 μCi/ml ^{3}H-thymidine. Radioautographs were prepared and the index of labeled nuclei (%) and the number of silver grains over the nucleus (label intensity) were calculated as described [2,6]. For determination of mitotic parameters the bioptates were fixed in cold Carnoy's fluid for 1 h, and paraffin sections were stained with hematoxylin. Mitotic index (‰) was determined in 5000 cells at magnification 900. The content of pathological mitoses (%) was determined according to classification [1].

The data were analyzed by the methods of variational statistics.

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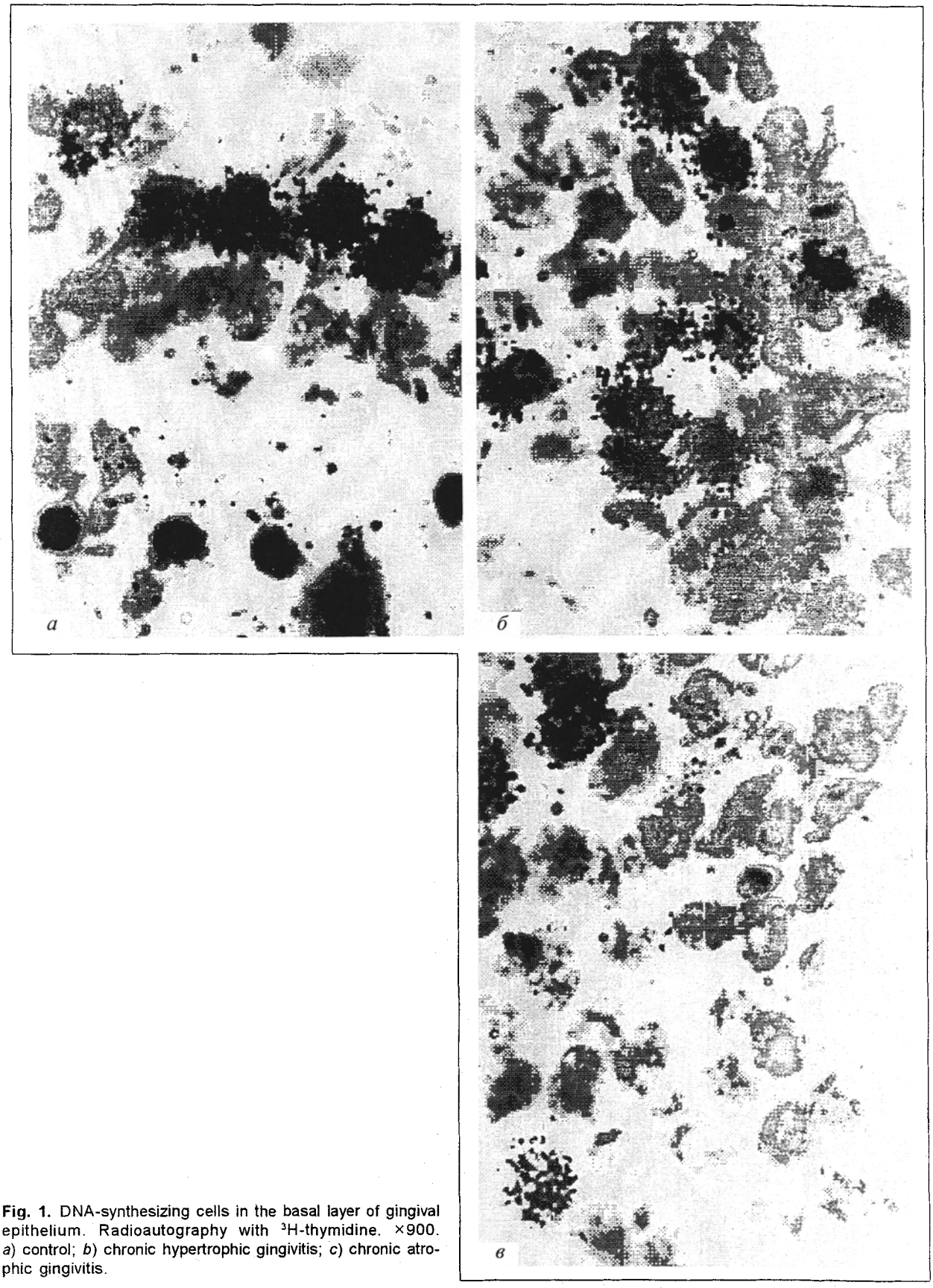

Fig. 1. DNA-synthesizing cells in the basal layer of gingival epithelium. Radioautography with ^{3}H-thymidine. ×900. *a*) control; *b*) chronic hypertrophic gingivitis; *c*) chronic atrophic gingivitis.

RESULTS

The rate of DNA synthesis in the basal epithelium cells increases in CCG and CHG and decreases in CAG in men and women compared with the control (Table 1). There were no statistically significant changes in the index of labeled nuclei, a parameter reflecting the number of DNA-synthesizing cells.

The mitotic index increased both in male and female patients; however, the difference from the control was statistically significant only in men with CAG and in women with CHG and CAG. The number of pathological mitoses also increased (Table 1).

It should be noted that all parameters of cell proliferation in men were lower than in women (Table 1). This points to higher intensity of regeneration in women and is probably associated with sexual dimorphism of gingival epithelium (Fig. 1).

Taking into consideration that the index of labeled nuclei was the same in men and women, it can be suggested that the differences in mitotic index are be due to asynchronous mitoses.

Increased label intensity at a relatively constant index of labeled nuclei attest to sufficient compensatory-adaptive reactions in CCG and CHG, while decreased label intensity points to suppression of reparative processes in CAG. Our data indicate that the maintenance of tissue homeostasis in gingival epithelium is due predominantly to modified rate of DNA synthesis, which may be typical of gingival mucosa. It was shown that in other segments of human and animal alimentary canal tissue homeostasis in the epithelium is maintained against the background of high label intensity and index of labeled nuclei [5,8,10].

A correlation between severity of gingivitis, intensity of morphological changes, and intensity of cell division has been established. This indicates that the ability to adequate adaptive reaction of gingival epithelium is preserved against the background of enhanced inflammatory-dystrophic processes. Increased mitotic index at a relatively constant index of labeled nuclei points to impaired differentiation of cells and their intense extrusion in chronic gingivitis. At the same time, increased mitotic index may be indicative not of the real increase in the number of dividing cells but of a prolonged mitosis. This suggestion is indirectly confirmed by a high level of pathological mitoses. It is noteworthy that the number of pathological mitoses increases considerably (1.5-2-fold) with the development of pathological changes during CCG, CHG, and CAG. This may lead to the accumulation of cells with karyotypic imbalance and the emergence of atypical cells in CAG. An increase in the level of pathological mitoses in this case may be caused by a significant decrease in the rate of DNA synthesis in epithelial cells. Presumably, the decrease in the label intensity reflects lowered activity of reparation in the cells, including reparation of mitotic apparatus. Thus, the amount of DNA-producing cells in gingival epithelium does not change in chronic gingivitis. This indicates that the reparative potential of epithelium

TABLE 1. Parameters of Cellular Proliferation in Human Gingival Epithelium in Various Forms of Chronic Gingivitis ($M \pm m$)

Parameter, sex	Control	CCG	CHG	CAG
Label intensity				
M	23.88±1.03	24.43±1.18	27.83±1.31*	17.05±0.73*
F	25.72±1.01	29.02±1.61	32.00±1.26*	20.90±1.59*
Both sexes	25.01±0.98	26.38±1.06	29.50±1.06*	21.31±1.26*
Index of labeled nuclei, %				
M	6.62±0.54	6.87±0.60	6.76±0.62	7.42±0.66
F	8.99±0.87	9.60±0.76	10.63±0.82	8.41±0.81
Both sexes	7.52±0.73	8.10±0.72	9.24±0.63	7.95±0.76
Mitotic index, ‰				
M	9.07±0.84	10.16±0.73	10.76±0.93	11.95±1.04*
F	13.30±0.69	12.87±0.61	15.91±1.02*	16.39±0.98*
Both sexes	11.71±0.56	11.78±0.59	14.03±0.77*	14.58±0.89*
Pathological mitoses, %				
M	4.97±0.35	5.51±0.44	6.76±0.46*	7.81±0.81*
F	6.37±0.42	6.46±0.52	8.42±0.54*	12.31±0.89*
Both sexes	5.91±0.31	6.10±0.44	7.14±0.54*	9.89±0.72*

Note. *$p<0.05$ in comparison with the control.

is preserved, although in some cells reparative processes are impaired.

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