PHARMACOLOGY AND TOXICOLOGY

Richlocaine in Combined Therapy of Periodontitis

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The results of combined therapy of chronic moderate generalized periodontitis can be improved by adding richlocaine to protocols of drug therapy: the drug reduces the time of treatment, prolongs remission, decreases the number of relapses, and stabilizes pathological processes in periodontal tissues.

Key Words: *richlocaine; periodontitis; combined therapy*

Improvement of the efficiency of prevention and treatment of periodontitis is an important problem of modern dentistry. High prevalence, different severity of clinical course, early loss of teeth, decrease of working capacity, and deterioration of the psychoemotional sphere determine medical and social significance of this problem [5].

Despite a great variety of drugs used now for the treatment of periodontal and buccal mucosa diseases, it is sometimes impossible to attain stable remission and stimulate the regeneratory processes in periodontal tissue. This necessitates the search for highly effective drugs for etiotropic, pathogenetic, and symptomatic therapy of periodontal diseases.

Bacteria play an important etiological role in the development of periodontal inflammations. Toxins and enzymes released by microorganisms activate cellular and humoral mediators and modulators of inflammation increasing vascular wall permeability, edema, and painful sensations.

The use of drugs with "double" (analgesic and antibacterial) effect in combined therapy of periodontitis attracts special interest in dentistry [2,4]. Richlocaine (1-allyl-2,5-dimethylpiperidol-4 benzoic ester hydrochloride), possessing local anesthetic, analgesic, antibacterial, antihistaminic, dermatoprotective (improving survival of tissues under conditions of reduced circulation), and immunocorrective effects, was synthesized at Laboratory of Organic Synthesis (Al' Farabi Kazakh State University) by Professor Sh. S. Akhmedova [1,3].

We studied the efficiency of richlocaine in combined therapy of periodontitis.

MATERIALS AND METHODS

Fifty-four patients aged 36-69 years (main and control groups) with chronic generalized periodontitis of medium severity (CGPMS) lasting for 5-20 years were examined and treated. The patients with irreparable occlusion disorders or abnormalities, few natural teeth (less than 6 per jaw), and parafunction of the masticatory muscles were excluded. The main and control groups were matched by age, sex, forms of periodontitis, other characteristics.

The main group (richlocaine therapy) consisted of 33 patients with CGPMS during exacerbation, of these 2 women and 3 men aged 36-39 years, 5 women and 4 men aged 40-49 years, 10 women and 9 men aged 50 and older. Six patients had a history of somatic diseases: chronic pharyngitis, chronic cholecystitis, peptic ulcer, and essential hypertension.

Control group (no richlocaine) consisted of 21 patients with CGPMS during exacerbation, of these 2 women and 1 man aged 36-39 years, 4 women and

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3 men aged 40-49 years, 7 women and 4 men aged 50 years and older. Four patients had a history of somatic diseases: peptic ulcer, essential hypertension, and coronary disease.

All patients were examined before, during, and after therapy by routine methods used in dentistry, including basic and accessory methods of examination (determination of the index of periodontal tissue status, evaluation of pathological mobility of teeth, *etc.*). Clinical efficiency of treatment was evaluated on day 15 of treatment and 1 and 6 months after it.

Objective data on patients with CGPMS included the presence of inflammations of the buccal mucosa and gingiva: color, relief, hyperemia, painfulness, edemas, stomatorrhagia, presence of fistulas, ulceration, abscesses, alteration of gingival configuration, hypertrophy and tightness of adhesion of interdental gingival papilla and gingival edge to the teeth, and denudation of the tooth necks.

Dental deposits were detected by staining with 1-2% methylene blue. Periodontal status was evaluated using periodontal (PI) and papillary-marginal-alveolar (PMAI) indexes. The status of periodontal tissues and efficiency of treatment were evaluated using Schiller—Pisarev, Ketchke, and Parm functional tests.

Sanitation of the oral cavity, repair of elements of traumatic occlusion of the periodontium and bacterial deposit retention, and revision of periodontal pouches were carried out in all patients, and routine drug therapy was prescribed. Immobilization (splinting and/or orthodontic treatment) or removal of mobile teeth were carried out if necessary.

Patients of the main group received, in addition to traditional therapy, a course of richlocaine injections under the buccal mucosa into the transitional maxillo-mandibular fold (0.2 ml 0.25% solution, for 10-14 days). In parallel, richlocaine (5% ointment) was applied (on turundae) into periodontal pouches and applications of 0.5% solution of this drug to the gingival mucosa (20 min) were made daily.

The treatment efficiency was evaluated by X-ray methods: intraoral spot roentgenography of the teeth and orthopantomography of the jaws using Cranex dc 2 SL-4/PT-10 (Soredex) device under the same physical and technological conditions.

X-ray studies were carried out before and 6 months after the end of combined treatment. The time course of changes and severity of pathological processes in bone tissue were evaluated by densitometry of roentgenograms using VideoTest-Master software (Morfologiya). Leveling of optical density of scanned X-ray images was carried out using Adobe Photoshop software (version 7.0). The data were analyzed using Excel applied software.

RESULTS

The treatment led to positive changes in both groups, but the results were better in the main group, in which traditional drug therapy for CGPMS was supplemented by richlocaine.

Acute inflammatory processes were arrested in patients of the main group by the second or third session. The patients reported alleviation of edema, stomatorrhagia, painful sensations, and the absence of unpleasant odor. The sensitivity to thermal and chemical irritants decreased. By day 15 no complications associated with the treatment were observed. No signs of abnormalities in the gingival mucosa were seen: it had natural color, relief, and shape, palpation was painless; probing of the gingival pouches caused no bleeding, gingival papillae were seen between the teeth, and the gingiva tightly adhered to hard dental teeth in the pericervical area of teeth. The amount of exudation in the periodontal pouches sharply decreased. Pathological mobility of teeth (I-II degree before therapy) decreased and approached the normal level. Periodontal indexes decreased and corresponded to the normal mean values (Table 1). Schiller—Pisarev, Parm, and Ketchke tests were negative in all patients of this group (before treatment Schiller—Pisarev test was positive and Parm and Ketchke tests were equal to 28.9 and 26.5%, respectively).

In the controls acute inflammatory processes were arrested by sessions 5-7. Pain, edema, and stomatorrhagia gradually decreased. By day 15 of observation the treatment was not associated with any complications. Exudation from the gingival pouches and pathological mobility of teeth decreased. However more than half of the patients had signs of pathological changes characteristic of periodontitis: partially

TABLE 1. Time Course of PMAI and PI in Patients with CGPMS Treated by Traditional Therapy Supplemented with Richlocaine ($M \pm m$)

Parameter	PMAI, %	PI
Before treatment	39.17±3.76	3.19±0.12
Control group		
on day 15	2.96±0.12*	1.29±0.08*
after 1 month	2.98±0.07*	1.30±0.27*
after 6 month	2.99±0.47*	1.32±0.06
Main group		
on day 15	6.27±1.02*	1.87±0.7*
after 1 month	7.35±1.34	2.37±0.89*
after 6 month	9.61±1.01*	7.44±1.73*

Note. Here and in Table 2: *$p<0.05$ compared to values before treatment.

TABLE 2. Densitometry of Roentgenograms in Patients with CGPMS Treated with Richlocaine in Addition to Traditional Therapy (*M*±*m*)

Parameter	Control group		Main group	
	before therapy	after 6 months	before therapy	after 6 months
Mean value	27.64±2.03	40.17±4.81*	27.41±1.97	48.39±3.91*
Standart deviation	4.12±1.07	5.70±0.33*	4.08±0.09	6.63±0.12*
Median	25.00±3.11	31.42±2.90*	26.00±2.82	49.00±3.04*
Pixels	14,567.00±154.28	21,084.00±205.64*	14,538.0±137.5	26,873.00±214.76*

hyperemic mucosa round dental necks, sometimes with hemorrhages; the gingival papillae were not always formed; tight adhesion of the mucosa to hard dental tissues was observed in just few patients. Periodontal indexes reduced (Table 1). Schiller—Pisarev test was weakly positive in 2 (9.53%) patients, Parm and Ketchke tests were negative in all patients.

No signs of chronic inflammation were detected 1 and 6 months after combined therapy in the main group. The patients had no complaints, gingival mucosa was pale pink, palpation was painless, the gingival edge relief was completely restored and tightly adhered to hard dental tissues in the pericervical part and between the teeth. Exudation from the periodontal pouches was absent, the depth of pouches decreased and was 2.27±0.34 mm (*vs.* 3.85±0.42 mm before therapy). No pathological mobility of teeth was detected. Pathological recession of the gingiva was 0.1-0.2 mm. Improvement of periodontal tissue status was confirmed by positive changes in PMAI and PI (Table 1). Schiller—Pisarev, Parm, and Ketchke tests were negative in all patients of this group.

Of 21 patients of the control group 12 (57.2%) patients still complained of tooth mobility, stomatorrhagia during tooth cleaning and eating hard food 1 and 6 months after a course of therapy. Objective examination of the oral cavity showed initial signs of inflammatory process in 9 (42.9%) patients; 2 (9.5%) patients had exudation from periodontal pouches (whose depth was 3.47±0.24 mm). First-degree pathological mobility of teeth was observed in 12 patients; pathological recession of the gingiva was 0.3-0.7 mm. Positive shifts in periodontal indexes were observed during and after treatment (Table 1). Schiller—Pisarev test was weakly positive in 12 patients, Parm and Ketchke tests were positive in 9.52 and 4.76% patients, respectively.

X-ray picture of CGPMS patients before treatment was characterized by widening of the periodontal fissure, destructive changes in bone tissue at the site of interalveolar septae (up to half of the root length), formation of osseous pouches, and osteoporosis.

During and after therapy (up to 6 months) a positive trend to stabilization of destructive processes of the alveolar processes in bone tissue, no new foci of bone destruction and bone pouches were observed in both groups. However X-ray images show more active reparative processes of bone structures in the main group in comparison with the control. The contours of the interface of destroyed alveolar septae between teeth were better discernible, the lumen in the periodontal fissure decreased significantly, foci of osteoporosis disappeared, and a trend to osteointegral rearrangement of bone structure was seen.

Densitometry of roentgenograms showed different optical density of bone structure of the alveolar processes in the main and control groups. More pronounced positive condensation of bone structure of the alveolar septae (concerning both the septae between the teeth and the body of the jaw) was observed in the main group in comparison with the initial X-ray images. In controls optical density of bone structure changed negligibly in comparison with the initial level (Table 2).

Hence, richlocaine added to traditional therapy for CGPMS improved treatment efficiency, prolonged remission, decreased the number of relapses, and stabilized pathological processes in the periodontal tissue.

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