

Each medication class has specific potential adverse effects on the cornea and ocular surface. Prostaglandin analogs are associated with both a higher prevalence and severity of obstructive meibomian gland dysfunction. Furthermore, prostaglandin analog therapy was shown to cause a higher rate of meibomian gland dysfunction in patients already receiving non-prostaglandin analog ocular hypotensive therapy, possibly worsening ocular surface disease. Beta blockers act on beta receptors in the lacrimal gland reducing basal tear turnover rate. Timolol has been found to alter the mucus composition in the tear film and also cause increased staining of the cornea and conjunctiva after one month of therapy. The commonly used alpha-adrenergic agonist brimonidine tartrate has a significantly higher incidence of ocular allergy compared to other topical medications and may predispose patients to ocular allergy from additional topical antiglaucoma drops. The carbonic anhydrase inhibitor dorzolamide has been found to increase corneal thickness, but the effect of dorzolamide on the corneal endothelium is still in question.

All patients underwent a clinical examination, Schirmer test, Norn test, vital staining with fluorescein and the functional state of the meibomian glands.

The signs of the “dry eye” syndrome were detected in 70.6% (42 eyes). Of these, 23.6% of patients (13 eyes) had meibomian gland dysfunction confirmed by Norn tests – 9.7 ± 0.1 . Dry eye features were seen as primary complaints of dry eye (foreign body sensation, burning, stinging, dryness, soreness, and heaviness of the lids, photophobia, or ocular fatigue). The result of Schirmer test (20.7 ± 0.8 mm) allows us to conclude the presence of hypersecretion as the initial manifestation of “dry eye”. The main changes pertained to the condition of the lipid layer of the tear film: an irregular thickness with normal thickness limited only to some small areas. When prescribing treatment for the “dry eye” associated with primary open-angle glaucoma, it is important to correct the defects of the lipid layer of the tear film. Using warm lid compresses and scrubs may be helpful. Dry eye symptoms may be treated with over-the-counter medications such as artificial tears, gels, and ointments.

In conclusion, our results show that a significant proportion of dry-eye patients have a coexisting glaucoma.

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TREATMENT AND PROPHYLAXIS OF PARASITIC BLEPHARONCONJUNCTIVITIS

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The cause of parasitic blepharoconjunctivitis is ticks of the genus *Demodex* - opportunistic pathogens (present in 90% of the adult population). In conditions of reduced immunity, under the influence of adverse external conditions and internal factors, diseases of the nervous, vascular, endocrine and digestive systems, metabolic disorders, demodicosis occur. Asymptomatic carrier of the parasite is possible. The tick parasitizes in the ducts of the sebaceous, meibomian glands and hair follicles. Only drug therapy of demodicosis is ineffective, as only the most superficial ticks die.

We use a comprehensive approach to the treatment of demodicosis blepharoconjunctivitis by sequential application of Spregal or Stop demodex gel on the skin of the eyelids and subsequent darsonvalization of the eyelids. The drugs should be applied to the front edge of the eyelids using an ear stick, without getting on medicine on the mucous membrane of the eye.

The method of darsonvalization has the following therapeutic effects: acaricidal and bactericidal – due to the action of spark discharge and ozone generated in the near electrode space of the apparatus for darsonvalization; analgesic and antipruritic effects – by increasing the sensitivity threshold of pain and tactile exteroceptors; immunostimulating effect also due to the action of a spark discharge, which stimulates phagocytosis, and the release of biologically active substances that stimulate the humoral part of the immune system.

Using this technique in the period of 2012-2020, we treated 56 patients using gel “Stop demodex” and 23 patients using Spregal. The course of darsonvalization with the specified means lasted 10 days with the subsequent break for two weeks and repeated treatment. This treatment regimen corresponds to the full life cycle of the mite (15 days), as all treatments act only on adults

ticks. Itching, swelling and redness of the eyelids after the first course of treatment decreased in 96.5% of patients using Spregal. If at primary eyelash microscopy in the microscope slide were revealed 8-15 ticks in the investigated area, then after the first course of treatment with Spregal their number decreased to 1-2 in the investigated area. After re-treatment, the percentage of negative microscopic eyelash tests approached 100%. Almost similar data were obtained when combining darsonvalization with topical use of gel "Stop demodex".

Darsonvalization of the eyelids gives a good therapeutic effect. This method involves contacting specific agents with the maximum number of parasites, even deep ones. In our opinion, the spark charge, due to the action on smooth muscle cells of meibomian and sebaceous glands, stimulates the release of their secretion together with the demodex mite, which is exposed to specific drugs previously applied to the skin.

To prevent recurrence of exacerbations of the disease, we recommend daily regular therapeutic eyelid hygiene. For this purpose it is necessary to carry out self-massage of eyelids about 1-2 minutes after a warm compress. The compress is usually performed using cotton swabs, immersed in hot water, squeezed and applied to closed eyelids for 1-2 minutes. Thermal procedures help to improve local metabolic processes and drain the excretory ducts of the meibomian glands.

Self-massage is performed after applying an indifferent eye gel to the eyelash growth area, which helps to clean the surface of the eyelids from toxic agents, scales and crusts.

Our proposed new combined method of treatment of demodicosis blepharitis by sequential application of specific drugs Spregal or Stop Demodex gel on the skin of the eyelids and subsequent darsonvalization of the eyelids is an easy-to-use, affordable and effective way to treat demodicosis.

Daily observance of therapeutic eyelid hygiene (self-massage with a cleansing gel after warm compresses) can significantly reduce the likelihood of exacerbation of demodicosis blepharoconjunctivitis.

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POLYMORPHISM N34S OF THE SPINK1 GENE IN PATIENTS WITH ACUTE PANCREATITIS

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The course of acute pancreatitis is stipulated by one and the same factor. An important role is played by genetically determined defence mechanisms aimed at preventing an intrapancreatic activation of enzymes. One of such basic mechanisms is the neutralizing effect of the secretory pancreatic trypsin inhibitor (the serine protease inhibitor of Kazal's type I - SPINK1).

The research involved 37 people with different forms of acute pancreatitis. Among them there were 25 men (67.6%) and 12 women (34.2%). An average age of the patients was $48 \pm 14,4$ years. The patients were divided into 2 groups. The first group included 17 patients with acute edematous pancreatitis. The second group comprised 20 patients with acute necrotizing pancreatitis.

The length of the amplicate of N34S polymorphism of the SPINK 1 gene consisted of 320 pairs of nucleotides (pn). In the presence of the 3rd exon of the nucleotide sequence of the mentioned gene of adenine in the 34th codon, the amplification splits by PstI restrictase into fragments, measuring 320 and 286 pn. In case of transversion A - G the site for PstI restriction was lost.

The presence of the favourable "wild - type" N - allele ("wild - type", Wt) was detected in the majority of the subjects – in 73,0% cases (27). The pathological "mutant" S – variant was identified in 27,0% of people (10). Also, there were 45.9% of the cases (17) of homozygous carriers of the "wild" NN - genotype (N34), NS - heterozygotes (N34S) - 51,4 % of the cases (19). One patient (2,7%) was a homozygous carrier of the mutant S - allele (SS - genotype, 34S). A distribution of the genotypes according to the polymorphic N34S variant of the SPINK1 gene among the examinees corresponded to expected Hardy – Weinberg's equilibrium ($p > 0,05$).

On distributing all the patients according to the etiological agent it was found out that the frequency of the NN - and NS - genotypes in patients with biliary pancreatitis involved 52,6% (10)