

This retrospective, observational, comparative study included 38 patients (38 eyes) who underwent concomitant phacoemulsification with posterior chamber intraocular lens implantation and high-speed (10,000 cuts per minute) 25-gauge transconjunctival sutureless PPV with segmentation and removal of combined fibrovascular membranes. After removal of the vitreous gel at the vitreous base, vitreous base shaving was performed under scleral depression, and blood clots in the peripheral vitreous skirt were also removed using this process. At the end of each surgery, air-fluid exchange was always carried out, and endolaser treatment and endotamponades were performed when required. In group 1 (20 patients, 20 eyes) after the air-fluid exchange, 0.05 ml 0.05% of tranexamic acid was injected from the limbus via the pars plana using a 30-gauge needle. For standard cases, we used air as endotamponade (9 eyes) but for complicated cases (intraoperative retinal tear and extensive fibrovascular tissue dissection), gas (C2F6) endotamponade (19 eyes) or silicone oil (10 eyes) was used. Topical antibiotics and steroids were prescribed postoperatively. The gas volume reduced to about 30% of the vitreous cavity 3 days after surgery and was reabsorbed completely in 10 days. Main outcome measure was the occurrence of recurrent early vitreous hemorrhage. Time points for postoperative examinations were first day, first week, and first month.

Patient characteristics were similar between both groups (intravitreal tranexamic acid and control (group 2)) at baseline and no statistically significant differences were noted between the groups. All 38 eyes were given intravitreal aflibercept within 10 days prior to surgery. On the first postoperative day the rate of rebleeding in the intravitreal tranexamic acid group was 10.0% (2 eyes), which is significantly lower than the control group (38.8%, 7 eyes, $p < 0.05$). The incidence of early manifest PDVH in first week was also significantly lower in the intravitreal tranexamic acid group than the group 2 ($p < 0.05$). Later, one month after a surgery, PDVH occurred in 3 eyes (15.0%) in group 1 and 7 eyes (38.8%) in group 2. So, the incidence of PDVH in the intravitreal tranexamic acid group was significantly lower than the control group.

So, as conclusion, we can suggest that intravitreal tranexamic acid is effective in reduction of the probability of early postoperative diabetic vitreous hemorrhage occurring in patients who received pars plana vitrectomy with removal of fibrovascular membranes due to PDR and may be recommended in clinical usage as a new method of prevention of postoperative vitreous hemorrhages.

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DRY EYE SYNDROME IN PATIENTS WITH GLAUCOMA

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Glaucoma is the second leading cause of blindness in the world and is expected to affect 79.6 million people by 2022.

Glaucoma medications can be associated with toxicities to the ocular surface, most often due to the nature of the preservative included in the medication; however, the incidence of toxicity can be mitigated by the use of preservative free medications, decreased preservative medications, or treatment of dry eye disease.

The aim of the study was to confirm the previously reported association between use of primary open-angle glaucoma eyedrops and corneal staining, suggesting that medications or both are damaging directly the ocular surface.

Materials and methods included the treatment of 30 patients (60 eyes): 20 women, 10 men aged 66 to 91 years (average age 68.5 ± 9.6 years old) with primary open-angle glaucoma. Glaucoma duration varied from 3 to 15 years. All patients were observed on the base of Chernivtsi regional hospital (Chernivtsi). The patients were administered the following topical hypotensive drops: prostaglandin analogues (Bimatoprost, Latanoprost, Tafluprost, Travoprost), α -adrenergic antagonists (nonselective and selective), selective α agonists (Brimonidine), carbonic anhydrase inhibitors (Dorzolamide, Brinzolamide) or combination of two of them.

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