cells and produce recurrent epithelial ulcerations, chronic stromal ulcers, deep stromal vascularization and corneal perforation, in this way leading to blindness. Acid ocular burns are produced by: sulfuric acid (battery acid, industrial cleaner), acetic acid (vinegar), hydrochloric acid (chemical laboratories), sulfurous acid (bleach, refrigerant, fruit and vegetable preservative). Alkali burns: ammonia (fertilizers, refrigerants), lye (drain cleaner), lime (plastic, mortar, cement, whitewash), potassium hydroxide (caustic potash), magnesium hydroxide (sparklers, incendiary devices). In our case, patient's burn was due to salicylic acid (a component used for preparing drops to treat dermatomycosis).

Case report. A 42 y.o. male presented to our clinic with right eye pain, redness and decrease of visual acuity for two weeks, when he accidentally instilled a drop of topical dermatomycosis medication (wich contains salicylic acid of 10%, ethanol 3%, phenol 1% and preservatives) considering it as artificial tears. That led to severe ocular pain, irritation, watering and photophobia. Clinical examination revealed: VA OD/OS = 0.01/0.67; at slit lamp biomicroscopy - diffuse conjunctival congestion with corneal epithelial defect of 6×5.7 mm involving the central visual axis with swollen rounded edges and surrounding area of corneal edema. After saline wash, the patient started on topical moxifloxacin 0.5%, dexamethasone 0.1%, vitamin C drops, hydroxy propyl methyl cellulose 0.3%, and carboxymethylcellulose gel 1% along with oral doxycycline 100 mg and vitamin C 500 mg., subconjunctival autologous serum and 2 amniotic membrane transplantation were performed. The defect healed leaving behind a macular corneal opacity after a period of 10 weeks, VA OD= 0,16.

Conclusions. 1. Salicylic acid and phenol are frequently used for most dermatological drugs. They affect the ocular surface, causing chemical burns. 2. The release of dermatological drugs similar to ophthalmic solutions in vials is a risk factor for confusing them, especially if the patient has visual impairment, is mentally deficient, or simply is in a hurry. 3. The treatment of chemical burns is very challenging and often ends with blindness.

Key words: ocular trauma, acid burn, corneal transplant

48. SURGICAL TREATMENT IN INDUCED OCULAR HYPERTENSION IN RABBIT

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Background. Nowadays, glaucoma imposes a major issue for public health, representing the second worldwide leading cause of blindness (WHO Resnikoff 2002). It is a group of complex and heterogeneous ocular diseases, characterized by progressive optical atrophy (Almasieh 2012; Yanoff, 2014; Salmon, 2020). Glaucomatous damage is irreversible; therefore understanding its pathology and selection of optimal management minimizes the risk of progression and development of visual loss. That is why the researches continue. We report a case of filtration surgery treatment in experimentally induced ocular hypertension.

Case report. Since for reliable tonometry in awake rabbits, it is advisable to keep the animals as quiet and unfrightened as possible, avoiding excessive manipulation and stimulation, we thought of using Tono-PenXL© Reichert at New Zealand rabbit and to take the measurement of normal IOP (intraocular pressure) after surface anesthesia. An ocular hypertension model in

rabbit was induced by using a model proposed by Hester (1987), Melena (1997), just because using other proposed methods found in literature can block the filtering device. The hypertension was obtained by a local subconjunctival injection of 0,7ml betamethasone suspension in one eye. The procedure was repeated for 3 weeks. The injections were done in aseptic conditions under local anesthesia. It was observed the elevated IOP after the last injection with corticosteroid. After obtained ocular hypertension, it was performed the filtration surgery by implantation of a new design model of antiglaucoma shunt and it was monitoring the IOP postoperative and the ocular status.

Conclusions. We aim to highlight the possibility of using a new device for glaucoma filtration surgery, its influence on IOP and ocular surface. Good results in the experimental implementation of this way of glaucoma surgery seem to be the most important step in treating this pathology except the classic trabeculectomy, which has also limitations.

Key words: glaucoma, surgery, experiment, shunt.

49. OCULAR MYASTHENIA GRAVIS: CASE REPORT

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Background. Myasthenia gravis (MG) is an autoimmune disease in which the patients' immune system, through the antibodies, attack the nicotinic acetylcholine receptors located on the postsynaptic neuro-muscular junction, resulting in fatigability and weakness of skeletal muscles. If weakness is limited only to the extrinsic ocular muscles and to the levator palpebrae superioris, the disease is called Ocular MG. However, ocular muscle weakness can be a debut symptom in the Generalized form of the MG (GMG) as well. Thus, the surveillance of the patient in early stage is essential, particularly during first 2 years, as most of them develop GMG within this period.

Case report. A 52-year-old man was admitted at the Neurology Department with weakness in the upper eyelids, expressed through the reduction of the palpebral fissures, gradually through the first half of the day (blepharoptosis), incapacity to fully open the eyes, moderate diffuse headache, anxiety and difficulty in falling asleep. He presented similar symptoms for about 15 years. In 2009, the patient did an electroneurography of the median nerve, where a positive decrement was registered and the diagnosis of Ocular Myasthenia Gravis was first mentioned. The patient was given treatment with Ipidacrinum, with no positive dynamics. After almost 10 years, in 2017, the patient's general condition worsened, he was not capable anymore of driving, his quality of life has decreased and he addressed the neurologist again. He is tested on the serum antibodies. Both the Anti-acetylcholine receptor (anti-AchR) antibodies and the Anti-muscle-specific tyrosine kinase (anti-MuSK) antibodies were found slightly positive (AchR Ab - 0.25 [normal value < 0.2]; MuSK Ab - 0.05 [normal value < 0.05]).After several months, the anti-AchR Ab raised up to 0.52nmol/L. The Tensilon (Neostigmine) test was performed and revealed only a week positive outcome: after administrating 1ml Neostigmine i/m, the palpebral fissures measured 4 mm, compared to 3 mm before the injection. Based on these borderline results, we confirmed the Ocular Myasthenia Gravis form as diagnosis and we added Prednisolone to the treatment, with moderate improvement of the symptoms.