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THE DAMAGE OF OCULAR SURFACE DUE TO UNCONTROLLED INTRAOCULAR PRESSURE IN NEOVASCULAR GLAUCOMA

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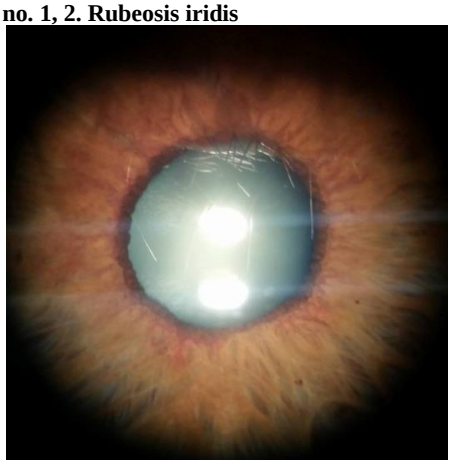
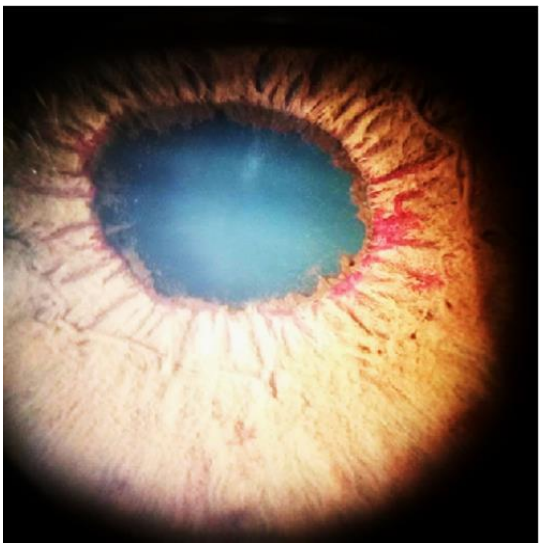
Abstract: *Neovascular Glaucoma (NVG) is a severe form of glaucoma characterized by neovascularization and the proliferation of fibrovascular tissue in the anterior chamber angle. Patients with NVG generally present with elevated intraocular pressure (IOP) and may experience severe pain. Ocular surface is affected by high IOP and can lead from moderated to marked conjunctival congestion that is frequently associated with edematous cornea. The aim of the study is to show how the high IOP can affect the ocular surface of the NVG patients and how we can treat and prevent the suffering. Materials and methods: We took in the study a number of 38 eyes from 35 patients with NVG in stage 3 with angle closure glaucoma, that presented high IOP and impaired ocular surface. Results and discussions: The ocular surface was damaged in patients that presented IOP between a minimum of 38 mmHg and maximum of 89 mmHg. The symptoms that patients presented were: conjunctival congestion in particular perikeratic, epithelial and stromal corneal edema, epithelial bubble, corneal ulcerations. Treatment followed rapid drop in IOP and the restoration and protection of ocular surface. The management of neovascular eye with high IOP was medical, laser and surgical. The restoration of ocular surface was made with lubricating hyperosmotic ophthalmic solutions, regenerative and protective agents. In all cases after the treatment was performed the ocular surface was restored. Conclusions: NVG is a very difficult pathology and is very hard to manage. The uncontrolled IOP in NVG patients affect the ocular surface and leads to complications. Long-term maintenance of normal intraocular pressure is important in NVG management but also in protecting the ocular surface.*

INTRODUCTION

Neovascular Glaucoma (NVG) is characterised by a severe tipe of glaucoma, described in the special literature like a malignant glaucoma. It is described by neovascularization and the proliferation of fibrovascular tissue in the anterior chamber angle.(1)

Figures no. 1, 2. Rubeosis iridis





Ocular surface (OS) is deeply affected by high IOP in NVG and can lead to marked mixt conjunctival congestion associated with edematous cornea (figure no. 1).(2.3)

Acute angle closure in GNV is an ophthalmic emergency that is the result of a sudden increase in IOP along

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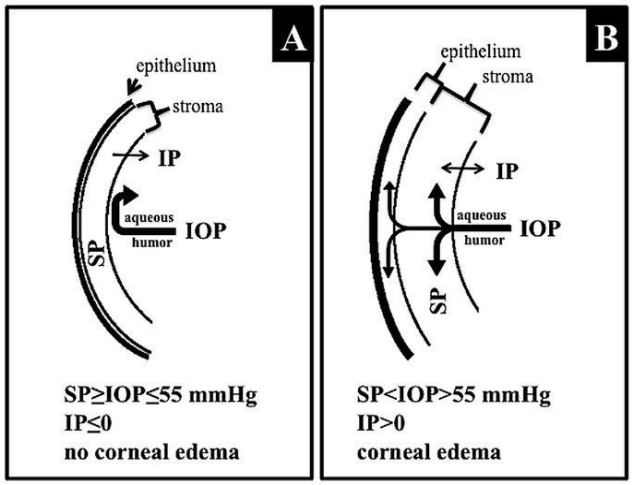
with the next symptoms and clinical signs, such as corneal edema, shallow anterior chamber, blurred vision, severe ocular pain or headache, nausea and vomiting sometimes.(3)

According to a large number of studies and publications, angle closure leads to a significant decrease in endothelial cell density.(2,4) Corneal endothelium can adapt to a gradual and modest increase in IOP, but this happens and it persists for an extended period of time, without developing major changes.(2,4) In contrast, corneal edema can be can induced by a rapid and transient increase of IOP.

An acute, sudden, and large increase in IOP has been suggested to modify the ultra-structural appearance of corneal endothelium by disrupting the cytoplasm and causing pycnosis, excrescences and even loss of cells but the mechanism is not well understood.(2) Increased IOP can affect endothelial cells through direct mechanical damage, impaired endothelial pump, and ischemic, oxidative stress.(2) Long-term high IOP induce deformation and affect endothelial function, reduce blood flow, induce hypoxia.(2,5)

Figure no. 3. (A,B) Mechanism of corneal edema



AIM

The aim of the study is to show how we can prevent and treat the suffering of the ocular surface in the NVG patients

MATERIALS AND METHODS

We took in the study a number of 38 eyes from 35 patients with NVG in stage 3, with angle closure glaucoma, presenting high IOP and impaired ocular surface (figures no. 4,5) We conducted ocular examination, IOP measurement, and imagistic investigations.

The etiology of NVG in the group was classified in diabetic retinopathy, central or branch vein occlusion and ocular ischemic syndrome.

RESULTS AND DISCUSSIONS

The ocular surface was damaged in patients that presented IOP between a minimum of 38 mmHg and maximum of 89 mmHg (figure no. 4). Corneal edema is one of the obvious clinical signs of this disease.

The main factor in the ocular surface damage in NVG is elevated IOP (figure no. 3).

The symptoms that patients presented were: conjunctival congestion in particular perikeratic, epithelial and stromal corneal edema, epithelial bubble, corneal ulcerations (figure no. 2).

Treatment algorithm followed aimed at dropping the high IOP with medical treatment applied locally, generally and the restoration and protection of ocular surface.

Figure no. 4. Cases of high IOP with corneal edema (A, B,C)and ulcer (D)

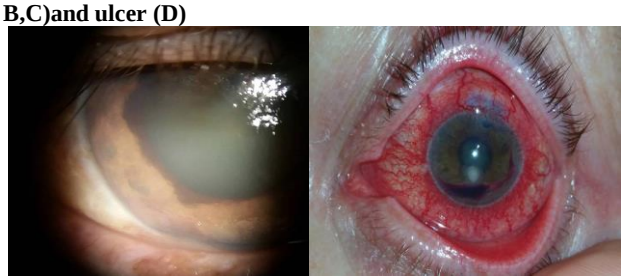
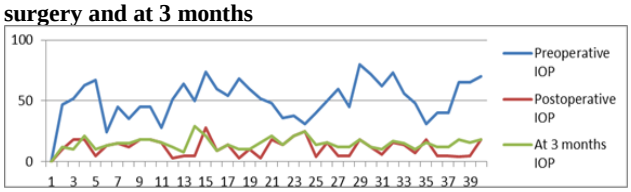


Figure no. 5. The values of the IOP before the surgery, after surgery and at 3 months

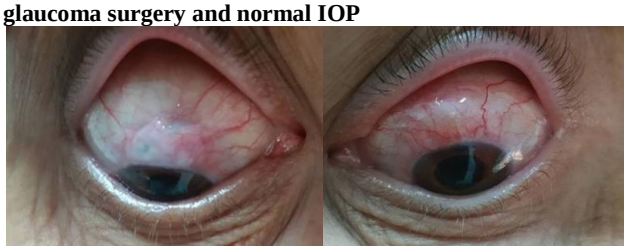


Local ocular hypotensive therapy has been the foirts step and it was consisted by fixed combinations of Carbonic anhydrase inhibitors and Beta blockers, Alpha-adrenergic agonist, and for putting at rest ciliary body and reduce the pain we administrated. Atropine, Cyclopentolate and anti-inflammatory topical agents (Steroids, Nonsteroids). Acetazolamide was used in systemic therapy with the purpose to drop the aqueous humor production and osmotic agents to reduce the vitreous volume. To reduce the inflammation and pain were used antalgic agents and anti -inflammatory agents. Protection and restoration of ocular surface with artificial tears, autologous serum, matriceal therapy, liposome therapy, ocular surface bioprotection, antibiotic drops, corticosteroids, nonsteroidal anti-inflammatory drops. Protective treatment**,** with therapeutic contact lenses.

After we succeed to drop the IOP we made intravitreal injections with anti-VEGF agents and panphotocoagulation when clear media was obtained. Intravitreal injection of anti-VEGF agents in patients with NVG reportedly causes reduced vascular permeability, decreased inflammatory reaction, loss of vascular function, and endothelial cell degeneration.(6,7,8)

Surgical therapy by trabeculectomy with application of antimetabolite local (Mitomycin C, 5-Fluorouracil) and Interferon alfa-2b was performed in all cases.(9,10,11,12)

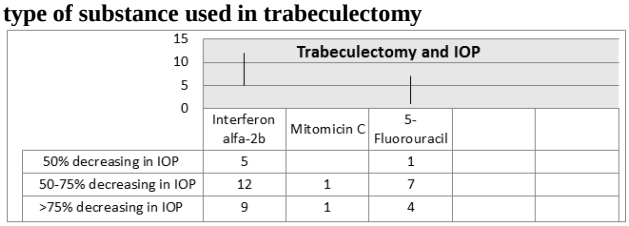
Figure no. 6. (A,B) Filtration bleb at 3 months after glaucoma surgery and normal IOP



If the algorithm treatment plan was in made in 3 directions, medical, laser and surgery. We noticed that the average IOP before surgery was very high, with values over 50 mmHG, and decreased by 40.57 mmHg after surgery at 3 days and was maintained with values under 40 mmHg 1 months later.

We observed an average increase of IOP (10-15 mmHg) at 3-month evaluation after this algorithm treatment and we repeated the anti-VEGF injections and measured IOP 2 weeks later We repeated the injections after 4 weeks depending of the value of IOP measured. In cases where visual function was preserved and we observed cataract progression, cataract surgery was performed with intraocular lens implantation.

Figure no. 7. Percentage of decrease in IOP reported in the type of substance used in trabeculectomy



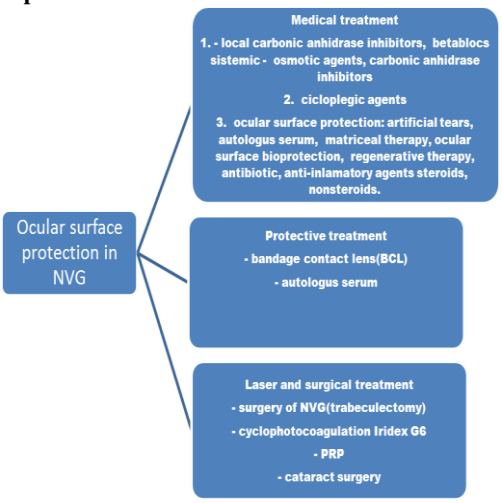
	Interferon alfa-2b	Mitomicin C	5-Fluorouracil		
50% decreasing in IOP	5		1		
50-75% decreasing in IOP	12	1	7		
>75% decreasing in IOP	9	1	4		

Figure no. 8. Ocular surface at 3 months

Evaluations of patients with NVG was made at 3 months or prior in cases where IOP was increased and ocular symptoms like decreased vision, corneal edema and pain were present. A part of the patients was lost from the study due to noncompliance or they passed away. In the cases with persistence of high IOP after performing algorithm treatment we succeed to maintain low values of IOP by completing the treatment process with injections of anti-VEGF followed by cyclophotocoagulation with IRIDEX Cyclo G6.

Ocular surface was restored in all cases that remained in the study because of the controlled IOP by this algorithm treatment.

Figure no. 9. Treatment algorithm of NVG and ocular surface protection



CONCLUSIONS

NVG is a very difficult pathology and is very hard to manage. The uncontrolled IOP in NVG patients affect the ocular surface and leads to complications. Uncontrolled IOP is the main risk factor implicated in the suffering of ocular surface. Long-term maintenance of normal intraocular pressure is important in NVG management but also in protecting the ocular surface.

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