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Neovascular Glaucoma, A Serious Condition

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Abstract: ***Neovascular glaucoma is serious sequela of an underlying ocular and/or advanced systemic disease. Retinal hypoxia and ischemia typically triggers the release of several known angiogenic factors, such as vascular endothelial growth factor (VEGF), which leads to retinal neovascularization, iris neovascularization (NVI) and anterior chamber angle neovascularization (NVA). Management of neovascular glaucoma consists of eliminating the underlying cause of the neovascularization and reducing elevated IOP. This is a case report of a patient, 1 year-old girl with symptoms of palpebral edema , conjuctival injection, irritation, from 4 days . Ocular examination revealed retinal edema with optic disc neovascularisation , elevated intraocular pressure, rubeosis iridis . B-scan showed thickness of the macular region and hiperecogenicity in the optic disc .MRI suggested a probable optic nerve glioma. Patient was treated with iv antibiotics, intraocular anti-VEGF injection, anti glaucomatous drugs, close follow-up. Differencial diagnosis was between neovascular glaucoma, optic nerve glioma, inflammatory neuritis.***

Keywords: neovascular glaucoma, optic nerve, glioma, IOP

1. Introduction

Neovascular glaucoma is a serious sequela of an underlying ocular and/or advanced systemic disease. The most common cause is diabetes mellitus , proliferative diabetic retinopathy.1.3.4.6-8. Other causes include central retinal vein occlusion, chronic uveitis, chronic retinal detachment, carotid artery occlusion, sickle cell disease, intraocular tumors, radiation therapy and trauma. 1.5-8. Retinal hypoxia and ischemia typically triggers the release of the several angiogenic agents, such as vascular endothelial growth factor (VEGF), which leads to retinal neovascularization, iris neovascularisation, and anterior chamber neovascularistion.1.2.5.6.11.Neovascular glaucoma can present without symptoms, although patients typically report redness, pain, photophobia, headache, decreased vision, though each case can be different. 2.9.10. Patients can also complain of nausea, vomiting, intense headache in cases of very high intraocular pressure .9.10.

Management of neovascular glaucoma consists of eliminating the underlying cause of the neovascularization and reducing the elevated IOP.6.

Medical management consists of inflammation control and topical IOP-lowering medications, such as beta-blockers, alpha agonists, and carbonic anhydrase inhibitors.Oral carbonic anhydrase inhibitors can be used in patents who can tolerate the associated side effects.1.Prostaglandins analogs should be used with caution, because they can perpetuate inflammation in patients with retinal ischemia and NVG (neovascular glaucoma).1.7

Topical corticosteroids can be used to control inflammation. Intravitreal injections of Avastin (bevacizumab,Genentech), Lucentis(ranibizumab,Genentech) and Eyelea (aflibercept, Regeneron) have been used to treat patients with proliferative retinal disease. 3.7.12.

2. Method & Materials

This is a case of a 1-year old girl, presenting in emergency room with symptoms of palpebral edema, conjuctival injection, irritation. Patient had ocular symptoms from 4 days. Parents referred a healthy child, with no ocular or general complaints before. On objective examination, child presented a relatively good health, non febrile, no vomiting. There were no hemorrhagic elements on the skin or mucosa. Heart had clear, rythmic tones, palpable abdomen, spleen and liver were within normal limits. Child was hospitalized first in the pediatric clinic and treated with iv antibiotics and non-steroid anti-inflammatory drugs.

Laboratory tests: complete blood count, assesment of erythrocyte sedimentation rate, measurements of levels of hepatic transaminases, bilirubinemia, glucose, LDH, uric acid, hemoglobin electrophoresis and G6PDH.

On ophthalmologic examination child had palpebral edema, ciliary conjuctival injection of the right eye, transparent cornea with a slight epithelial edema, corneal diameter 13 mm, shallow anterior chamber, mydriasis, iris neovascularisation, transparent crystalline lens and vitreous, IOP 40 mmHg. On fundus examination there was evidence of retinal edema in the inferotemporal region and around the optic disc, with a grey-white reflex, neovascularisation of the optic disc. Left eye had no problem . IOP (OS) 20,6 mmHg.

Recomandation :

- -B-scan
- -MRI
- -serologic laboratory test- TORCH
- -topical antiglaucomatous therapy : sol. timolol, sol azopt, twice a day.

3. Results

All laboratory tests were within normal limits, except for CMV-IgG- (positive) and Rubella IgG-(positive).

B-scan showed clear vitreous , no hiperecogenicity in retina except for a thickness in the macular region and a hiperecogenicity in the optic nerve disc, optic nerve cone clearly visible.

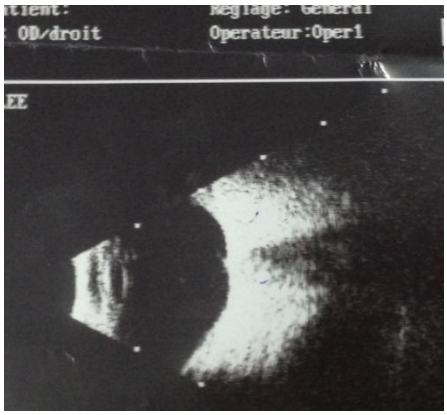

Figure 1: B-scan showing hiperecogenicity in the optic disc, optic cone visible

MRI showed a lesion in the level of optic nerve with enhancement after iv contrast injection , probably optic nerve glioma or inflammatory optic neuropathy.

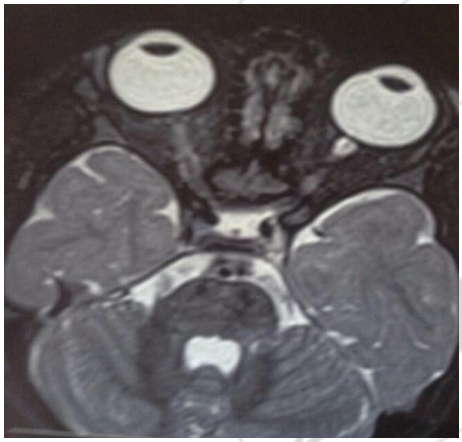

Figure 2:.MRI showing lesion in the optic nerve with enhancement after iv contrast injection

One week after intravenous antibiotic and topical therapy there was resolution of palpebral edema and reduction of intraocular pressure (IOP) -28mmHg. Patient was recomanded to start intraocular anti-VEGF treatment (Lucentis) once a month and repeat MRI to evaluate the optic nerve lesion.

4. Conclusion

NVG can lead to total vision loss because of the high intraocular pressure and anterior /posterior segment involvement. Correct diagnosing of the condition dictates treatment of the raised IOP and the underlying cause.6. In younger patients, additional tests should be considered based on clinical presentation and after ruling out common systemic vascular diseases (diabetes, hypertension, hyperlipidemia, carotid occlusive disease). These tests include hemoglobin electrophoresis antinuclear antibody (ANA), lupus anticoagulant, rapid plasma regain (PRP), fluorescent treponemal antibod absorbed (FTA-ABS), rheumatoid factor, factor V Leiden, protein C and S levels, Lyme titers, angiotensine converting enzyme levels (ACE), chest X-ray,etc.11. Differential diagnosis sometimes can mislead to treatments not related to the primary condition (neovascular glaucoma). In our case, differential diagnosis was between inflammatory optic neuropathy (ION) and optic nerve glioma.

Inflammatory optic neuropathy (ION) is a condition comprising subacute visual loss, pain and an early response to systemic steroids.13.14. Our patient had retinal changes (optic disc neovascularisation, retinal edema) not tipical for inflamatory neuritis , except for the MRI findings.13.14.15.16.17.18.

Optic nerve glioma is a tumor that grows in different parts of the brain. It can affect one or both optic nerves or the optic chiasm.18. The symptoms are due to the tumor growing and compressing the optic nerve or the nearby structures. Symptoms are :vision loss in one /both eyes, squinting, proptosis. Recommended exams are head-CT/MRI of the head and visual field testing. In our patient visual field test can not be done because of the child age. MRI findings suggest us a close follow-up for the patient, since this is a slow growing tumor and the condition remains stable for a long period of time. Treatment varies with the size of the tumor and the general health of the patient. The main goal of treatment is to cure the disorder, relieve symptoms, and improve vision.

As a result , neovascular glaucoma presents significant management challenges. For most of the patients the visual prognosis remains poor.6.7. It is very important eleminating the ischemic cause with the use of anti-VEGF agents, thus relieving patients from the painful symptoms, and providing any chance of vision for them.

References

[1] Olmos LC, Lee RK. Medical and surgical treatment of neovascular glaucoma. Int Ophthalmol Clin. 2011 Summer; 51(3):27-36.
[2] Shazly TA, Latina MA. Neovascular glaucoma: etiology, diagnosis and prognosis. Semin Ophthalmol. 2009 Mar-Apr;24(2):113-21.
[3] Bahar I, Yeung SN, Sella R, Slomovic A. Anterior segment uses of bevacizumab. Curr Opin Ophthalmol. 2012 July;23(4):303-16.
[4] Kuang TM, Ling Liu CJ, Chou CK, Hsu WM. Clinical experience in the management of neovascular glaucoma. J Chin Med Assoc. 2004 Mar;67(3):131-5.
[5] Al-Aswad L. Treating neovascular glaucoma: a surgeon summarizes the signs, symptoms and appropriate treatment for this problematic disease. Rev Ophthalmol. Available at: www.revophth.com/content/d/glaucoma_management/i/1313/c/25277/. Accessed March 27, 2014.
[6] Sivak-Callcott JA, O'Day DM, Gass DM, Tsai JC. Evidence-based recommendations for the diagnosis and treatment of neovascular glaucoma. Ophthalmology. 2001 Oct;108(10):1767-76.
[7] Tsai JC, Shields MB. Neovascular glaucoma: current concepts and management. Glaucoma Today. 2006 May/June: 36-42.

[8] SooHoo JR, Seibold LK, Kahook MY. Recent advances in the management of neovascular glaucoma. Semin Ophthalmol. 2013 May;28(3):165-72.
[9] Yazdani S, Hendi K,. Intravitreal bevacizumab (Avastin) injection for neovascular glaucoma. J Glaucoma. 2007 Aug;16(5):437-9.
[10] Rhee DJ (ed.). Glaucoma: Color Atlas & Synopsis of Clinical Ophthalmology. 1st ed. New York: McGraw Hill; 2003:326-8.
[11] Alexander LJ (ed.). Primary Care of the Posterior Segment. 3rd ed. Spain: McGraw Hill. 2002:409-37.
[12] Chang JH, Garg NK, Lunde E, et al. Corneal neovascularization: an anti-VEGF therapy review. Surv Ophthalmol. 2012 Sep;57(5):415-29.
[13] Hickman S.J., Dalton C.M., Miller D.H., Plant G.T. Management of acute optic neuritis. Lancet. 2002;360:1953–1962. [PubMed]
[14] Kinnunen E. The incidence of optic neuritis and its prognosis for multiple sclerosis. Acta Neurol Scand. 1983;68:371–377. [PubMed]
[15] Brusa A., Jones S.J., Plant G.T. Long-term remyelination after optic neuritis: a 2-year visual evoked potential and psychophysical serial study. Brain. 2001;124:468–479. [PubMed]
[16] Werring D.J., Bullmore E.T., Toosy A.T. Recovery from optic neuritis is associated with a change in the distribution of cerebral response to visual stimulation: a functional MRI study. J Neurol Neurosurg Psychiatry. 2000;68:441–449. [PubMed]
[17] Slamovits T.L., Rosen C.E., Cheng K.P., Striph G.G. Visual recovery in patients with optic neuritis and visual loss to no light perception. Am J Ophthalmol. 1991;111:209–214. [PubMed]
[18] Huson, Susan Mary; Hughes, Richard Anthony Cranmer (1994). The neurofibromatoses: a pathogenetic and clinical overview. London: Chapman & Hall. 1.3.2:9. ISBN 0-412-38920-7.