We immediately discontinued the administration of latanoprost and started application of topical diclofenac sodium four times daily. Four months later, visual acuity in his right eye improved from 0.7 to 1.2, and the micropsia disappeared. Although the retinal capillary telangiectasis was essentially unchanged, slit-lamp biomicroscopic fundus examination showed that the macular edema was improved, and OCT showed a decrease in the retinal thickness (Fig. 2B).

Comments

Cystoid macular edema associated with latanoprost usually develops in eyes with certain risk factors such as the presence of uveitis or a history of cataract surgery or diabetes mellitus.1,2 Our patient had neither uveitis nor any history of ophthalmic surgery or diabetes. The relationship between the period of topical application of latanoprost and the transient cystoid macular edema suggests that latanoprost induced cystoid macular edema in the phakic eye with juxtafoveal telangiectasis. Since prostaglandin F 2α causes relaxation and an increase in the permeability of blood vessels in various organs,4 latanoprost, a prostaglandin F 2α analog,1,2 could induce relaxation and increase the permeability of the retinal capillaries. Although the presence of juxtafoveal retinal telangiectasis was not detected before his visit to our hospital, it is unlikely that the administration of latanoprost for only 3 weeks induced the development of this retinal vascular disease, based on available literature. We hypothesized that retinal capillary changes were present subclinically until the development of significant macular edema, which was aggravated by the latanoprost administration. In eyes with juxtafoveal retinal telangiectasis, the retinal capillaries thicken and the capillary endothelial cells become abnormal.5 These factors probably contributed to the breakdown of the blood-retinal barrier by prostaglandins.

The findings in our case suggest that the use of latanoprost might have aggravated the macular edema associated with juxtafoveal telangiectasis. Therefore, latanoprost use should be carefully considered in eyes with juxtafoveal retinal telangiectasis, and, if it is used, the macula requires careful monitoring.

Key Words: juxtafoveal retinal telangiectasis, latanoprost, macular edema

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References


Bilateral Neovascular Glaucoma Due to Multicentric Castleman Disease

Multicentric Castleman disease (MCD) is a rare lymphoproliferative disorder of lymphadenopathy.1 Some studies have demonstrated vascular occlusive changes such as epistaxis, oral hemorrhage, and brainstem infarction due to the hyperviscosity syndrome associated with MCD.2,3

Irregular overproduction of interleukin-6 from affected lymph nodes is implicated in the pathogenesis of Castleman disease.4 Hyperviscosity syndrome sometimes induces central retinal vein occlusion (CRVO),5 but there has been no report of neovascular glaucoma (NVG) associated with MCD.

Case Report

A 45-year-old man was admitted to our hospital in April 2003 because of an increased blood serum level of immunoglobulin G (IgG), 6425 mg/dl; IgM, 267 mg/d; and IgA, 1335 mg/d. He complained of headache, nasal hemorrhage, skin rash on his axilla and abdomen, and multiple axillary lymph node swellings. Chest X-ray and computed tomography showed bilateral hilar lymphadenopathy. Pathological analysis of the lymph node specimen obtained by pulmonary biopsy with a thoracoscope revealed follicular hyperplasia with dense infiltration of plasma cells in the interfollicular areas. The light microscopic features were characteristic of the plasma type seen in Castleman disease. Despite treatment with methylprednisolone and plasmapheresis therapy, the hyperviscosity syndrome continued.

In October 2004, the patient became aware of visual disturbance, and he complained of pain and progressive visual loss in the right eye. Intraocular pressure (IOP) was 50 mmHg. Neovascularization and peripheral anterior synchiae (PAS) were observed throughout the whole angle. A sausage-like appearance of the retinal vein, blot- and flame-
shaped retinal hemorrhages, macular and disk edema, and several soft exudates were observed (Fig. 1), indicating CRVO. Panretinal photocoagulation (PRP) and retinal cryotherapy in the peripheral area were not successful in lowering IOP, and finally the right eye developed absolute glaucoma.

The same retinal changes started to appear in the left eye in March 2005, and became prominent in May. Despite plasmapheresis, steroid therapy, and the application of humanized anti-interleukin-6 receptor monoclonal antibody, fluorescein fundus angiography (FFA) showed diffuse fluorescein leakage from the retinal vessels, markedly distended and tortuous veins, total occlusion of blood capillaries, and a wide avascular area (Fig. 2).

Because of the rapid deterioration of vision in the patient's right eye and the abnormalities shown by FFA in the left eye, PRP in the left eye was started for a total of 6776 spots of 400-µm burns until the occurrence of iris rubeosis.

IOP remained normal until August, and then it increased to 30mmHg and did not decrease despite full medical treatment. Visual acuity in the left eye was unchanged at 0.02. Neovascularization was observed on the iris and on the whole circumference of the angle. PAS was not observed. Combined surgery for cryotherapy in the inferior half of the peripheral retina, trabeculectomy, and double plate Molteno implantation with tube ligation on the anterior chamber side were performed in October 2005. IOP increased to 30mmHg 7 months after the combined surgery. Cutting of the tube ligation by argon laser lowered IOP to the normal level, which has been maintained for 13 months to date. Iris and angle neovascularization have not been observed. PAS is still not observed, and the visual acuity in the left eye remains unchanged at 0.03.

**Comments**

It seems important to document that plasmapheresis, steroid therapy, and treatment with humanized anti-interleukin-6 receptor monoclonal antibody, which have been reported as effective therapy for MCD, could not prevent the occurrence of NVG in our case. Although the patient received more than 6000 burns during early PRP despite having an edematous retina, this did not succeed in lowering IOP. This finding suggests that NVG in MCD may be caused by hypoxia, not only in the posterior part of the eye but also in the anterior part of the eye, in addition to the fact that hypoxia is accelerated by the high IOP itself. Therefore, in such a difficult case of NVG, physicians should consider early treatment for both high IOP and hypoxia of the eye. In our case, early PRP treatment followed by combined surgery for retinal cryopexy in the peripheral area and glaucoma drainage implant prevented progression to blindness in this patient's left eye.

**Key Words:** Castleman disease, central retinal vein occlusion, hyperviscosity syndrome, neovascular glaucoma

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