Case Report

Retino-choroidal ischemia in central retinal vein occlusion

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Abstract

A 41-year-old gentleman with insulin dependent diabetes had decreased vision in the right eye due to non-ischemic central retinal vein occlusion with macular edema. One month following intravitreal ranibizumab, he developed retino-choroidal ischemia with further loss of vision. Authors show the fluorescein angiographic transition from non-ischemic central retinal vein occlusion to retino-choroidal ischemia.

Keywords: Central retinal vein occlusion, Macular edema, Retinochoroidal ischemia

Introduction

The common causes for visual decrease in central retinal vein occlusion are macular edema, macular ischemia and vitreous hemorrhage. Early treatment may be required to improve vision in macular edema before irreversible photoreceptor damage occurs. Retinal vein occlusion is associated with varying amounts of retinal ischemia or macular edema that results in the expression of vascular endothelial growth factor (VEGF). Various reports and short-term studies in the literature have shown that intravitreal bevacizumab and ranibizumab are effective and safe in the treatment of macular edema secondary to retinal vein occlusion. 1,2

We report a case of central retinal vein occlusion with macular edema that developed retino-choroidal ischemia following intravitreal ranibizumab.

Case report

A 41-year-old gentleman came with complaints of decreased vision in the right eye since 2 weeks. He gave history of undergoing Laser-Assisted in situ Keratomileusis (LASIK) in both eyes 6 years ago. He is a known diabetic since 5 years on conventional insulin therapy. On examination, his visual acuity was 0.2 in the right eye and 1.0 in the left eye. Anterior segment examination was unremarkable and there was no afferent pupillary defect in both eyes. Intraocular pressure was 16 mmHg in both eyes. Dilated fundus examination showed clear ocular media in both eyes and the left eye was unremarkable. Right eye showed scattered blot retinal hemorrhages, tortuous and congested venules with cystoid macular edema. Disk showed cup disk ration (CDR) 0.3 with healthy neuroretinal rim in both eyes. A diagnosis of central retinal vein occlusion with cystoid macular edema in the right eye was made.

His lab test showed erythrocyte sedimentation rate (ESR) 08 mm/hr, HbA1c 10.6%, serum homocysteine 10.28 micromol/L, slight increase in total cholesterol (5.57 micromol/L) and low density lipoproteins (LDL) cholesterol (3.63 micromol/L). He was referred to the endocrinologist for management. Fundus fluorescein angiography (FFA) and optical coherence tomography (OCT) were done. The right eye FFA showed delayed venous filling with dilated and tortuous venules. Late phase showed pooling of dye in petaloid

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pattern around the fovea with late perivenous staining (Fig. 1). The left eye showed normal study. OCT of the right eye showed elevated foveal contour with extensive intraretinal cystic changes and serous foveal detachment. The central macular thickness was 592 microns and foveal thickness was 619 microns in the right eye. Based on this evaluation, he was diagnosed to have non-ischemic central retinal vein occlusion with cystoid macular edema in the right eye.

Three weeks after the control of diabetes, he underwent intravitreal ranibizumab (0.5 mg) in the right eye. Post-injection period was uneventful. One month later, he came with complaints of decreased vision in the right eye since 10 days. His visual acuity was 0.1 in the right eye and 1.0 in the left eye. Anterior segment examination was unremarkable in both eyes. Intraocular pressure was 16 mmHg in the right eye and 15 mmHg in the left eye. Dilated fundus examination showed attached retina, scattered retinal hemorrhage and an area of outer retinal opacification was seen in the macula involving the fovea (Fig. 2). No gross macular thickening was observed. An impression of resolved CRVO and acute branch retinal artery occlusion or possible choroidal ischemia was made in the right eye. He was advised FFA, OCT and advised cardiological opinion. He underwent FFA, which showed macular infarct with whole of temporal retina showing lack of arteriolar and venular perfusion as well as choroidal non-filling of dye suggestive of both outer retina-choroid and inner retinal ischemia in the right eye. The retinal arterioles appeared straightened and attenuated. The venular filling was differential between the nasal and temporal side of the retina across the optic disk (Fig. 3). Left eye angiographic phases were all within normal limits. OCT showed widening of foveal contour and intraretinal edema was evident. An impression of retino-choroidal ischemia was made. His cardiological evaluation was normal and he was put on aspirin, lipitor and continued on antidiabetic treatment.

Three months later, his visual acuity was counting fingers 1.5 meters in the right eye. The fundus examination was same as before.
Discussion

Central retinal vein occlusion is one of the most common retinal vascular diseases and often associated with moderate to severe visual loss. It has been reported that monthly intravitreal ranibizumab for macular edema from retinal vein occlusion contributes to greater improvement in vision related function compared to sham treated patients through 6 months. It has also found to be safe and effective with significant impact on visual function. No new safety events were identified with long-term use of ranibizumab and rates of serious adverse events potentially related to treatment were consistent with prior ranibizumab trials. All available data, suggest that intravitreal ranibizumab provided an effective treatment for macular edema secondary to central retinal vein occlusion.

Retino-choroidal infarction is an uncommon ophthalmic complication in central retinal vein occlusion. The persistent visual loss in our patient is caused by ischemic damage to the retina and choroid. Reported cases of retinal choroidal ischemia have been caused following intralesional steroid injection, during the treatment of acute lymphoblastic leukemia and in pregnancy induced hypertension and disseminated intravascular coagulation.

The risk factors for central retinal vein occlusion are systemic hypertension, diabetes mellitus and open angle glaucoma. In women, the risk of occlusion decreased with the use of postmenopausal estrogens and increased with higher erythrocyte sedimentation rates. Cardiovascular disease, electrocardiographic abnormalities, history of treatment of diabetes mellitus, higher blood glucose levels, lower albumin-globulin ratios, and higher alpha-globulin levels were associated with increased risk only for ischemic CRVO. Higher total plasma level of homocysteine was also found to be an independent risk factor for central retinal vein occlusion (odds ratio 13) and thrombophilia. We do know that progressive retinal vein occlusion can cause extensive ischemia of the retina, however, choroidal ischemia occurs as a result of defective retro-ocular circulation. This can only happen when blood flow to the choroidal perfusion is affected.

Choroidal vascular compromise can acutely occur in malignant hypertension and pre-eclampsia. Perfusion abnormality depends primarily on the focal effect on choriocapillaries or effect on larger arterioles. Inflammatory conditions like giant cell arteritis and Wegener's granulomatosis can also cause inflammatory occlusion of choriocapillaries or occlusion of intra-orbital part of ophthalmic artery so does vaso-occlusive diseases in a variety of diseases. This condition results in varieties of choroidal infarction or choroidal ischemia.

In the present case, the cause for retino-choroidal ischemia could not be determined. However, it is possible that such an undesirable vascular event could have happened due to occlusion of short posterior ciliary arteries with reasons not known to us. The only risk factor was uncontrolled blood sugar (HbA1c 10.6%) at the time of presentation.

This case demonstrates the fluorescein angiographic characteristics of retino-choroidal ischemia occurring after the treatment for central retinal vein occlusion and highlights that such an event can occur in patients with central retinal vein occlusion.

Conflict of interest

The authors declared that there is no conflict of interest.

References


