Combined Cilioretinal Artery Occlusion and Central Retinal Vein Occlusion - A Case Report

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Cilioretinal artery occlusion present in 5% of patients with central retinal vein occlusion giving an erroneous picture of a poor visual prognosis. However, in majority of patients the occlusion is transient as demonstrated in the case history given below.

A 37 year old male with no systemic illness presented with sudden loss of vision in his left eye. On examination, best corrected visual acuity was 6/6, N₆ in the right eye and CF ½ m in the left. Applanation tonometry was 16 mm Hg in both eyes. Anterior segment was within normal limits. Dilated fundus evaluation showed dilated and tortuous veins in the left with few superficial haemorrhages and an area of whitening in the superior macular region below the superotemporal vessel. The right eye was normal (Fig 1a & b).

The patient was started on Tab. Trental 400 mg twice daily and dorzox eye drops 3 times. A digital fluorescein fundus angiogram was done which revealed a non-ischaemic central retinal vein occlusion in the left eye with cilioretinal artery occlusion (Fig 2a & b). The right eye was within normal limits (Fig 3). Blood investigations including ESR, serum lipid profile, ANA and LE cell were performed. He was advised a cardiology consultation following which inj. heparin was given. On review after 2 days, his vision and fundus were status quo. All investigations including cardiology work up were within normal limits. On review after one month, his vision had recovered to 6/6 B in the left with near complete resolution of the central retinal
vein occlusion and complete resolution of the cilioretinal artery occlusion (Fig. 4).

**Discussion**

Cilioretinal artery usually enters the retina from the temporal aspect of the optic disc separate from the central retinal artery and can be seen clinically in about 20% of eyes. Fluorescein angiographically, they are visible in approximately 32% of eyes. In a normal fluorescein angiographic sequence, they usually fill with the choroidal circulation, about 1-2 seconds before filling of the retinal artery.

Ophthalmoscopically, a cilioretinal artery obstruction appears as an area of superficial retinal whitening along the course of the vessel. The following clinical variants have been described: 1) isolated cilioretinal artery obstruction 2) cilioretinal artery obstruction associated with central retinal artery obstruction 3) cilioretinal artery obstruction associated with anterior ischemic optic neuropathy. Cilioretinal artery obstruction along with central retinal vein obstruction makes up just greater than 40% of cases of cilioretinal artery obstruction.

The venous obstruction are generally non ischemic and therefore do not usually lead to rubeosis iridis and neovascular glaucoma. However, it is possible for a cilioretinal artery obstruction to be difficult to detect in the presence of an ischemic central retinal vein obstruction causing the incidence of rubeosis iridis to be falsely low in this subgroup with cilioretinal artery obstruction. Approximately 70% of these eyes achieve 20/40 or better vision with the venous obstructive component accounting for the greatest degree of visual loss. Fong and colleagues have noted that 5% of patients with central retinal vein obstruction also have cilioretinal artery occlusion. The reasons for this association are unclear. Reduced hydrostatic pressure in the cilioretinal artery, as compared to the central retinal artery, may predispose the cilioretinal artery to stasis and thrombosis in the setting of increased hydrostatic pressure within the retinal venous system. In addition, swelling of the optic disc may compromise the cross sectional area of the cilioretinal artery and lead to reduced flow. According to Poiseuille's law, the flow within a blood vessel is proportional to the fourth power of the radius of the vessel. Thus flow within a vessel with twice the radius of a second vessel will be 16 times that with the similar vessel.

The systemic work up for causes of cilioretinal artery obstruction is similar to that for central retinal artery. An extensive work up for the embolic sources is probably not indicated however for cases of associated with CRVO.

Ocular treatment is not usually given. Studies have shown that eyes with combined CRVO and CRAO with recent visual loss respond well to superselective ophthalmic artery fibrinolytic therapy with urokinase. Another study showed no alternation on course of the disease with intravitreal tissue plasminogen activator.

In this case, the obstruction resolved completely in one months time with return of vision to 6/6.

**References**