

Central Serous Chorioretinopathy-A Review

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Abstract—Central serous chorioretinopathy is a retinal disorder characterised by an idiopathic serous neural retinal detachment in the macular region. We report a case series of two patients who presented with diminution of vision. On further evaluation with fundus examination and Optical coherence tomography they were diagnosed with central serous chorioretinopathy. The aim of this review article is to summarize the etiopathogenesis and evolving treatment options in central serous chorioretinopathy.

I. INTRODUCTION

Central serous chorioretinopathy (CSCR) is accumulation of fluid between the neurosensory retina and retinal pigment epithelium. It most commonly affects young and middle aged men and patients with type A personalities. The incidence of CSCR is 5–6 per 100 000 people¹.

Case series

Case 1- We report a case of 38 years old male who presented to eye outpatient department with complaint of decreased vision left eye for three days. Patient was not a known case of

hypertension or diabetes. There was no history of intake of antitubercular drug or any other drug intake. Patient was smoker and non-alcoholic. General physical and systemic examination of patient was normal. On ocular examination visual acuity in right eye was 6/6 and in left eye was 6/60 and with +1.5DS 6/9. Intraocular pressure in right eye was 18 mm of hg and in left eye was 16 mm of hg. Anterior segment of both the eyes were normal. Fundus examination (Figure 1) of right eye was normal whereas in left eye in macula there was circumscribed round elevation of 2 disc diameter obliterating the foveal reflex.

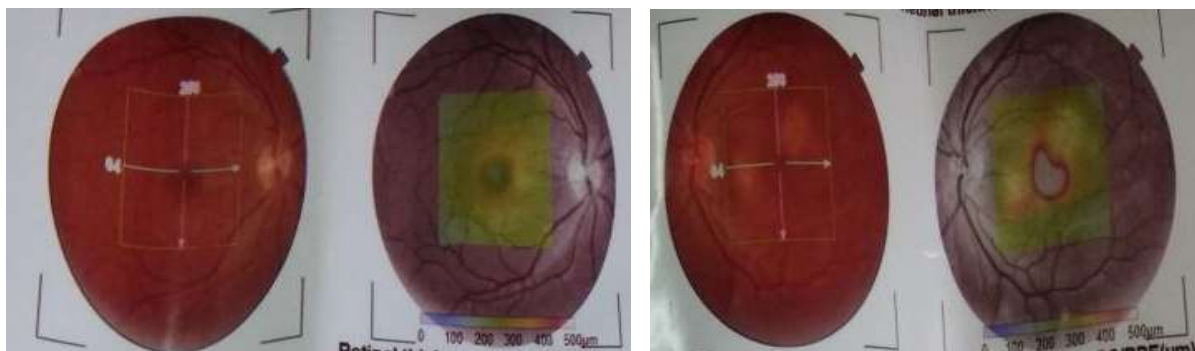


Fig. 1. Fundus photograph showing right eye normal and in left eye showing circumscribed round elevation at macula.

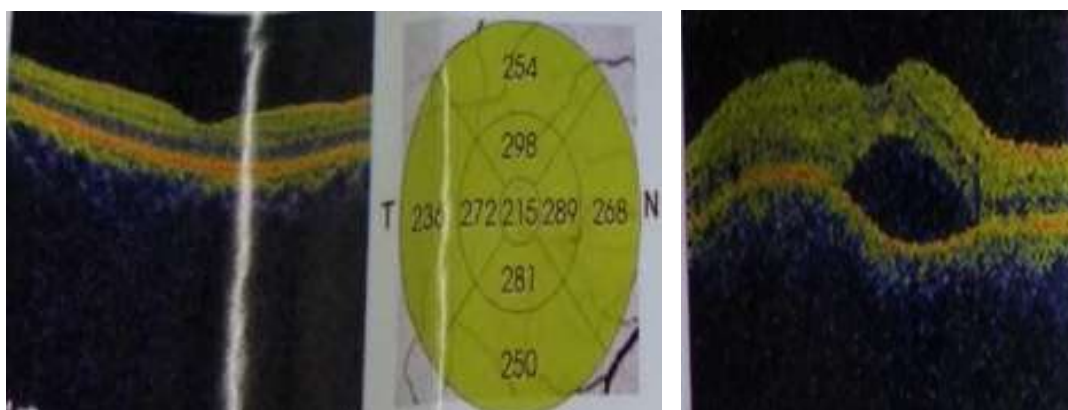


Fig. 2. OCT of right eye normal and left eye showing serous retinal detachment of neurosensory retina at macula.

Patient was started on topical nepafenac 0.1% and after three months follow up vision improved to 6/9 in left eye.

Case 2 Another patient 28 years old male presented to eye outpatient department with chief complaints of blurring of

vision right eye for six days. Patient was nonsmoker and non-alcoholic. Patient was nonhypertensive, nondiabetic. There was no history of tuberculosis or any drug intake. General physical and systemic examination of patient was normal. On

ocular examination visual acuity in right eye was 6/6 partial and in left eye was 6/6. Intraocular pressure in right eye was 12 mm of hg and in left eye was 16 mm of hg. Anterior segment of both the eyes was normal. Fundus examination of left eye was normal whereas in right eye in macula there was

circumscribed round elevation obliterating the foveal reflex. Optical coherence tomography of left eye (Figure 3) was normal whereas in right eye there was serous retinal detachment of neurosensory retina at macula.

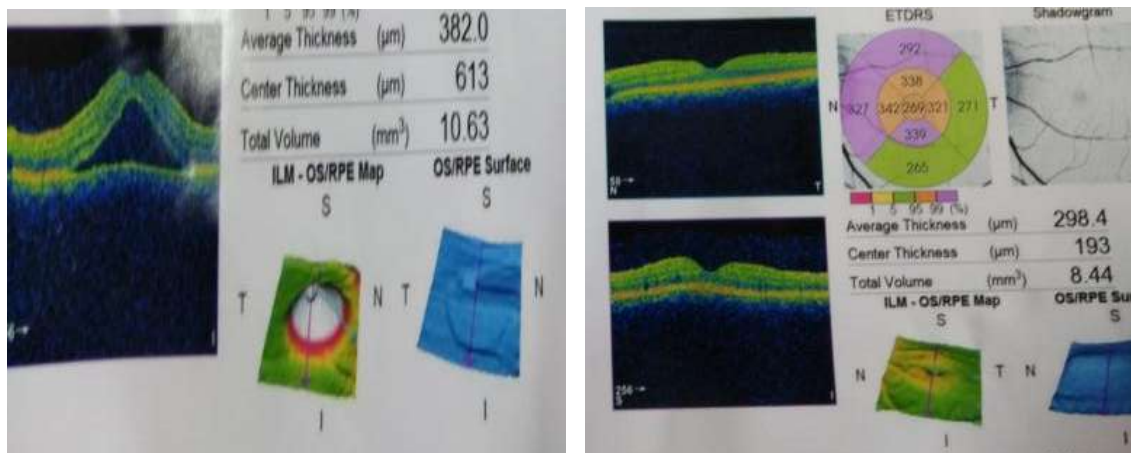


Fig. 3. OCT of right eye showing full thickness detachment of neurosensory retina and left eye normal macula.

Patient was started on topical nepafenac 0.1% but unfortunately patient was lost to follow up.

II. DISCUSSION

The disease was first recognised by Albrecht Von Graefe in 1866 when he termed it central recurrent retinitis.² In 1967, Gass described the pathogenesis and clinical features and named it idiopathic central serous choroidopathy.³ CSCR most commonly occurs in middle age between 20–50 years affecting males more than females.

Central serous chorioretinopathy occurs due to the disruption of the ionic pump of the retinal pigmented epithelial cells or breakdown of the blood-retinal barrier at the retinal pigment epithelium level which leads to accumulation of subneural retinal fluid between neurosensory retina and retinal pigment epithelium.

Another theory is hyperpermeability of choroidal vasculature which cause increased tissue hydrostatic pressure in the choroid and lead to mechanical disruption of the RPE barrier and allows entry of fluid in the subretinal space. The hyperpermeability of choroid can be caused due to stasis, ischemia or inflammation which is evident with the staining of the inner choroid in midphase ICGA⁴.

The systemic risk factors associated with CSCR are hypertension, gastroesophageal reflux disease, systemic lupus erythematosus, pregnancy, type A personality, emotional stress, membranoproliferative glomerulonephritis type II, helicobacter pylori infection, autoimmune disorders, tobacco and alcohol use, organ transplantation. Drugs which are associated with CSCR are psychopharmacologic medications, corticosteroids, antacids and antireflux medications, antibiotics, sildenafil citrate, antihistamines. CSCR is of two types acute and chronic. Acute CSCR is also known as typical or classic CSCR. In this type there is mild to moderate loss of visual acuity, few focal leaks and it resolve spontaneously.

Chronic CSCR is also known as diffuse retinal pigment epitheliopathy. It most commonly occurs in older age, more than 6 months duration detachment at posterior pole. Visual prognosis is poor in chronic CSCR because of cystoids macular oedema, foveal atrophy, subretinal fibrosis, and choroid neovascularisation. Patients may present with unilateral blurred vision, metamorphopsia micropsia, impaired dark adaptation, color desaturation, and relative scotoma. Visual acuity ranges from 6/6 to 6/60, usually correctable with hyperopic correction. In the present study the first patient had visual acuity of 6/60 in left eye and in second patient visual acuity was 6/6 (partial). On fundus examination there is absence of foveal reflex and yellowish discoloration due to increased visibility of retinal xanthophylls. Fundus Fluorescein Angiography shows one or more hyperfluorescent leaks at the level of RPE. Inkblot pattern is seen in approximately 90% of cases. The dye spreads symmetrically to all sides and slowly and evenly stains the subretinal detachment. In approximately 10% of the cases, smokestack pattern is seen. The dye rises within the detachment and then diffuses upward laterally in a mushroom or umbrella like fashion. This occurs because of the convection currents and with increased protein concentration in the subretinal fluid⁵.

Fundus autofluorescence shows patchy area of autofluorescence in macular area because of intraretinal and subretinal precipitates. Autofluorescence occurs due to collection of the shed photoreceptors or abnormal accumulation of lipofuscin⁶. Optical coherence tomography is a noninvasive technique that can demonstrate the presence of subretinal fluid and thickening of the choroid

As CSCR resolve spontaneously over time, 6–12 weeks most often the initial treatment of choice is observation. We managed our patient conservatively with topical nepafenac drops. The diagnosis of CSCR is clinical, with confirmation by fluorescein angiography (FA), fundus autofluorescence

(FAF) and OCT. In recurrent or persistent cases, the treatment depends on the location of the lesion. If the lesion is focal and extrafoveal, treatment is photocoagulation but in cases in which the leak is subfoveal or the leakage pattern is diffuse, photodynamic therapy is beneficial⁷. Vascular endothelial growth factor (VEGF) is one of the major cytokines that induce vascular hyperpermeability. Some studies have supported the role of intravitreal injection of anti-VEGF bevacizumab in improving the symptoms of CSCR by blocking the activity of VEGF^{8,9}.

III. CONCLUSION

Recent advances and imaging techniques have made it possible to diagnose CSCR at the early stage thereby preventing photoreceptor damage. These patients also require detailed ocular and systemic evaluation as systemic diseases too have been implicated in the etiology of CSCR.

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