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Case Study

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Clinical Findings and Management of Central Serous Retinopathy

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ABSTRACT

Central Serous Chorioretinopathy is an idiopathic disorder characterized by localized serous retinal detachment and retinal pigment epithelial detachment secondary to leakage from the choriocapillaris through one or more hyper permeable retinal pigmented epithelium sites.

Central serous chorioretinopathy occurs when fluid builds up under the retina. This can cause distortion to the vision similar to the symptoms the patient experience in this case. This is a case report of a 34-year-old male who presented with clinical signs and symptoms and was managed accordingly.

KEY WORDS

Central Serous Retinopathy (CSR), Meibomian Gland Dysfunction (MGD), Elevated Macular Retina, Tigroid Fundus, Thalasemia.

Introduction:

Central Serous Chorioretinopathy (CSR) is an idiopathic disorder characterized by localized serous retinal detachment and retinal pigment epithelial detachment (PED) secondary to leakage from the choriocapillaris through one or more hyperpermeable Retinal Pigmented Epithelium (RPE) sites.[1]

Changes are most often confined to the macula and are associated with leakage of fluid through the RPE into the subretinal space. Patients with CSR often experience loss of central vision, central scotoma, micropsia, metamorphopsia, decreased color vision, and abnormalities in contrast sensitivity. Visual acuity may be only moderately reduced and there may be a hyperopic shift.[2]

Numerous risk factors have been associated with CSR, the most common being glucocorticoid use. Given the strong association between CSR and steroids their use should be avoided whenever possible. Furthermore, patients with CSR should be questioned about their use of all forms of steroids, including products that may contain steroids (e.g., skin creams, joint injections, nasal sprays, inhalants, and other commonly overlooked forms of glucocorticoid), as these could be contributing factors. Pregnancy is a recognized risk factor for CSR. Plasma cortisol levels are elevated during pregnancy, particularly during the third trimester. Pregnancy-associated CSR tends to present as white sub retinal exudation that usually resolves spontaneously after delivery.[2]

Another risk factor traditionally associated with CSR is psychological stress. Other associations include systemic hypertension, gastroesophageal reflux disease, and the use of alcohol or sympathomimetic agents, although the latter requires further confirmation.[2]

Case Presentation:

Mr. X, a 34-year-old Chinese male presented to University College Sedaya International (UCSI) University Optometry Clinic on 6th August 2019 with a complaint of distorted vision in the left eye for two years ago. The distortion occurred gradually. Patient reported that the distortion was on and off (intermittently) and persisted for a few months before it resolved. He claimed that there was no treatment done, and that the distortion had occurred previously and was associated with decrease in contrast and brightness in the particular eye.

Ocular history revealed the patient had an eye examination two years ago at an optical store in Kuala Lumpur, Malaysia. He was prescribed glasses for distance and had no other abnormal findings. The patient was spectacle lens wearer for more than 20 years, and his current glasses were two years old (he claimed that he was comfortable wearing them). Patient reported that he had no history of wearing contact lenses nor any history of ocular disease/infection, surgery or injury. He was a designer by occupation and often engaged in heavy Visual Display Unit usage. The patient was not under any medications or supplements.

Patient had his medical examination 1 year ago and no abnormal findings were reported. His overall health history was unremarkable except Thalassemia which was diagnosed by a physician 20 years ago. Patient had no history of injury, surgery or any infections from head to toe.

Family history revealed that his mother also had Thalassemia and retinal detachment. Both of his parents were myopic without any ocular or systemic diseases.

Examinations and Findings:

The distance unaided visual acuity (VA) was 2/36 and 6/36 for right eye and left eye respectively. The distance habitual visual acuity was 6/6 and 6/9 for right eye and left eye respectively. The near unaided visual acuity was N8 at 36 cm and the near habitual visual acuity was N5 at 36 cm for both eyes.

Preliminary examinations and findings included Stereopsis of 100 sec of arc with Titmus fly test and on Hirschberg test the corneal light reflex was in center and symmetrical in both the eyes. Pupil was equal in size, round regular and reacting to light. Ocular Motility and Confrontation tests showed no abnormalities. A Colour Vision with pseudoisochromatic Ishihara Plates was normal in both eyes. Keratometry revealed astigmatism of 0.875D and 0.25D in the right eye and left eye respectively. Retinoscopy findings were -3.00DS/-0.50DC × 060 (VA: 6/6) in the right eye and -0.75DS/-0.50DC × 135 (VA: 6/9) in the left eye. Subjective refraction was then conducted over the objective refraction, and it revealed refractive errors of -2.75DS/-0.75DC × 040 (VA: 6/6) in the right eye and -0.75DS/-0.75DC × 150.

Visual acuity was 6/9 in the left eye. The present glass prescription of the patient was -2.75DS/-0.75DC × 040 right eye and -0.50DS/-0.75DC × 150 left eye, with the visual acuity of 6/6 and 6/9 respectively. As patient's primary complaint was distorted vision, an Amsler Grid test was performed. The test showed no abnormalities for the right eye, but a distortion on temporal portion of the chart for the left eye was evident.

Slit lamp examination showed Meibomian bland blockage grade-1 and in upper eye lid and grade-2 in inferior eyelid on right eye, similarly grade-0.5 on superior and inferior eyelid of left eye. It was found mild conjunctival concretion on inferior palpebral conjunctiva of left eye only. The Bulbar conjunctiva redness in right eye was grading 0.5 and grading-1 on left eye. A palpebral conjunctiva of right eye was grading 0.5 and left eye is grading-1; Palpebral conjunctiva of right eye and left eye upper and lower lid is grade-1 and grade-1.5 respectively. On examination of Van Henrick test in right eye was G3/G3 and in left eye was G3/G3 respectively. A tear film was also measured by Tear Break Up Time (TBUT) test. Which was 6 sec for right eye and left eye 5 sec respectively.

Fundus Examination:

Right eye showed optic nerve head crescent and tigroid appearance of the posterior retina, while left eye showed thickened or elevated macular retina with the presence of drusens. Cup disc ratio was 0:3 and arterial vein ratio was 2:3 for both eyes.

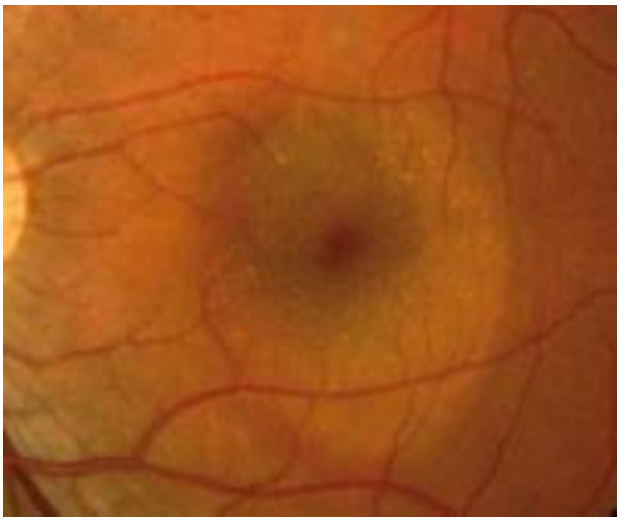

Figure 1. Fundus photo of the right eye (OD) and left eye (OS).

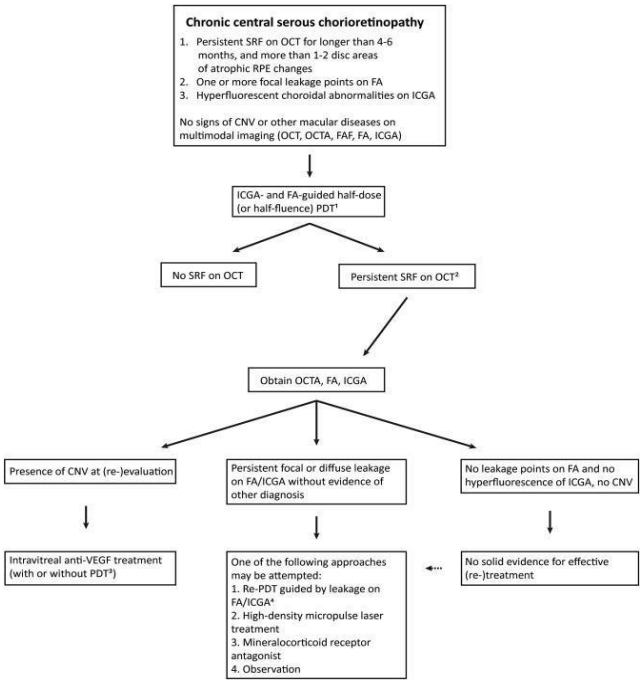

Figure 2. Differential diagnosis based on patient's complaints and fundus image

Diagnosis and Assessment:

The patient was diagnosed with having low compound myopic astigmatism both eye (OU). Other diagnoses included Meibomian Gland Dysfunction (MGD) both eye (OU) right eye (OD) > left eye (OS), moderate dry eye both eye (OU), optic nerve head crescent and "tigroid" appearance of the posterior retina right eye (OD), thickened or elevated macular retina with the presence of drusens left eye (OS).

Management and Outcome:

No new prescription was given as there were no significant changes. The patient was advised for warm compression 3 times a day for meibomian gland blockages and was advised to continue using artificial tears as necessary. Visual hygiene (20-20-20 rule) was suggested to reduce stress due to prolonged VDU usage. The patient was given Amsler Grid to monitor the progression of distortion at home and was referred to ophthalmologist (Vitreoretinal Specialist) for further evaluation (i.e. Optical Coherence Tomography and Fundus Fluorescein Angiography) regarding the changes in the retina with a provisional diagnosis of suspected central serous retinopathy.

Further Evaluation/Diagnosis by Ophthalmologist (Vitreoretinal Specialist):

A vitreoretinal specialist confirmed the diagnosis of central serous retinopathy based on optical coherence tomography images and kept the patient under observation for 3 consecutive months. The diagram below presents the management plan for the patient.

Discussion:

Acute classic CSR is characterized by a short clinical course with spontaneous resolution within 3–6 months with near-normal visual recovery. Recurrences are known in 30–50% of all cases.[3, 5] Chronic CSR, also termed diffuse retinal pigment epitheliopathy (DRPE) is seen in a few cases. It is characterized by a chronic

course lasting more than 12 months typically affecting individuals above 50 years of age. Such cases may have permanent visual impairment due to progressive RPE atrophy and photoreceptor degeneration.[3, 5]

Bullous CSR is a rare presentation characterized by larger and more numerous areas of serous retinal and RPE detachments often confused with bullous retinal detachment. Differential diagnosis of CSR includes other entities which may produce serous detachment of sensory retina in the macular area. These include optic pit, idiopathic polypoidal choroidal vasculopathy, macular hole with serous detachment, choroidal tumours and pigment epithelial detachment (PED).[5, 7]

CSR undergoes spontaneous resolution in 80 to 90% of cases. Visual acuity returns to normal or near-normal within 3 to 6 months. Discontinuation of steroids if possible should be done at the earliest. Lifestyle changes to reduce stress in life should be adopted. Laser photocoagulation is indicated in long-standing cases (more than 6 months), recurrent CSR with visual loss and permanent loss of vision in the other eye due to this condition.[8]

Contraindications include the cases having leaks near or within the foveal avascular zone. Photodynamic therapy (PDT) may be beneficial for those with severe disease not amenable to conventional laser treatment, e.g., with subfoveal leaks and chronic cases. Anti-VEGF can be considered if choroidal neovascularization develops. [9, 11]

Conclusion:

This report presents a case of central serous retinopathy. Most people with CSR regain their vision. However, having chronic CSR can cause changes to the vision in the long term, but much can be done to help make the most of the remaining vision and adapt to any changes.[12, 13] Patients with CSR who are under corticosteroids should discontinue their use if possible but only after checking with their prescribing physician to ensure it is safe to stop.[14, 15] If the affected eye is the eye with better visual acuity, then one may need to make changes by using aids to make the most of the remaining sight. This may mean making things bigger, using brighter lighting or using colour to make things easier to see.[16] A low vision assessment can explore these things. The eye care professional should make a referral to a local low vision service for an assessment.[10] Management of CSR, in particular the chronic one, is challenging. Although focal laser and PDT are current standards of care in chronic CSR, micro-pulse laser is a good alternative in instances when verteporfin is not available. Some systemic treatments may benefit patients with chronic CSC by decreasing the rate of therapy-induced complications.[19]

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