Central serous retinopathy a disease overview case study examples

Health & Medicine, Nursing

Central serous retinopathy (CSR), also known as central serous chorioretinopathy, is characterized by separation of the two major layers of the retina (the innermost layer of the eye): the inner neurosensory layer (which contains cells that transmit electrical impulses to the occipital lobe) and the retinal pigment epithelium (RPE) (the outer layer of the retina that contains the photoreceptor [light-receiving] cells). The mechanism of this condition is leakage of serous (serum-like) fluid from a portion of the choroid (the layer of the eye just outside the retina) called the choriocapillaris (a network of small blood vessels located in that portion of the choroid that is responsible for providing nutrition to the retina) through the retinal pigmentary epithelium, lifting and detaching the neurosensory layer from it. There are two types: the first, in which there are localized, isolated areas of detachment due to serous leaks and the second, a diffuse form with detachment overlying areas of generalized RPE dysfunction (Oh).

Causes

CSR is thought to be due to dysfunction of the choroidal blood vessels, leading to abnormal functioning of the overlying RPE. This may be due to high levels of circulating cortisol and catecholamines, causing disordered autonomic regulation of the choroidal vessels (Jampol et al. 1765). Hypertension, sleep apnea, alpha-type personalities and pregnancy are associated with CSR (Tittl et al. 63). Various medications, such as corticosteroids, sildenafil (Viagra) and psychotropic medications have been associated with CSR. The strongest association appears to be with corticosteroids (Carvalho-Recchia et al. 1834).

History

The patient usually complains of the sudden onset of vision loss and metamorphopsia (image distortion), especially micropsia (perceiving objects as smaller than they really are), decreased central vision, scotomata (blind spots), loss of contrast perception and decreased color saturation (Oh).

Physical Findings

Examination shows subtle detachment of the neurosensory retina from the RPE. Less commonly, detachment of the RPE from the choroid may be seen. Fluorescein angiography (injection of a fluorescent dye) demonstrates either focal or generalized hyperfluorescence at the site of neurosensory layer detachment. An imaging technique called optical coherence tomography (OCT) reveals subretinal fluid and neurosensory and RPE detachments (Oh).

Treatment

No pharmacologic treatment for CSR has been proven effective. Anti-anxiety drugs and antidepressants may worsen the condition. Corticosteroids are to be avoided, as they may actually worsen the condition. Beta-blockers, such as propranolol, have been reported to be useful and have a theoretical basis for being effective (Tatham et al. 145).

Laser photocoagulation shortens the course of an episode of CSR and decreases the risk of recurrence but does not improve ultimate visual outcome. It is used for serous retinal detachments due to CSR of more than four months duration, for

recurrence in an eye with visual deficits from prior episodes of CSR, when

the opposite eye has visual defects from CSR and when occupational needs demand a quicker recovery (Burumcek et al. 616). Photodynamic therapy (laser treatment after injection of a light sensitive medication such as verteporfin) can clot the leaking choroidal vessels and has proven useful in promoting more rapid resorption of subretinal fluid and return of visual acuity (Lai et al. 1834).

Outcome

Focal serous retinal detachments resolve spontaneously in most cases, with 80-90 % of patients returning to visual acuity of 20/25 or better. Even these patients, however, may continue to experience image distortion problems, such as dyschromatopsia (reduced ability to distinguish colors), metamorphopsia, decreased contrast and nyctalopia (night blindness). However, recurrence risk in the same eye is 40-50 %, with some of these patients going on to have recurrent or chronic serous detachments with progressive atrophy of the RPE and permanent vision loss to the level of 20/200 or worse (Yap et al. 1996).

Works Cited

Burumcek, E., et al. “ Laser Photocoagulation for Persistent Central Serous Retinopathy:
Results of Long-Term Follow-Up.” Ophthalmology 104. 4 (1997): 616-22. Print.

Carvalho-Recchia, C. A., et al. “ Corticosteroids and Central Serous Retinopathy.”
Ophthalmology 109. 19 (2002): 1834-37. Print.

Jampol, L. M., et al. “ Involvement of Corticosteroids and Catecholamines in the

Pathogenesis of Central Serous Chorioretinopathy: A Rationale for New Treatment Strategies.” Ophthalmology 109. 10 (2002): 1765-66. Print.

Lai, T. Y., et al. “ Safety Enhanced Photodynamic Therapy with Half Dose Verteporfin for

Chronic Central Serous Chorioretinopathy: A Short Term Pilot Study.” British Journal of Ophthalmology 90. 7 (20062): 1834-37. Print.

Oh, Kean Theng. “ Central Serous Chorioretinopathy.” Medscape. 2 Nov. 2011. Web.

18 Feb. 2012.

Tatham, A., et al. “ The Use of Propranolol to Treat Central Serous Chorioretinopathy:

An Evaluation by Serial OCT.” Journal of Ocular Pharmacologic Therapy 22. 2 (2006): 145-49. Print.

Tittl, M. K., et al. “ Systemic Findings Associated with Central Serous Chorioretinopathy.”

American Journal of Ophthalmology 128. 1 (1993): 63-68. Print.

Yap, E. Y., et al. “ Long-Term Outcome of Central Serous Chorioretinopathy.” Archives of

Ophthalmology 114. 6 (1996): 689-92. Print.

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www. kenteyesurgery. co. uk