



## A review of acute central serous chorioretinopathy

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ARTICLE INFO	ABSTRACT
<p><b>Article type</b> Review article</p> <p><b>Article history</b> Received: 8 Apr 2014 Revised: 18 Apr 2014 Accepted: 20 Apr 2014</p> <p><b>Keywords</b> Acute visual loss Central serous chorioretinopathy Serous retinal detachment</p>	<p>Central serous chorioretinopathy is a common cause of visual morbidity. It is characterized by idiopathic serous retinal detachment in macular or paramacular regions. The symptoms of the CSC include decreased vision, micropsia and metamorphopsia. The prognosis of the disease is good and almost 90% of patients obtain visual recovery in a few months. However, in less than 5% of patients the chronic disease with poor prognosis is developed.</p> <p>The acceptable approach is to observe patients with acute central serous chorioretinopathy, because central serous chorioretinopathy is self-limited. The pathophysiology of central serous chorioretinopathy is not clear and not well understood. Therefore, various medical treatments have been suggested such as propranolol, indomethacin, bevacizumab, acetazolamide, mifepristone, labetalol, etc. However, wait and watch would be the most recommended management of the central serous chorioretinopathy.</p>

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### Introduction

#### *Acute Central Serous Chorioretinopathy*

In 1866, Von Graefe defined central serous chorioretinopathy (CSC) as a “idiopathic detachment of the macula”(1). This disease often affects young adults in the age range of 25-50. CSC often manifests unilaterally which could be bilaterally. Various symptoms

of the CSC are micropsia, blurred vision, impaired dark adaptation in visual acuity, impaired color vision and relative scotoma after sleep. Unilateral metamorphopsia is the classic sign of the CSC disease (2). Some patients may develop hyperopia (due

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to anterior displacement of photoreceptors in fovea) (2). CSC causes are not yet fully understood and few studies have explained pathophysiology process in CSC (3). CSC is a retinal disease due to idiopathic accumulation of serous fluid under the retina in the macular area (2).

A favorable prognosis is usually predicted for more than 80-90% of patients which recovers within a few months. However, More than 50-40% of patients have relapsing disease (4). The recurrence rate of the disease in the same eye is more than 30% (5). Situations with increased stress hormones (corticosteroids and epinephrine) are associated with CSC including stress, systemic corticosteroid therapy, pregnancy, Cushing's disease, etc (6). Type A personality is another risk factor of CSC (7). CSC incidence rate is estimated to be 6-5 cases per 100,000 people. This disorder is more common in Caucasians and Hispanics, particularly Asian but is less common in African-American (6). The incidence rate of the disease is 9.9 per 100,000 men and 1.7 per 100,000 women approximately (8). CSC is the most common cause of retinopathy after the age-related macular, retinal branch vein occlusion and diabetic retinopathy (6).

CSC diagnosis is based on clinical evidences. In most cases, the quite conclusive OCT (optical coherence tomography) and angiography is necessary to confirm the diagnosis and assess the extent and severity of retinal dysfunction and disease follow-up (9). CSC has various forms in angiography, which include expansive dot, diffuse leak, and Smoke Stack (6,9).

In CSC disease, subretinal fluid would be observed on fundoscopic examination. The fluid is usually clear but can be fibrinoid. The chronic form is more superficial retinal separation than the acute form. Specific changes occur in the retina of patients

with chronic CSC such as retinal pigment epithelium changes and cystic changes within the retina of severe cases (6).

### ***Pathophysiology of CSC***

Among the factors associated with CSC, corticosteroids (exogenous and endogenous) are associated with the occurrence rate of CSC (10). In an experimental study, multiple injections of corticosteroids and epinephrine have lead to CSC in adult monkeys (11).

Other factors that are associated with CSC include psychological stress, type A personality, systemic hypertension, gastroesophageal reflux disease (GERD), pregnancy, organ transplantation, SLE, smoking, alcohol consumption, glomerulonephritis and autoimmune diseases (6,12). Some studies reported an association between H. pylori infection and CSC occurrence (13,14). Psychological medications, methamphetamine and sildenafil are associated with CSC (6).

Therefore, psychiatry history and drug history are very important in medical evaluation of CSC patients. The blood-retinal barrier and retinal pigment epithelium is responsible for the development of abnormal subretinal fluid in CSC (15). Angiography evidence showed a choroid blood circulation disorder (12).

### ***Differential Diagnosis of CSC***

Clinical and paraclinical studies are very beneficial in CSC diagnosis. Some of differential diagnosis parameters are listed in Table 1. Wet age-related macular degeneration (AMD) finding could be presented by neurosensory macular detachment the same as CSC (16). Optical coherence tomography is the most recommended diagnostic procedure in CSC (17). Multifocal electroretinography has been used to connect the signs of CSC with sections of reduced retinal

**Table 1.** Differential diagnosis of central serous chorioretinopathy

Choroidal Neovascularization due to
<ul style="list-style-type: none"> <li>• Exudative AMD (age-related macular degeneration)</li> <li>• Multifocal choroiditis</li> <li>• Degenerative myopia and Angioid streaks</li> </ul>
Vogt- Koyanagi- Harada disease
Macular hole
Idiopathic serous RPE (Retinal pigment epithelium) detachment
Choroidal tumors
<ul style="list-style-type: none"> <li>• Hemangioma</li> <li>• Metastasis</li> <li>• Melanoma</li> </ul>
Acute lymphocytic leukemia(ALL)

function and to examine the response to therapy (3). Vogt-Koyanagi-Harada (VKH) disease is a granulomatous uveitis, which is characterized with exudative retinal detachment (9).

### Treatments

CSC disease cures spontaneously in most cases and it is a self-limited disease. Therefore, most of the physicians follow up their patients without treatment (18). Laser treatment of retina and photodynamic therapy are the recommended treatment methods for the disease (1,9).

Trimepranol as a non-specific beta-blocker, with the dose of 5 mg twice per day, is reported as a influential medication in the treatment of 84.6% of cases in Chrapek et al. study (19).

In another report, the subretinal serous fluid in CSC decreased with applying acetazolamide (20).

In one case report, a patient with CSC was treated with administration of mifepristone, an antagonist of progesterone (21).

According to the different reports of a great A meta-analysis in 2013, which

studied the efficacy of intravitreal avastin in the treatment of CSC, it failed to obtain any positive association between using avastin and the treatment of CSC (22). Antifungal medications such as ketoconazole has been also used for the treatment of CSC (23).

### Conclusion

CSC is described by an exudative serous macular detachment. This disease is a self-limited disease and most of the time just wait and follow-up method is the most recommended management. However, clinical and paraclinical studies should be performed for rolling out other differential diagnosis. Less than 50% of patients will experience relapse periods. Therefore, pharmacotherapy and patient follow-up are more important than disease treatment methods.

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### Conflict of Interest

The authors declare no conflict of interest.

### References

1. Tarantola RM, Law JC, Recchia FM, et al. Photodynamic therapy as treatment of chronic idiopathic central serous chorioretinopathy. *Lasers Surg Med.* 2008;40:671-675.
2. Yanoff M, Duker JS, Augsburger JJ. *Ophthalmology.* 3 ed. Philadelphia: Mosby Elsevier; 2009.
3. Marcuson J, Riley T. Central serous chorioretinopathy. *Optometry.* 2008;79:241-251.
4. Yap EY, Robertson DM. The long-term outcome of central serous chorioretinopathy. *Arch Ophthalmol.* 1996;114:689-692.
5. Bujarborua D, Chatterjee S, Choudhury A, et al. Fluorescein angiographic features of asymptomatic eyes in central serous chorioretinopathy.

- thy. *Retina*. 2005;25:422-429.
6. Wang M, Munch IC, Hasler PW, et al. Central serous chorioretinopathy. *Acta Ophthalmol*. 2008;86:126-145.
  7. Yannuzzi LA. Type A behavior and central serous chorioretinopathy. *Trans Am Ophthalmol Soc*. 1986;84:799-845.
  8. Kitzmann AS, Pulido JS, Diehl NN, et al. The incidence of central serous chorioretinopathy in Olmsted County, Minnesota, 1980-2002. *Ophthalmology*. 2008;115:169-173.
  9. Tasman W, Jaeger EA. *Duane's Ophthalmology*. 7 ed. Philadelphia: Lippincott Williams & Wilkins; 2001.
  10. Caccavale A, Romanazzi F, Imperato M, et al. Central serous chorioretinopathy: a pathogenetic model. *Clin Ophthalmol*. 2011;5:239-243.
  11. Yoshioka H, Katsume Y, Akune H. Experimental central serous chorioretinopathy in monkey eyes: fluorescein angiographic findings. *Ophthalmologica*. 1982;185:168-178.
  12. Spitznas M. Pathogenesis of central serous retinopathy: a new working hypothesis. *Graefes Arch Clin Exp Ophthalmol*. 1986;224:321-324.
  13. Giusti C. Association of *Helicobacter pylori* with central serous chorioretinopathy: hypotheses regarding pathogenesis. *Med Hypotheses*. 2004;63:524-527.
  14. Misiuk-Hojlo M, Michalowska M, Turno-Krecicka A. *Helicobacter pylori*--a risk factor for the development of the central serous chorioretinopathy. *Klin Oczna*. 2009;111:30-32.
  15. Marmor MF. New hypotheses on the pathogenesis and treatment of serous retinal detachment. *Graefes Arch Clin Exp Ophthalmol*. 1988;226:548-552.
  16. Giovannini A, Scassellati-Sforzolini B, D'Alto-brando E, et al. Choroidal findings in the course of idiopathic serous pigment epithelium detachment detected by indocyanine green videoangiography. *Retina*. 1997;17:286-293.
  17. Beger I, Koss MJ, Koch F. Treatment of central serous chorioretinopathy: MicroPulse photocoagulation versus bevacizumab. *Ophthalmologie*. 2012;109:1224-1232.
  18. Klein ML, Van Buskirk EM, Friedman E, et al. Experience with nontreatment of central serous chorioidopathy. *Arch Ophthalmol*. 1974;91:247-250.
  19. Chrapek O, Spackova K, Rehak J. Treatment of central serous chorioretinopathy with beta blockers. *Cesk Slov Oftalmol*. 2002;58:382-386.
  20. Gonzalez C. Serous retinal detachment. Value of acetazolamide. *J Fr Ophthalmol*. 1992;15:529-536.
  21. Nielsen JS, Bachhawat A, Jampol LM. A case of chronic severe central serous chorioretinopathy responding to oral mifepristone: update. *Retina*. 2008;28:1363.
  22. Chung YR, Seo EJ, Lew HM, et al. Lack of positive effect of intravitreal bevacizumab in central serous chorioretinopathy: meta-analysis and review. *Eye (Lond)*. 2013;27:1339-1346.
  23. Meyerle CB, Freund KB, Bhatnagar P, et al. Ketoconazole in the treatment of chronic idiopathic central serous chorioretinopathy. *Retina*. 2007;27:943-946.