# Central Serous Chorioretinopathy Clinical Presentation


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## History

Patients with central serous chorioretinopathy typically present with acute symptoms of visual loss and metamorphopsia (especially micropsia). Other symptoms include decreased central vision and a positive scotoma.

The decreased vision usually is improved by a small hyperopic correction.

Other clinical signs include a delayed retinal recovery time following photostress, loss of color saturation, and loss of contrast sensitivity.

## Physical

Clinical examination shows a serous retinal detachment but no subretinal blood. The neurosensory retinal detachment may be very subtle, requiring contact lens examination for detection.[17]

Pigment epithelial detachments, RPE mottling and atrophy, subretinal fibrin, and, rarely, subretinal lipid or lipofuscinoid flecks also may be seen.[18]

## Causes

Previous hypotheses for the pathophysiology have included abnormal ion transport across the RPE and focal choroidal vasculopathy. The advent of ICG angiography has highlighted the importance of the choroidal circulation to the pathogenesis of central serous chorioretinopathy (CSCR).

ICG angiography has demonstrated both multifocal choroidal hyperpermeability and hypofluorescent areas suggestive of focal choroidal vascular compromise. Some investigators believe that initial choroidal vascular compromise subsequently leads to secondary dysfunction of the overlying RPE.[19]

Type A personalities and systemic hypertension may be associated with CSCR, presumably because of elevated circulating cortisol and epinephrine, which affect the autoregulation of the choroidal circulation.[20, 21]

Studies using multifocal electroretinography have demonstrated bilateral diffuse retinal dysfunction even when CSCR was active only in one eye. This supports the belief of diffuse systemic effect on the choroidal vasculature.[3]

Allibhai et al reported an association with sildenafil citrate and CSCR.[22] Fraunfelder and Fraunfelder further evaluated the association of CSCR with sildenafil.[23] The authors reviewed 1500 cases of sildenafil associated ocular side effects. Eleven of these cases described men with CSCR. The symptoms resolved following cessation of sildenafil in 8 of 11 patients but recurred in 3 of these patients upon restarting sildenafil. They determined that a causal relationship could not be determined as a result of the cyclic nature of CSCR though patients with refractory CSCR should consider cessation of sildenafil use.

Another hypothesis involves activation of the mineralocorticoid pathway. Inappropriate or over-activation of mineralocorticoid pathways in ocular tissues could lead to vasodilation in the choroid. This pathway would also link CSCR to co-morbidities such as systemic hypertension and psychological stress. Animal models stimulated by aldosterone demonstrate clinical findings similar to acute CSCR.[24]

The complement cascade has also been implicated in chronic CSCR. Certain polymorphisms of complement factor H and variants in the C4B have been associated with chronic CSCR in certain populations.[25, 26]

Systemic associations of CSCR include organ transplantation, exogenous steroid use, endogenous hypercortisolism (Cushing syndrome), systemic hypertension, sleep apnea, systemic lupus erythematosus, pregnancy, gastroesophageal reflux disease, and use of psychopharmacologic medications.[27, 28, 21, 29, 4, 30] Carvalho-Recchia et al showed in a series that 52% of patients with CSCR had used exogenous steroids within 1 month of presentation as compared with 18% of control subjects.[6] Haimovici et al evaluated systemic risk factors for CSCR in 312 patients and 312 control subjects.[31] Systemic steroid use (odds ratio [OR], 37.1) and pregnancy (OR, 7.1) were most strongly associated with CSCR. Other risk factors included antibiotic use, alcohol use, untreated hypertension, and allergic respiratory disorders.

Differential Diagnoses

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