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Review Article

Advances in the treatment of central serous chorioretinopathy

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Abstract

Central serous chorioretinopathy is a disease that is partly understood. Novel advancements have led to further understanding of the disease, and have identified choroidal dysfunction as the principal element in CSCR development. New imaging tools have aided in better monitoring disease response to various treatment models. Enhanced depth imaging optical coherence tomography, in particular, has helped in observing choroidal thickness changes after various treatment models. To date, photodynamic therapy and focal laser remain the main stay of treatment. More understanding of disease pathophysiology in the future will help in determining the drug of choice and the best management option for such cases.

Keywords: CSCR, Central serous chorioretinopathy, Central serous retinopathy, Photodynamic therapy, Corticosteroids, Treatment

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Introduction

Central serous chorioretinopathy (CSCR) is an acquired chorioretinal disorder that was first described by Von Graefe in 1866 as recurrent central syphilitic retinitis.[1] Other names used to describe this disease entity include capillarospastic central retinitis, central angiospastic retinopathy, central serous retinopathy, and central serous pigment epitheliopathy.[2,3]

CSCR usually affects middle-aged men between the ages of twenty and fifty years.[4–6] It has also been associated with type A personality, or those who are experiencing psychological stress.[5,7] It has been also linked to use of sympathomimetic agents, corticosteroid use in any form, endogenous high levels of corticosteroids, and some psychopharmacologic agents.[8–13] Smokers tend to have poorer vision and need longer period for visual rehabilitation.[14]

Clinical features

Patients with CSCR most commonly complain of metamorphopsia, micropsia, blurred vision, and mild dyschromatopsia in the affected eye. On fundus examination, typical signs include a round well-demarcated detachment of the neurosensory retina at the macula. Pigment epithelial detachment (PED) of variable size can also occur and can be single or multiple. The subretinal fluid (SRF) can be clear or turbid/fibrinous. The turbid fluid may even form in the sub-retinal pigment epithelial (sub-RPE) space.[15,16] In chronic CSCR or in patients with old resolved disease, RPE mottling, atrophy, and clumping might be observed.[17–19] In addition, yellow dots that are thought to represent phagocytosed photoreceptor outer segments are frequently seen just over the inner surface of the RPE.[18] Other atypical CSCR presentations include bullous neurosensory retinal detachment, inferior neurosensory detachment with atrophic tracts, and multifocal CSCR.[2,20,21]

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Investigations for CSCR include fluorescein angiography (FA) which may show 'ink blot' pattern of leakage or the less common 'smoke stack' appearance that mimics a mushroom cloud.[22] In addition, dye pooling in the sub-RPE space can be seen in cases of PED. Diffuse leakage or multiple leaking points can be seen in recurrent, chronic, or multifocal CSCR. Indocyanine green (ICG) angiography may demonstrate dilated choroidal vasculature corresponding to the site of CSCR with choroidal hyperpermeability in the late phase.[23,24] Optical Coherence Tomography (OCT) can demonstrate the neurosensory detachment and areas of PED. Enhanced depth imaging (EDI) OCT can show the thickened choroid in the areas corresponding to the neurosensory detachment.[25]

Pathophysiology

Notwithstanding the well-identified clinical picture of CSCR, its pathophysiology is inadequately understood. Proposed theories include choroidal hyperpermeability which overloads the RPE pumping mechanism responsible for keeping the subretinal space dry and may – in some instances-lead to decompensation and SRF collection.[8,26,27] RPE dysfunction is another theory for developing CSCR in which focal damage of adjacent RPE cells or even a single RPE cell leads to reversal of ion pumping mechanism and resulting in fluid accumulation in the subretinal space.[28,29] Marmor hypothesized that a dysfunction in the RPE metabolic transport system is needed in order to accumulate subretinal fluid in CSCR due to focal RPE defects.[30]

An alternative theory is a combined choroidal and RPE dysfunction.[31] In addition, there is a close relationship between CSCR and both endogenous and exogenous corticosteroids suggesting a role in pathogenesis.[9–12] It has been suggested that corticosteroids might sensitize the choroidal blood vessels or RPE to the effects of catecholamines;[32] furthermore, corticosteroids have certain genomic effects on adrenergic receptor gene transcription and expression which can result in an increase in the number of adrenergic receptors.[33,34]

Zhao et al. were the first to demonstrate that blocking the aldosterone upregulated the endothelial vasodilatory potassium channels that prevented aldosterone-induced choroidal thickening. This is suggestive of the presence of mineralocorticoid receptors in the choroidal vasculature which might be involved in the pathophysiology of CSCR.[35]

Treatment

CSCR is usually a self-limiting disease with spontaneous resolution within 3–4 months with overall good visual outcome.[36–38] However, recurrences are seen in up to 50% of patients within the first year.[39]

Chronic CSCR diseases are the cases in which there are diffuse RPE changes without evident detachment in most cases.[9] However it is sometimes difficult to clinically differentiate a chronic disease from a recurrent episode of CSCR. Spaide identified chronic CSCR as serous macular elevation detected microscopically or by OCT, and is associated with RPE atrophic areas and subtle leaks or ill-defined staining on FA.[15] These recurrences or chronic neurosensory detachments may lead to RPE atrophy or hypertrophy with irreversible loss of visual function.[40–44]

Given all of the above, observation can be regarded as a first-line approach in newly diagnosed cases of less than 3 month duration.[7] In addition, risk factors should be addressed to increase the chance of spontaneous resolution. This includes discontinuing exogenous corticosteroids intake in any form – if possible-, and life style modification for patients with type A personality traits.[7,9,10,45–48] On the other hand, different modalities of treatment for CSCR exist. These treatments are reserved for chronic CSCR, recurrent CSCR, single CSCR attack of more than 3 month duration, and if the fellow eye suffered from permanent visual loss due to a previous episode of CSCR whether acute or chronic. We will discuss below, current, emerging, and advances in therapeutics for CSCR.

Laser photocoagulation

Applying laser photocoagulation to the leaking RPE guided by FA has been shown to hasten resolution of the neurosensory detachment in CSCR. Xenon laser was used at first followed by krypton laser; currently argon laser is more widely used.[49–51] Many level one evidence studies have demonstrated faster resolution of SRF in patients who underwent laser photocoagulation compared to control eyes.[52–54] Nevertheless laser photocoagulation does not influence the final visual outcome or rate of recurrence.[6,54,55]

Therefore, argon laser photocoagulation is an effective treatment for acute CSCR with clearly defined focal leakage point as seen on FA given that the leakage is not subfoveal or juxtafoveal. Still, side effects such as permanent scotoma, laser scar enlargement, and laser induced CNV can still occur (Fig. 1).

Micropulse diode laser photocoagulation

This method of treatment uses subthreshold diode laser energy in order to minimize retinal damage. It is similarly effective in CSCR with point source leakage but not in eyes with diffuse leakage, and leaves no clinically detectable laser-induced damage.[56–58] Since there is no visible endpoint to diode micropulse laser (DMPL) application, ICG enhanced DMPL can be used to identify treated areas with post-treatment ICG angiography.[59]

To date, only one randomized clinical trial (RCT) assessed DMPL versus argon laser photocoagulation in acute CSCR.[60] Patients in both groups had complete resolution of SRF at 12 weeks of follow-up. All patients had no scotomas in the DMPL group compared to 3 out of 15 patients in the argon laser group who had persistent scotomas. Contrast sensitivity was also significantly better in the DMPL group.[60]

Transpupillary thermotherapy

Transpupillary thermotherapy (TTT) is a 810 nm long-pulse low-energy diode laser. It works by raising the temperature of the choroid and outer retina while sparing the inner retina and photoreceptors to some degree, but the exact mechanism is not clear.[61] First described in 2005 by Wei, short-term encouraging visual and anatomical outcomes have been observed in CSCR with subfoveal leaks, multiple or diffuse leaks, and recurrent atypical CSCR with PEDs.[61–64] More well structured RCTs with long-term results are required to establish the role of TTT in the treatment of CSCR.