# Medical Management of Central Serous Chorioretinopathy

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The term central serous Chorioretinopathy (CSC) refers to a localized serous detachment of the neurosensory retina or a detachment of the retinal pigment epithelium (RPE) at the macular area. It is usually an idiopathic, unilateral, self limiting disease affecting young to middle aged men. Risk factors include Type A personality, psychological stress, smoking, use of steroids in any form.

The diagnosis of CSC can be done by a dilated fundus examination combined with multiple imaging modalities like the OCT, FFA or ICG. With the advent of newer imaging modalities fresh insight has been gained in the possible pathology of CSC and hence the possible use of various drugs has been explored for managing the condition medically. Studies based on OCT analysis have demonstrated choroidal thickening in eyes with CSC suggesting CSC primarily involves vascular deregulation in choroids; a target area for majority of the drugs discussed.

Acute CSC usually has good visual outcome. However in certain cases permanent visual loss can occur secondary to photoreceptor damage or RPE atrophy[1]. There are certain specific indications for treatment of CSC which include

- Single CSC attack of more than 3 months duration
- Recurrent CSC
- Chronic CSC/ diffuse retinal pigment epitheliopathy
- If the fellow eye suffered from permanent visual loss due to CSC
- For rapid rehabilitation in patients with special occupational demands for binocular visual function

Apart from resolution of sub-retinal fluid, CSC treatment is aimed at shortening the time to visual rehabilitation, decreasing the rate of recurrences and a possible effect on restoration of visual function.

Discussed below are a few agents which have been demonstrated to have a significant effect in managing CSC medically.

## ANTI CORTICOSTEROID THERAPY

### A. Mineralocorticoid Receptor antagonist

Corticosteroids are a known risk factor in occurrence of CSC. An increase in corticosteroid levels either endogenously or by external use can be responsible[2]. Corticosteroids refer to both glucocorticoids and mineralocorticoids. Glucocorticoid binds to both, the Glucocorticoid receptor and the Minerlocorticoid receptor (MR). Primarily located in kidneys , recent studies have confirmed that MR is expressed in choroidal vasculature as well. Eplerenone a selective MR antagonist originally used for treatment of hypertension and congestive heart failure has been found to be effective in medical management of chronic CSC[3]. Popular MR antagonist are eplerenone and spironolactone. The affinity of eplerenone is 10-20 fold less than that of spironolocatone for MR , however it has much higher specificity and is also devoid of antiandrogenic effects of spironolactone, hence more popular.

Dose: 25mg/day for a week followed by 50 mg/day for 3 months-6 months depending on response

Contraindication: liver or kidney disease, pregnancy, concomitant use of drugs which increase serum potassium level

While on treatment patient can experience sedation, fatigue, also serum potassium and blood pressure need to be monitored.

### B. Rifampicin

It is a mainline antibacterial drug used in treatment of tuberculosis and leprosy. It induces cytochrome P450 thus altering metabolism of steroid leading to a decrease in plasma level of steroids[4]. Since its first described role in medical management of CSC in 2010 various studies have successfully reported its role in resolution of subretinal fluid in CSC.

Dose: Oral Rifampin is administered in a dose of 300 mg twice per day for 3 months

Being an inducer of cytochrome P450 complex enzymes, Rifampin has multiple drug interactions and should be cautiously used on patients taking other medications.

Common side effects of include elevation in liver enzymes, blood urea nitrogen (BUN) elevation, serum uric acid elevation, gastrointestinal upset, rash, flushing, anorexia, and flu-like symptoms. Rarely it can also cause hepatitis, renal failure, Stevens–Johnson syndrome.

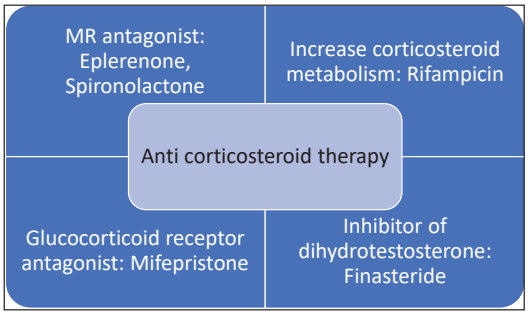
### C. Mifepristone

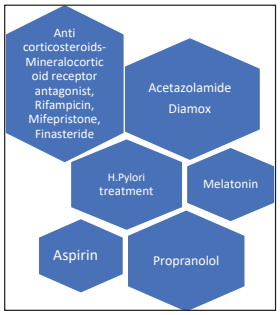
It is a high affinity anti progesterone drug and a glucocorticoid receptor antagonist. Popularly used as an abortifacient, its rationale for use in CSC is based on the fact that it lowers endogenous cortisol production[5].

**D. Finasteride-** the role of androgens such as testosterone has also been postulated in pathophysiology of CSC. Finasteride, a 5 alpha reductase inhibitor prevents conversion of testosterone to dihydrotestosterone which is more potent than testosterone[6].

## ACETAZOLAMIDE

It is a carbonic anhydrase inhibitor, popularly known as Diamox, originally used to control intraocular pressure in glaucomatous eyes. In CSC it enhances subretinal fluid absorption by causing acidification of the subretinal space by modulation of carbonic anhydrase 4 in retinal pigment epithelium (RPE) and an increase in retinal adhesiveness[7]. Acidification of the subretinal space is responsible for the




increased fluid resorption from the retina through the RPE. There is no influence on the final visual outcome or the recurrence rate however the resolution of subretinal fluid is clinically enhanced by the use of this drug.

Dose- 250 mg per day as a slow release formulation

Contraindication – allergy to sulpha drugs

Side effects from the medication, include paresthesias, nervousness, and gastric upset.

## MELATONIN

In humans, melatonin participates in the regulation of sleep, seasonal disorders, and aging. Besides the pineal gland, melatonin is also biosynthesized in the retina, where it behaves as an endogenous neuromodulator and has an inhibitory effect on glucocorticoid action. Melatonin and its metabolites act as a direct and indirect antioxidant, scavenging free radicals, stimulating antioxidant enzymes, and enhancing the activities of other antioxidants[8].

Its usual dose is 3 mg three times a day for a month. It is fairly well tolerated drug with no adverse reactions and is safe in pregnancy.

## PROPRANOLOL

Originally used for management of hypertension, propranolol has been found to have a positive effect on absorption of SRF[9]. Oral propranolol is administered at a dose of 40 mg twice a day

## LOW DOSE ASPIRIN

There is a state of hypercoagulability and increased platelet aggregation in CSC secondary to elevated steroids which can cause a focal, transitory, or permanent occlusion of the choroidal vasculature. Aspirin possesses platelet antiaggregant effects and therefore the rationale behind use of low dose aspirin in CSC. Used at a dose of 100 mg per day, certain studies have documented beneficial effet of this drug in chronic cases of CSC[10].

## H. PYLORI TREATMENT

There has been found a higher prevalence of H.pylori infection in patients with CSC. Resolution of CSC has been found to be directly correlated with eradication of H.Pylori infection[11]. For treatment of H.Pylori infection Triple therapy is advisable which includes combination of two antibiotics (clarithromycin, metronidazole or amoxycillin) along with a PPI- proton pump inhibitor.

## ANTI VEGF TREATMENT

This group of drugs have shown inconsistent results in treatment of CSC. The rationale behind their use is that they have a number of effects that reduce vascular leakage, including the upregulation of tight junctions between endothelial cells and reduction of vascular fenestrations. Systemic side effects are minimal, the ocular side include inflammation, vitreous haemorrhage and endophthalmitis which is rare. Bevacizumab[12], ranibizumab[13] and aflibercept (CONTAIN study)[14] are the usual drugs used.

Although none of the agents listed above have a direct effect on final visual outcome, they all have been found to have a positive effect on early resorption of SRF as demonstrated on OCT and FFA findings. Cases of recurrent, chronic CSC warrant additional treatment apart from observation in order to hasten resolution of neurosensory detachment in CSC.

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