Central serous retinopathy (CSR) is characterised by acute reduction in central vision associated with serous detachment of the neurosensory retina in posterior pole. The condition has also been known as central serous chorioretinopathy (CSCR) and central serous choroidopathy (CSC).

Most descriptions of CSR refer to the classic acute CSR. It is increasingly recognised that there are different clinical subtypes of CSR. Though CSR occurs more frequently in the third to fourth decade, it also affects the elderly. However, neovascular age-related macular degeneration (AMD) needs to be ruled out in the elderly.

CSR is known to be an idiopathic condition though risk factors like stress, hypertension, corticosteroids, adrenergic drugs, smoking and pregnancy are reported associations with acute CSR. There is a male preponderance with a male:female ratio of 10:2. Unilateral involvement and single episode is more frequent; however, up to a third of the patients experience recurrent episodes.

Clinical types
CSR can be described as per three observed clinical patterns.

Acute CSR: This is the classic description of CSR as mentioned above. A patient with acute CSR presents with blurred vision, reduction in contrast sensitivity and colour vision, metamorphopsia and a small hyperopic shift. Classically there is a circular serous detachment of central retina, sometimes with dull yellow deposits and in some cases with serous RPE detachment (Figure 1). The amount of serous fluid varies and visual deficit may range from 6/9 to 6/60. Sometimes eccentric CSR can be found. The serous detachment may increase gradually, however, most cases see spontaneous resolution of fluid with improvement in visual acuity within three months. It is not uncommon to have some residual visual deficit. The retina may develop stippled retinal pigment epithelium (RPE) atrophy and focal pigmentation in the area.

Acute persistent CSR: Some of the acute CSR cases have persistent subretinal fluid beyond three months with persistent visual symptoms; however, the visual acuity remains stable. Such cases often experience fluctuating levels of subretinal fluid, with some cases resolving and others becoming chronic CSR.

Chronic CSR: Chronicity based on the duration of CSR (e.g. > three or six months) has been described in studies on the treatment of CSR, but some of these cases may be of acute persistent CSR. There are distinct morphological changes observed in cases of chronic CSR especially the characteristic widespread RPE changes, often with pigment clumps and areas of retinal atrophy; with chronic shallow subretinal fluid or fibrosis seen best on the optical coherence tomography (OCT) scans. These RPE changes are referred to as diffuse retinal pigment epitheliopathy (DRPE). Some chronic CSR patients have gravitational ‘descending tracts’ in inferior retina with RPE changes. There is increased risk of CNV in chronic CSR. Patients with chronic CSR experience visual deterioration with time due to photoreceptor loss, hence the visual prognosis is less favourable.

Pathophysiology
The hypothesis of the focal RPE leak can be supported by fundus fluorescein angiography (FFA) appearance of ‘smoke stack’ and ‘ink blot’. RPE pump dysfunction has also been postulated with reversal of fluid movement in to the retina.

The indocyanine green (ICG) angiography and imaging of choroid with HD OCT (EDI/Swept source) has led to better understanding of the pathology in CSR. The increased permeability of the choroid is noted on ICG angiogram, so is increased choroidal thickness on enhanced OCT scans.
These would be consistent with the theory of raised hydrostatic pressure in the choroid as the primary cause. We have observed polypoidal vascular changes in the choroid in cases of CSR, often correlated with focal leaks on ICG angiograms. These vascular changes appear to be the source of leakage and a possible reason for increased choroidal thickness, as both seem to resolve with focal photodynamic therapy (PDT) of the polypoidal lesions.

**Investigations**
Serial OCT scans provide the best information as to the evolution of CSR. This can be supplemented by retinal angiography especially combined FFA and ICG angiography. EDI OCT can offer additional information as to the choroidal thickness change in CSR.

**Management**
Acute CSR cases can be observed as spontaneous resolution is expected, usually in three months. For acute persistent CSR, a conservative approach can be adopted. However, where there is significant visual disturbance and/or earlier visual rehabilitation is needed, treatment with PDT should be considered. Focal argon laser treatment for extrafoveal focal leaky spots can be considered, however, its use has been infrequent due to potential side effects of blind spots, risk of choroidal neovascularization (CNV) formation and enlargement of burn.

For chronic CSR cases, PDT has been shown to be an effective treatment. The PDT has been applied using various strategies such as reduced fluence (by either reducing power of laser or duration of laser exposure) or reduced dose of verteporphpin injection. In published reports, the PDT was applied to the area of choroidal hyperpermeability. The Bradford targeted PDT (tPDT) protocol involves full dose verteporphpin injection followed by targeted focal PDT to individual choroidal polypoidal changes for the duration of 60 seconds. The tPDT is guided by a combined ICG FFA, which helps to localise focal spots of choroidal vasculopathy. Vasoocclusion of the polypoidal change is observed with remodeling of the choroidal vasculature and resolution of subretinal fluid and restoration of retinal architecture.

Following PDT, patients can be followed up at three monthly intervals and if there is no recurrence can be discharged after six months. If required, tPDT can be repeated. There are some reports of anti-vascular endothelial growth factor (antiVEGF) injection treatment for CSR; however, there is lack of conclusive evidence for its use.

**Complications of CSR**
The common sequel of CSR is mild pigmentary changes in the macula. Rarely there may be subretinal fibrosis affecting the macula especially with chronic CSR cases. Some CSR may be complicated by development of CNV and are at risk of sudden loss of vision. Such patients should be managed with antiVEGF injection treatment as required. It is increasingly recognised that macula clinics have undiagnosed CSR cases which seemingly have ‘poor response’ to the AMD treatments received. Such cases may benefit from further investigation with ICG and alternative treatment such as tPDT as necessary.

For optimum management, clinical subtypes of CSR should be identified. New technology has helped define various phenotypes; however, there is a need for further research for better understanding of CSR and its management.


Figure 1. Acute CSR with circular serous detachment and yellow deposits. An OCT scan shows a PED within serous detachment.