Central Serous Chorioretinopathy

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6

Core Messages

- Central serous chorioretinopathy is seen mainly in middle-aged males, and is particularly linked to stress and corticosteroids
- Leaks are seen from the level of the retinal pigment epithelium
- There is an underlying choroidal vascular hyperpermeability
- Many cases resolve spontaneously
- Thermal laser and photodynamic therapy are treatment options

6.1 Introduction

Central serous chorioretinopathy (CSC) is characterized by an idiopathic circumscribed serous retinal detachment that is usually confined to the posterior pole caused by leakage of fluid though the retinal pigment epithelium. Some patients have a more chronic version of the disease that can often have descending tracts of fluid inferiorly. Eyes with CSC do not have signs of intraocular inflammation, accelerated hypertension, infiltration or infarction of the choroid or retinal pigment epithelium [47]. The disease was first described as recurrent central retinitis by von Graefe [55], and later by Horniker [26] as capillaro-spastic central retinitis. Horniker believed patients with this condition had a constitutional angioneurosis causing angiospasm and exudation. Gilford and Marquardt [19] termed the disorder central angiospastic retinopathy, and they too believed that the disorder was due to an angioneurotic diathesis. The name central serous retinopathy was adopted by Bennett [4]. Our understanding of the disease was greatly increased through the use of fluorescein angiography. Maumenee [35] first described the leak through the retinal pigment epithelium seen during fluorescein angiography. Gass [13] expanded the description of the fluorescein angiographic findings and named the condition idiopathic central serous choroidopathy. Over time it has been common to refer to the condition as central serous chorioretinopathy.

6.2 Systemic and Ocular Risk Factors

Central serous chorioretinopathy shows certain common demographic features [8, 13, 19, 20, 26, 35, 47, 48, 51, 55]. Although it has been described as occurring in young adults, two large studies found the mean age of affected patients to be mid to late 40s. Male patients substantially outnumber female patients, with a ratio reported in older studies of at least 6:1 [13, 19, 20, 26, 35, 47, 51, 55]. Subsequent studies have shown

that the male:female ratio is less than 3:1 [24, 53]. CSC seems notably severe in certain races, particularly in patients of Hispanic and Asian descent. CSC has been stated to be uncommon in African-Americans, but some authors disagree with this contention [9]. In Western countries CSC appears to be more common in patients with hyperopia or emmetropia, although this association may not be true in other regions, particularly Japan. Patients with CSC frequently have had a preceding stressful event [18] and are likely to be socially well-integrated men, mostly white collar workers or self-employed [46]. Many patients with CSC are self-motivated, pressure themselves to succeed, and seem to internalize stress.

Tittl and associates, in a retrospective case-control study of 230 patients, found that use of corticosteroids (used by 9.1 % of patients), psychotropic medications and the presence of hypertension were risk factors for central serous chorioretinopathy [53]. Haimovici and co-workers [24] found in a retrospective case-control study of 312 patients the use of corticosteroids (used by 14.4 % of patients), pregnancy, antibiotic use, alcohol use, untreated hypertension, and allergic respiratory disease were associated with CSC. Later a smaller, but prospective, case-control study confirmed the finding that corticosteroids are a risk factor for the development of CSC [30]. Particularly severe CSC can occur in patients who have had organ transplants and are being treated with medications to prevent rejection such as corticosteroids [16, 42] or in women who are pregnant [15]. Ocular findings thought to be specifically related to organ transplantation [17] were later described in patients being treated with corticosteroids who never had organ transplantation, but who did have corticosteroid induced CSC [28]. Many patients with CSC have elevated 24-h urine corticosteroids, which may contribute to the pathogenesis of disease [23]. Excessive use of sympathomimetic agents has also been associated with CSC [36]. In addition, in one study the plasma concentrations of epinephrine and norepinephrine were found to be higher among CSC patients than in controls [52].

Summary for the Clinician

- **Middle-aged males**
- **Hyperopic or emmetropic**
- **Stress**
- **Corticosteroid use common**

6.3 Presenting Symptoms

The most common symptoms of CSC are decreased and distorted vision. The visual acuity is usually reduced to between 20/30 and 20/60, and can be partially corrected with a low plus lens. Some patients, particularly those with severe or recurrent disease, have visual acuities as low as 20/200. With the onset of the neurosensory detachment, patients describe symptoms of metamorphopsia, micropsia, persistent after images, altered colour vision, and a central dimness in vision that may have grey, or sometimes, a purple cast. Younger patients with CSC usually have unilateral involvement, while older patients are more likely to have bilateral involvement. Patients with inferior detachments from gravitating fluid can have superior visual field defects.

Summary for the Clinician

- **Decreased or distorted vision**
- **Improvement in vision with a small plus lens**
- **Older patients are more likely to have bilateral disease**

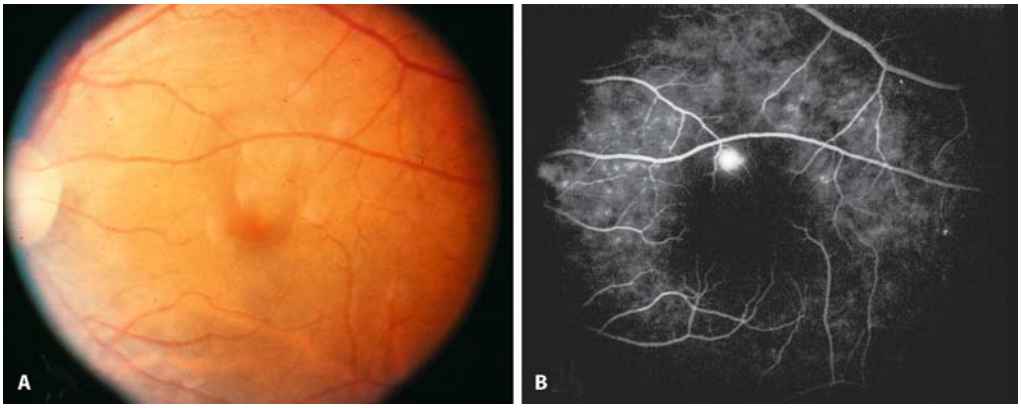


Fig. 6.1. **A** This patient had a localized serous detachment of the macula secondary to a focal leak (**B**)

6.4 Ocular Findings of Classic CSC

There are three different types of presentation for CSC. The first and most common is a solitary, localized neurosensory detachment in the posterior pole, and has been referred to as classic or acute CSC. By biomicroscopic examination a blister of clear fluid is seen elevating the macula (Fig. 6.1). The base of the detachment is ringed by light reflexes where the sloping retina reflects light back to the observer. Observation of turbid subretinal fluid, often, and subretinal blood almost always, suggests a diagnosis other than CSC. Serous retinal pigment epithelial detachments (PEDs) are commonly seen in association with CSC. They form when the RPE cells, and their associated basement membrane, separate from the underlying Bruch's membrane. Serous PEDs are seen as smooth, circumscribed, orange-coloured elevations with a slightly darker rim. When the slit beam of the biomicroscope illuminates a serous PED, particularly from the side, the entire PED emanates a characteristic glow. Prominent PEDs are found in three conditions: CSC, CNV, particularly occult CNV, and polypoidal choroidal vasculopathy, a variant of CNV. Some patients may have the deposition of what has been termed subretinal fibrin. The subretinal fibrin is greyish-white feathery edged plaque that occurs over "energetic" leaks. Frequently there may be a concomitant underlying PED with the fibrin accumulating radially around the top of the PED making a ring appearance. On ultrasonographic evaluation the choroid does not appear thickened.

Summary for the Clinician

- **Acute focal leak from the RPE seen during fluorescein angiography**
- **Multifocal choroidal hyperpermeability seen during ICG**
- **Circumscribed round or oval detachment**
- **Pigment epithelial detachments common**
- **Often younger patients**

6.5 Angiographic Findings of "Classic" CSC

Fluorescein angiography in acute cases of classic CSC demonstrates one or several hyperfluorescent leaks at the level of the reti-

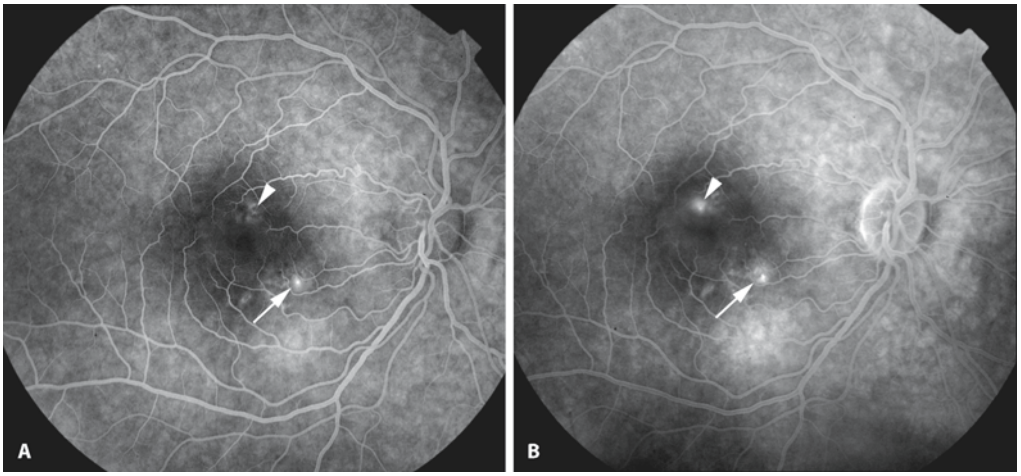
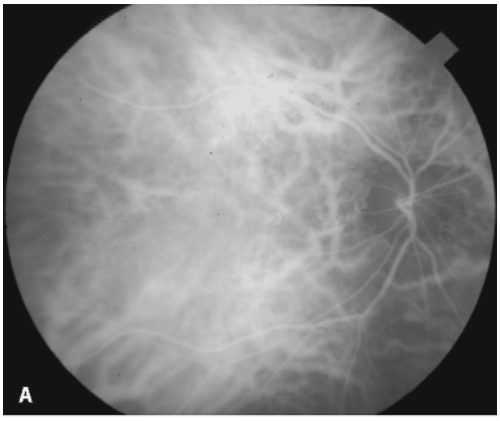
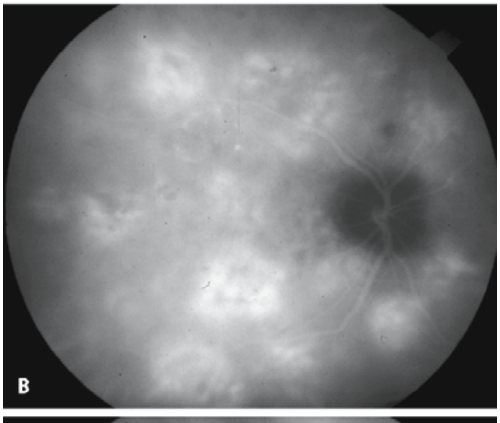


Fig. 6.2. **A** This patient developed a macular detachment from a solitary leak (*arrow*). Note the pigmentary change in the superior macula (*arrowhead*). **B** While waiting for spontaneous resolution of the leak in **A**, the patient noticed a worsening of his symptoms. Reimaging with fluorescein angiography revealed an additional leak in the superior macula (*arrowhead*)

nal pigment epithelium (RPE). In a minority of cases (10 %) the dye rises up under the neurosensory detachment as a "smokestack" leak. This pattern is thought to be related to the increased concentration of protein in the fluid accumulating in the detachment [45]. The new fluid entering into the detachment has less protein and consequently a lower specific gravity. The newly entering fluid rises up and then spreads out when it reaches the dome of the detachment. A more commonly seen pattern of dye leakage is manifested as a small blot-like leak that increases in size during the angiographic evaluation (Fig. 6.2). In the later phases of the angiogram the dye diffuses throughout the fluid and is seen to pool within the detachment. Smokestack leaks are usually associated with larger areas of retinal detachment. In any case focal leaks are somewhat more common nasally than temporally, superiorly than inferiorly [51].

Indocyanine green (ICG) angiography demonstrates patchy areas of choroidal vascular hyperpermeability (Fig. 6.3) [40, 43, 49]. These areas are best seen in the mid-phases of the angiogram, and appear localized in the inner choroid. With time the liver removes the indocyanine green from the circulation, and the dye that has leaked into the choroid appears to disperse somewhat, particularly into the deeper layers of the choroid [49]. This produces a characteristic appearance of hyperfluorescent patches in the choroid with silhouetting of the larger choroidal vessels in the later phases of the ICG angiographic evaluation. The total area of choroidal vascular hyperpermeability was seen to be correlated with age and with the type of CSC. Patients with CSC but no fluorescein leakage have areas of underlying choroidal vascular hyperpermeability just the same. Younger patients may have PEDs as a forme fruste of CSC in that underlying choroidal hyperpermeability may cause elevations of the RPE without creating breakthrough leaks (Fig. 6.4).





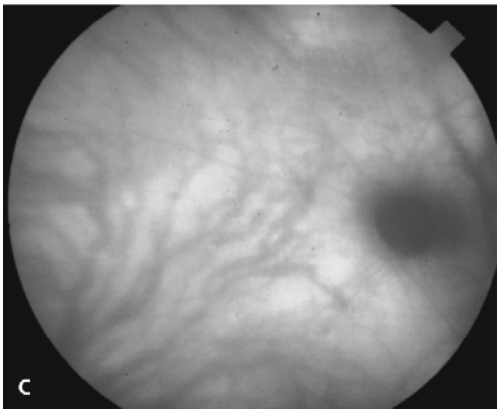
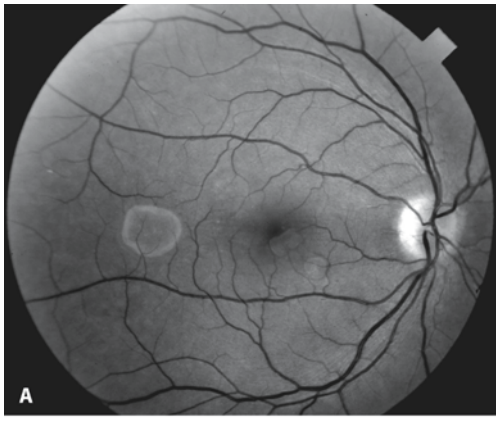
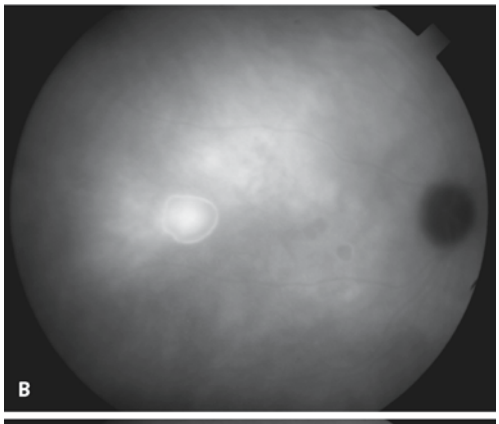


Fig. 6.3 A–C. Typical ICG angiographic findings of CSC. **A** Initially after injection the dye is seen in the vessels. By the midphase of the angiogram the dyes leaks out into clouds of hyperfluorescence. **C** With time the dye is removed from the circulation and the dye that has leaked out from the vessels diffuses outward and posteriorly. The larger choroidal vessels are seen in silhouette





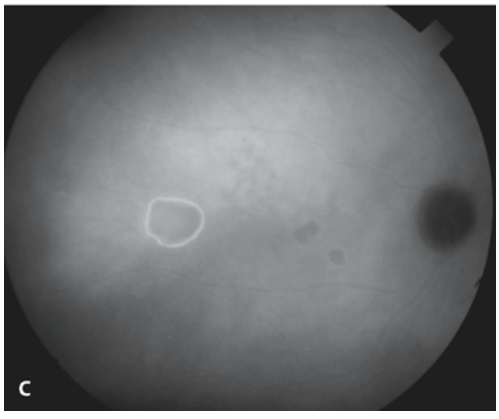


Fig. 6.4 A, B. This patient was treated with oral prednisone and developed an alteration in his vision in the right eye. He had a PED (**A**), which was on top of an area of choroidal vascular hyperpermeability (**B**). The later phases of the angiogram show the PED as a ring of hyperfluorescence, a common finding for larger PEDs (**B**, **C**)

Summary for the Clinician

- **The classic form is the most common presentation of CSC and is usually simple to diagnose**

6.6 Chronic CSC

A second principal presentation of CSC shows widespread alteration of pigmentation of the RPE in the posterior pole that appears to be related to the chronic presence of subretinal fluid. This variant of CSC has been termed "diffuse retinal pigment epitheliopathy" (DRPE) or "chronic CSC" [49]. Just as central serous chorioretinopathy has had many names during its history, so has this more chronic variant. DRPE is related to not only a past history of CSC, but also to the age of the patient at the time of diagnosis [48]. Patients with DRPE generally have a more pronounced loss of visual acuity, and may have permanent loss of visual acuity to the level of legal blindness.

Summary for the Clinician

- **Numerous chronic leaks from the RPE seen during fluorescein angiography**
- **Multifocal choroidal hyperpermeability seen during ICG**
- **Broad shallow detachment**
- **Pigment epithelial detachments less common**
- **Often older patients**
- **Often misdiagnosed as being something else**

6.7 Ocular Findings of DRPE

Patients with DRPE have relatively flat, broad detachments. Close examination of the retina by slit lamp biomicroscopy may show thinning of the retina and possible cystic changes within the retina. There are often RPE alterations that are manifest in three different ways. The RPE can show atrophy where there is a loss of pigmentation and increased visibility of the underlying larger choroidal vessels. This atrophy is readily seen with autofluorescence photography. The RPE can have areas of focal hyperpigmentation. Finally some patients may have RPE hyperplasia to the point where they develop bone spicules. The intervening subretinal fluid is clear, but it is not uncommon to see flecks of subretinal lipid. This feature may suggest the presence of occult choroidal neovascularization (CNV) when in fact the diagnosis is CSC. The broad areas of detachment in chronic CSC can occupy the posterior pole and tracts of fluid may descend inferiorly toward the equator.

6.8 Angiographic Findings of DRPE

The various diffuse areas of disturbance of the RPE are easy to see with fluorescein angiography. These areas have a granular hyperfluorescence due to relative atrophy of the involved RPE and associated subtle, indistinct leaks (Fig. 6.5). There may be dependent retinal detachments into the inferior fundus with associated atrophic tracts of the RPE (Fig. 6.6) [56]. These patients also may have capillary telangiectasis, capillary non-perfusion [1], and secondary neovascularization associated with the chronic detachments. Because of the widespread alteration in pigmentation and chronically reduced visual acuity, these patients are sometimes misdiagnosed as having an inherited retinal or macular dystrophy. ICG angiography of DRPE shows the same type of widespread choroidal vascular hyperpermeability as patients with typ-

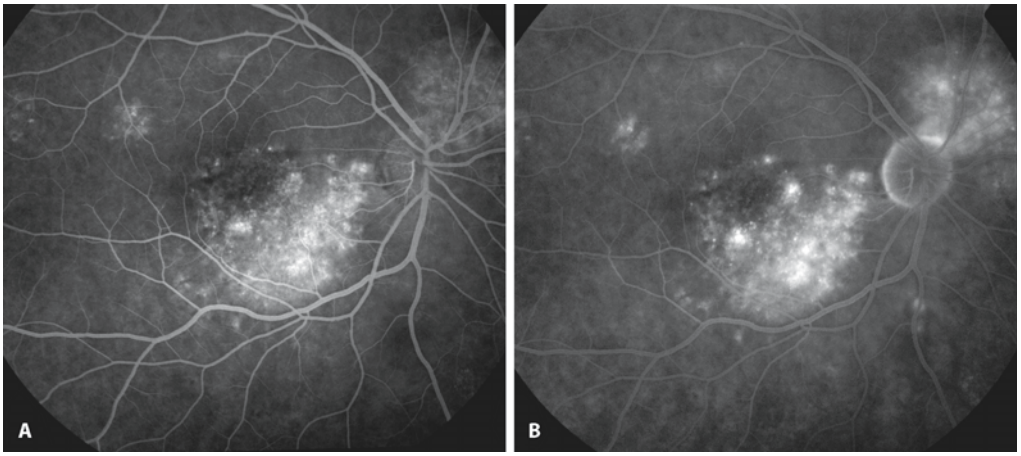


Fig. 6.5 A, B. This patient had DRPE with granular hyperfluorescence (**A**). Later in the angiogram subtle leakage can be seen from a number of points (**B**)

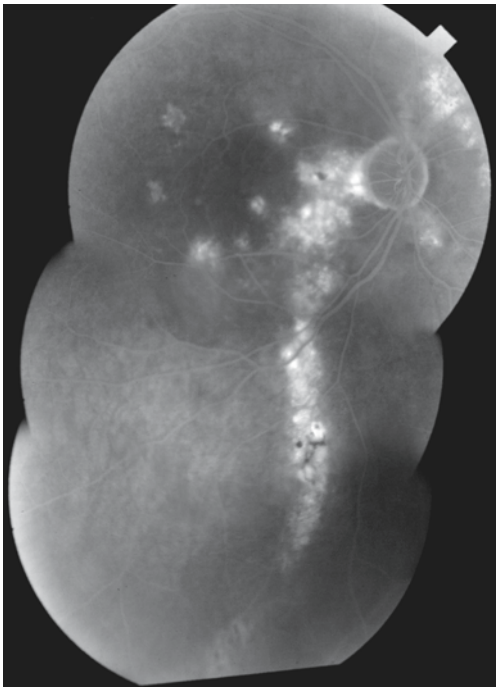


Fig. 6.6. A common finding in chronic CSC is the descending tract of fluid

ical CSC have, except the number and area of hyperpermeability seem to be greater in patients with DRPE.

Summary for the Clinician

- **DRPE is not uncommon. Because DRPE may cause a variety of fundus changes, it has been diagnosed as other conditions**

6.9 Bullous Detachment of the Retina Secondary to CSC

There is an additional, but rare form of CSC that causes bullous retinal detachments (Fig. 6.7). Although most patients have one to three leaks seen during fluorescein angiography, in an unusually severe variant of CSC some patients have numerous, exuberant leaks, which are not necessurily in the macular region, multiple PEDs and bullous retinal detachments that extend into the inferior periphery of the fundus [14]. Several reports of this condition originated in Japan, where this variant seems more common [1, 40, 54]. Bullous serous retinal detachments have also been reported in patients who have had organ transplantation [12]. Patients with bullous detachment have the same findings during ICG angiography

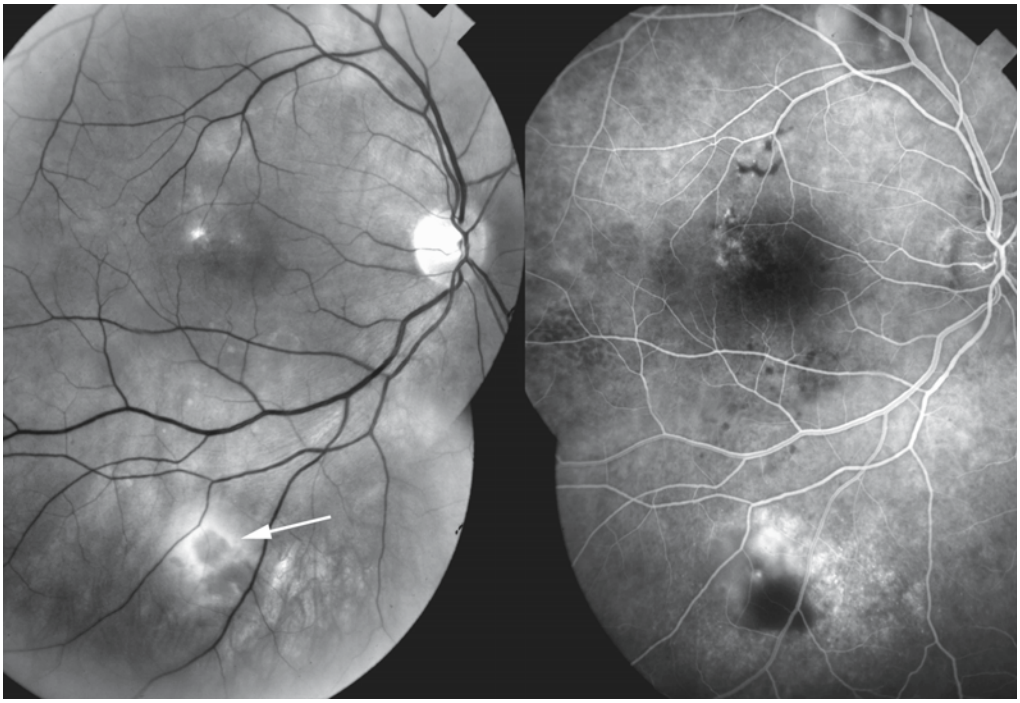


Fig. 6.7. Bullous detachment variant of CSC. Note the area of subretinal fibrin surrounding a PED (*arrow*)

that patients with classic CSC and DRPE do, except the number and size of areas of choroidal hyperpermeability are greater.

6.10 Subretinal Deposits

Patients with CSC, of any variety, may have deposition of subretinal material that occurs in three main forms [15, 27]. The first is subretinal fibrin and the second lipid. A third deposit can be seen in almost every patient with CSC lasting more than a few months (Fig. 6.8). These are small white dots that form on the outer retinal surface. Some of the patients have been initially suspected of having retinitis, choroidal tumours, or CNV because of the subretinal deposits. The small white dots probably represent macrophages with phagocytized outer segments.

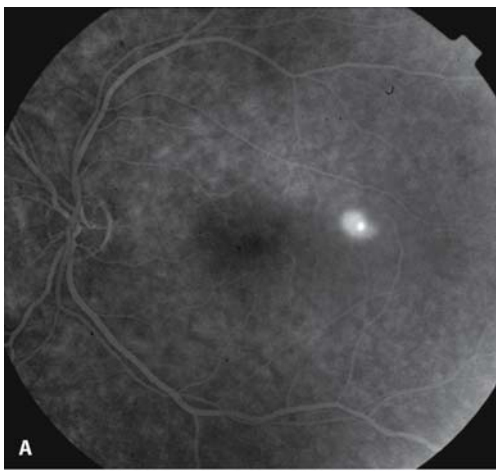


Fig. 6.8. **A** This patient had a serous detachment related to a focal leak. He had punctuate subretinal deposits as seen in stereophotographs (**B**). Fig. 6.8 B see next page

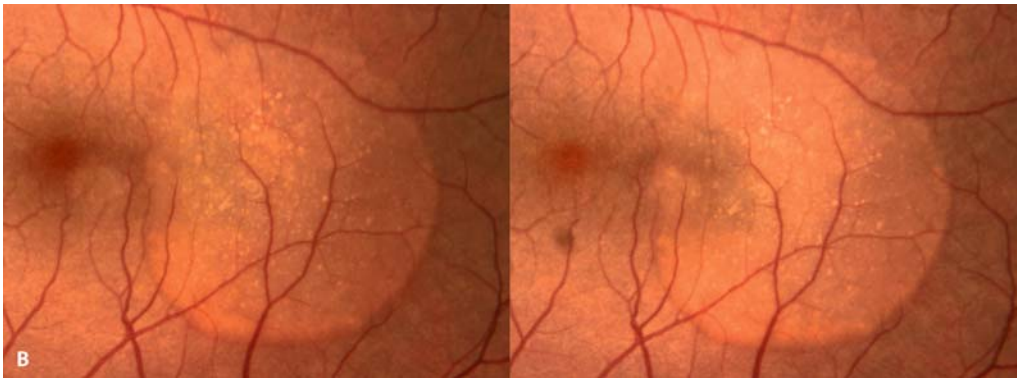


Fig. 6.8 B.

6.11 Differential Diagnosis

The principal condition that needs to be differentiated from CSC is choroidal neovascularization, particularly occult CNV. The ocular findings of CNV share many similarities with those of CSC: both groups of patients may have neurosensory detachments, PEDs, mottled depigmentation, hyperpigmentation, areas of RPE atrophy, and subretinal deposits of fibrin and lipid [48]. Patients with CNV, though, have thickening at the level of the RPE, notched PEDs, and subretinal or subpigment epithelial blood, findings not seen in CSC. In addition, eyes with CNV generally have coexistent ocular findings related to the generation of new blood vessel growth. These factors include punched-out chorioretinal scars in the presumed ocular histoplasmosis syndrome, lacquer cracks and areas of choroidal atrophy in pathological myopia, breaks in Bruch's membrane in cases of choroidal rupture, and drusen and pigmentary clumping in patients with age-related macular degeneration.

The CNV secondary to proximal causes such as chorioretinal scars or choroidal ruptures generally has "classic" findings on fluorescein angiography. These cases demonstrate a lacy vascular pattern of hyperfluorescence that shows increasing leakage and staining throughout a fluorescein angiographic evaluation. Occasionally a specific feeder vessel can be seen extending from the chorioretinal scar. The fluorescein angiographic findings of exudative age-related macular degeneration may be much more difficult to differentiate from CSC. Although AMD may present with classic CNV, where the new vessels are easily demonstrable during fluorescein angiography, the vast majority present with "occult" CNV. ICG angiography of CSC demonstrates multifocal choroidal vascular hyperpermeability, usually bilateral, that has specific temporal and topographical characteristics. The hyperpermeability in CSC is most evident in the mid-phases of the angiographic evaluation. The later phases of ICG angiography show dispersion of the dye with negative staining of the larger choroidal vessels. CNV, on the other hand, shows a unilateral, unifocal area of hyperfluorescence that usually shows progressively increasing contrast with the surrounding choroid in the later phases of the angiogram. ICG angiography may provide important information to help rule out the presence of occult CNV.

The differential diagnosis of CSC also includes a variety of infiltrative conditions, inflammatory diseases, congenital ocular abnormalities, and rhegmatogenous retinal detachments. Infiltrative conditions, such as leukaemia, amelanotic melanoma or metastatic disease, generally have a different colour than the surrounding normal choroid, demonstrate thickening of the choroid by ultrasonography, and do not have serous PEDs. Eyes affected with inflammatory conditions such as posterior scleritis or Harada's disease show signs of intraocular inflammation, like iritis or vitritis, have patches of yellowish discolouration in the posterior pole, demonstrate staining of the optic nerve head during fluorescein angiography, and have thickening of the choroid by ultrasonography. Extraocular symptoms, such as headache, neck stiffness, and vomiting are common in Harada's disease. Patients with optic nerve pits may have a serous detachment of the macula, but the optic nerve problem is generally readily visible. The macular elevation in patients with optic nerve pits generally appears as a bilaminar detachment of the macula. There are no leaks from the level of the RPE during fluorescein angiography in patients with optic nerve pits. Rhegmatogenous retinal detachments may cause elevation of the macula, but they have an associated retinal hole or tear and do not have leaks visible during fluorescein angiography.

Summary for the Clinician

- **Choroidal neovascularization is the most important disease to rule out**
- **Occult CNV usually has thickening at the level of the RPE, and may have associated blood**
- **ICG angiography is useful in differentiating DRPE from occult CNV**

6.12 Pathophysiology

With the advent of fluorescein angiography, ophthalmologists had a more precise method of diagnosing and evaluating CSC. Fluorescein angiography demonstrates a site or sites of fluorescein leakage in cases of active CSC. With cessation of these leaks the detachment was seen to regress. This suggested, at least to some observers, that the leak seen during fluorescein angiography represented fluid coming from the choroid into the subretinal space through a defect in the continuity of the RPE. The fluorescein, contained in the choroidal fluid, was brought into the subretinal space with the bulk fluid flow going from the choroid toward the retina.

The balance of the tissue oncotic and hydrostatic pressures ordinarily causes fluid flow from the retina toward the choroid. In experimental models, injury or destruction of the RPE was seen to speed the resorption of subretinal fluid [39]. These findings suggested that a simple defect in the integrity of the RPE alone could not explain the findings seen in CSC. To help explain the findings of CSC based, in part, on findings from animal models, several newer theories were postulated. One theory stated that what appeared to be leaks at the level of the RPE were in fact not necessarily active leaks, but were areas where dye diffused into the subretinal space [33]. The neurosensory detachment was thought to be secondary to widespread areas of RPE dysfunction. This theory did not clearly elucidate why the areas of RPE dysfunction occurred or why CSC spontaneously improves, as it frequently does. The theory also did not explain why patients with CSC frequently develop PEDs, or why laser treatment to a "leak" causes a rapid resolution of the neurosensory detachment. Another theory

suggested that a focus of RPE cells, losing their normal polarity, pumps fluid from a choroid to retina direction, causing a neurosensory detachment [50]. This theory could not explain the presence of PEDs, subretinal fibrin, or how a few RPE cells pumping in the wrong direction could overcome the pumping ability of broad areas of surrounding RPE cells.

Integration of the clinical findings of CSC with the ICG angiographic abnormalities of the choroidal circulation in patients with CSC led to new theoretical considerations. During ICG angiography the choroidal circulation appears to have multifocal areas of hyperpermeability [22, 25, 41, 43, 44, 49]. These areas of hyperpermeability may arise from venous congestion. Excessive tissue hydrostatic pressure within the choroid from the vascular hyperpermeability may lead to PEDs, disruption of the retinal pigment epithelial barrier, and abnormal egress of fluid under the retina. In past studies leaks demonstrable at the level of the RPE invariably are contiguous with areas of choroidal vascular hyperpermeability [22, 25, 40, 41, 43, 44, 49]. On the other hand, most areas of hyperpermeability are not associated with actual leaks. These areas of hyperpermeability without leaks may affect the size, shape, and chronicity of any overlying neurosensory detachment by inducing changes in the ability of the overlying RPE to pump.

Theoretical considerations about why the choriocapillaris would develop increased permeability have been described elsewhere. Increased circulating epinephrine and norepinephrine levels have been found in patients with CSC. Administration of sympathomimetic compounds have been associated with CSC in humans and a CSC-like condition in monkeys, which actually were also given corticosteroids as well. It is possible to postulate that sympathomimetic compounds or corticosteroids, either endogenous or exogenous, alter the permeability of the choriocapillaris directly, or through secondary means such as affecting the autoregulation of the choroidal vessels. However, most theories about CSC and corticosteroids will have to be revised because of a simple observation: patients receiving intravitreal triamcinolone do not seem to develop CSC. The author has given hundreds of injections to patients and has not seen one case of induced CSC. Although the rate of developing CSC with corticosteroid use is not known, CSC is a relatively common disease. Clearly systemic administration of corticosteroids can lead to CSC, but whatever the physiologic alterations systemic administration causes, local administration does not appear to do so with anywhere near the same frequency. It may be that the local concentration is so high CSC inducing alterations do not occur. However, intravitreal corticosteroids eventually dissipate, leaving very low concentrations.

Summary for the Clinician

- **Many theories of pathogenesis**
- **Each theory is based on information known about the physiology at the time**
- **Choroidal vascular permeability appears to lead to increased hydrostatic pressure with breakthrough of fluid through RPE**
- **Corticosteroids and sympathomimetics induce CSC, suggesting that altered choroidal vascular permeability is induced by these compounds**
- **However, intravitreal triamcinolone has yet to be associated with CSC, suggesting a systemic route to the eye is required to produce CSC**

6.13 Histopathology of CSC

Knowledge of the histopathology of CSC is limited. Neurosensory detachment with subretinal and subpigment epithelial deposition of fibrin has been reported. A model of exudative detachment has been produced in monkeys with repeated injection of corticosteroids and epinephrine [37, 38, 60, 61]. While the monkeys developed neurosensory detachments, they demonstrated a leakage pattern on fluorescein angiography that appeared more like accelerated hypertension than just simple central serous chorioretinopathy.

Optical coherence tomography (OCT) because of its high resolution can provide optical biopsies, in effect. Although OCT has been commonly used to determine the presence of subretinal fluid, it can provide more information. Retinal atrophy has been seen in some patients. However, by normalizing the foveal thickness in one eye by that in the normal fellow eye a fairly linear inverse relationship between visual acuity and foveal thickness was found. In addition the ability to visualize finer anatomic details such as the external limiting membrane was much less in patients with lower levels of visual acuity, suggesting anatomic alterations occur that are associated with decreased acuity [10]. Patients with a history of chronic detachment and poor visual acuity after reattachment may have cystoid spaces within the retina, a condition that has been termed cystoid macular degeneration [29].

6.14 Natural Course

The large majority of patients with CSC spontaneously resolve and experience an almost complete restoration of vision. Patients frequently notice a slight permanent decrease in visual acuity, brightness, or colour discrimination in the affected eye, and may also notice a slight distortion in their central vision. Some patients have resolution of their neurosensory detachment, but regain only part of their central vision. These patients may have suffered photoreceptor damage, atrophy and irregular pigmentation of the underlying RPE, or have subretinal fibrosis.

Recurrence of CSC is not uncommon, and occurs in 40–50 % of patients [20, 31]. Some of these patients will go on to have recurrent focal leaks while others will inexorably progress to DRPE. Secondary CNV may occur, particularly in patients over 50 years of age [48].

Summary for the Clinician

- **Most patients with classic CSC will spontaneously improve and retain good acuity**
- **Recurrences are common in classic CNV**
- **DRPE is usually chronic or recurrent acute and many patients eventually lose significant acuity**

6.15 Treatment

Each treatment technique for CSC has been based to a certain extent on proposed mechanisms of pathophysiology at the time. The resultant treatment approaches for CSC have been varied, to say the least, and have usually been examined as part of

uncontrolled studies. Medical treatments have included diet modification, antihistamines, carbonic anhydrase inhibitors, beta-blockers, enzyme therapy, acupuncture, corticosteroids, non-steroidal anti-inflammatory agents, stellate ganglion blocks, and antiviral medications. No randomized controlled study has shown any drug to be useful in the treatment of CSC. Because of the suggestion that CSC may be related to abnormal levels of circulating adrenaline, the use of β-blockers has been suggested as a treatment. A small study suggested a possible benefit [2], but the findings have not been confirmed with either a larger study or a randomized trial. Adrenaline stimulates α- and β-receptors; blocking only β-receptors would allow unopposed α-stimulation. This might produce unwanted vascular constriction. Numerous reports have suggested that corticosteroids are associated with the production or worsening of CSC.

6.16 Photocoagulation Therapy

Photocoagulation by means of sun-gazing, xenon arc photocoagulation, and direct and indirect photocoagulation using a variety of different types of lasers has been performed. The most commonly studied modality in the treatment of CSC has been laser photocoagulation. The principal goal behind photocoagulation is to reduce the leakage through the RPE and cause a resolution of the subretinal fluid with improvement of visual acuity. Laser photocoagulation to the site of leakage seen during fluorescein angiography shortens the duration of macular detachment in patients with typical CSC, but does not appear to affect the final visual acuity [5, 6, 11, 21, 32, 59]. Laser photocoagulation appeared to reduce the rate of recurrence in some studies [6, 59], but not in others [6, 11, 21]. Approximately 1% of Japanese patients treated with laser photocoagulation for CSC develop choroidal neovascularization, which can have dire consequences [34]. The rate may be higher in Caucasian patients.

Because of the unfavourable risk:benefit ratio, laser photocoagulation generally is reserved for those patients with symptoms for greater than 4 months that are located greater than 375 µm from fixation and the need and desire for laser photocoagulation. If the leak is located well away from the central macula, then there is less reason to hesitate in giving laser photocoagulation. A detailed examination of the patient and the fluorescein angiogram for the presence of choroidal neovascularization is essential.

Summary for the Clinician

- Mechanism of action for laser not really known
- Decreases duration length of disease, final acuity not different
- May decrease rate of recurrence
- Use very mild photocoagulation
- CNV most important side effect of thermal laser

6.17 Methods of Photocoagulation

The laser photocoagulation of typical CSC begins with a patient with CSC and decreased visual acuity, who is unhappy with his or her vision, and has the need, occupational or otherwise, for improved visual acuity. A factor that may encourage laser photocoagulation is a history of CSC in the fellow eye with an unfavorable outcome. Serial fluorescein angiograms should show a leak or leaks that are in the same position from one angiogram to the next. The closest most central leak should be greater than 375 µm from the point of fixation.

The laser is set for a spot size of 200 µm and a power of 100–150 mW and an application time of 0.1–0.2 s. With a recent angiogram as guidance the more peripheral leaks are treated first. The amount of laser uptake varies with the amount of subretinal fluid present, the degree of pigmentation of the RPE, which is variably pigmented in areas of chronic subretinal fluid, the degree of RPE detachment, and the laser wavelength used. The leakage point is treated as well as a small surrounding region of normal RPE. Great care should be taken to obtain only a dull grey coagulation to avoid the possibility of secondary choroidal neovascularization.

The patient should be seen 2 weeks after treatment, and every 2 weeks for the next few follow-up visits. The subretinal fluid generally takes a few weeks to resorb. The visual symptoms start to abate with diminution of the subretinal fluid, but the time it takes for the patient to regain final visual acuity seems proportional to the amount of time the retina was detached. The initial follow-up examinations are to evaluate the patient for choroidal neovascularization.

If the patient is seen to have hemorrhage, increased turbidity of the subretinal fluid, or thickening at the level of the RPE in or adjacent to the area of laser treatment, secondary choroidal neovascularization should be suspected. The patient should have a repeat fluorescein angiogram at that point to help in establishing the diagnosis. Secondary CNV generally causes a nodular or crescent shaped area of hyperfluorescence under or adjacent to the area of previous laser photocoagulation. If the original site of treatment was sufficiently extrafoveal, it is possible to discover and treat secondary CNV, in many cases, before the neovascularization extends under the fovea. The CNV may be treated with thermal laser if sufficient room exists or with PDT.

Summary for the Clinician

- **Photocoagulation is not difficult, but a small percentage of patients may develop CNV. The patient needs to understand the risks and benefits of treatment.**

6.18 Photodynamic Therapy

DRPE represents a challenge to treat because of the diffuse nature of the problem. There are generally a number of subtle or indistinct leaks, usually distributed over a region. Grid laser photocoagulation to an area with these small leaks appeared to cause a decrease in the amount of subretinal fluid present [57], but did not cause a long-term change in the visual acuity. Several groups have investigated the use of photodynamic therapy (PDT) with verteporfin for more chronic forms of CSC [3, 7, 58]. Generally PDT causes the subretinal fluid to decrease or resolve completely. Recurrences of subretinal fluid occur, but are responsive to retreatment with PDT. Our group found that if a patient had very poor acuity before treatment the probability of improvement was limited even if the retina flattened. The treatment spot for the PDT was aimed at treating regions of choroidal vascular hyperpermeability seen during ICG angiography responsible for the fluid leakage into the macula. It probably is not necessary to use ICG angiography since useful information can be obtained with conventional fluorescein angiography. Avoidance of directly treating the central foveal may help reduce the possibility of unwanted side effects, such as inducing foveal atrophy with the PDT. Although the usual dose of verteporfin is commonly used, it may be possible to reduce the dose of medication, possibly resulting in de-

creased costs. On occasion PDT has been used for typical acute leaks. Because of the high cost of PDT, its use has been limited in classic CSC to those patients with focal leaks near the centre of the fovea where laser photocoagulation may induce excessive harm.

Summary for the Clinician

- **Used principally to treat DRPE**
- **Causes marked regression of subretinal fluid**
- **Visual acuity improvement not common if the patient starts with very low acuity**
- **Appears to decrease the amount of subretinal fluid in patients with DPRE. Long-term benefit is not known at present**

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