Other Vascular Disorders

13

Contents

13.1 Introduction

There are a number of conditions which stimulate neovascularisation of the retina with subsequent complications such as vitreous haemorrhage and tractional retinal detachment from pathological separation of the vitreous. The most common is severe diabetic retinopathy but also retinal vein occlusion, sickle-cell retinopathy and retinal vasculitis.

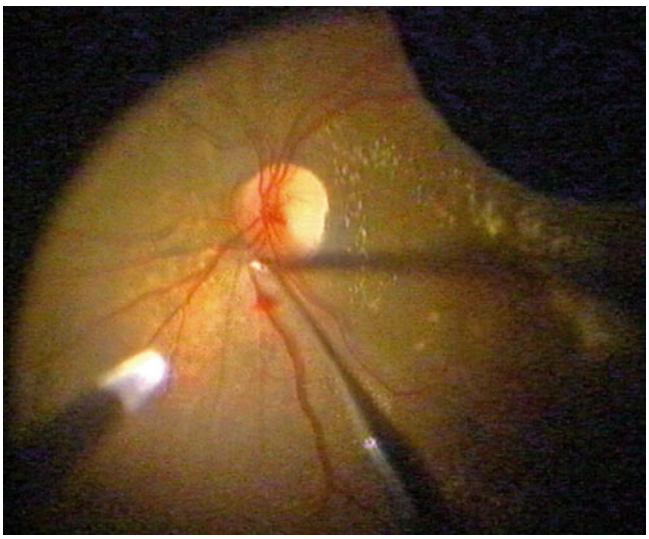

Fig. 13.1 It is possible to dissect the overlying artery off the underlying occluded retinal vein in a branch retinal vein occlusion to try to relieve the blockage

13.2 Retinal Vein Occlusion

Retinal vein occlusions (RVO) are the second commonest vascular events in the eye after diabetic retinopathy. The eye is unusual in suffering from occlusion of the veins more often than arteries. Retinal vein occlusion can be divided into two groups, branch retinal vein occlusion, BRVO, and central retinal vein occlusion, CRVO (which includes hemiretinal vein occlusion). BRVO occurs most commonly where a retinal arteriole crosses over a venule. Sharing an adventitial sheath in the presence of the thickened arteriolar wall may compress the thinner walled venule resulting in occlusion. In CRVO, pathological evidence suggests the site of obstruction is situated at the lamina cribrosa (Green et al. 1981). A number of innovations have been designed, but none are of proven benefit.

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New methods:

Chorioretinal anastomosis	CRVO
Intravitreal steroid injection	CRVO and BRVO
Pars plana vitrectomy	CRVO and BRVO
Arteriovenous decompression	BRVO
Radial optic neurotomy	CRVO
Tissue plasminogen activator	CRVO

13.2.1 Chorioretinal Anastomosis

Chorioretinal anastomosis using argon laser has been successful improving vision in one-third of eyes in the non-ischemic variant of the disorder in selected patients but has been associated with frequent complications (Browning and Antoszyk 1998; McAllister et al. 1998; McAllister and Constable 1995). This therapy is not generally used in ischemic CRVO because of a high complication rate in particular neovascularisation.

13.2.2 Arteriovenous Decompression

Grid macular laser therapy in BRVO can control macular oedema, with 60 % of treated cases retaining 20/40 vision or better at 3 years (Branch Vein Occlusion Study Group 1984) and reduce the incidence of vitreous haemorrhage by half (Branch Vein Occlusion Study Group 1984, 1986). In BRVO, the site of occlusion can easily be visualised and is thought to occur as a result of arteriosclerosis in the media of the artery giving rise to a compressive effect on the adjacent vein. At the arteriovenous crossing, the artery is generally located anterior to the occluded vein, within a common adventitial sheath. Charles attempted to relieve the blockage by dissecting the arteriole off the venule at the arteriovenous crossing in one patient (Osterloh and Charles 1988). Recently, the technique has been revisited with reported success in 10 out of 15 patients, with an average gain of four lines of vision (Opremcak and Bruce 1999). In a non-randomised comparative study, patients receiving intervention performed better with 75 % doubling their visual angles compared to 40 % with conventional treatment (Mason et al. 2004). Opremcak has designed an instrument for the blunt dissection of the arteriole from the venule by inserting a spatulated knife between the blood vessels. Potential complications include retinal tear or detachment, vitreous haemorrhage, retinal gliosis at the incision site, arcuate scotoma and cataract. Resolution of macular oedema following sheathotomy has been confirmed using optical coherence tomography (OCT) (Fujii et al. 2003). There are also theoretical reasons whereby the localised ischemia of the retina in BRVO might be improved by the PPV procedure because removal of the vitreous gel may allow oxygenation of the retina from other sites in the eye with good blood flow (Cringle et al. 1992). Indeed, some investigators have tried PPV and gas alone (Saika et al. 2001).

Table 13.1 Difficulty rating for arteriovenous decompression

Difficulty rating	High
Success rates	Unknown
Complication rates	Low
When to use in training	Late

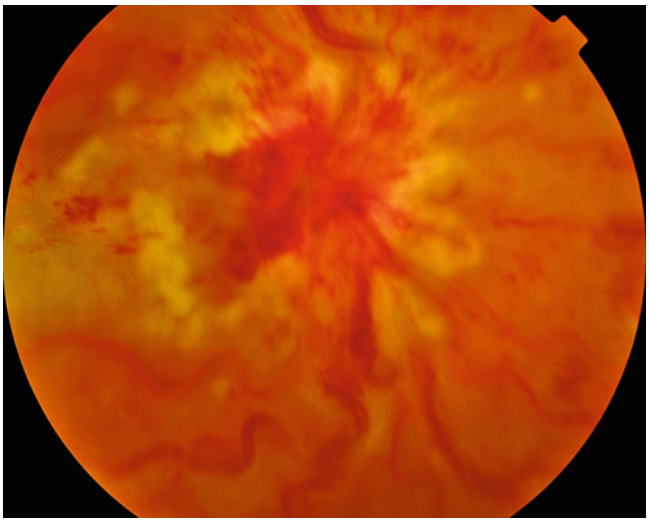
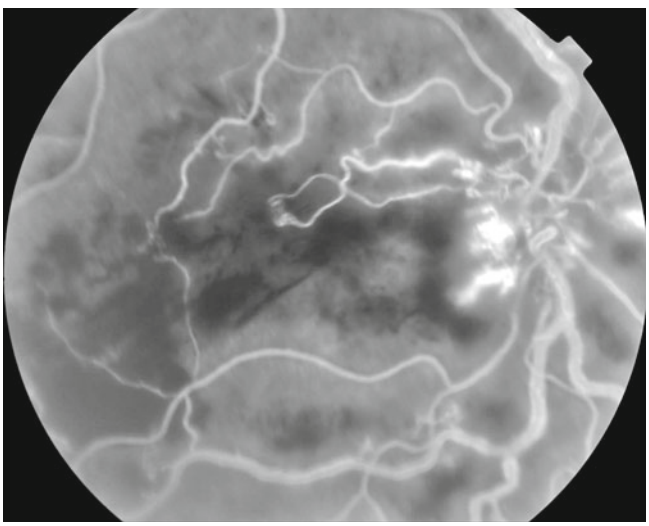


Fig. 13.2 CRVO can be excessively severe as in this patient (see Fig. 13.3)



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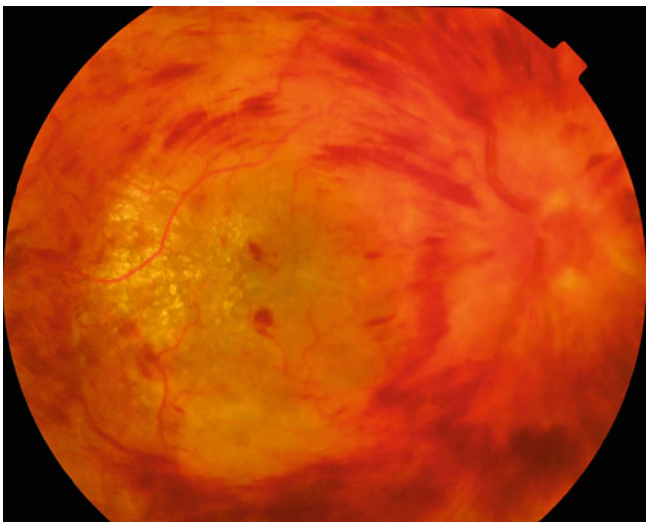

Fig. 13.4 A 13-year-old girl with CRVO in whom the onset was associated with a bout of diarrhoea on ski trip and a short-haul plane journey. She had a familial cholesterolaemia

Table 13.2 Difficulty rating for RON

Difficulty rating	Low
Success rates	Unknown
Complication rates	Low
When to use in training	Late

13.2.3 Radial Optic Neurotomy

Usually, CRVO is associated with severe irreversible visual loss and with improvement of vision in only 20 % (The Central Vein Occlusion Study Group 1997). Incision of the optic nerve on the nasal side (radial optic neurotomy) has been described (Opremcak et al. 2001; Williamson et al. 2003; Garcia-Arumi et al. 2003) but is of unproven benefit as yet despite large case series (Opremcak et al. 2001, 2006). It is thought that the neurotomy may help blood flow in the central retinal vein by relieving pressure on the vein as it exits the lamina cribrosa, assuming that CRVO is a 'neuro-vascular compression syndrome', resulting from increased pressure within the confined space of the scleral outlet. Intravitreal pO_2 is severely reduced in CRVO and may be increased by vitrectomy (Williamson et al. 2009a).

13.2.4 Intravitreal Steroid and Anti-VEGF Agents

Cystoid macular oedema (CMO) is a major cause of visual loss in RVO. Injections of varying doses of triamcinolone into the vitreous cavity (1–21 mg) have been investigated for reversing CMO in uveitis, postcataract surgery, retinal vein occlusion, telangiectasia and diabetes (Jonas et al. 2002, 2003, 2004, 2005a, b; Spandau et al. 2005; Alldredge and Garretson 2003; Antcliff et al. 2001; Benhamou et al. 2003; Bynoe and Weiss 2003; Conway et al. 2003; Martidis et al. 2001; Greenberg et al. 2002; Martidis et al. 2002; Degenring and Jonas 2003; Degenring et al. 2003; Williamson and O'Donnell 2005). Dexamethasone pellets can be inserted into the vitreous and have shown some efficacy for improved vision over 6 and 12 months (Haller et al. 2010), but as with triamcinolone, repeated injections are required.

Table 13.3 Difficulty rating for intravitreal steroid injection

Difficulty rating	Low
Success rates	Slight
Complication rates	Low
When to use in training	Early

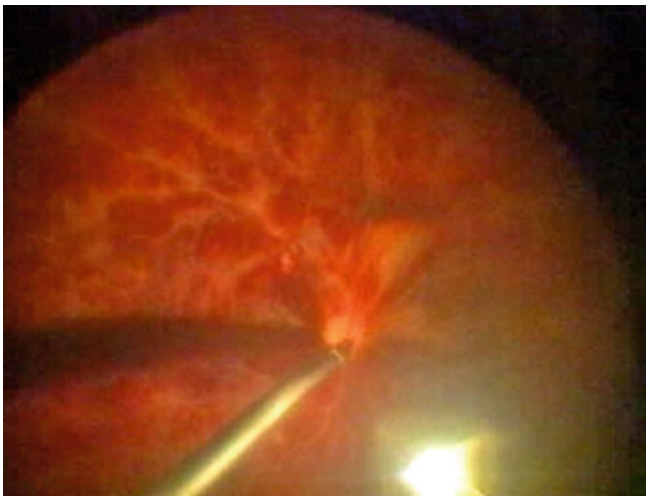

Fig. 13.5 Incising the optic nerve in radial optic neurotomy is of uncertain worth in the treatment of central retinal vein occlusion

The steroid stabilises the leaky vascular endothelium reducing the extracellular fluid accumulation perhaps by downregulating vascular endothelial growth factor (VEGF) (Jonas and Sofker 2001). In many cases, the feasibility of steroid is lessened because the duration of action is only a few months, requiring the administration of repeated injections in chronic conditions. Unfortunately, those repeat injections are associated with a reduced or absent response with time (Williamson and O'Donnell 2005). Dexamethasone has, however, been associated with less frequent IOP elevation than triamcinolone injection which in the later can be severe and require surgery for IOP control.

Anti-VEGF agents have been used widely for cystoid macular oedema from many causes including RVO. Bevacizumab reduces CMO and improves vision with low complication rates but requires repeat injection. Anti-VEGF agents are useful also for controlling neovascular glaucoma.

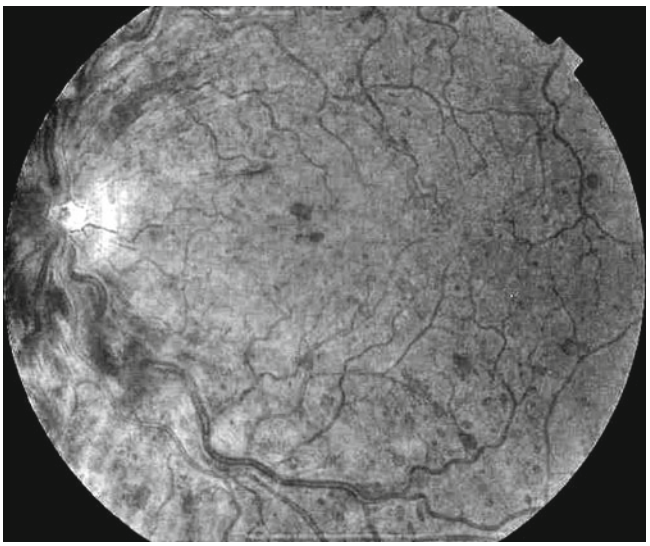

Fig. 13.6 Intravitreal triamcinolone in a patient with central retinal vein occlusion; the steroid gradually disappears and the CRVO resolves (see Figs. 13.7–13.12)

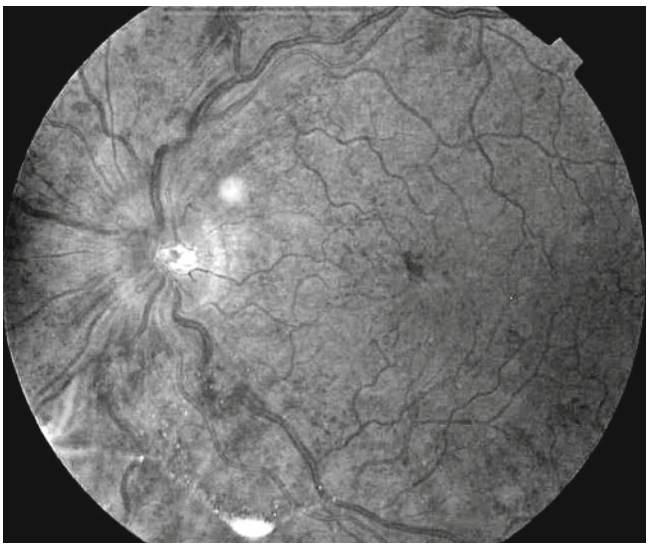

Fig. 13.7 See previous figure

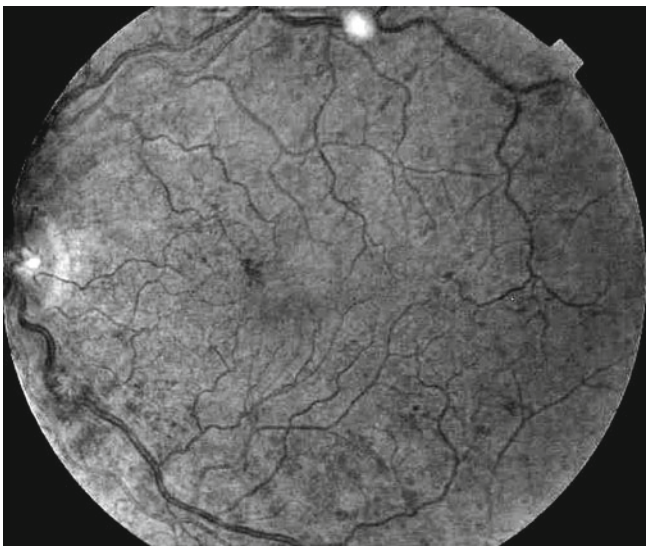

Fig. 13.8 See Fig. 13.6

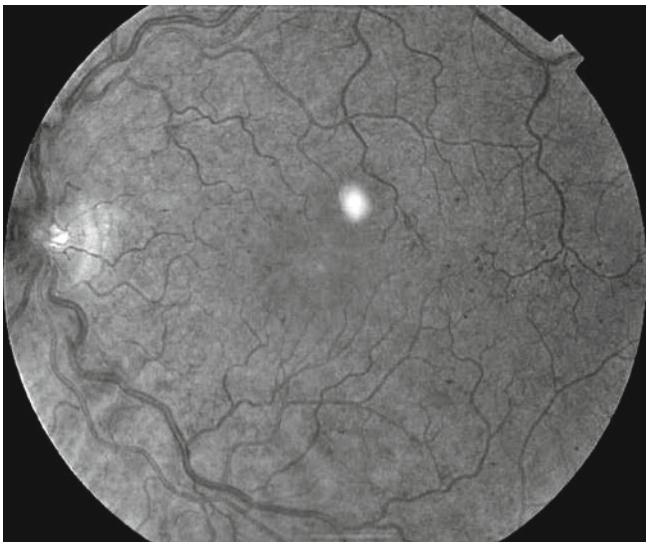

Fig. 13.9 See Fig. 13.6

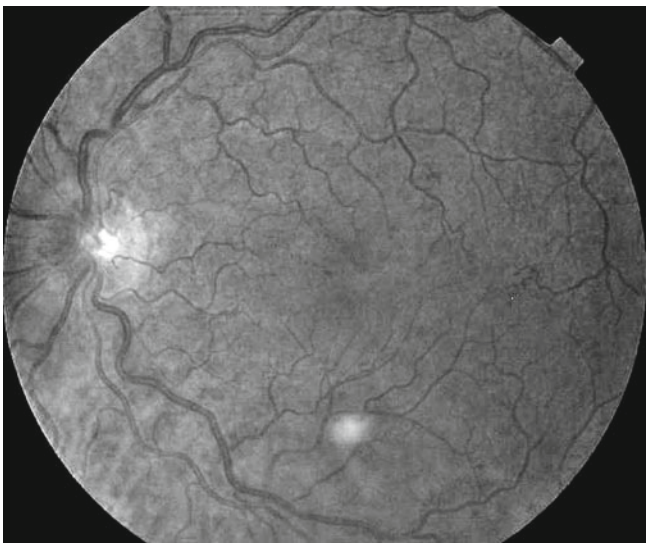

Fig. 13.10 See Fig. 13.6

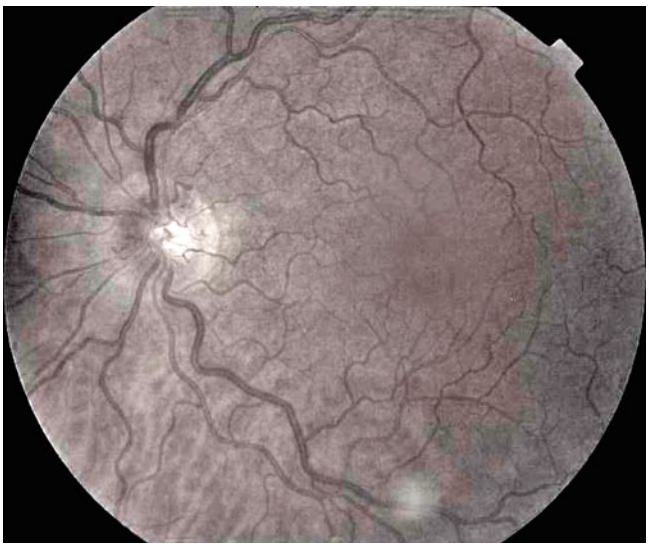

Fig. 13.12 See Fig. 13.6

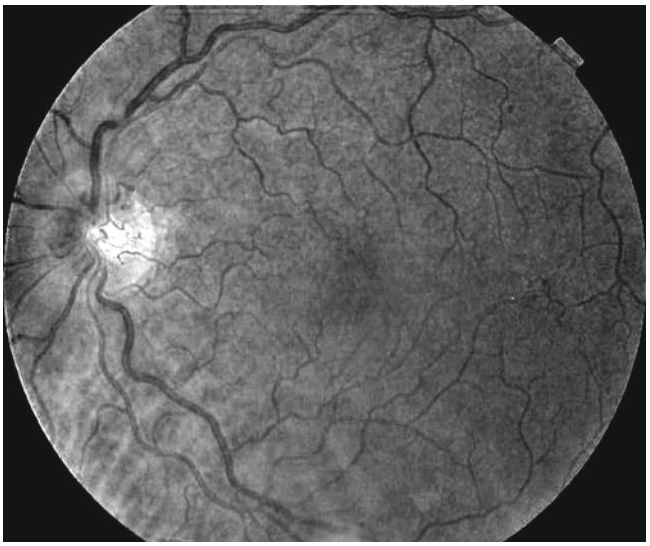

Fig. 13.11 See Fig. 13.6

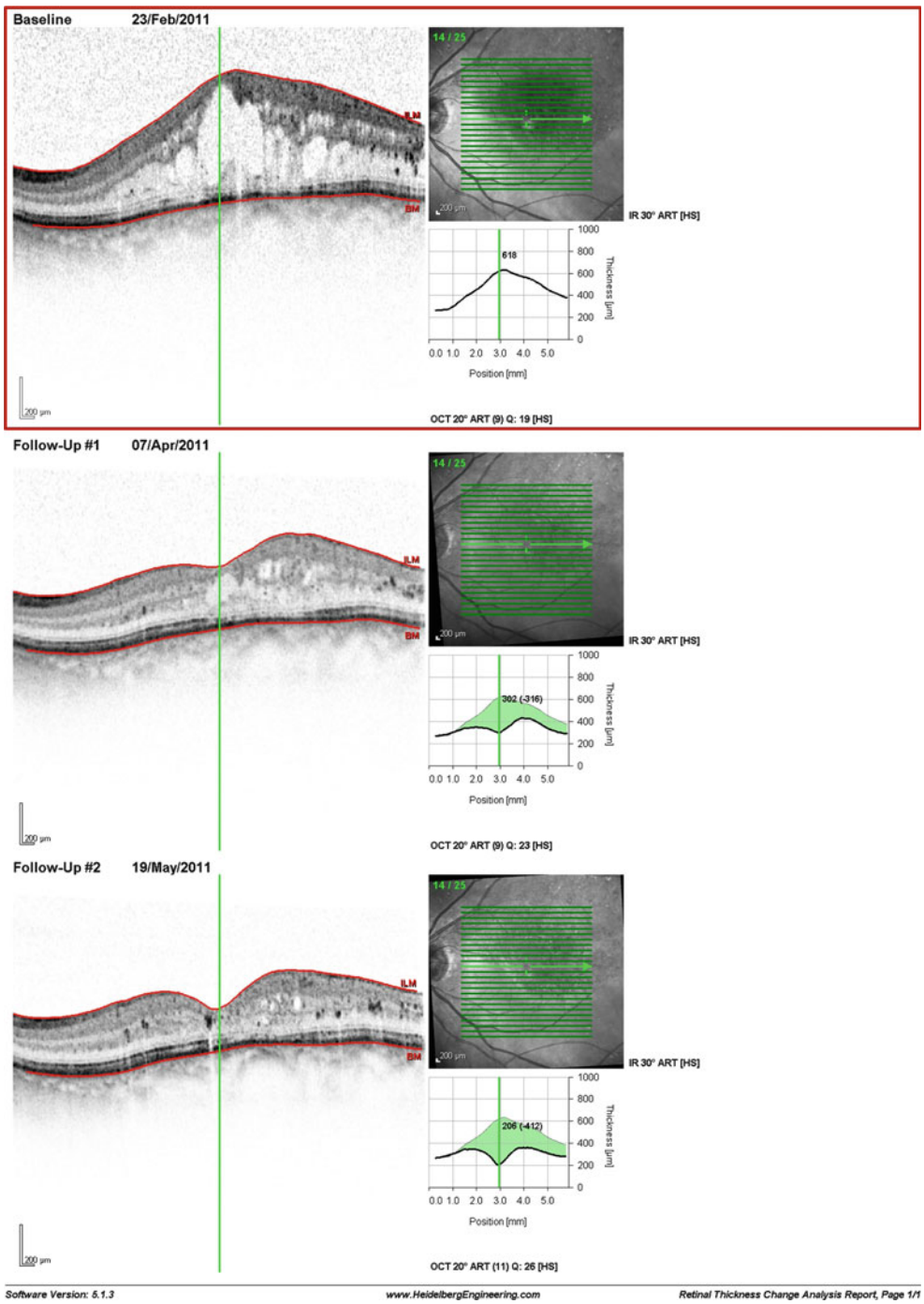

Fig. 13.13 CMO of the retina from BRVO, reduced by the intravitreal injection of bevacizumab

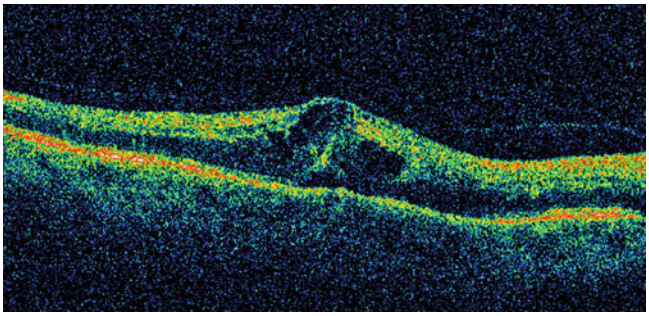

Fig. 13.14 CMO from CRVO can be treated by Avastin as in this patient; however, it does not aid recanalisation of the vein, and these patients may suffer later loss of vision even without return of the CMO. Notice how in this sequence of OCT the vitreous can be seen to separate a common accompaniment to acute retinal ischaemia (see Figs. 13.15–13.16)

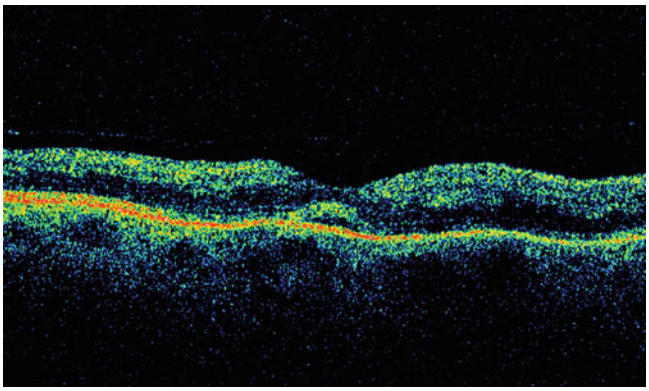

Fig. 13.15 See previous figure

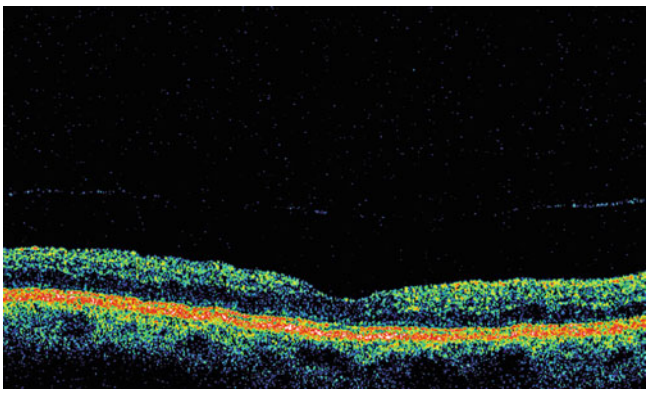

Fig. 13.16 See Fig. 13.14

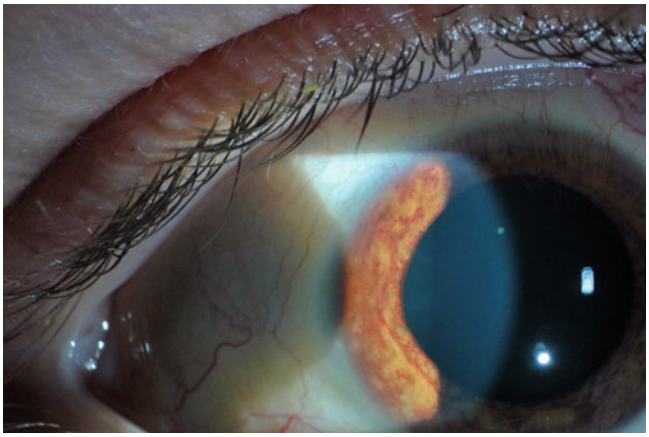

Fig. 13.17 Iris neovascularisation after CRVO. The neovascularisation can be reversed by injection of anti-VEGF into the vitreous. This may temporarily reduce the IOP to allow PRP or other measures such as PPV in an attempt to reverse the ischaemia. Repeated injections can be used to prolong the duration of residual vision in the eye (for up to 2 years), although these patients will usually eventually suffer further loss of vision from retinal ischaemia and CMO

13.2.5 Tissue Plasminogen Activator

TPA may not cross the vasculature to enter the central retinal vein; therefore, investigators have attempted to insert TPA directly into the vein (Weiss 1998) using a 33-G cannula. The technique does not seem to have had a major effect on visual acuity, and vitreous haemorrhage is common (Weiss and Bynoe 2001). More recently, Weiss has added intravitreal steroid injections to try to improve success rates (Bynoe and Weiss 2003).

13.3 Sickle-Cell Disease

13.3.1 Introduction

The sickle-cell haemoglobinopathies result from an abnormality in the beta chain of the haemoglobin molecule. They are hereditary disorders that cause red blood cells to take on a sickle shape. These blood cells are rigid and pass with more difficulty through blood vessels causing vascular occlusion in multiple organs including the retina. There are chronic haemolytic anaemia and vaso-occlusive crises and a number of clinical features in the eye (Clarkson 1992). Sickling occurs more in hypoxic or acidotic conditions.

13.3.2 Types of Sickle-Cell Disease

- Sickle-cell trait (Hb AS)
- Sickle-cell anaemia, homozygous sickle-cell disease (SS disease)
- Sickle-cell disease, heterozygous sickle-cell C disease (SC disease)
- Sickle-cell thalassaemia disease (S-thal disease)
- Systemic investigations

13.3.3 Systemic Investigation

Ask for a history of systemic crisis, medications, racial and family history and check haemoglobin electrophoresis and full blood count.

13.3.4 Inheritance and Race

It is an autosomal incomplete dominant condition (inheritance is similar to recessive inheritance, e.g. 1 in 4 chance of SS if both parents AS), but the S gene can have effects if combined with the C gene or thalassaemia gene.

The sickle gene is present in approximately 8 % of the black population in the USA but can be higher or lower depending on geographical location. The gene is thought to have originated in West Africa and become more prevalent because of its protective effect in falciparum malaria (shorter living red blood cells and relative hypoxia may be detrimental to the infection). The gene is also seen in eastern Mediterranean and Middle Eastern patients. SC disease is traditionally thought to develop more retinal complications; however, the complications are also seen in SS disease. Most cases of retinopathy seem to appear between 20 and 40 years and stabilise or regress thereafter.

13.3.5 Systemic Manifestations

- Painful vaso-occlusive crisis
- Acute chest crisis
- Anaemia
- Leg ulcers
- Bacterial infections
- Arthritis and swelling in the hands and feet
- Bone necrosis
- Splenomegaly
- Hepatomegaly
- Heart and lung damage

13.3.6 Ophthalmic Presentation

The blood cells occlude the retinal blood vessel causing hypoxia stimulating neovascularisation. These may bleed or produce traction on the retina which may be implicated in retinal break formation and retinal detachment. The process also causes secondary changes in the vitreous which stimulates epiretinal membrane formation and macular holes. Retinal complications are seen in 43 % of patients aged between 20 and 30 years with sickle cell (Downes et al. 2005). In one study, blindness in patients with proliferative vitreoretinopathy was seen in 12 % (Condon et al. 1984). However, in a large study of young patients in Jamaica (307 patients with SS and 166 with SC), followed for 20 years up to the age of 26 years, only two patients had sight threatening disease, one patient suffering irreversible sight loss in one eye and one patient with a successfully treated RRD (Downes et al. 2005). A description of the natural history of the condition has demonstrated a moderate risk of vitreous haemorrhage (5.3 %) and macular lesions (4.6 %) and a low risk of retinal detachment (2 %) over a mean follow-up of 6.3 years (Clarkson 1992).

The asymptomatic patient:

- Black sunburst spots
- Iridescent spots
- Retinal haemorrhages (salmon patches)
- Sea fan neovascular proliferation

The symptomatic patient (Brazier et al. 1986):

- Vitreous haemorrhage
- Tractional retinal detachment (TRD)
- Rhegmatogenous retinal detachment (RRD)
- Macular epiretinal membrane (ERM)
- Macular hole

The conjunctival and optic nerve head blood vessels show characteristic segmentation of blood columns in the homozygous sickle-cell disease (SS). There may be comma-shaped vessels on the bulbar conjunctiva and iris atrophy. Spontaneous hyphaema can occur and cause raised intraocular pressure and has a risk of rebleeding. Surgical intervention is sometimes required.

The optic nerve may show dark red spots or clumps on the surface.

The macula

- Chronic retinal ischaemia.
- Macular epiretinal membrane.
- Macular hole.
- An association with angioid streaks has been described.

The peripheral retina

- Non-proliferative changes.
- Venous tortuosity.

- Salmon patch haemorrhage, round or oval intraretinal haemorrhages in the mid-periphery, initially bright red then fading to the colour of salmon flesh.
- Black sunburst, hypertrophy of the retinal pigment epithelium with spiky border.
- Iridescent white spots.
- Proliferative changes, Goldberg classification (Goldberg 1971).
- Peripheral arteriolar occlusion in the far periphery.
- Arteriolar venular anastomosis seen at the junction of the perfused and non-perfused retina. Best seen on angiography, flat on the retina and non-leaking.

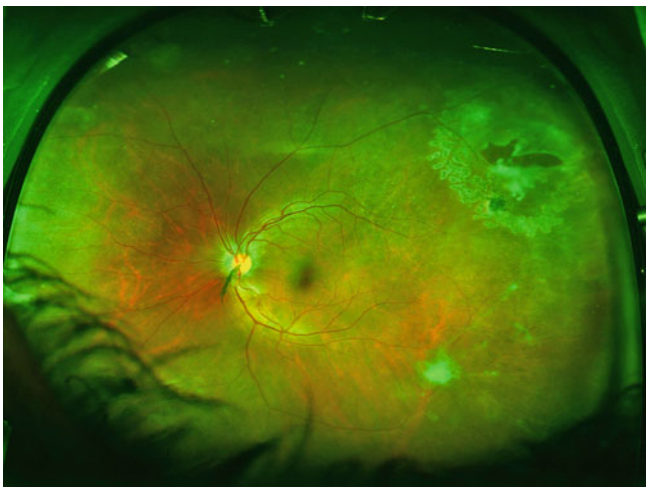

Fig. 13.18 A large retinal tear in a patient with sickle was found 6 months after PVD symptoms and was treated with laser

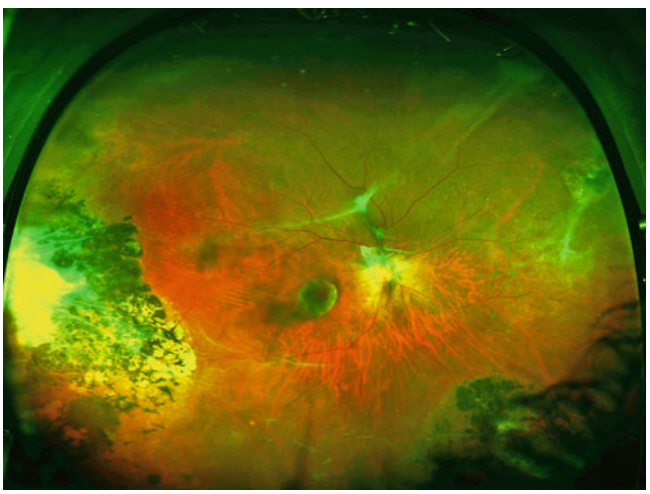

Fig. 13.19 A long-standing atrophic macular hole in a patient with sickle retinopathy

- Neovascular proliferation in the far periphery giving a 'sea fan' appearance. These often auto-infarct and are commonest in SC disease.
- Vitreous haemorrhage.
- Retinal detachment either tractional or rhegmatogenous.

13.3.7 Laser Therapy

The use of scatter laser as a means of regressing neovascularisation is controversial because of the high incidence of auto-infarction of the retinal proliferation in 30 % (Fox et al. 1993). Occlusion of the feeder vessels supplying sea fans has been employed; feeder vessel laser has been associated with retinal break formation and choroidal neovascularisation but has been shown in a small randomised study to reduce the risk of vitreous haemorrhage (Condon et al. 1984). It has been used to treat a patient with exudative retinal detachment apparently from sickle-cell retinopathy.

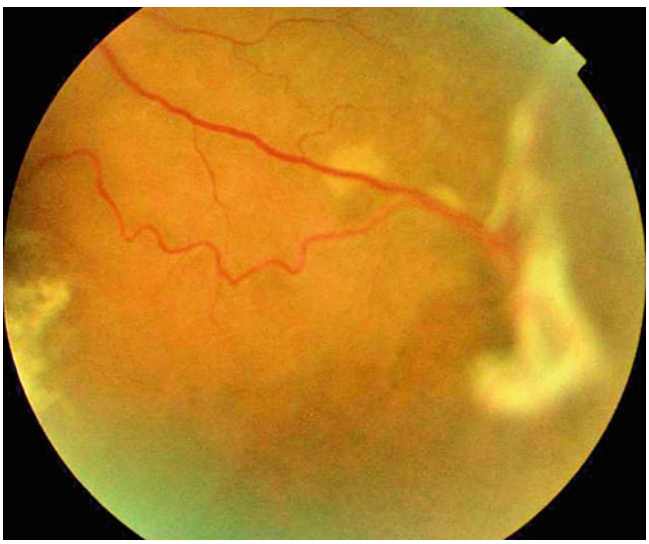

Fig. 13.20 Patients with sea fans in the periphery can present with vitreous haemorrhage or retinal detachment. Although these patients are difficult to operate on, success rates tend to be high. The sea fans are very adherent to overlying vitreous and cannot be dissected and must be segmented, but the neovascular process is inactive, so the eyes tend to respond to surgery well without laser photocoagulation

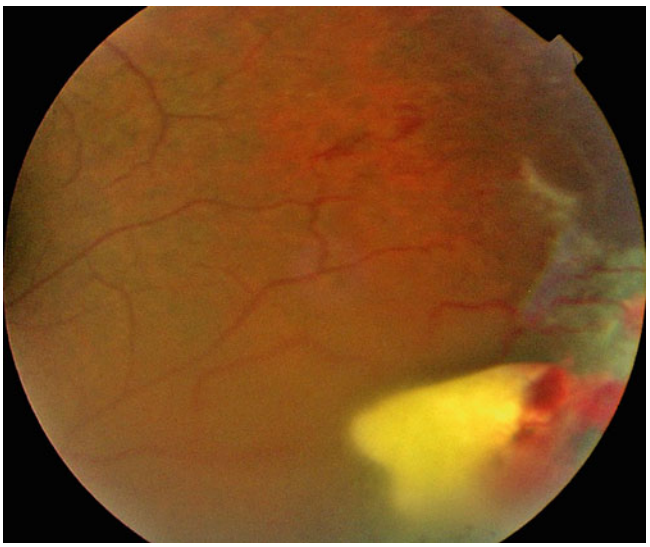

Fig. 13.21 A sea fan complex with thick membrane and blood, with leakage seen on FFA. These do not dissect off the retina and should be segmented

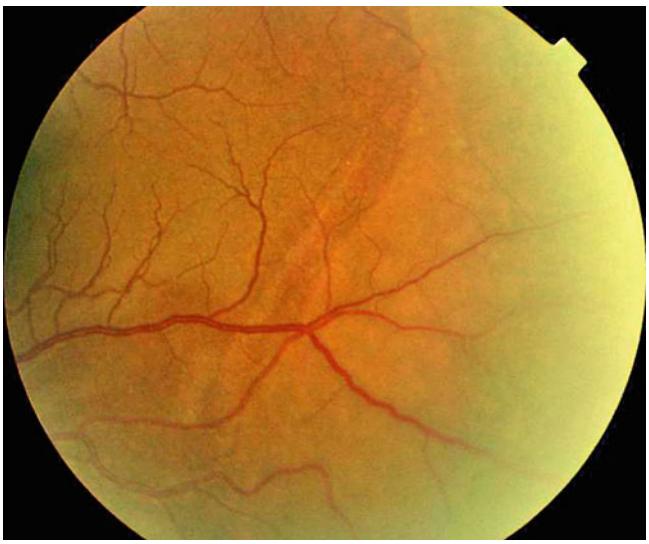

Fig. 13.23 Because the vitreous is often attached to sickle-cell patients, not all retinal detachments will proceed or progress, and these can be watched on occasion carefully. Such retinal detachments may spontaneously regress (see Fig. 13.24)

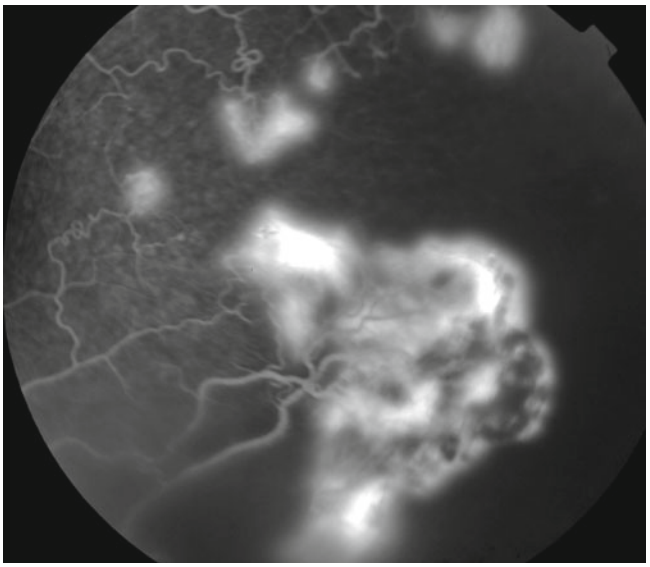

Fig. 13.22 A fluorescein angiogram of a sea fan complex showing leakage from the neovascularisation and far peripheral capillary drop out

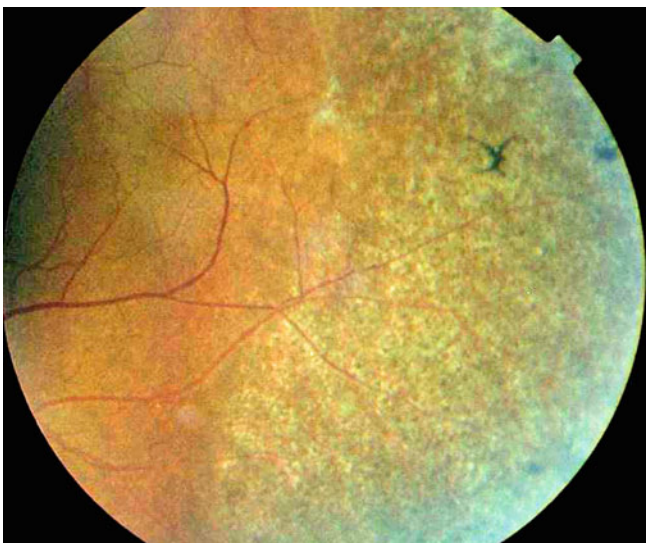

Fig. 13.24 See previous figure

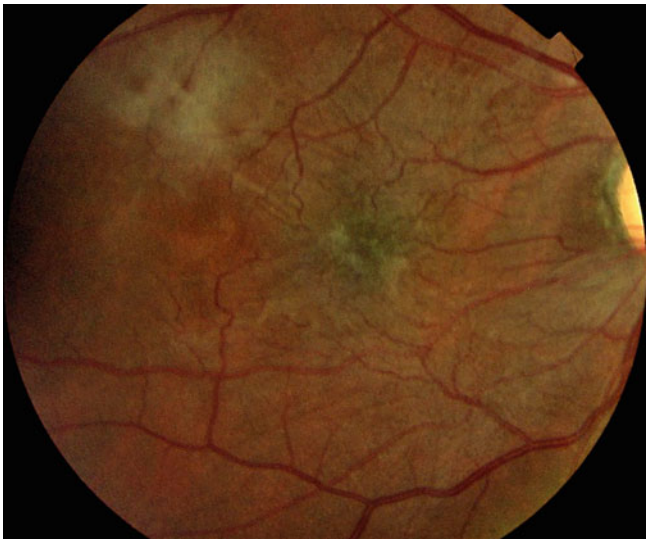

Fig. 13.25 Macular ERM can appear in sickle-cell retinopathy usually with an attached vitreous gel presumably because of changes in vitreal structure secondary to the peripheral retinopathy

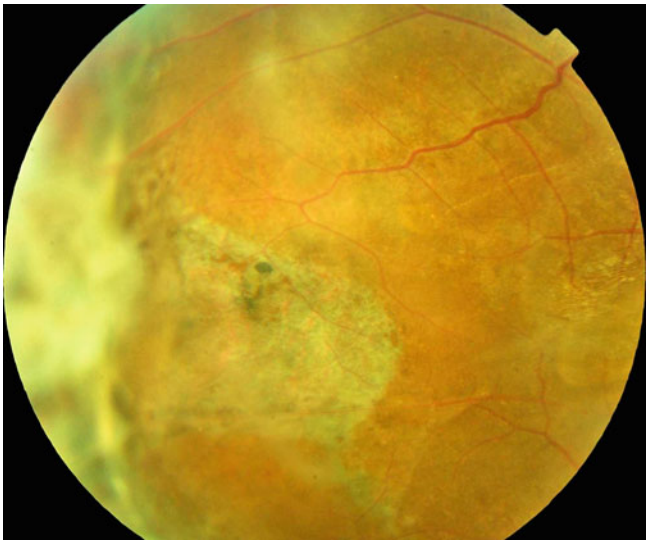

Fig. 13.27 Tractional retinal detachments can occur with sickle cell. Any membrane that can be should be dissected and removed, but often membrane is adherent to sea fans and must be left behind

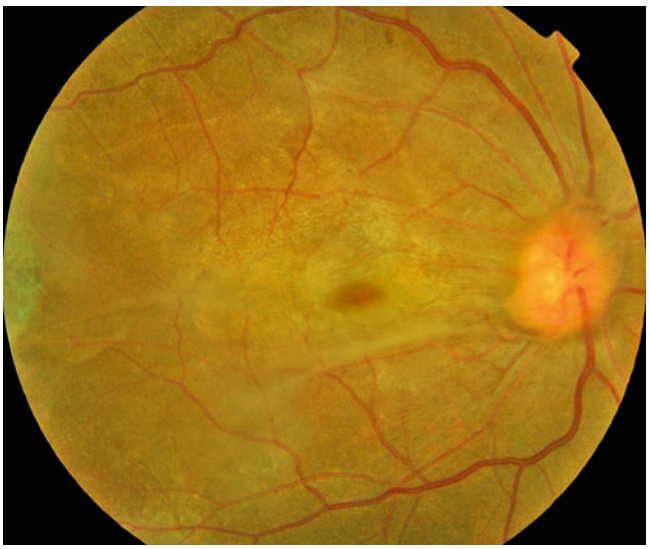

Fig. 13.26 The sickle-cell retinopathy may cause ERM

13.3.8 Surgery

Patients with vitreous haemorrhage, TRD, RRD and macular disorders such as ERM and macular holes can be treated with pars plana vitrectomy (Williamson et al. 2009b). Perioperative complications are frequent, in particular, a high incidence of iatrogenic tears formation during PPV especially around sea fan complexes. Sea fans can be safely left in situ and a segmentation technique used if necessary to remove vitreal attachments. There is no need to try to induce regression of the sea fans with scatter laser or feeder vessel laser. Cataract formation after surgery is uncommon perhaps relating to the young age of many of the patients or to the relative ischaemia of these eyes which may protect against cataractogenesis as has been suggested in diabetic retinopathy.

There is less use of scleral buckling or scatter photocoagulation (which has been used both preoperatively and perioperatively). Both interventions are associated with the development of anterior segment ischaemia in these patients (Cohen et al. 1986), a complication rarely seen today (Osterloh and Charles 1988).

Not all patients with vitreoretinal complications from sickle-cell retinopathy require surgery. Vitreous haemorrhage can be allowed to clear without detriment and TRD observed without apparent progression in some patients. Indeed, patients may show spontaneous improvement.

Exchange transfusion once used before surgery is no longer considered necessary and has been abandoned.

Note: Avoid carbonic anhydrase inhibitors (e.g. acetazolamide) in case of the induction of acidosis.

Some suggest avoiding topical epinephrine and related drugs in case of exacerbating local ischaemia.

Avoid scleral buckling in case of anterior segment ischaemia.

13.3.9 Visual Outcome

In general, the risk to vision is low, and most complications are responsive to surgery. After the age of 40 years, the condition usually stabilises. In one study, outcomes for

visions were good with 10 of 18 eyes achieving 6/12 vision or better and 15 eyes (83 %) with improved vision postoperatively.

13.3.10 Screening

The need for surveillance is doubtful because:

- There is doubt over effectiveness of prophylactic therapy, for example, scatter laser (in contrast to diabetic retinopathy where it is of proven benefit)
- The relatively low prevalence of sight-threatening complications
- That sight-threatening complications present symptomatically and usually progress slowly
- That sight-threatening complications respond well to surgery if necessary

13.3.11 Survival

- Sickle cell-anaemia (homozygous SS), the median age at death is 42 years for males and 48 years for females (Platt et al. 1994).
- Sickle-cell haemoglobin SC disease, the median age at death is 60 years for males and 68 years for females.
- 18 % of the deaths occur in patients with presence of organ failure, usually renal.
- 33 % die during an acute sickle cell crisis (78 % had pain, the chest syndrome or both; 22 % had stroke).

13.4 Retinal Vasculitis

Retinal neovascularisation can occur and cause vitreous haemorrhage or tractional retinal detachment. The former can be surgically removed, but care is required because of accentuated vitreoretinal adhesions and usually an attached vitreous gel. PRP is usually not required but increase in immunosuppressive cover may help the new vessels to regress. Traction retinal detachment tends to be associated with severe subretinal exudation and cholesterol crystal formation. Dissection of traction membranes is difficult and visual recovery is frequently poor.

13.5 Central Retinal Artery Occlusion

In experimental surgery, the embolus in central retinal artery occlusion (CRAO) has been surgically removed by incising the wall of the artery over the embolus (Garcia-Arumi et al. 2006). The embolus exits the artery spontaneously or by grasping the embolus with forceps. Only a few patients have been described.

13.6 Summary

Vitrectomy methods are established to treat the ocular complications of diabetic retinopathy. The methods of membrane dissection require new skills for delamination of the membranes off fragile retina. The same skills can be applied to other similar conditions.

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