Anterior segment ischaemia following laser photocoagulation in a patient with systemic lupus erythematosus

BRADLEY F JOST,† R JOSEPH OLK,† ARNALL PATZ,† STUART L FINE,† AND ROBERT P MURPHY†

From the †Department of Ophthalmology, Washington University School of Medicine, St Louis, Missouri, and the ‡Wilmer Ophthalmological Institute, Johns Hopkins University, Baltimore, Maryland, USA

SUMMARY The authors report the occurrence of occlusive retinal arterial disease and retinal neovascularisation in a 44-year-old woman with systemic lupus erythematosus (SLE). Two days after uncomplicated panretinal photocoagulation the patient developed an acute anterior segment ischaemic syndrome. To our knowledge this complication has not been reported in any other patient following laser photocoagulation or in association with SLE.

Systemic lupus erythematosus (SLE) is a systemic disease of unknown aetiology in which the basic pathogenic mechanism is deposition of immune complexes, activation of complement, and resulting inflammation.1 The ocular manifestations of SLE include involvement of the lids, cornea, retina, and optic nerve. Retinal haemorrhages and cotton-wool spots are the most common manifestations of retinal involvement.2 Patients have been reported to develop occlusive retinal arterial disease,5657 with retinal neovascularisation occurring in some cases.56 Anterior segment ischaemia is manifest by pain, reduced vision, corneal oedema with striate keratopathy, and acute iridocyclitis. Posterior synechiae, peripheral anterior synechiae, iris atrophy, cataract, and even phthisis bulbi can develop after an episode of anterior segment ischaemia. This complication has been reported following various operative procedures such as muscle surgery,1215 retinal detachment surgery,1619 cyclocryotherapy,1721 and extracapsular cataract extraction with posterior chamber intraocular lens implantation.24 It has also been described in association with systemic diseases including occlusive cerebrovascular disease25 and chronic myelogenous leukaemia.26 Patients with sickle cell haemoglobinopathy are at particular risk of developing anterior segment ischaemia following retinal detachment surgery.2728

In this report we describe the development of acute anterior segment ischaemia following panretinal photocoagulation for retinal neovascularisation in a patient with SLE. To our knowledge this syndrome has not been reported following panretinal photocoagulation or in association with SLE or laser photocoagulation in any other patient.*

Case report

A 44-year-old white woman presented to the Retinal Vascular Center of the Wilmer Ophthalmological Institute with a two-week history of blurred vision in the right eye. A diagnosis of SLE had been made at the National Institute of Health six years previously, and the patient had been on varying doses of prednisone since that time. Her past medical history was significant, with deep-vein thrombophlebitis and a pulmonary embolus six years prior to her presentation at Wilmer, and two mild strokes: the first stroke two years previously, associated with right hemiparesis; and the second just four months previously, involving mild paresis of the right arm. The patient was taking 15 mg of prednisone daily at the time of her examination.

The best corrected visual acuity was 10/200 in the

*This patient has been reported as part of a large series of patients with SLE. (Jabs DA, Fine SL, Hochberg MC, et al. Severe retinal vaso-occlusive disease in systemic lupus erythematosus. Arch Ophthalmol 1986; 104: 558-63.)

Correspondence to R Joseph Olk, MD, 4949 Barnes Hospital Plaza, East Pavilion Suite 17413, St Louis, MO 63110, USA.
Right eye and 20/20 in the left eye. The pupillary, external, and extraocular motility examinations were all normal. Slit-lamp examination revealed mild ciliary injection for 360° in each eye. There was no cellular reaction or flare in the anterior chamber, no iris neovascularisation was detected, and no lens opacity was seen. The intraocular pressure was 8 mmHg in the right eye and 10 mmHg in the left eye.

Ophthalmoscopy of the right eye revealed mild optic disc pallor with marked attenuation of the retinal arterioles and periarteriolar sheathing (Figs. 1A, B). There were several areas of intraretinal haemorrhage and cotton-wool exudation. There were multiple patches of neovascularisation and non-perfused retina with marked arteriolar attenuation in the mid periphery. The left eye also showed arteriolar narrowing with periarteriolar sheathing, intraretinal haemorrhage, and focal non-perfusion (Fig. 2). No discrete areas of neovascularisation were identified in the left eye. Fluorescein angiography confirmed the presence of significant areas of retinal non-perfusion with associated retinal neovascularisation (Fig. 3).

The patient underwent peripheral scatter argon photocoagulation in the right eye. Only topical anaesthesia was used, and she received 1011 500 μm
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spot size applications of 0.1 second duration varying in power from 250 to 400 milliwatts (Fig. 4). A Goldmann three-mirror contact lens was used and the procedure was completed within 30 minutes.

Two days after this treatment the patient presented with an acutely inflamed, tender right eye (Fig. 5). Her visual acuity was counting fingers. The eye had 4+ conjunctival injection and moderate chemosis, with marked corneal oedema and striae. The anterior chamber was deep, with 4+ suspended red blood cells, which were layering in the filtration angle and on the corneal endothelium. The aetiology of the anterior chamber haemorrhage was unknown, though the authors suspect that perhaps a few undetected twigs of iris neovascularisation or pre-existing dilated iris vessels may have ruptured and caused the haemorrhage. There was also a marked fibrinoid response with early formation of posterior synechiae. Ocular tension by applanation was 2 mmHg in the right eye, compared with 15 mmHg in the left. Acute anterior segment ischaemia was diagnosed and topical prednisolone acetate 1% every four hours and atropine sulphate 1% twice daily were prescribed. The patient was admitted to the Rheumatic Disease Unit for examination and treatment of her active SLE. Her systemic involvement

Fig. 4  Immediate post-laser appearance of the right eye.

Fig. 5  Appearance of the right eye two days after argon laser photocoagulation. There is a pronounced conjunctival reaction with corneal oedema and suspended red blood cells in the anterior chamber. Early posterior synechiae formation and a marked fibrinoid response are also present.

Fig. 6A  Ten-week post-treatment appearance of the right eye. Fine iris neovascularisation can be seen as well as pigment clumping on the anterior lent capsule and posterior synechiae. There is a sector of iris atrophy from the 3 o’clock to 5 o’clock position. B: Iris angiography confirms the presence of rubeosis.
was controlled with 80 mg prednisone per day and a
three-month course of azathioprine.

On follow-up examination 10 weeks after treat-
ment the patient had visual acuity of 5/200 in the
right eye and 20/20 in the left eye. The cornea and an-
terior chamber were clear. Sector iris atrophy, sup-
porting the diagnosis of anterior segment ischaemia, was
present from 3 to 5 o’clock (Fig. 6). Mild rubeosis
was noted at the pupillary margin, with posterior
synechiae, diffuse angle neovascularisation, and
early formation of peripheral anterior synechiae
(Figs. 6A, B). Ophthalmoscopy revealed involution
of the retinal neovascularisation in the right eye.
Examination of the left eye showed progression of
retinopathy, with further arteriolar attenuation and
increased cotton-wool exudation (Fig. 7).

Discussion

The frequency with which ocular findings are seen in
association with SLE is highly variable and depends
on the activity of the systemic disease. The early
literature reported retinal haemorrhage and cotton-
wool spots in 10 to 28% of patients.26-33 However,
Gold et al.2 examined 61 outpatients with SLE and
found no retinal haemorrhages and only two (3-3%)
patients with cotton-wool exudates. This lower inci-
dence of retinal findings was attributed to better
systemic control of the disease. It is believed that
the retinopathy of SLE is seen more frequently in acutely
ill patients.2,34 Our patient had heightened systemic
activity of her SLE in association with her active
retinopathy.

A less common and more serious ocular manifesta-
tion of SLE is occlusive retinal arterial disease.2-11

The precise aetiology of the retinal arterial occlusions
is obscure. Histopathological study of the central
nervous system (CNS) in patients with SLE has
revealed numerous microinfarcts secondary to occlusive
disease of small arteries and arterioles.35 These
vessel walls did not have significant inflammatory cell
infiltration but had undergone fibrinoid degeneration
or hyalinisation with necrosis. Histopathological
studies of the retina have demonstrated thrombosed
arterioles with thickened intimas but have not shown
any evidence of active vasculitis.11 36 36 Local deposi-
tion of antigen-antibody complexes, embolism, or in-
situ thrombosis may all play a part in the occlusive
arterial disease of SLE. Gold et al.5 suggested that the
occlusive retinal vascular disease seen in patients
with SLE may be associated with CNS lupus. It is
noteworthy that our patient had two transient
ischaemic attacks while she had active retinal arterial
occlusive disease.

Retinal neovascularisation has been seen in
association with the retinal arterial occlusive disease
of SLE.5-4 Neovascularisation of the iris and anterior
chamber angle has also been seen in an SLE patient
with active retinal occlusive disease.3 Vitreous
haemorrhage may occur,1,6 and laser photocoagula-
tion has been successful in causing regression of the
neovascularisation and preventing vitreous haemorr-
hage.6

Anterior segment ischaemia is a syndrome of acute
ocular pain, reduced vision, corneal oedema with
Descemet’s folds, marked anterior chamber cells
and flare which can lead to iris atrophy, posterior
synechiae, peripheral anterior synechiae, cataract,
and even phthisis bulbi. This syndrome has been seen
following retinal detachment surgery17-21 27 28 and is
believed to be related to circulatory compromise of the
long posterior ciliary arteries,19 detachment of the
extraocular muscles, or venous outflow insufficiency
secondary to vortex vein compression by an en-
circling element.20

Extraocular muscle surgery has also been associ-
ated with anterior segment ischaemia.12-16 Inter-
ruption of the anterior ciliary arteries within the
rectus muscles has an aetiological role in these
cases. Anterior segment ischaemia may follow cyclo-
ycryotherapy.22,23 It has been suggested that this
complication results from further reduction in blood
flow to an already relatively ischaemic eye induced
by the acute cryogenic vascular injury of cyclocryo-
therapy.22 Hyperviscosity30 and generalised cerebro-
vascular insufficiency25 have also been reported to
cause anterior segment ischaemia.

Anterior segment ischaemia has not been pre-
viously described in association with either retinal
photocoagulation or SLE. There is no evidence in
this case that the laser photocoagulation caused the
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Anterior segment ischaemia. Furthermore, it would be reasonable to assume that our patient was experiencing an element of anterior segment circulatory compromise prior to the photoocoagulation. Widespread arterial and arteriolar occlusions are seen in SLE, and this patient was already showing significant retinal circulatory disturbance.

The patient did not have a retrobulbar anaesthetic or a prolonged operative procedure, two factors that may have affected ocular circulation. However, the brief time in which the Goldmann lens was pressing on the eye may have impaired the circulation enough to cause anterior segment ischaemia in an eye already ischaemic from active occlusive arterial disease. Choroidal infarction following panretinal photoocoagulation for proliferative sickle retinopathy has been reported.37 In this situation the pressure of the contact lens was believed to impair the choroidal circulation.

Concomitant with our patient’s retinal arterial occlusive disease was evidence of CNS and systemic disease activity. This supports Gold et al’s3 observation that retinal occlusive disease is associated with CNS and systemic disease activity. SLE patients with active retinopathy should be examined for possible systemic disease activity, so that potentially life threatening complications can be prevented or appropriately treated. In addition SLE patients with retinal arterial occlusive disease should be carefully examined for signs of anterior segment ischaemia.

Care should be taken to avoid exacerbating any pre-existing ischaemia when performing operative procedures, including retinal photoocoagulation, on these patients. The authors would suggest avoiding any undue pressure on the contact lens, performing panretinal photoocoagulation in short, multiple sessions, and if possible avoiding the use of retrobulbar anaesthesia.

References


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