persisted subretinal haemorrhage could have been responsible for the poor visual prognosis after PDT,[10] except the progressive enlargement and disciform transformation of the CNV.

We report a case of increased and persisted subretinal haemorrhage after PDT for CNV secondary to angioid streaks. The visual prognosis was poor despite retreatments of PDT.

References

1 Mansour AM. Systemic associations of angioid streaks. *Ophthalmologica* 1993; **207**: 57–61.
2 Puig J, Garcia-Arumi J, Salvador F, Sararols L, Calatayud M, Alforja S. Subretinal neovascularization and hemorrhages in angioid streaks. *Arch Soc Esp Oftalmol* 2001; **76**: 309–314.
3 Karacorlu M, Karacorlu S, Ozdemir H, Mat C. Photodynamic therapy with verteporfin for choroidal neovascularization in patients with angioid streaks. *Am J Ophthalmol* 2002; **134**: 360–366.
4 Shaikh S, Ruby AJ, Willams GA. Photodynamic therapy using verteporfin for choroidal neovascularization in angioid streaks. *Am J Ophthalmol* 2003; **135**: 1–6.
5 Ladas ID, Georgalas I, Rouvas AA, Gotsis S, Karagiannis DA, Moschos M. Photodynamic therapy with verteporfin of choroidal neovascularization in angioid streaks: conventional versus early retreatment. *Eur J Ophthalmol* 2005; **15**: 69–73.
6 Gelisken F, Inhoffen W, Karim-Zoda K, Grisanti S, Partsch M, Voelker M *et al*. Subfoveal hemorrhage after verteporfin photodynamic therapy in treatment of choroidal neovascularization. *Graefes Arch Clin Exp Ophthalmol* 2005; **243**: 198–203.
7 Theodossiadis GP, Panagiotidis D, Georgalas IG, Moschos M, Theodossiadis PG. Retinal hemorrhage after photodynamic therapy in patients with subfoveal choroidal neovascularization caused by age-related macular degeneration. *Graefes Arch Clin Exp Ophthalmol* 2003; **241**: 13–18.
8 Schmidt-Erfurth U, Schlotzer-Schrehard U, Cursiefen C, Michels S, Beckendorf A, Naumann GO. Influence of photodynamic therapy on expression of vascular endothelial growth factor (VEGF), VEGF receptor 3, and pigment epithelium-derived factor. *Invest Ophthalmol Vis Sci* 2003; **44**: 4473–4480.
9 Gelisken F, Inhoffen W, Schneider U, Partsch M, Kreissig I. Retinal pigment epithelial tear after photodynamic therapy for choroidal neovascularization. *Am J Ophthalmol* 2001; **131**: 518–520.
10 Toth CA, Morse LS, Hjelmeland LM, Landers III MB. Fibrin directs early retinal damage after experimental subretinal hemorrhage. *Arch Ophthalmol* 1991; **109**: 723–729.

M-P Shyong[1,2], S-J Chen[3], F-L Lee[3], Y-P Tsao[4] and W-M Hsu[3]

[1]Institute of Clinical Medicine, National Yang-Ming University, Taipei, Taiwan, ROC

[2]Su-Ao Veterans Hospital, I-Lan, Taiwan, ROC

[3]Department of Ophthalmology, Taipei Veterans General Hospital, Taipei, Taiwan, ROC

[4]Department of Ophthalmology, Mackay Memorial Hospital, Taipei, Taiwan, ROC

Correspondence: F-L Lee, Department of Ophthalmology, Taipei Veterans General Hospital, No. 201, Sec. 2, Shi-Pai Rd, Taipei 112, Taiwan, ROC
Tel: +886 2 2875 7325;
Fax: +886 2 2876 1351.
E-mail: fllee@vghtpe.gov.tw

Eye (2006) **20,** 1420–1422. doi:10.1038/sj.eye.6702278; published online 28 April 2006

Sir,

Acute subretinal macular haemorrhage following an accidental electrical shock

Ocular complications of electrical trauma are widely reported. This presentation of a severe macular subretinal haemorrhage following an electrical shock has not been described previously.

Case report

A 58-year-old man presented with a 24 h visual deterioration in his left eye following an accidental electrical shock (230 V AC) to his left forearm, lasting a few seconds. There was no relevant past ocular or medical history. His left visual acuity was reduced to counting fingers. Fundus examination showed a large, elevated subretinal haemorrhage at the macula (Figure 1), with no other abnormalities. The right eye was normal, with no risk factors for choroidal neovascularisation such as drusen or retinal pigment epithelial changes. Fluorescein and indocyanine green angiography were not performed. The haemorrhage took several months to resolve (Figure 2). At 2 years, a dense macular scar remains (Figure 3) with visual acuity of counting fingers. There are no lens opacities or other abnormalities noted.

Comment

The first documented case of electrical trauma to the eye was reported by St Yves in 1722 when a field worker developed cataracts following a lightning strike.[1] Since then, ocular complications of electrical trauma have been widely reported.

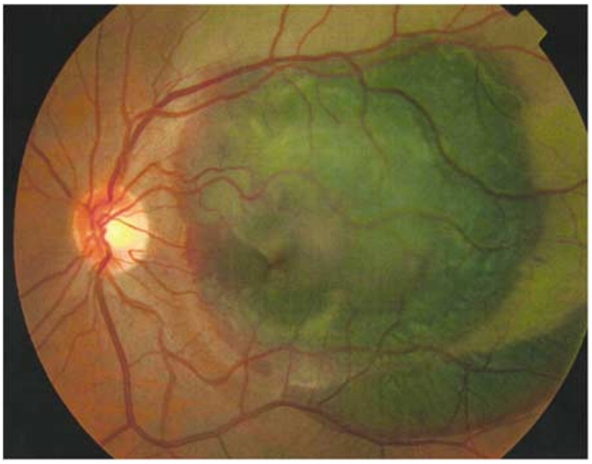

Figure 1 Large, elevated subretinal haemorrhage at the macula, 24 h following trauma.

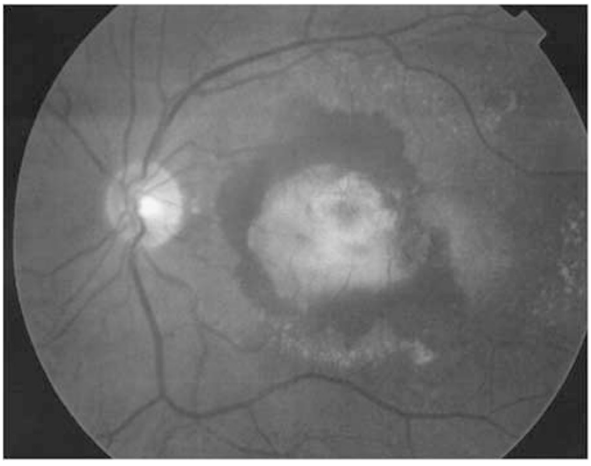

Figure 2 Resolving subretinal haemorrhage.

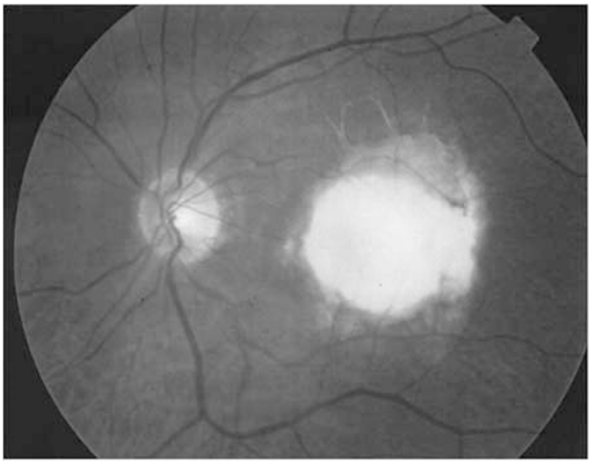

Figure 3 Macular scar remains 2 years following injury.

Tissue damage from electricity may occur from its direct transmission, conversion to thermal energy, or ischaemia caused by vascular constriction. The extent of damage is influenced by numerous factors including voltage, duration of contact, tissue sensitivity, and area of contact.[2] Cases have been described following contact varying from 220 to 80 000 V.[3]

The presence of ocular sequelae, which can affect any structure, depends on the proximity of the route travelled by the electrical current.[2] The latency of complications following trauma secondary to lightning is shorter when compared to those following contact with lower voltages.[4]

Anterior subcapsular cataracts are most common, developing from 1 month to 2 years after the injury.[3] These range from fine vacuoles to linear or mossy opacities and may regress.[5] The contact area is usually the head or neck.[2]

The retina and choroid are thought to be affected by ischaemia resulting from coagulation and necrosis of the vasculature. Macular oedema, retinal pigment epithelium changes, macular holes, retinal haemorrhages, and detachments have been described, occurring months to years after the original insult.[2,5] This necessitates serial ophthalmologic examinations.

Subretinal haemorrhages have been associated with conditions such as systemic hypertension and blood dyscrasias, including idiopathic thrombocytopaenic purpura and von Willebrand's disease.[6,7] The use of aspirin or warfarin increases the risk of subretinal haemorrhage, especially in patients with age-related macular degeneration.[8,9] These conditions were not present in this case, although polypoidal choroidal vasculopathy could not be excluded as the cause of this haemorrhage, due to the lack of angiography.[10]

Our patient was managed conservatively. Other approaches include vitrectomy and pneumatic displacement using intravitreal sulphur hexafluoride or perfluoropropane gas. These aim to minimise damage by removing the blood from the macula. Both techniques can be used with tissue plasminogen activator, which liquefies and facilitates reabsorption of the thrombus.[11]

To the best of our knowledge, this is the first reported case of subretinal macular haemorrhage following electrical trauma. The acute history and low voltage involved is also unusual, when compared to previous reports.

Acknowledgements

None of the authors has any proprietary interests or research funding.

References

1 Duke-Elder S, Mac Faul PA. *System of Ophthalmology*, Vol XIV, Injuries, Part 2—Non-Mechanical Injuries. Henry Kimpton: London, 1972, pp 813–835.
2 Miller BK, Goldstein MH, Monshizadeh R, Tabandeh H, Bhatti MT. Ocular manifestations of electrical injury: a case report and review of the literature. *CLAO J* 2002; **28**(4): 224–227.
3 Reddy SC. Electric cataract: a case report and review of the literature. *Eur J Ophthalmol* 1999; **9**(2): 134–138.
4 Biro Z, Pamer Z. Electrical cataract and optic neuropathy. *Int Ophthalmol* 1994; **18**(1): 43–47.
5 Wiesinger H. Electrical injuries to the eye. *Albrecht Von Graefes Arch Klin Exp Ophthalmol* 1975; **193**(1): 67–79.
6 Shah PA, Yang SS, Fung WE. Idiopathic thrombocytopenic purpura with massive subretinal hemorrhage. *Arch Ophthalmol* 2005; **123**(11): 1612–1613.
7 Herrmann WA, Lohmann CP, Demmler-Hackenberg M, Gabel VP. Von Willebrand's disease type I as a cause for subvitreal, retinal and subretinal haemorrhages. *Graefes Arch Clin Exp Ophthalmol* 2005; **243**(4): 383–385.
8 Superstein R, Gomolin JE, Hammouda W, Rosenberg A, Overbury O, Arsenault C. Prevalence of ocular haemorrhage in patients receiving warfarin therapy. *Can J Ophthalmol* 2000; **35**(7): 385–389.
9 el Baba F, Jarrett II WH, Harbin Jr TS, Fine SL, Michels RG, Schachat AP *et al*. Massive hemorrhage complicating age-related macular degeneration. Clinicopathologic correlation and role of anticoagulants. *Ophthalmology* 1986; **93**(12): 1581–1592.
10 Ciardella AP, Donsoff IM, Huang SJ, Costa DL, Yannuzzi LA. Polypoidal choroidal vasculopathy. *Surv Ophthalmol* 2004; **49**(1): 25–37.
11 Tennant MT, Borrillo JL, Regillo CD. Management of submacular hemorrhage. *Ophthalmol Clin North Am* 2002; **15**(4): 445–452.

SE Liyanage, S Khemka and DV de Alwis

Croydon Eye Unit, Department of Ophthalmology, Mayday Hospital NHS Trust, Thornton Heath, UK

Correspondence: SE Liyanage, Croydon Eye Unit, Department of Ophthalmology, Mayday Hospital NHS Trust, London Road, Thornton Heath, CR7 7YE, UK
Tel: +44 208 401 3477;
Fax: +44 208 401 3489.
E-mail: sidath@doctors.org.uk

Eye (2006) **20,** 1422–1424. doi:10.1038/sj.eye.6702279; published online 10 February 2006

Sir,

The impact of national diabetic retinopathy screening on ophthalmology: the need for urgent planning

We read with interest the article 'The impact of national diabetic retinopathy screening on ophthalmology: the need for urgent planning' by S Harding *et al*.[1] They report the need for a systematic screening system in view of the introduction of the National Service Framework recommendations. We would like to describe a scheme used in King's College Hospital and University Hospital Lewisham, which has been adapted to streamline the service.

Methods

Photographic screening images are graded by a trained level 1 screener. Screen-positive images (evidence of sight-threatening retinopathy, ungradeable image due to media opacity or evidence of other eye disease) are then reviewed by a more experienced, level 2 screener. If they are confirmed to be positive, they would normally be referred to the HES at this stage. In our scheme, these images are instead passed to the referring retinal specialist for further advice. Confirmation of diagnosis results in acceptance into the HES (with guidance on urgency). If HES review is not required then they receive either annual or 6 monthly recall in the screening service. Allocation of patients with cataract or other eye diseases to alternative clinics ensures that appropriate and timely follow-up is achieved without overloading the retinal specialist clinics.

Results

Of the 2260 patients screened from November 2004 to April 2005, referral was requested on 186 patients (8.2%). Of those, 94 (50.5%) were accepted for further examination for possible early treatment with laser. The most common reason that referral was not required was early maculopathy with no clinically significant macular oedema, fundal lesions of no consequence, and previously treated, inactive maculopathy.

Conclusion

This method has allowed for a 50% reduction of the referrals to the HES, thus allowing space for an ever-increasing population of diabetics.

We continue to use this method to reduce the referral rate; however, it has the added advantage of providing helpful feedback allowing further development of experience for the screeners, therefore continually improving the service we can offer to patients.