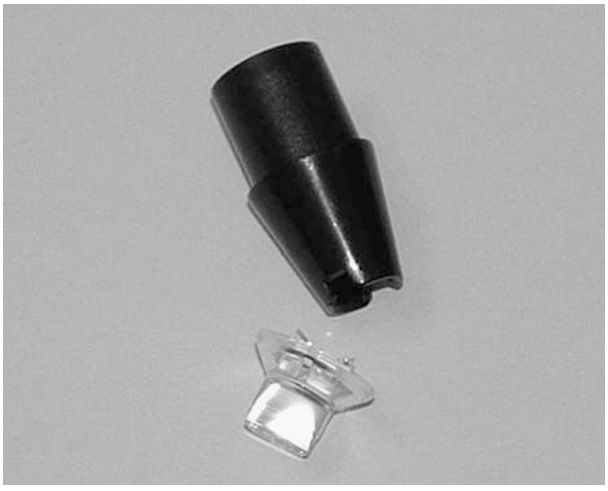

Figure 1 Illustration of TONOSAFE prism and holder.

Figure 2 Blood agar plate from plating of holder showing the growth of *Staphylococcus aureus* following the evaluation by Staphareux.

The sterile holder (TONOSAFE) was also plated after use with each successive patient ($n = 10$). The next day, the process was repeated, but the holder was disinfected with alcohol wipes between patients (as was standard in the traditional Goldmann tonometer).

Normal skin flora was grown on doctor's blood agar plates. This included *Staphyloccocus aureus* and coagulase-negative *Staphylococcus*. These organisms were shown to be present on the plates of the holder even after the first patient (see Figure 2). We believe that they had transferred from patient to holder through the doctor's hands. Alcohol wiping of holder removed all organisms.

Comment

Disposable tonometers have been advocated as a better replacement for Goldmann tonometers as they 'eliminate the need to clean and disinfect prisms.'[2] This audit has shown that bacteria can be transferred to the holder and are transferred between patients through the clinicians' hands (despite handwashing). The transmission of *S. aureus* (including MRSA) presents a potential risk for infection control, particularly in the elderly population.[5] Although this is unlikely to be of pathological significance, these results contradicts the manufacturer's claims regarding their product.

We believe that even the disposable holders should be cleaned with alcohol wipes between patients to reduce this risk. The original reason for introducing disposable prisms was to eliminate transfer. These results indicate that this risk has not been averted.

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The abstract has been accepted as a poster for Royal College of Ophthalmologists Congress in Liverpool 2008.

Eye (2009) **23,** 1474–1475; doi:10.1038/eye.2008.163; published online 6 June 2008

Sir,

Posterior scleritis presenting with simultaneous branch retinal artery occlusion and exudative retinal detachment

Posterior scleritis is a potentially blinding but frequently underdiagnosed condition. Serous retinal detachment (SRD) is a common finding,[1] whereas vascular occlusions are rare.[2–4] We report simultaneous SRD and retinal arterial occlusion as a presentation of posterior scleritis, successfully treated with systemic corticosteroids.

Case report

A 26-year-old lady presented with painful diminution of vision OD for a week. She had a similarly painful visual

loss OS 2 years back, which was permanent in spite of some unspecified treatment taken elsewhere. There was no history of any systemic illness affecting skin, joints, or organ systems accompanying or following the visual loss in the run-up to the current episode. Her best-corrected visual acuity (BCVA) was 2/60 OD and 6/36 OS. Intraocular pressures were normal, and anterior segment was unremarkable OU, except a relative afferent pupillary defect OD. Fundus examination OD revealed optic disc oedema with SRD at the posterior pole, retinal whitening superotemporally (Figure 1a), and peripheral choroidal infiltrates (not shown). Fundus OS revealed extensive chorioretinal scarring over macula (Figure 1b). Neither eye revealed vitreous cells or flare.

Fluorescein angiography OD showed delayed arteriovenous transit in the superotemporal branch retinal artery (Figure 1c). Late phases showed dye leakage from the disc and pooling in detachment spaces. B-scan ultrasonogram revealed grossly thickened sclera with sub-Tenon's fluid (Figure 1d). After obtaining a detailed history related to previous illness, current symptoms, or drug intake, a complete systemic evaluation including mucocutaneous, musculoskeletal, respiratory, cardiovascular, gastrointestinal, and genitourinary systems was performed by the in-house physician. As the patient had an uneventful history for the preceding 2 years and an unremarkable review of systems, general screening investigations were ordered, including erythrocyte sedimentation rate, a complete haemogram (including total and differential leukocyte counts), tuberculin skin test, chest X-ray, urinalysis, and serological tests such as *Treponema pallidum* haemagglutination antigen, rheumatoid factor, C-reactive protein, and antinuclear antibody. The investigations were not suggestive of any systemic infectious, rheumatic, or vasculitic disease. In the absence of anterior scleritis, episcleritis, and keratitis, systemic infections were a remote possibility in our patient.[5] The most common infections—herpes zoster, syphilis, and tuberculosis[5]—were further ruled out by the absence of typical mucocutaneous findings and negative investigations, as mentioned above. Neuroretinitis—most commonly caused by *Bartonella* infection—was also a differential diagnosis because of the presence of disc oedema, SRD, and subretinal infiltrates.[6] Absence of features such as a history of exposure to cats, systemic flu-like illness, a benign course, granulomatous or ulcerative conjunctivitis, and regional lymphadenopathy ruled against cat-scratch disease.[6]

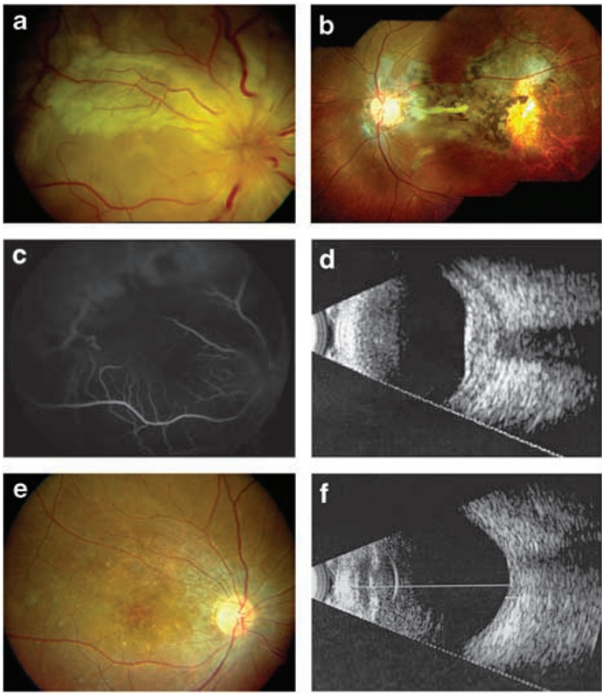

Figure 1 Clinical, angiographic, and ultrasonographic presentation, and post-treatment resolution of posterior scleritis with retinal artery occlusion and exudative detachment (a) Fundus view of the right eye, showing optic disc oedema, venous dilatation, serous retinal detachment, and segmental pallor along the superotemporal arcade, rendered less prominent by the turbidity of the subretinal fluid. (b) Left fundus showed the evidence of resolved inflammation with severe chorioretinal scarring, retinal pigment epithelial atrophy, and pigment migration. (c) Midphase fluorescein angiogram shows delayed transit of dye from the superotemporal branch retinal artery, with empty corresponding capillaries and vein. The faint choroidal hyperfluorescence is probably indicative of associated inflammatory activity. (d) B-scan ultrasound through the right optic nerve shows gross hyper-reflective thickening of the retina-choroid-sclera complex (5.06 mm), fluid in the sub-Tenon's space (T-sign), and squaring of the optic nerve shadow. (e) 4 months later, the fundus shows complete resolution of the inflammatory and vascular sequelae, with residual granular pigmentary stippling of the posterior pole. (f) B-scan confirms the resolution of scleral thickness (1.39 mm) and sub-Tenon's fluid, with normalised optic nerve shadow.

After carefully ruling out all the infectious aetiologies, the patient was treated with intravenous methyl prednisolone (15 mg/kg/day) for 3 days, followed by oral corticosteroids (1 mg/kg/day). After 2 weeks, BCVA improved to 6/60 with decreased scleral thickening. At 16 weeks, BCVA was 6/9 OD, with a brisk pupillary reaction. There was complete clinical and sonographic resolution of scleritis (Figure 1e and f), with no residual field defect.

Comment

Posterior scleral inflammation can extend into the optic nerve and retinal vessels, resulting in vascular occlusions:[7] cilioretinal artery occlusion, retinochoroidal infarction, and combined retinal vascular occlusion have been reported.[2–4] We are unaware of any report describing simultaneous retinal arterial occlusion with SRD in posterior scleritis. Ocular pain, SRD, disc oedema, and choroidal infiltrates were important clues; ultrasonography clinched the diagnosis. Although macular lesions and visual loss mandate aggressive therapy, a third of the patients suffer further loss of vision.[1] We obtained a rapid anatomic and functional resolution of scleritis and its sequelae with early diagnosis and aggressive treatment. This was of critical importance in a patient who had already suffered visual loss probably due to the same cause in the fellow eye, as indicated by similar antecedent symptoms and chorioretinal sequelae. Indeed, nearly half of the

patients with scleritis develop bilateral disease, 50% of whom have delayed onset in the fellow eye, mostly of the same type of scleritis.[8] A high index of suspicion may uncover this sight-threatening but treatable condition.

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Eye (2009) **23,** 1475–1477; doi:10.1038/eye.2008.217; published online 11 July 2008

Sir,

Black or negative flashes in posterior vitreous detachment a transient symptom before lightning flashes commence

Occasionally a patient with medical training will experience the symptoms of a disorder that may provide a unique opportunity to determine the exact nature of the symptom. This has occurred in the past with lightning flashes from posterior vitreous detachment (PVD) because Moore published the findings of his own photopsia in 1947.[1] We describe an ophthalmologist colleague who suffered bilateral PVD with specific symptoms of black flashes for a brief period before the onset of the typical lightning flashes. One of the authors (LW) a 51-year-old female ophthalmologist with −5.5 D right eye and −6.0 D left developed acute PVD in the left eye. The first symptoms she noticed were black coloured flashes in the inferotemporal periphery. These were momentary, vertically orientated and occurred in a flickering pattern before subsiding and then returning again. After 6 h, they were replaced by white flashes and floaters. She was examined by THW and found to have a PVD with Weis ring. Seven years later, she developed in the right eye a similar pattern of black flashes for 2 h before they were replaced by white flashes and floaters from PVD (confirmed by THW). We suspect that many patients forget or disregard this initial symptom because these flashes are soon replaced by white lightning flashes. Since the presentation by LW, we have on specific questioning found black flashes to be present in other individuals with PVD. The symptom may be due to traction on the axons of the surface of the optic nerve by the vitreous as the PVD process creates the Weis ring thus interrupting the signal from the retina. The fact that the PVD has not yet occurred is suggested by the appearance of floaters and white lightning flashes only after the black flashes have disappeared. Evidence that the PVD process can be traumatic to the optic nerve head can be seen in some patients who develop haemorrhages on the surface of the optic nerve head after PVD.[2,3] This is the first description of black photopsia in PVD that we are aware of.

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Eye (2009) **23,** 1477; doi:10.1038/eye.2008.209; published online 4 July 2008

Sir,

Successful surgical treatment of optic disc pit maculopathy

Congenital optic disc pit (ODP) with associated maculopathy is a rare anomaly with unknown pathogenesis. However, since the remarkable observations of Lincoff, it is widely accepted that fluid originating from ODP creates a schisis-like separation of the neuroretina and subsequently breaks through into the subretinal space.[1]

We report successful surgical management of this pathology with emphasis on drainage of subretinal fluid.