patient with high BP and unilateral acute-angle closure glaucoma without any other end-organ damage.

Case report

A 22-year-old man presented with severe pain in the left eye without any significant past ocular history. The right eye examination was within normal limits and the left eye had visual acuity and intraocular pressure (IOP) of 20/400 and 55 mm Hg, respectively. The refractive error was plano in the right and −3.00 in the left eyes. Anterior chamber of the left eye was significantly shallower and gonioscopy revealed Shaffer grade 4 (OD) and 0 (OS). Fundoscopy showed optic nerve head swelling, soft exudates, generalized narrowing of arteries, and venous engorgement (Figure 1). On the basis of these findings, we checked BP and found it to be 250/160 mm Hg. There were no signs or symptoms of hypertensive encephalopathy and the brain MRI was normal. The patient was admitted for intravenous anti-hypertensive medications. Because of the high BP, mannitol could not be administered and the IOP was lowered to 28 mm Hg using intravenous lidocaine (0.8 mg/kg) and ocular massage.[3] Although fundoscopy showed no choroidal detachment, ocular sonography revealed choroidal thickening and choroidal effusion (Figure 2). Peripheral iridoplasty was carried out and systemic and topical steroid, topical anti-glaucoma, and cycloplegic medications were started.

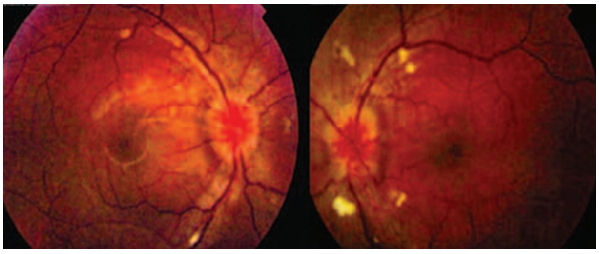

Figure 1 Fundoscopy showing optic nerve head swelling, soft exudates, generalized narrowing of arteries, A-V nicking, and venous engorgement in both eyes.

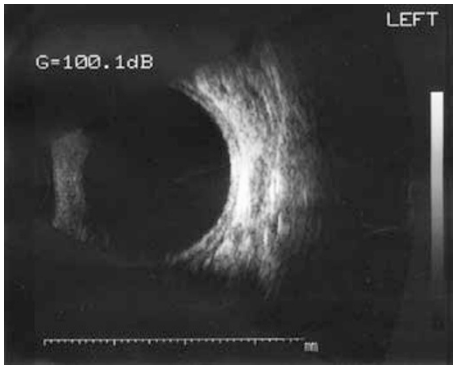

Figure 2 Left eye sonography showing choroidal thickening and choroidal effusion.

After 2 months, the patient's visual acuity returned 20/20 (uncorrected) and IOP was 18 mm Hg without medication. A complete laboratory work-up for possible causes of hypertension was negative.

Comment

The increased BP creates mechanical stress and endothelial injury that can lead to leakage from choroidal vessels and accumulation of fluid in the sub-retinal space.[4] The leaked fluid may result in choroidal effusion and anterior rotation of ciliary body and myopia, as happened in this case.

A PubMed search for 'acute angle-closure glaucoma', 'hypertension' yielded no relevant publications. We cannot attribute with absolute certainty the acute angle-closure glaucoma as end-organ damage of hypertensive crisis. Our aim was rather to sensitize the scientific community to this possibility.

Conflict of interest

The authors declare no conflict of interest.

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Eye (2010) **24,** 1629–1630; doi:10.1038/eye.2010.114; published online 27 August 2010

Sir,

An unusual case of recurrent endogenous *Klebsiella* endophthalmitis

We report an unusual case of recurrent endogenous *Klebsiella* endophthalmitis.

Case report

A 58-year-old HIV-negative male with diabetes mellitus, intravenous drug use, and *Klebsiella pneumoniae* sepsis complained of floaters of the right eye following 6 days of intravenous ceftriazone. Visual acuity of the right eye was 6/120 with 3+ anterior chamber cells, posterior

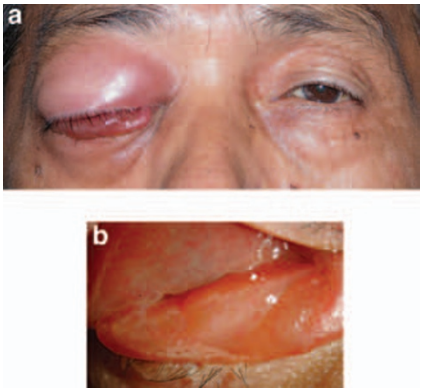

Figure 1 (a) Recurrent episode showing proptosis of the right eye. (b) Slit lamp examination showing conjunctival chemosis.

synechiae, but no hypopyon. Fundal examination showed vitritis and choroidal abscesses. The left eye was normal.

Intravitreal vancomycin and ceftazidime were given following vitreous tap that showed no bacterial growth. Intravenous ceftriaxone was continued for another 4 weeks with topical ciprofloxacin 0.3%. Dexamethasone 0.1% was added at 4 weeks. The inflammation improved and topical medications were stopped at 3 months. At 4 months, visual acuity of the right eye was 6/30, with quiet anterior chamber, mild vitreous haze, a small choroidal scar, but no active lesions.

After 1 month, he developed severe right panopththalmitis (Figure 1a and b). Visual acuity of the right eye was hand motions with oedematous cornea, 4+ anterior chamber cells, with a hypopyon. IOP was 34 mm Hg. Fundal examination revealed multiple vitreous opacities with no choroidal abscesses. B-scan ultrasound showed thickened sclera. He was given intravitreal vancomycin and ceftazidime, topical ciprofloxacin, glaucoma medications, and intravenous ceftriaxone following vitreous tap, which was positive for *Klebsiella pneumoniae*, sensitive to ceftriaxone and ciprofloxacin. The septic workup was negative. Unfortunately, he rapidly worsened and the right eye was eviscerated 8 days later due to increasing pain, proptosis, and refractory glaucoma.

Comment

Recurrence following successful treatment of endogenous endophthalmitis (EE) is unknown. Our patient was well for 2 months before presenting as a relentless panophthalmitis. Although corticosteroids can decrease tissue destruction from the host inflammatory response, its use in EE remains controversial with visual outcomes ranging from no perception of light to better than 6/60.[1–3] In our patient, the use of topical steroids as well as his diabetes mellitus may have allowed the organism to become sequestered rather than being totally eradicated, hence permitting a recurrence, which proved to be more fulminant than the first episode.

In conclusion, despite apparently successful treatment of EE, ophthalmologists should be aware of possible recurrence, especially in patients who had received corticosteroids.

Conflict of interest

The authors declare no conflict of interest.

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Eye (2010) **24,** 1630–1631; doi:10.1038/eye.2010.95; published online 18 June 2010