

eyes (Figure 2b). Features typical of IPT, such as reduced macular transparency and superficial white crystals, had developed since the previous visit (Figure 2b). Changes were similar in both eyes. Fluorescein angiography showed telangiectatic changes in the early phase as well as diffuse–late hyperfluorescence (Figure 2c–f). Innerlamellar holes seen in OCT in 2004 were still present and somewhat larger (Figures 2g and h).

Comment

The clinical findings in IPT are most obvious in the retinal blood vessels, however, it is possible that the primary defect lies in other retinal components, such as the retinal pigment epithelium, neural cells (including photoreceptors) or Muller/glia. The inner-lamellar holes may result from the neuroretinal decay rather than being of exudative origin since they were already present before vascular changes and are not visible during fluorescein angiography. The precession of typical vascular changes of IPT by the less well-recognised neuronal changes, as demonstrated in this case, suggests that photoreceptor damage may contribute to the early pathogenesis of the disease.

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Conflict of interest

Dr Mark Gillies is Executive Scientific Manager of the MacTel Research Project.

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Sir,

Recreational use of 3,4methylenedioxymethamphetamine ('ecstasy') relieving symptoms of posterior scleritis

Posterior scleritis is an uncommon form of scleral inflammation. While the prevailing consensus is that scleritis is an immune-mediated disease,¹ its precise pathogenesis remains enigmatic.

We describe a 23-year-old man with intermittent painful right eye and vision loss since 3 years due to posterior scleritis, which resolved when taking recreationally 3,4-methylenedioxymethamphetamines (MDMA, 'ecstasy').

Case report

A healthy 23-year-old man was referred to our hospital with pain and severe loss of visual acuity in the right eye (20/400) since 3 months. History revealed similar episodes during the last 3 years, which resolved partially when using ecstasy on a recreational basis. Since the patient had stopped ecstasy abuse 3 months ago, symptoms worsened.

Echographic evaluation showed choroidal thickening and oedema in Tenon's space in the right eye (Figure 1a). Clinical examination revealed a swollen optic disc surrounded with chorioretinal folds (Figure 1b). Pupillary reflexes and ocular motility were normal.



Figure 1 (a) B-scan echographic examination of the right eye showing choroidal thickening and oedema in Tenon's space at presentation; (b) red-free image of the right optic nerve surrounded by chorioretinal folds; and (c) pattern visual evoked potential (pVEP) showing half of the height of the amplitudes in the right eye compared to the left eye, and normal latencies.



Figure 2 Goldmann visual field analysis tested with object V4, I4, and I2 (a) at presentation, showing normal peripheral limits in the left eye; (b) constriction of the peripheral limits and a central scotoma in the right eye; (c) after 3 months of high-dose corticosteroid regimen, the left eye stayed within normal limits; and (d) right eye examination showed almost normalization with, however, still a central scotoma.

Goldmann visual field revealed a central scotoma (Figure 2a) and colour vision was discretely disturbed on the red–green axe. Latencies were normal, but amplitudes were smaller on pattern visual evoked potential (VEP; Figure 1c). Examination of the left eye was unremarkable.

Treatment with oral steroids was instituted (1 mg/kg), and tapered slowly. Visual acuity recovered to 20/25 and the central scotoma regressed over 3 months (Figure 2b and c).

Comment

The role of cellular immune dysfunction in posterior scleritis is suggested by immunopathological findings showing a predominance of T cells, mostly CD4 + lymphocytes, infiltrating the scleral fibers.¹ Oral corticosteroids are the treatment option when nonsteroidal inflammatory drugs fail.

Ecstasy is a psychostimulant drug that acts on the central nervous system. Amphetamine isomers have been described to induce acute nonarteritic ischaemic optic neuropathy.² Ecstasy also activates the hypothalamic pituitary adrenal axis, which induces a significant rise in cortisol plasma concentrations and a depression in immune function.^{3–5}

In summary, we present here a patient with long-standing, unrecognized posterior scleritis. Reminiscent episodes following recreational use of ecstasy were most probably due to a depression in cellular immunity. It is important for physicians to recognize the atypical course of some eye diseases such as posterior scleritis because of ecstasy abuse.

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Sir,

Prenatal diagnosis of dacryocystocele

Congenital dacryocystocele is a benign solitary mass arising from narrowing or obstruction of the nasolacrimal system during natal development. Its prenatal diagnosis, using sonography, is straightforward. CT and MRI are of benefit only if the diagnosis is unclear.¹ It is usually detected during the third trimester. Many lesions resolve spontaneously or after minimal intervention.

Case report

We report a case of a healthy male neonate with a congenital dacryocystocele, which was diagnosed pre-natally. The mother was a 30-year-old primigravida with no history of consanguinity. A routine screening ultrasound test at 20 weeks of gestation was normal. At 33 weeks' gestation, she underwent a second routine ultrasound scan. Both surveys were performed using an ATL 3000 ultrasound machine (Philips Medical Systems, Bothell, WA, USA). At this second examination, a unilateral 10 mm hypoechogenic mass was located inferiomedially to the right orbit. The differential diagnosis for a medial canthal mass includes dacryocystocele, capillary hemangioma, solid dermoid,



Figure 1 Prenatal ultrasound scan (at 33 weeks' gestation) that demonstrates the globe, the dacryocystocele (arrow), and the nose.



Figure 2 Postnatal photograph (a) immediately after birth showing the dacryocystocele; a bluish, cystic, non-tender, firm mass inferior to the right medial canthal tendon and (b) spontaneous resolution of the dacryocystocele 24 h later with no further intervention.

dermoid cyst, encephalocele, meningoencephalocele, nasal glioma, lymphangioma, and heterotopic brain. Further examination did not reveal any other pathological findings and demonstrated normal fetal facial anatomy (Figure 1). Repeated scans demonstrated no change in the mass size. On the 39th week of gestation, a vacuum extraction delivery was performed. Gestational weight was 2105 g. His Apgar score was 9/10. The diagnosis of congenital dacryocystocele was confirmed postnatally (Figure 2a). The infant had no signs of epiphora, dacryocystitis, facial cellulitis, or airway obstruction. The lesion resolved spontaneously 24 h postnatally with no further intervention (Figure 2b).

Comment

Prenatal diagnosis of dacryocystocele is very important because of the possibility of accompanying pathologies such as anterior encephalocele, teratoma, hemangioma, glioma, or rhabdomysarcoma.² A retrospective study of congenital dacryocystoceles showed complete resolution in 90% of cases following surgery, favouring early surgical intervention.³ Another retrospective study reported spontaneous resolution in 16.7% with a recurrence rate of 22% after probing.⁴ We suggest conservative treatment initially. If spontaneous resolution does not occur within 24 h, then the nasolacrimal probing is the treatment of choice. Surgical intervention may benefit those who are suffering from dacryocystitis, facial cellulitis, breathing difficulty, recurrences, and failure of digital massage, or probing.

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