Figure 2 Clinical photograph demonstrates a formed anterior chamber after Nd:YAG-laser peripheral iridotomy (white arrow).

In conclusion, C_3F_8 migration into the anterior chamber, inducing a pupillary block glaucoma is a complication that has not been reported in phakic eyes. An early Nd:YAG laser iridotomy is a suitable way to reduce IOP in such cases.

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doi: 10.1136/bjo.2006.091918

Accepted for publication 5 February 2006

Did not receive any financial or material support to conduct this study.

The authors have no conflicts of interest or proprietary interest in any of the instruments or topics presented in this manuscript.

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Acute retinal pigment epitheliitis associated with intravenous bisphosphonate

Zoledronate is a newer bisphosphonate, primarily used for the treatment of tumour induced hypercalcaemia and skeletal related events in advanced bone malignancy. Bisphosphonates have been associated with a number of ocular side effects including conjunctivitis, episcleritis, scleritis, uveitis, and visual blurring.¹ We report a case of acute retinal pigment epitheliitis following the use of intravenous zoledronate. To our knowledge bisphosphonates have not

previously been associated with acute retinal pigment epitheliitis and could find no reference to it in computerised search utilising Medline and Pubmed.

Case report

A 47 year old glazier noted visual blurring and metamorphopsia within a day following intravenous infusion of zoledronate. One month after the infusion and onset of symptoms, his best corrected visual acuity was 6/5 in each eye. There was no vitritis or anterior uveitis. Fundus examination showed subtle clumps of hyperpigmentation surrounded by a paler halo of hypopigmentation in discrete clusters around both maculas at the level of the retinal pigment epithelium (RPE), more visible in the left eye (fig 1). The Amsler grid showed metamorphopsia corresponding to the area of RPE disturbance. The optic discs, retinal vessels, and surrounding retina were normal. Optical coherence tomography was normal. Fundus fluorescein angiography revealed an area of hypofluorescence corresponding to the area of hyperpigmentation, surrounded by an area of hyperfluorescence in the early phase and in the arteriovenous phase (fig 2), while fading in the late phase, with no leakage (fig 3). A detailed history, systems review, and comprehensive investigations were negative.

Comment

Acute retinal pigment epitheliitis (ARPE) or Krill's disease was first described by Krill and Deutman as a benign, acute self limiting inflammatory disorder of the RPE without systemic association, with reduced visual acuity, metamorphopsia, and subtle changes at the level of the RPE.² Clusters of lesions with hyperpigmented centres, surrounded by paler halos, at the posterior pole have been reported as early as 1 month after the onset of symptoms.³ These lesions become less

Figure 1 Colour fundus photograph (A) right eye, (B) left eye. These show subtle clumps of hyperpigmentation surrounded by a paler halo of hypopigmentation in discrete clusters around both maculas at the level of the retinal pigment epithelium, more visible in the left eye.

Figure 2 Fundus fluorescein angiogram, arteriovenous phase (A) right eye, (B) left eye, showing areas of hypofluorescence corresponding to the area of hyperpigmentation, surrounded by an area of hyperfluorescence.

distinct with time leaving an area of subtle pigment mottling. Although most recover over several months Chittum and Kalina describe one case with a persistent small scotoma.³

Acute retinal pigment epitheliitis is thought to represent areas of RPE inflammation with breakdown of the pigment epithelial barrier and subsequent leakage of serous

Figure 3 Fundus fluorescein angiogram, late phase (A) right eye, (B) left eye, illustrating fading hyperfluorescence with no leaking.

fluid.² Viral causes including hepatitis C have been postulated.²⁻⁴

Bisphosphonates have been associated with many ocular adverse effects, the majority being inflammatory; anterior and posterior uveitis, episcleritis, scleritis, and optic neuritis.⁵ Blurred vision is well documented and listed by the World Health Organization as a "certain" ocular adverse effect of bisphosphonates⁵ although, the cause of this has not been identified.

Drug induced immunological or toxic reaction have been hypothesised to be responsible for an acute inflammatory reaction in the case of pamidronate.⁶

Unfortunately, most ophthalmic toxicology data rely on voluntary post-marketing surveillance systems and case reports.⁵ Applying the World Health Organization Causality Assessment Guide⁷ this case of acute retinal pigment epitheliitis is a possible adverse reaction of zoledronate as there was a plausible time relation to drug administration and absence of other drugs or chemicals or of concurrent disease that could explain the adverse effect.

The main limitations of our report are that this is a single case, the eyes were not examined until 1 month after the drug infusion and subsequent onset of symptoms including no examination before the bisphosphonate infusion, and the patient did not undergo a re-challenge test.

Further reports from similar cases are required to confirm these findings. It is important that ocular symptomatology occurring in association with the use of novel medicines is diagnosed and data drawn to the attention of physicians.

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doi: 10.1136/bjo.2006.092189

Accepted for publication 7 February 2006

Commercial interest: None of the authors have a commercial interest in the drug mentioned or its competitors.

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Retinal haemorrhages in a preterm infant following screening examination for retinopathy of prematurity

Examination induced retinal haemorrhage in preterm infants is uncommon. It may occur independently of retinopathy of prematurity (ROP) associated neovascularisation. Retinal haemorrhages due to ROP tend to occur on the surface of the neovascular ridge, which represents an arteriovenous shunt formed by the anastomosis of primitive retinal vessels.¹ However, ROP related retinal haemorrhages occurring posteriorly,² as well as vitreous haemorrhage,³ have also been described in advanced disease. Other common causes of retinal haemorrhage in infancy such as birth⁴ and shaken baby syndrome should be excluded. Retinal haemorrhage in a premature infant after RetCam photography has been reported.⁵ We report retinal haemorrhages in a premature infant immediately following examination for ROP.

Case report

A 1416 g, 33 week gestation infant was transferred to our centre after developing necrotising enterocolitis at 6 days old. Ventilatory support was required briefly following subtotal colectomy at 50 days old. In accordance with Canadian screening guidelines,⁶ this infant had significant risk of developing ROP based on birthweight criteria. However, he was referred by the attending neonatologist for his first ROP screening at 45 weeks postconceptional age. Initial examination, conducted by a paediatric ophthalmology fellow, showed no ROP in the left eye and two areas of right eye vitreous haemorrhage (superonasally and inferonasally). No retinal haemorrhages were seen. On examination by the staff ophthalmologist 10 minutes later, a moderate number of intraretinal dot and blot haemorrhages and a few flame haemorrhages were observed in the right posterior pole, predominantly peripapillary (fig 1). There was no vascular tortuosity or disc swelling. The left eye remained normal. Both examinations were performed with a lid speculum and Flynn scleral depressor (Bausch and Lomb, Rochester, NY, USA) to rotate the eye.

Figure 1 Intraretinal haemorrhages, predominantly peripapillary.

Figure 2 RetCam II fluorescein angiography showing peripheral areas of telangiectasia.

RetCam II (Clarity Medical Systems, Inc, Pleasanton, CA, USA) images showed no additional bleeding. Investigations to screen for intrauterine infections and coagulopathy were normal. Intracranial haemorrhage was absent.

To facilitate visualisation, RetCam fluorescein angiography was performed the following day under general anaesthesia to look for vascular anomalies underlying the areas of vitreous haemorrhage as well as those not immediately visible on standard examination. This demonstrated 3 clock hours of telangiectasia peripherally in the right eye (fig 2) consistent with zone 3 immature retinal vasculature without leakage. Inferionasally, there appeared to be peripheral retinal vascular fronds directed towards the vitreous haemorrhage. Subsequent RetCam examinations documented resolution of the retinal haemorrhages with persistence of the vitreous haemorrhage at 14 weeks. No demarcation lines or ridges were seen.

Comment

RetCam photography has been implicated in the development of retinal haemorrhages in a premature infant. A 50 day old infant, status post 25 week prematurity, developed two episodes of unilateral retinal haemorrhages following RetCam examination on two separate occasions.³ Immature fragile retinal vessels, made more susceptible to rupture by hypoxia and poor central control of circulatory autoregulation, were postulated as likely mechanisms. A 24 week premature infant with bilateral stage 3 zone II ROP examined at 35 weeks postconceptional age, developed multiple flame-shaped intraretinal haemorrhages after intravenous sedation and cardiopulmonary resuscitation (CPR).⁷ While the authors suggested the role of CPR, they cautioned that pre-existing ROP and immature retinal vasculature might have predisposed to vessel rupture. In both cases, it may be that the premature retinal vasculature, particularly in the setting of ROP, functions differently from that in older infants. Examination induced retinal haemorrhages appear to have a predilection for areas posterior to the vascularised avascular retina junction occurring in moderate numbers without subretinal involvement. Our patient's vitreous haemorrhage may have occurred at a time when the vascular abnormalities were more active and the vessels more immature. It has been suggested that poor vascular autoregulation in sick neonates, or the absence of a mature autonomic system in preterm infants, can expose