LETTERS TO THE EDITOR

Recurrent uveitis in a patient with adult onset cyclic neutropenia associated with increased large granular lymphocytes

EDITOR—A 35-year-old white man was referred with a 1 month history of recurrent bilateral uveitis. The patient had a history of cyclic neutropenia beginning at age 19. Severe neutropenia (total neutrophil count as low as 68 cells $\times 10^{3}$), lasting about 3 days, was documented to occur at the time of clinical symptoms. Prednisone produced a marked diminution in episodes of symptomatic neutropenia. At age 35, the patient developed iritis in his left eye during a ‘crisis’. This was treated with 0.1% dexamethasone phosphate solution. The anterior chamber inflammatory reaction subsided, but 1+ cells persisted in the vitreous despite an injection of methylprednisolone 40 mg into the sub-Tenon’s capsule. The fundus was normal. An extensive immunological examination and HLA-B27 determination were negative or normal. The uveitis resolved as the crisis subsided. Recurrent episodes of uveitis in the left eye occurred episodically, but always at the time of neutropenic crisis and despite topical and systemic antiinflammatory therapy.

Development of cataract reduced the visual acuity to counting fingers at 1 foot. Cataract excision, with posterior chamber lens implantation resulted in stable visual acuity of 20/30 (5 year follow up) despite continued episodes of iritis.

COMMENT

Cyclic neutropenia is a rare disorder characterised by regular oscillations in blood neutrophil counts, as well as other blood cells.1 Cycling occurs regularly every 21 days and lasts 3–6 days in most of the cases.2–5 The patient described here presented with cyclic episodes of neutropenia occurring every 18–25 days and lasting for 3–5 days. The patient’s neutrophil counts were repeatedly below 800 cells $\times 10^{3}$ at the changes in other leucocyte or platelet counts were observed.

During neutropenic episodes, patients may experience a wide variety of symptoms, from malaise and low grade fever to severe life threatening gastrointestinal infections and, although there is moderate morbidity associated with the disease, its course is usually benign.6 Conjunctivitis has been described. Iridocyclitis associated with neutropenic cy- cling and refractory to therapy has not been described previously. Extensive and repeated laboratory investigations for an underlying cause of the uveitis in our patient were negative except for large granularlymphocytes and an IgG antineutrophil antibody, both characteristic of cyclic neutropenia.

Many therapeutic strategies have been used in the management of patients with cyclic neutropenia. Splenectomy, testosterone, etiocholanolone, and endothelin have been employed, all to some therapeutic effect.7 The response to alternate day low dose prednisone therapy has been particularly successful for patients with the adult onset of the disease.8 Our patient was managed with 5–25 mg of prednisone every other day, with significantly reduced symptomatology and neutrophil cy- cling. More recently, treatment has been reported with granulocyte-macrophage colony stimulating factor (GM-CSF)9 and granulocyte-colony stimulating factor (G-CSF).10 Although a direct cause-effect relationship between the recurrent uveitis present in this patient and adult onset cyclic neutropenia cannot be definitively established, the existence of different immune system abnormalities (for example, proliferation of large granular lymphocyte cells, inversion of the CD4:CD8 ratio, presence of different autoantibodies) in patients with cyclic neutropenia and large granulocyte lymphosarcoma may contribute to the recurrence of uveitis and its refractoriness to conventional therapy.

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REFERENCES


Delayed acute retinal necrosis after herpetic encephalitis

EDITOR—The herpes viruses family, especially herpes simplex virus (HSV) and varicella zoster virus (VZV), has been associated with acute retinal necrosis (ARN) and these viruses also represent an important cause of encephalitis.7 Cases of retinitis in patients with a history of previous herpetic encephalitis, although rare, have been reported8; a delay between the diagnosis of encephalitis and the onset of ocular symptoms has been reported to be as long as from 1 to 5 months,9 but an interval of up to 5 years has been recorded.10 We report here two patients who developed the clinical picture of ARN 7 and 17 years after having had herpetic encephalitis.

CASE REPORTS

Case 1

CH, a 27-year-old white woman presented with a 10 day history of deterioration of vision in her left eye. Seven years previously she had been diagnosed as having HSV encephalitis, confirmed by electron microscopy and viral culture of a brain biopsy specimen. The sequelae of HSV encephalitis included right homonymous hemianopia. Visual acuities were 6/5 right eye and light perception left eye. The right eye was normal. Examination of the left eye revealed intense vitritis, and retinal thickening with extensive peripheral retinal necrosis and patches of central retinal necro- sis. The optic disc was swollen and there was an inferior retinal detachment, extending to the macula. There was extensive retinal vascularitis and haemorrhage. ARN was diagnosed on clinical grounds and she was treated with intravenous acyclovir, 10 mg/kg three times a day which was reduced to 400 mg five times daily during 12 weeks. After this period the dose was reduced to 400 mg five times daily, which she is still taking.

Vitreal biopoly and lumber puncture were performed. No virus was found in the CSF; however, using techniques based on polymerase chain reaction (PCR) the vitreous specimen revealed the presence of HSV-1 and VZV DNA. The right eye has remained stable and the patient has retained the left hemifield of vision in the right eye with visual acuity of 6/5.

Case 2

GP, a 17-year-old white man presented with a 1 week history of blurring of vision in his left eye. Vision in the right eye had been poor since childhood as a result of optic atrophy. HSV encephalitis had occurred at 1 week of age, which was diagnosed clinically and confirmed on aspiration of fluid from a skin vesicle.

Visual acuities, on presentation, were of counting fingers in both eyes. The right eye revealed sector iris atrophy temporally and optic atrophy. The left eye had significant vit- ritis, peripheral retinal necrosis, and disc swelling. No iris atrophy was present in this eye and there were no records of optic nerve or periperal retinal involvement before the current presentation.

A clinical diagnosis of ARN was made and the patient was treated with intravenous acyclovir 10 mg/kg three times daily and oral prednisolone (initially 80 mg/day). Unfortunately a vitreous sample was not available for PCR analysis.

After 1 week on intravenous acyclovir he was given a course of oral acyclovir for 12 weeks (800 mg five times a day) which was then reduced to a maintenance dose of 400 mg five times a day. At a last examination he had visual acuities of hand movements in his left eye, and there was no inflammatory activ- ity in either eye.

COMMENT

Delay between the onset of viral encephalitis and ARN has been mentioned previously in the literature. Intervals of 1 and 5 months have been reported with the mention in one case of a 5 year interval,12 but without any other information regarding the clinical and diagnostic aspects of both the encephalitis and ARN. In our patients the delays were 7 years.
and 17 years respectively and the occurrence of ARN in these patients was particularly dev-
astating as both had pre-existing visual impairment related to previous encephalitis. The explanation for such prolonged intervals include the possibility of persistent latent virus in the CNS with spread to the eye via the optic nerve, or the presence of latent virus in the ocular tissue since the time of primary infection. Cases with asymptomatic peripheral chorioretinal lesions have been reported and these lesions were felt to be an indication of persistence of the virus in the retina. Treatment in the acute phase is by systemic acyclo-
vir, which has a potent antiviral action against vesi
culoviruses, and herpes simplex. Acyclovir is only effective against actively replicating virus, and cannot eradicate latent virus, such that any protective effect is questionable. The risk of further reactivation of the virus in our patients is unclear. For this reason, and especially taking into consideration the severe visual sequelae from their herpetic encephali-
tis and the further loss of vision after the episode of ARN, both patients have been maintained on low dose acyclovir (400 mg five times a day).

Considering that significant visual loss usually results from retinal and optic nerve involvement as a late sequela of congenital HSV infection and, also, potentially from involvement as a late sequela of congenital cytomegalovirus infection, visual sequelae from their herpetic encephali-
tis may occur. We report on successful treatment of methanol (methyl alcohol) induced blindness with late steroids and vitamin B-1.

CASE REPORT
A 40-year-old man was admitted to our eye department with sudden bilateral visual loss. He was generally healthy except that he was a smoker and drank alcohol regu-
larly. During a period of 4–10 days before admission, the patient consumed pure meth-

anol used for cleaning metal electrical wires. One day before admission, the patient com-
plained of visual loss and pain; however, a complete medical exami-

ination in the emergency room was inconclusive and his vision was not affected. On admission, physical and ophthalmological examination were normal. Ophthalmic examination revealed visual acuity of hand movements in the right eye and 50 cm counting fingers in the left eye. Ocular examination was normal except for mild dilatation of the pupils which reacted sluggishly to light. No funduscopic abnor-
malities were observed. One day after admission, visual acuity diminished to no light per-
ception in the right eye and remained as counting fingers in the left eye, with a large central scotoma. Relative afferent papillary defect was detected in the right eye. Visual evoked response (VER) was undetectable in the right eye and prolonged latency was detected in the left eye (Fig 1). Computed tomogra-

phic analysis of the brain and the electroencephalogram were normal. Blood laboratory tests were nor-
mal with no evidence of metabolic acidemia. Nine to 15 days after methanol ingestion, no significant recovery was evident. The conventional treatment with bicarbonate was longer than effective. Medical therapy was initiated including prednisone 60 mg/day by mouth and an intramuscular injection of vitamin
B-1 100 mg/day. Three days after initiat-
ing the treatment, visual acuity, visual field analysis, and VER demonstrated evidence of signifi-
cant improvement in both eyes (Fig 1B). The patient was treated with vitamin B-1 injections for 20 days and with oral prednisone for 1 month with gradual tapering off. Seven weeks later the visual acuity had fully recovered in both eyes to the 20/20 level. Visual fields of both eyes showed only slight concentric constriction with no scotoma; VER demonstrated normal patterns on both eyes (Fig 1C).

COMMENT
Methanol intoxication may be associated with severe damage to the visual pathways, poten-
tially leading to permanent blindness. In an outbreak of methanol intoxication involving 320 people, Benton et al stated that in cases of visual impairment or blindness, if vision did not return to normal within 6 days, recovery was not complete and the vision deteriorated. Other reported cases of methanol intoxication also demonstrated the guarded prognosis of visual loss. Thus, prolonged visual malfunc-
tion is considered an unfavourable prognostic sign, and vision almost never fully recovers. Delayed recovery is rare, and usually not complete.

Figure 1 Visual evoked response (VER) with pattern reversal recorded during the acute phase, initial therapy, and after recovery. (A) Day 1, visual acuity, right eye no light perception, left eye counting fingers 50 cm. No structured pattern was recorded from the right eye and prolonged latency was recorded over the left eye with a very low amplitude. These recordings indicate a non-functional right optic pathway and severe damage to the left optic nerve. (B) Day 7, after 3 days of treatment, visual acuity was right eye hand movement and left eye 20/100. A definitive pattern is evident from both sides, however, latency is still prolonged corresponding to the recovery process of the optic pathways. (C) Seven weeks after methanol blindness visual acuity recovered in both eyes to 20/20. VER over the right eye demonstrates a residual prolonged latency whereas the VER pattern of the left eye returned to normal.
for we assume that he did not consume alcohol during this period—that is, ethylism did not play a role in his clinical symptoms.

The rationale for using steroids in methanol toxic neuropathy was based on the clinical experience with this drug being effective in other forms of optic neuropathies. The potential therapeutic effect of steroids on methanol optic neuropathy might be a reduction in the oedema of the nerve sheaths caused by the histotoxic anaemia as suggested by Sharpe et al.1 Steroids are usually not used to treat III effects of ethylism. Vitamin B-1 is given for treatment of Wernike-Korsakoff syndrome, following prolonged ethanol consumption. We assumed that since vitamin B-1 is highly effective in ethanol intoxication, it might well be effective in methanol intoxication. Other ocular associated pathologies in which thiamine may be effective are nutritional amblyopia, and Leigh’s disease (infantile necrotising encephalomyopathy), both irrelevant in this case.

In conclusion, the combination of steroids and vitamin B-1 was highly effective in treating severe methanol optic neuropathy. We do not know whether using only one of the drugs might be sufficient but, since the risk of permanent blindness is high, we recommend using this combination in the event of methanol intoxication.

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Intracranial plasmacytoma presenting with optic nerve compression

EDITOR,—Neurological complications are not uncommon in patients with multiple myeloma; however, an isolated intracranial plasmacytoma is rare. We describe a patient with multiple myelomatosis and a single intracranial lesion presenting with optic nerve compression.

CASE REPORT

A 73-year-old woman had symptoms of intermittent blurring of vision in the right eye for several months and had consulted her optometrist, who had reassured her. She subsequently presented to her general practitioner with a 3 month history of left upper arm and right lower chest ‘rib’ pain, anorexia, weight loss, malaise, and fatigue. Skeletal survey revealed multiple extramedullary lesionalisations, bilaterally, with associated lytic bone destruction at each site. There was also evidence of lysis of the transverse process of T1 and facet complex of C7 on the right. She was referred urgently for further investigation.

On admission examination revealed no focal neurological deficit and investigation with inferior albinus scotoma was noted on the right. There were minimal cortical lens opacities and funduscopy did not indicate any optic disc hyperaemia, swelling, or pallor. An urgent cranial magnetic resonance imaging was requested and this revealed a smoothly outlined mass in the right middle cranial fossa at the apex of the orbit compressing the optic nerve and enhancing with gadolinium (Fig 2); this mass appeared to arise from the dura mater and was the only intracranial mass detected. A diagnosis of intracranial plasmacytoma secondary to multiple myeloma was made and she was referred for radiotherapy to the lesion. Six weeks later visual acuity was 6/9 N 5 right and left eyes, the right RAPD was barely detectable, and optic disc appearances remained normal.

COMMENT

Multiple myeloma accounts for 1% of all malignancies and 10% of haematological malignancies. Systemic signs and symptoms are due to pathological plasma cell proliferation, secondary metabolic alteration, and associated immunological dysfunction. Plasmacytomas are tumours comprising a proliferation of abnormal plasma cells; they demonstrate monoclonal intracellular immunoglobulins using immunohistochemical staining techniques, distinguishing them from plasma cell granulomas which show a polyclonal pattern of several different heavy and light chains.

Neurological complications in multiple myeloma were first classified by Clark1 into: (a) cranial nerve palsies; (b) intracranial tumour syndromes; and (c) intraorbital syndromes.

Intracranial tumour syndromes tend to present with multiple neurological symptoms and signs, occasionally with those of a space occu-}

Figure 1 Grey scale representation of the generalised constriction of the patient’s right eye central visual field as assessed by a Humphrey visual field analyser 24-2 full threshold program.

Figure 2 T1 weighted, gadolinium enhanced, axial magnetic resonance image illustrating post-enhancing lesion on the right compressing the intracranial optic nerve. (The bright image on the left was non-enhancing and reported as a probable old venous thrombus.)


Conjunctival and lacrimal sac pigmentation by kohl (eyeliner)

EDITOR.—Abnormal pigmentation of the conjunctiva and lacrimal sac was noted in 10 patients using kohl (eyeliner, surma) that was made in India. This eyeliner is used by millions in the Middle East, Asia, and Africa. The median age of nine women and one man was 52 years (range 40–64 years). All patients had used kohl for many years. The two patients with conjunctival pigmentation had bilateral, diffuse pigmentation of the fornical and tarsal conjunctiva of the upper and lower eyelids in the form of discrete, punctate black deposits (Fig 1). The other eight patients presented with epiphora and had chronic dacryocystitis associated with obstruction of the lacrimal passages, including the lacrimal canaliculi and common lacrimal canaliculus. Three of these patients also had dacryocystitis fistulas. During dacryocystorhinostomy, focal to diffuse black pigmentation of the lacrimal sac was observed in seven individuals, in scar tissue obstructing the common canaliculus in one, in the subcutaneous tissues around the fistula in one, and in the perioseum of the lacrimal sac fossa in another. The black pigmentation of the lacrimal sac resembled to some extent malignant melanoma, except for the absence of a mass. Preoperatively, a dark contiguous pigmentation was observed around the cutaneous fistula site in one patient. Histologically, the fine to coarse extracellular pigment was often surrounded by a chronic inflammatory infiltrate, mainly lymphocytes with a few plasma cells and neutrophils.

Energy dispersive analysis of x rays of all biopsy specimens revealed a high lead content in the patient’s tissues (Fig 2). Other less frequent elements included silicon, carbon, sulphur, silver, magnesium, iron, and aluminium. The kohl used by the patients was also tested and showed the same elements as detected in their biopsy specimens.

Radiographic studies of the biopsy specimens from a pigmented lacrimal sac showed a diffuse and dense radiopacity, corresponding to the high metallic content, mainly lead in the tissue.

COMMENT

In this study, the findings of a similar composition in the patient’s tissues and the kohl used in the area of origin strongly support the clinical diagnosis of kohl pigmentation in the conjunctiva and lacrimal drainage system. The various elements in kohl that cause tissue pigmentation include lead, silver, iron, carbon, and magnesium. Specifically, black pigmentation of the lacrimal sac by kohl may be confused with malignant melanoma if the surgeon is not aware of this entity. A more serious medical problem than pigmentation in our patients is the finding of lead in their tissues. In the past three decades, several well documented cases of lead poisoning (plumbism) have occurred in England among Indian and Pakistani children as a result of applying lead-containing kohl to their eyes. In our study, five patients who were tested for lead toxicity were normal. The reason for this discrepancy is probably related to the dose and site of kohl application. All of our adult patients applied the eyeliner to the eyelid margins for cosmetic reasons while in children a larger amount of kohl is usually instilled into the conjunctival sac for therapeutic reasons.

It is interesting to note that a biopsy specimen from the pigmented lacrimal sac that contained lead showed diffuse radiopacity when tested radiologically. This finding may be confusing to the radiologists and/or ophthalmologists if they are not aware of this problem. Dacryocystitis in those patients with lacrimal sac pigmentation is most probably the result of trachoma, which is very common in the Middle East.


The opinions or assertions contained herein are the private views of the authors and should not be construed as being official or as representing the views of the Department of the Army or the Department of Defense.

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Table 1 Clinical data for nine patients with kohl pigmentation

<table>
<thead>
<tr>
<th>Patient No, age (years), sex</th>
<th>Site of pigmentation</th>
<th>Clinical diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1, 60, F</td>
<td>Conjointiva, both eyes</td>
<td>Conjunctival pigmentation, cataract</td>
</tr>
<tr>
<td>2, 64, F</td>
<td>Conjointival, both eyes</td>
<td>Conjunctival pigmentation, cataract</td>
</tr>
<tr>
<td>3, 44, F</td>
<td>Common lacrimal</td>
<td>Chronic dacryocytosis, both eyes; common canalicular occlusion by pigmented scar, right eye; upper and lower canalicular occlusion, left eye</td>
</tr>
<tr>
<td>4, 52, M</td>
<td>Lacrimal sac, right eye</td>
<td>Chronic dacryocytosis with non-expressible mucocoele, right eye</td>
</tr>
<tr>
<td>5, 50, F</td>
<td>Lacrimal sac, right eye</td>
<td>Chronic dacryocystitis with mucocoele and dacrocystocanulius, right eye; blockage of lower canaliculus right eye; common canaliculus right eye and common canaliculus left eye</td>
</tr>
<tr>
<td>6, 50, F</td>
<td>Lacrimal sac, left eye</td>
<td>Chronic dacryocystitis, both eyes; partial common canalicular occlusion, both eyes</td>
</tr>
<tr>
<td>7, 40, F</td>
<td>Lacrimal sac, left eye; subcutaneous scar tissue around the fistula</td>
<td>Chronic dacryocystitis with fistula, left eye; pigmentation of subcutaneous tissue around the fistula left eye</td>
</tr>
<tr>
<td>8, 55, F</td>
<td>Lacrimal sac, left eye; periostium of lacrimal sac fossa, left eye</td>
<td>Blockage of upper and lower canaliculi, right eye; blockage of common canaliculus; congenital fistula from skin to common canaliculus</td>
</tr>
<tr>
<td>9, 56, F</td>
<td>Lacrimal sac, left eye</td>
<td>Common canalicular obstruction, both eyes</td>
</tr>
<tr>
<td>10, 55, F</td>
<td>Lacrimal sac, right eye</td>
<td>Chronic dacryocytosis, right eye</td>
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An unusual case of map-dot epithelial dystrophy

Editor,—Map-dot epithelial dystrophy, also called Cogan’s microcystic dystrophy, is a well known and relatively frequent clinical entity. The microscopic basis for the biomicroscopic appearance of the typical superficial corneal opacities and epithelial erosions are intraepithelial cysts and a multilaminated or bilaminated aberrant epithelial basement membrane. Therefore, the term epithelial basement membrane dystrophy has been introduced by some authors.

We report a patient with Cogan’s corneal dystrophy with histologically proved erythrocyte inclusions in the intraepithelial cysts and a conspicuous clinical appearance not before described.

CASE REPORT

In March 1995 a 48-year-old white man reported to our clinic with typical symptoms of recurrent epithelial microerosions mainly on his right eye. He complained of early morning pain and reduced vision for a few weeks but could not recall an earlier ocular trauma. He did not wear contact lenses. Biomicroscopically, bright red intraepithelial inclusions in the corneal epithelium were found in the right eye (Fig 1A); there was no fluorescein staining. The left eye showed a few whitish, map-dot-like epithelial opacities.

Figure 1 (A) Slit-lamp photograph of right eye. Note red blood-like colour of the inclusions in the map, dot, and fingerprint-like epithelial alterations of the cornea of the right eye (arrowheads). There was no fluorescein staining suggesting that the erythrocytes had already been entrapped intraepithelially. (B) Lower and (C) higher magnification photomicrographs of the corneal abrasion material demonstrating intraepithelial cyst with cell debris, keratin, and numerous erythrocytes (arrowhead) (trichrome stain).

Figure 2 (A) Photomicrograph of the corneal epithelium with bilaminated, intraepithelial basement membrane with folds (arrowhead). Note pale swelling of underlying oedematous basal epithelial cells (periodic acid Schiff, ×500). (B) Electron micrograph (×3240) illustrating basement membrane fold with bilaminated, redundant basement membrane. (C) Shows the more loosely arranged superficial layer (1) and the more densely packed second layer (2) beneath it, both containing randomly oriented fibrils in a finely granular matrix (contrasted with uranylacetate and lead citrate, ×9360).
There was no corneal or limbal neovascularisation and only a slight ciliary flush. The corneal stroma, the endothelium, and the anterior chamber were without peculiarities. The eyelid margins showed signs of mild chronic blepharitis, and the tear film was unstable. The visual acuities were 6/6 left eye and 6/7 right eye.

As the clinical appearance on the right eye was unknown to us and to exclude, for example, a bacterial infection, bacterial cultures, conjunctival scrapings, and focal superficial stripping of the corneal epithelium in some of the areas of the red alterations were performed by means of a hockey knife. The bacterial cultures were negative. Therefore, we only prescribed artificial tears.

After a few weeks treatment was stopped because the patient no longer had any symptoms. Biomicroscopically, the red inclusions had completely disappeared without leaving scars. However, there were still typical map-dot epithelial opacities in both eyes. The visual acuity of the right eye had normalised to 6/6.

**PATHOLOGICAL FINDINGS**

The light microscopic examination of the stripped corneal epithelium revealed intraepithelial microcysts containing entrapped cellular debris, keratin, and numerous red blood cells (Fig 1B and C). In addition, a thickened bilaminar aberrant basement membrane with folds was found intraepithelially, visible with periodic acid Schiff staining (Fig 2A). Electron microscopy again displayed a redundant, bilaminar basement membrane of the epithelial cells with folds (Fig 2B). On microscopy there was no hint of a bacterial infection.

**COMMENT**

We are not aware of any case of map-dot epithelial dystrophy or other corneal disease published previously with intraepithelial blood inclusions. It is useful to know of this corneal alteration because of the differential diagnosis of bacterial infection. It is also interesting because this case shows that blood cells can be included intraepithelially in the cornea under special circumstances despite regular blinking and washing of the ocular surface by the tear film.

The microscopic findings are compatible with previous histological reports on map-dot epithelial dystrophy. The aberrant intraepithelial basement membrane may obstruct the normal migration of maturing epithelial cells leading to the disintegration of these cells and the formation of microcysts which may rupture eventually and form corneal erosions.

In our case, initially there must have been temporary microerosions, presumably due to ruptured epithelial microcysts to which erythrocytes were attached, which were then incorporated into the epithelial microcysts when the corneal epithelium closed again. The site of origin of the erythrocytes within the intraepithelial cysts is unknown, although haemorrhage from the eyelid is a possibility. After a few weeks the red inclusions were no longer visible, probably because the entrapped erythrocytes had been phagocytised by macrophages.

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CORRESPONDENCE

Intraocular pressure changes after peribulbar injections with and without ocular compression

EDITOR,—Ten years on from Davis and Mandel’s description of the technique, and despite the popularity of the method, the exact role of, and rationale for, ocular compression in peribulbar anaesthesia remains somewhat obscure. Whatever the facts of the matter this would appear particularly undesirable since a body of evidence from clinical,1 cadaveric,2 and animal3 work would suggest certain potential risks associated with its employment. In their recent paper, published in this journal in May 1996,4 Bowman et al correctly admit that comparison of their results with those obtained by other workers is difficult since so many published studies attempt to draw conclusions from surgeons with widely differing practice. Final clarification of the controversy awaits. While conducting a study of a separate but related aspect of peribulbar anaesthesia, we were interested to establish the details of current consultant practice and surveyed 60 consultant members of the Scottish Ophthalmic Club with 42 (70%) questionnaires returned. Of consultants surveyed 55% were using peribulbar, 23% retrobulbar, 17% sub-Tenon’s, and 5% topical anaesthesia. Of those using peribulbar techniques, 62% considered compression with the balloon essential. In this group the minimum acceptable time for compression was quoted as between 5 and 20 minutes (average 9 minutes). Despite the theoretical risks from oculo-compression, especially in eyes with actual or potentially impaired optic nerve head blood flow, thankfully there is a relative paucity of reports of serious adverse effects in clinical practice.

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Author’s reply

EDITOR,—We knew that the use of oculocompression after peribulbar anaesthesia is not universal and we were interested to learn from Ellis and Kearns’s survey of the Scottish Ophthalmic Club that only 62% of the users of peribulbar anaesthesia considered oculocompression essential. We wonder whether those users of peribulbar anaesthesia who do not employ ocular compression were aware of our findings of the immediate mean pressure rise of 11.44 mm Hg with an injectate volume of 10 ml. We disagree with Ellis and Kearns that the rationale of the technique remains obscure: we have shown that oculocompression for 20 minutes with the Honan’s balloon after peribulbar anaesthesia produces a mean fall in intraocular pressure of 11.05 mm Hg. As Schipper points out, lower volumes of anaesthetic do not produce such a significant pressure rise. Oculocompression may not be necessary with smaller volumes of anaesthetic or topical anaesthesia. However, we continue to recommend the use of oculocompression to reduce the ocular pressure if the technique of peribulbar anaesthesia reported by Davis and Mandel is employed.

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First day follow up for routine phacoemulsification?

EDITOR,—We would certainly concur with Whitefield et al1 that an important reason why patients decline day case surgery is the need for an examination the next day. We would also question this necessity following uncomplicated sutureless phacoemulsification with lens implant. However, we feel it may be unsafe practice if patients are not examined at all in the early postoperative period.

Firstly, intraocular pressure can be raised following cataract surgery,2 and indeed it was in three cases in this study. The maximum pressure rise tends to occur 5–7 hours postoperatively3 and so if patients were examined then many more may well be found to have raised pressure. For most patients this transient ocular hypertension is not associated with significant morbidity, but central retinal artery and venous occlusions and non-arteritic anterior ischaemic optic neuropathy4 have been reported following cataract surgery and this pressure spike may be a significant factor.

It may be better, therefore, to examine the patient on the same day as surgery before discharge. The use of a prophylactic ocular anti-hypertensive agent would also be recommended. An early examination would also allow a significant wound leak to be detected.5 In the study by Tufail et al6 it was concluded that no complications were missed by examining the patients on the same day as surgery that would have been detected the next day.

Iris prolapse and endophthalmitis are both very rare (0.3%)7 and so would not be expected to be detected by a study of this size (100 patients). Often these conditions do not present on the first postoperative day, with endophthalmitis typically presenting 48–72 hours postoperatively.8 A fibrinous uveitis, which may be a warning of impending endophthalmitis, again is often worse a few days after surgery rather than at day 1. It is possible some of these cases could be screened for with a visit 3 days after surgery. It is our practice, however, to ask the patient to return as an emergency if symptoms change, especially increasing pain, rather than seeing all patients at day 3.

Our policy is to examine on the same day as surgery, before discharge, and again at 2 weeks

Reply

EDITOR,—Bowman and coauthors’ demonstrated in their well performed study that there is a marked rise in intraocular pressure (IOP) after peribulbar anaesthesia. They injected a total of 10 ml of the anaesthetic, divided, into two different locations. The pressure could be lowered again with the help of oculopressure.

I have been using peribulbar anaesthesia for intraocular procedures for some years now, injecting 1.5 ml of a mixture of 2% lignocaine and 0.5% bupivacaine in the upper fornix, then 1.5 ml in the lower fornix. The anaesthetic is very good and sufficient even for long and complicated operations. By using these relatively small quantities of local anaesthetics, the IOP rises only slightly, thus preventing complications and making ocular compression unnecessary. I would recommend this technique for most intraocular procedures.

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Author’s reply

EDITOR,—We would certainly concur with Whitefield et al1 that an important reason why patients decline day case surgery is the need for an examination the next day. We would also question this necessity following uncomplicated sutureless phacoemulsification with lens implant. However, we feel it may be unsafe practice if patients are not examined at all in the early postoperative period.

Firstly, intraocular pressure can be raised following cataract surgery,2 and indeed it was in three cases in this study. The maximum pressure rise tends to occur 5–7 hours postoperatively3 and so if patients were examined then many more may well be found to have raised pressure. For most patients this transient ocular hypertension is not associated with significant morbidity, but central retinal artery and venous occlusions and non-arteritic anterior ischaemic optic neuropathy4 have been reported following cataract surgery and this pressure spike may be a significant factor.

It may be better, therefore, to examine the patient on the same day as surgery before discharge. The use of a prophylactic ocular anti-hypertensive agent would also be recommended. An early examination would also allow a significant wound leak to be detected.5 In the study by Tufail et al6 it was concluded that no complications were missed by examining the patients on the same day as surgery that would have been detected the next day.

Iris prolapse and endophthalmitis are both very rare (0.3%)7 and so would not be expected to be detected by a study of this size (100 patients). Often these conditions do not present on the first postoperative day, with endophthalmitis typically presenting 48–72 hours postoperatively.8 A fibrinous uveitis, which may be a warning of impending endophthalmitis, again is often worse a few days after surgery rather than at day 1. It is possible some of these cases could be screened for with a visit 3 days after surgery. It is our practice, however, to ask the patient to return as an emergency if symptoms change, especially increasing pain, rather than seeing all patients at day 3.

Our policy is to examine on the same day as surgery, before discharge, and again at 2 weeks

Reply

EDITOR,—Bowman and coauthors’ demonstrated in their well performed study that there is a marked rise in intraocular pressure (IOP) after peribulbar anaesthesia. They injected a total of 10 ml of the anaesthetic, divided, into two different locations. The pressure could be lowered again with the help of oculopressure.

I have been using peribulbar anaesthesia for intraocular procedures for some years now, injecting 1.5 ml of a mixture of 2% lignocaine and 0.5% bupivacaine in the upper fornix, then 1.5 ml in the lower fornix. The anaesthetic is very good and sufficient even for long and complicated operations. By using these relatively small quantities of local anaesthetics, the IOP rises only slightly, thus preventing complications and making ocular compression unnecessary. I would recommend this technique for most intraocular procedures.

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Author’s reply

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Our policy is to examine on the same day as surgery, before discharge, and again at 2 weeks
and if all is well the patient is discharged for refraction by their optician, as no further intervention is required. If one suture has been used this is removed at this 2 week visit.

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A case of Leber’s hereditary optic neuropathy with elevated blood levels of lactate and pyruvate

EDITOR,—Leber’s hereditary optic neuropathy (LHON) is a maternally inherited disease characterised by acute bilateral loss of central vision. We report a patient with LHON who had elevated lactate and pyruvate levels in blood and cerebrospinal fluid (CSF).

CASE REPORT

A 22-year-old man noticed blurred vision in both eyes. Corrected visual acuity was 0.04 in his right eye and 0.05 in his left. Slit-lamp examination and intraocular pressures were normal. Pupillary light reflex was full in both eyes.

Fundus examination revealed slightly hyperaemic optic discs. There was marked disc palor over the next 2 months. Fluorescein angiography showed hyperfluorescent area over the optic discs. Therewasmarkeddiscpa

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Accelerated ocular hypertensive response to topical steroids in children

EDITOR,—Topical steroids are well recognised to elevate intraocular pressure (IOP) in adults who are sensitive to their effects.1 However, less is known of the steroid response in children.2 We report an accelerated ocular hypertensive response to topical steroids in two siblings.

CASE REPORT

A 6-year-old boy underwent bilateral lateral rectus recession for left divergent squint. The operation was uneventful and no subconjunctival steroid injection was given at the end of the operation. He was discharged with Maxirol (0.1% dexamethasone and neomycin) six times per day. When reviewed on the fourth postoperative day, he complained of photophobia and recurrent vomiting for the previous 2 days. Examination revealed mild ciliary injection with IOP of 44 and 46 mm Hg (measured by the application tonometry) in the right and left eyes, respectively. Gonioscopy showed open angles without any development anomalies, normal iris structure, and contour. The optic nerves were normal. Topical steroid was immediately withdrawn and ocular hypotensives administered. The IOP fell to 15 mm Hg bilaterally the next day and remained within normal limits after all medications were tailed off over the ensuing days. Both the visual acuity and visual field remained normal at the latest follow up, 1 year after the steroid treatment.

COMMENT

There was no family history of glaucoma and both parents were normotensive with normal optic nerves. Steroid provocation tests were initiated but subsequently discontinued at the parents’ demand when they grew alarmed by the positive steroid response in their other child (see below). The parents’ IOP remained normal 3 days into the provocation test when it was aborted. This test was, however, successfully completed in their 3-year-old daughter. It helps to exclude ocular hypertension secondary to squint surgery itself and tests for a familial predisposition to steroid responsiveness. Additionally, we asked if fluorometholone would produce less ocular hypertension in the second child. One eye was randomly assigned to receive Maxitrol while the other received fluorometholone, both six times a day. Investigators were masked as to the treatments given. IOP was measured with a Tono-pen (Men
tor), at 5 to 6 pm. The mean of three readings was recorded. Steroids were planned for withdrawal when the IOP doubled that of the baseline IOP. Just as in her brother, IOP rose rapidly to double the pretreatment value within 48 hours in the Maxitrol treated eye (Table 1). She exhibited a 66% and 33% IOP rise after a single day of instillation of Maxitrol and fluorometholone, respectively. On ceasing steroid treatment, the IOP returned to normal within 5 days. This abbreviated course of IOP elevation and recovery in this otherwise normal child strongly supports steroids as a cause of ocular hypertension in both children.

The cause of this exaggerated steroid response in children remains unknown. The relatively immature chamber angle in young children, which has been suggested to be completely developed only at 8 years, may predispose them to the effects of topical steroids.3 Support for this argument comes from a rabbit model, in which only the younger animals suffered steroid induced glaucoma, while older rabbits were resistant to its effects.4 As the response in young children can be both severe and rapidly in onset, we suggest close monitoring of IOP in the first week of topical steroid treatment under the age of 8. Moreover, with the possibility of strong familial tendency, siblings should also be tested and cautioned accordingly.

Table 1 Ocular hypertensive response to topical steroids in the patient’s 3-year-old sister

<table>
<thead>
<tr>
<th>Day</th>
<th>Maxitrol</th>
<th>Fluorometholone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1*</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>Day 2</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>Day 3</td>
<td>18</td>
<td>18</td>
</tr>
<tr>
<td>Day 4</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td>Day 8</td>
<td>11</td>
<td>11</td>
</tr>
</tbody>
</table>

*Baseline IOP was taken on day 1. †All drugs were withdrawn when the IOP doubled the baseline reading.

Correspondence, Notices

Dennis S C Lam
Alvin K K Kwok
Department of Ophthalmology and Visual Sciences, The Chinese University of Hong Kong, Prince of Wales Hospital, Shatin, Hong Kong

Sejin Chew
Singapore Eye Research Institute, Singapore

Day 1* Day 2 Day 3 Day 4 Day 8

Fluorometholone
Maxitrol

12
12
16
20
24†
16
12

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Feasibility of automated visual field examination in children

EDITOR,—We have read with great interest the recent paper written by Safran et al on the feasibility of automated visual field examination in children between 5 and 8 years of age. It is very important to know that all the children at this age did remarkably well regarding both the duration of the automated visual field examination and the reliability of the answers, using an Octopus 2000R perimeter with a two level strategy. However, a preliminary familiarisation with a specially designed adaptation program was found to be mandatory with children aged 7 years or under.

I would like to point out our good experience with the Humphrey 120 full field screening test in children with previous retinopathy of prematurity at the age of 10 years. There was no need for a preliminary adaptation phase, and this test was found to be reliable and reproducible in all these children. The data are presented in our paper, published in the BJO in 1995.

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YUVAL YASSUR
Department of Ophthalmology, Rabin Medical Center, Beilinson Campus, Petah Tikva, 49100, Israel

NOTES

Glaucoma Society (UK & Eire)
DAVID COLE TRAVEL FELLOWSHIP
The David Cole Travel Fellowship, instituted by Merck Sharp and Dohme in memory of Professor David Cole, will assist a visit to a hospital or research centre during the academic year starting 1 October 1997. The award will be equivalent to £2000. The purpose of the award is to enable the successful applicant to gain experience and knowledge in pursuit of a specific project related to glaucoma.

THE GLAUCOMA SOCIETY (UK & EIRE) RESEARCH GRANT
The Glaucoma Society (UK & Eire) research grant, sponsored by the International Glaucoma Association will be available for a research project clinically orientated to glaucoma for 1997. The award will be equivalent to £2500.

The grant may be used towards salary or project expenses or for buying equipment. These awards are available to both medical graduates and non-medical scientists resident in the United Kingdom or Ireland. They may be held concurrently with other awards.

For further details and application forms please contact: Dr S Nagaiaheman, Secretary, The Glaucoma Society (UK & Eire), Glaucoma Unit, Moorfields Eye Hospital, City Road, London EC1V 2PD.

The closing date for applications is the 31 May 1997. The successful candidate will be informed by August 1997.

Association for Research in Vision and Ophthalmology (ARVO)
The Association for Research in Vision and Ophthalmology (ARVO) is holding its annual meeting on 11–16 May 1997 at the Fort Lauderdale Convention Center, Fort Lauderdale, Florida, USA. Further details: ARVO, 9650 Rockville Pike, Bethesda, MD 20814-3998. (Tel: (301) 571-1844; fax: (301) 571-8313.)

30th Panhellenic Ophthalmological Congress
The 30th Panhellenic Ophthalmological Congress organised by the Hellenic Ophthalmological Society will be held at the Astir Palace Hotel, Vouliagmeni on 28 May to 1 June 1997. Further details: T Kouris, CT Congress, Creta Travel, 19 Amerikis 106 72, 5714448; fax: +49 (0) 7071 293746; email: ingrid.kreissig@uni-tuebingen.de

5th International Symposium on Ocular Circulation and Neovascularisation
The 5th International Symposium on Ocular Circulation and Neovascularisation will be held on 15–19 September 1997 in Kyoto, Japan. Further details: Professor Dr Masanobu Uyama, Secretary General of the Organising Committee, Department of Ophthalmology, kansai Medical University, Moriyuchi, Osaka 570, Japan. (Tel: 81-6-997-3475.)
INSTRUCTIONS FOR AUTHORS

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The British Journal of Ophthalmology is an international journal covering all aspects of clinical ophthalmology and the visual/ophthalmic sciences. Contributors should consider the widely varying readership and write clear, simple articles with the minimum of technical detail. Space in the journal is limited and should therefore be as concise as possible. One page of text is approximately 1000 words.

Manuscripts should be sent to the editor who selects them on the basis of their suitability for the journal and of reports from independent referees. Manuscripts are acknowledged on receipt and the majority (>80%) are sent for review. Those that are not reviewed are returned to the author as rapidly as possible so that they may be submitted elsewhere.

Manuscripts may be processed by section editors who deal with specific areas of ophthalmology including surgical retina, medical retina, neuro-ophthalmology, glaucoma, paediatric ophthalmology, ocular motility, orbital disease, anterior segment disease, oncology, lens, optics and visual sciences, laboratory sciences, pathology, and immunology. A minimum of two referees, chosen for their specific expertise, review each article.

Papers are accepted on the understanding that they have not been and will not be published elsewhere, and that there are no ethical problems with the work described. If requested, authors shall produce the data upon which the manuscript is based for examination by the editor.

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(a) Clinical science
Articles on clinical topics are research reports of a general or specialised nature comprising approximately 3000 words and 4-6 display items (Figures and Tables).

(b) Laboratory science
Articles on ophthalmic or visual sciences are research reports of experimental work generally of the same size as clinical research reports. Laboratory science papers will be included in a designated section of the journal.

Both types of original article should include the following: title; key words (up to four); address and with which author address for correspondence; structured abstract (approx 200 words, headings ‘Aims/background’, ‘Methods’, ‘Results’, and ‘Conclusion’); introduction; materials and methods; results and discussion; sections; references and acknowledgements; legends for display items (Figures and Tables).

REVIEW ARTICLES

Substantive review articles will be included under the section ‘Perspective’ and will address any aspect of clinical or laboratory ophthalmology. Review articles will be approximately 3000-5000 words in length including references and may contain display items (Figures and Tables). Most review articles are commissioned but uninvited reviews are welcomed, and the Editor is recommended. All reviews are subject to independent refereeing.

LETTERS TO THE EDITOR
Case reports will be published as ‘Letters to the editor’. These are normally 500–600 words written in the form of a letter with a maximum of two display items (Figures and Tables). The letter should include an introductory section (without heading), the case report (heading: Case report) and a comment (heading: Comment), plus a maximum of 10 references.

CORRESPONDENCE
Letters are normally constructed in the form of scientific correspondence and are usually 200–300 words.

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Manuscripts will be received on the understanding that they have not been and will not be published elsewhere while under editorial review. Manuscripts may be subject to editorial revision with the author’s agreement. All communications should be sent to the Editor, British Journal of Ophthalmology, Department of Ophthalmology, University of Aberdeen Medical School, Foresterhill, Aberdeen AB25 2ZD, Scotland, UK. (Tel: 01224 663812; Fax: 01224 663832.)

Manuscripts must be submitted in triplicate, and typed double spaced on one side of the paper only, with one inch margins. Each author must sign the covering letter as evidence of consent to publication. Revised manuscripts should be submitted as hard copy and on disk. Detailed instructions will be sent to authors on invitation to revise.

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Recurrent uveitis in a patient with adult onset cyclic neutropenia associated with increased large granular lymphocytes

ALEJANDRO RODRIGUEZ, ROBERT A YOOD, THOMAS J CONDON and C STEPHEN FOSTER

Br J Ophthalmol 1997 81: 415
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