Acute angle closure glaucoma following the use of intranasal cocaine during dacryocystorhinostomy

Many thousands of dacryocystorhinostomies (DCRs) are performed by ophthalmologists routinely without problems. Postoperative pain and nausea may wrongly be attributed to wound pain and post-anaesthetic nausea. Medical and nursing staff need to be aware of the potential for intranasal cocaine to precipitate acute angle closure glaucoma (AACG). We report two cases of AACG following the use of intranasal cocaine and subcutaneous lignocaine (lidocaine) with adrenaline during DCR surgery. We believe this to be the second report of such cases.

Case report

Two women, a 67 year old and a 75 year old, developed right sided AACG immediately after ipsilateral DCR surgery. Both patients were treated successfully for AACG. Cocaine is a known mydriatic and can induce angle closure glaucoma in either patient.

Acute angle closure glaucoma is best avoided by using adrenaline in the local anaesthetic solution with lignocaine and adrenaline 1:200 000. The mydriatic effect of the adrenaline in the local anaesthetic solution provides anaesthesia and haemostasis, making the use of cocaine desirable in lacrimal drainage surgery. Its use in DCR surgery was first described by Dupuy-Dutemps and Bourgener in 1922. We have routinely used cocaine for our DCR surgery, and emphasise the need to remain cognisant of its possible local and systemic side effects. Cocaine is well absorbed from mucosal surfaces, reaching plasma levels similar to those achieved with intravenous administration. In adults, 50–95 mg is a psychotomimetic dose. The maximum allowable dose is 3 mg/kg; 1 g or 10 ml of a 10% solution represents a fatal dose for an adult. Usually, 80–200 mg of cocaine is administered intranasally in DCR surgery. Systemic cocaine toxicity has been reported in patients undergoing DCR surgery under general anaesthesia.

AACG was described in the early 20th century with the use of cocaine as a cornel mydriatic. More recently, a case of AACG associated with ipsilateral intranasal cocaine abuse was reported. In 1999, Hari et al described a single case of AACG following the use of cocaine in DCR surgery. Our two case reports strongly suggest that the use of intranasal cocaine to aid DCR surgery may precipitate AACG.

It is highly probable that the mydriasis which precipitated the attack of AACG was caused by the intranasal cocaine rather than the low concentration of subcutaneous adrenaline (1:200 000). There are two possible mechanisms of cocaine entry into the eye during DCR. Inadvertent transfer of cocaine from the surgeon’s glove to the patient’s conjunctival sac is possible if fresh gloves are not used between cocaine nasal packing and the infiltration of local anaesthetic. Alternatively, residual cocaine in the nasal cavity has direct access to the conjunctival sac after the lacrimal sac anastomosis in the DCR procedure.

Other potential causes of AACG include the mydriatic effect of the adrenaline in the lignocaine 1%, 1:200 000 adrenaline, and atropine given intravenously during general anaesthesia.

At this concentration, subcutaneously administered adrenaline is unlikely to have precipitated the acute angle closure glaucoma. Atropine was not used during anaesthesia in either patient.

Given the apparent rarity of this proposed event, we cannot definitively conclude that lacrimal surgeons should change their approach to patients undergoing DCR surgery. However, it is important to remain cognisant of this potential complication and educate both nursing staff and medical trainees in ophthalmology and otolaryngology as to this potential complication. In addition, it would be reasonable to pad only the wound and not occlude the eye, which may have allowed the origin of the pain in our cases to be more readily identified. Postoperative pain and nausea following DCR surgery should not be automatically attributed to wound pain and post-anaesthetic nausea. A diagnosis of AACG needs to be excluded. It may be prudent to assess routinely anterior chamber depth in high risk eyes before DCR surgery. If there is any suspicion of an eye anatomically predisposed to AACG both nursing and medical staff should be made aware of the potential complication. A prophylactic peripheral iridectomy may be indicated.

G A Wilsec
Ocular Plastics Unit, Prince of Wales Hospital, University of New South Wales, Sydney, Australia

M J Vose
Ocular Plastics Unit, Manchester Royal Eye Hospital, Manchester, UK

I C Francis, S Sharma, M T Coroneo
Ocular Plastics Unit, Prince of Wales Hospital, University of New South Wales, Sydney, Australia

Correspondence to: Professor Minas T Coroneo, Ocular Plastics Unit, Eye Clinic, Level 4, Prince of Wales Hospital, High St, Randwick, NSW, 2031, Australia; m.coroneo@unsw.edu.au

Accepted for publication 10 April 2002

References


Presumed hypersensitivity to minocycline and conjunctival infiltration

Minocycline is a widely prescribed systemic antibiotic for acne. Its mechanism of action is complex and not only antimicrobial in nature. Other properties of minocycline include decreased chemotaxis of polymorphonuclear leukocytes, a modification of the complement pathways, an inhibition of the polymorphonuclear leukocyte chemotactic factor, and inhibition of lipase production in Propionibacterium acnes. In ophthalmology, minocycline is used for the treatment of rosacea. Although commonly considered to be a safe drug, there have been an increasing number of reports regarding systemic adverse reactions to minocycline during the past few years. Adverse effects to minocycline range from minor allergic reactions to death. The only ocular side effects reported to date have been cases of presumed minocycline induced scleral pigmentation. We report a case of conjunctival infiltrates, asthma exacerbation, and hypereosinophilia associated with the concurrent use of minocycline.

Case report
A 28 year old woman received oral minocycline 100 mg once daily, for 29 days for the treatment of acne, without any other topical or systemic medication. She had a history of multiple allergies and asthma since early childhood, but no ocular history. During the first day of treatment, she developed asthena, a mild fever of 38°C, and severe asthma. She was hospitalised. Thirty days after the beginning of minocycline treatment, she reported redness and burning of both eyes. Visual acuity was 20/20 in each eye. Several slightly elevated intracorneal nodules were seen in both eyes at the 12 o’clock position, with injection of the superficial and deep episcleral vessels (Fig 1). The tear film and the cornal epithelium were normal. There was no inflammation of the anterior chamber or of the posterior segment. Laboratory tests showed a severe eosinophilia: 2.12 × 10^9/l and 4.0 × 10^9/l at 15 and 30 days, respectively, after the onset of oral minocycline. Treatment of the patient’s asthma included systemic prednisone initiated at 0.5 mg/kg/day. Cessation of minocycline led to rapid remission of asthma. Despite no topical treatment for her ocular findings, the conjunctival infiltration disappeared, and the ocular symptoms resolved. Laboratory tests showed a rapid decrease of the eosinophilia: 1.06 × 10^9/l and 0.7 × 10^9/l at 3 and 9 days, respectively, after cessation of minocycline.

A conjunctival biopsy was performed 39 days after the onset of minocycline treatment. Histopathology showed no modifications of the conjunctival epithelium, but oedema of the stroma. Vessels were surrounded by mononuclear lymphocytes and eosinophils. May-Grumwald-Giemsa staining confirmed eosinophilic polymonuclear cells. Such inflammatory aggregates were also seen between vessels but not inside the vessel lumens (Fig 2).

Comment
Reports of adverse effects of minocycline include both hypersensitivity reactions and autoimmune disorders. Hypersensitivity reactions usually occur within a few weeks after the onset of treatment and may lead to exfoliative dermatitis, eosinophilic pneumopathy, pericarditis, nephropathy, lymphadenopathy, pseudoinfectious reactions, and blood eosinophilia (that is >0.6 × 10^9/l). Autoimmune disorders usually present after exposure to minocycline for 1 year or more and include autoimmune hepatitis, lupus, and vasculitis. Pigmentations of the skin, fingernails, bones, and teeth have also been described in relation to the use of minocycline. Seven cases of scleral pigmentation presumed to have been induced by oral minocycline treatment have been reported.

Exacerbation of asthma and eosinophilia similar to our observation have been previously reported. The definite temporal association with conjunctival infiltration strongly suggested that minocycline therapy was the causative agent in our case. The patient’s clinical findings did not show any abnormality of the superficial corneal epithelium near the limbus or any tear film abnormalities, as observed in a superior limbus keratoconjunctivitis-type reaction. Neither the transient nature of asthma and eosinophilia, nor the biopsy specimen was suggestive of Chung-Stauss disease in our patient. Different ocular manifestations, such as splinter haemorrhages and arterial emboli have been reported in the hyper eosinophilic syndrome (eosinophilic counts greater than 1.5 × 10^9/l), linked to chorioternal thromboembolic disease. No thromboembolic manifestations were observed in the conjunctiva of our patient.

To our knowledge, our observation is the first report of a drug induced eosinophilic conjunctival infiltration.

C Parc, A P Brézin, I Nataf
Department of Ophthalmology, Cochin University Hospital, Paris, France

D Dusser
Department of Pneumology, Cochin University Hospital, Paris, France

L Moachon
Department of Pharmacology, Cochin University Hospital, Paris, France
Importance of molecular testing in dominant optic atrophy

Juvenile onset dominant optic atrophy (DOA) is the most common inherited optic atrophy with a variable prevalence of between 1 in 10 000 (Denmark) and 1 in 50 000. The majority of cases have been shown to have mutations in the OPA1 gene on chromosome 3. Reduced visual acuity of insidious onset, temporal pallor of the optic disc, centrocaecal scotoma, and generalised dyschromatopsia are the key clinical features. Recent studies, however, have shown that penetrance within families is much lower than first realised and the assignment of status using clinical criteria alone can result in misdiagnosis. This is well illustrated by the following family.

Fifteen family members were asked about perceived visual difficulties before measurement of visual acuity, colour vision (City University Test), and visual field analysis (Humphry 24-2). Fundal examination and photography were performed. Blood was taken for screening of the OPA1 coding region using single stranded polymorphism analysis (SSCP) and automated DNA sequencing as previously described.

Seven family members were diagnosed as having optic atrophy from clinical tests alone (Fig 1). Where visual disability was recognised the age of onset ranged from 5.5 to 20 years (Table 1). Eight family members were classed as affected from molecular testing. All individuals exhibited an abnormal banding pattern on SSCP analysis of exon 27 (Fig 2). Sequencing revealed a four base pair deletion of TTAG at the start of exon 27 (nt2708-2704). This deletion has been described previously and causes a frameshift resulting in two amino acid substitutions (VA903Gl, Arg904Asp) and a premature stop codon.

Dominant optic atrophy may be the result of mutations in genes other than OPA1. However, where DNA linkage analysis or mutation screening implicates the OPA1 gene we feel that all family members should be offered molecular testing. In DOA it is well recognised that the clinical manifestation of the same mutation within a family can be very variable. This is well illustrated in Table 1. Genetic screening is the only reliable way of identifying individuals at risk of passing on the disease. The combination of SSCP and DNA sequencing resulted in an alteration of the status of individual II:7 from unaffected to affected. If clinical tests alone had been relied upon, this individual would have most likely received incorrect genetic advice.

Table 1

<table>
<thead>
<tr>
<th>Individual</th>
<th>Age (years)</th>
<th>Age of onset (years)</th>
<th>Snellen visual acuity</th>
<th>Centrocaecal field defect</th>
<th>City University binocular score</th>
<th>Temporal pallor</th>
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<tr>
<td>I:1*</td>
<td>73</td>
<td>–</td>
<td>6/18 6/18 N N</td>
<td>Tritan</td>
<td>Y Y</td>
<td></td>
</tr>
<tr>
<td>II:3*</td>
<td>50</td>
<td>14</td>
<td>6/12 6/18 Y Y</td>
<td>Tritan</td>
<td>Y Y</td>
<td></td>
</tr>
<tr>
<td>II:5*</td>
<td>49</td>
<td>20</td>
<td>6/12 6/36 Y Y</td>
<td>Tritan</td>
<td>Y Y</td>
<td></td>
</tr>
<tr>
<td>II:7</td>
<td>48</td>
<td>–</td>
<td>6/6 6/6 N N</td>
<td>Normal</td>
<td>N N</td>
<td></td>
</tr>
<tr>
<td>II:8</td>
<td>50</td>
<td>–</td>
<td>6/6 6/6 N N</td>
<td>Normal</td>
<td>N N</td>
<td></td>
</tr>
<tr>
<td>III:2*</td>
<td>28</td>
<td>–</td>
<td>6/18 6/18 Y Y</td>
<td>Generalised</td>
<td>Y Y</td>
<td></td>
</tr>
<tr>
<td>III:3*</td>
<td>23</td>
<td>7</td>
<td>6/36 6/18 Y Y</td>
<td>Generalised</td>
<td>Y Y</td>
<td></td>
</tr>
<tr>
<td>IV:3*</td>
<td>19</td>
<td>12</td>
<td>6/18 6/12 Y Y</td>
<td>Normal</td>
<td>Y Y</td>
<td></td>
</tr>
<tr>
<td>IV:7*</td>
<td>6</td>
<td>5.5</td>
<td>6/12 6/9 N N</td>
<td>Generalised</td>
<td>Y Y</td>
<td></td>
</tr>
</tbody>
</table>

Y = Yes; N = No. *Classified as affected from clinical tests alone.
A 64 year old man presented to the corneal service complaining of glare at night which had worsened over a 12 month period. He was found to have a pancorneal epithelial crystalline keratopathy with normal Snellen acuity (0.49 g/24 hours), a hypercellular bone marrow aspirate (70–80% plasma cells), and a single lytic bone lesion in the left iliac crest.

He was entered into the MRC Myeloma Trial (Myeloma VII) and was randomised to receive ABCM (adriamycin, BCNU, cyclophosphamide, melphalan). However, his renal function rapidly deteriorated (creatinine 714) and despite the use of weekly cyclophosphamide his renal function deteriorated and required haemodialysis. A month later a decerebration followed when the dose was increased to 100 mg daily; however, a peripheral neuropathy developed and the thalidomide reduced to 50 mg on alternate days and latterly to 50 mg twice weekly. Within 7 months of starting the thalidomide, the paraprotein had completely disappeared and the crystalline keratopathy had resolved (Fig IC, D). Visual acuity at this time was 6/9 right eye and 6/18 left eye, due now in large part to the presence of early posterior subcapsular lens opacity.

**Comment**

Corneal crystalline deposits may occur in cystinosis, multiple myeloma, and other monoclonal gammapathies, infective crystalline keratopathy, Schöny’s central crystalline dystrophy, Bietti’s marginal crystalline dystrophy, gout, chronic renal disease, lipid keratopathy, and chrysisia.1

Crystalline keratopathy occurs only rarely in association with multiple myeloma and monoclonal gammapathies.1 2 With the exception of Waldenstrom’s macroglobulinaemia, the crystals are composed of IgG light chains (usually κ but occasionally λ). The crystals may be deposited in the epithelium, as in the case we present, where they have been reported to lie both between and within the epithelial cells,1-3 and also within the cells of the corneal stroma,4 5 conjunctiva, and lens.6-7

It is not known why crystallisation occurs so infrequently in these conditions or why clinical appearances are so diverse, but factors including the structure, chemical properties and concentration of the paraprotein or its metabolites,6 and the local tissue environment are likely determinants. The effect of concentration cannot be disputed as the crystals tend to disappear as the paraprotein concentrations fall; however, the rarity of these corneal crystalline depositions suggests that the chemical and tertiary structures of the proteins or their metabolites is critically important.

Although it is reported that myeloma associated crystalline keratopathy can disappear in response to chemotherapy8 9 and plasma exchange there are no previous reports of resolution following the use of thalidomide in refractory disease.

The mechanisms of action of thalidomide are complex and poorly defined but include immunomodulatory and anti-inflammatory effects, the modulation of TNFα, T cell proliferation, increased production of IL2 and IFN-γ and modulation of WBC migration and chemotaxis. The drug also has important anti-angiogenic effects which may be related to modulation of TNFα or a direct inhibitory effect upon angioproliferation. Thalidomide is also sedative and hypnotic and its use for these latter effects gained infamy in the 1960s.

Used as single treatment for refractory or relapsed myeloma thalidomide represents one of the most significant advances in myeloma management in the past 20 years. Around a third to two thirds of patients are said to respond to thalidomide, typically within 2 months. The response is marked by a significant (25% to a least 90%) reduction in paraprotein and Bence-Jones proteins with some,
Iris coloboma and a microdeletion of chromosome 22: del(22)(q11.22)

A 22q11 microdeletion is described in a girl with ocular coloboma associated with velocardiofacial syndrome (VCFS). Microdeletion syndromes are a heterogeneous group of disorders caused by deletion of specific regions of chromosomal DNA that are not visible using standard chromosome analysis. Microscopic chromosome deletions are being recognised increasingly as a cause of congenital abnormality and clinical genetic syndromes. Laboratory confirmation of these conditions requires specialist molecular testing that must be requested on the basis of clinical suspicion. Del(22)(q11.22) microdeletion is among the most common of the microdeletion syndromes and is characterised by cardiac malformations (particularly outflow tract), craniofacial features, cleft palate, thymic hypoplasia, and hypoparathyroidism. The severity of the condition can be very variable and is now recognised as the basis of several independently described syndromes: DiGeorge syndrome (DGS), conotruncal face anomaly syndrome, and velocardiofacial syndrome (Shprintzen syndrome).1 The “velo” in VCFS refers to the palatal abnormalities such as cleft palate that are sometimes present. The characteristic facial appearance of del(22)(q11.22) is a prominent nose, broad nasal root and bridge, with prominent eyes and mouth.

Figure 1 Bilateral iris coloboma with clinical microphthalmos in a girl with velocardiofacial syndrome. Note the upslanting and narrow palpebral fissures, wide nasal root and bridge, with prominent nose, and small mouth.

Iris coloboma and a microdeletion of chromosome 22: del(22)(q11.22) anomalies of which iris coloboma and anal atresia are the best known, is characterised cytogenetically by an extra bisatellited marker chromosome.2 This chromosome represents an inverted duplication of part of chromosome 22 (inv dup(22)). The CES critical region overlaps with the proximal area of chromosome 22q2 deleted in VCFs or DGS. Clearly, this region of chromosome 22 is significant in the aetiology of some ocular colobomas. Ophthalmologists should be aware of these syndromes and the importance of recognising that eye anomalies such as iris coloboma might be the presenting feature of a systemic developmental disorder. Microdeletion syndromes should be considered even in the presence of a normal macroscopic chromosome report.

Figure 2 Second and third toe syndactyly in a girl with velocardiofacial syndrome and del(22)(q11.22).

References

Amniotic membrane transplantation for necrotising conjunctival ulceration following subconjunctival atropine injection

Conjunctival necrosis has been previously reported after subconjunctival injection of several antimicrobial agents and corticosteroids.3 Atropine is used widely as a form of eyedrops for mydriasis, cyclopia, and pain and inflammation control. We
describe a patient who developed severe conjunctival necrosis associated with scleral melting after subconjunctival atropine injection and which was treated by amniotic membrane transplantation.

**Case report**

A 30 year old male patient visited our clinic because of severe pain and redness in his left eye for a day. He had recently been diagnosed with uveitis associated with circular posterior synchiae in his left eye, and which was managed by his previous physician with a subconjunctival injection of 0.4 ml of atropine (10 mg/ml, atropine sulphate, powder dissolved in saline, Mallinkrodt, St Louis, MO, USA) to lyse the posterior synchiae. Upon consultation at our institute, he was found to have marked conjunctival injection and chemosis all around the limbus. Three days after the atropine injection, large areas of necrosis developed around the whole limbus (Fig 1). White necrotic material was noted on the underlying sclera and episclera. The underlying sclera in the inferotemporal bulbar conjunctiva showed a considerable area of melting (2 x 3 mm).

We decided to manage the defect by amniotic membrane transplantation to the sclera because of the large extent of the defect and the scleral involvement. Debridement of the necrotic conjunctiva and episclera was done and the defect was covered with an amniotic membrane graft, which was obtained from women undergoing elective caesarean section and preserved at −70°C. The amniotic membrane was placed basement membrane side up and the excess membrane was trimmed. The edges of them were sutured to the borders of the conjunctiva and the limbus with 10-0 nylon. Topical 0.3% ofloxacin and 1% prednisolone acetate were prescribed every 2 hours postoperatively.

For the next week, the patient improved steadily, having less pain and redness. After a month, the necrotised defect was re-epithelialised completely without inflammation, scar, or symblepharon formation (Fig 2). The eye drops were tapered to four times daily for 2 weeks, twice daily for more 2 weeks, and then stopped.

**Comment**

Subconjunctival atropine has been a useful management for the equine ophthalmic disorder including equine recurrent uveitis, many ulcerative keratopathies, and presurgical and postsurgical cataract extraction. In humans, however, the procedure of subconjunctival injection with atropine has been used infrequently. Our case shows that subconjunctivally delivered atropine should be approached with some degree of caution in humans. The necrotising conjunctival ulceration in this case covered a large area and was also associated with scleral melting. It was rapidly healed after amniotic membrane transplantation without scleral perforation or symblepharon formation. Transplanted amniotic membrane has been known to provide a thick basement membrane to facilitate epithelial migration, play a part in decreasing inflammatory and protease activity, and function as an anatomic barrier, keeping the adhesive surfaces apart. In our case, some combination of the mentioned characteristics would work for treating a severe necrotising conjunctival ulceration with the potential risk of symblepharon and perforation.

K Y Seo, C Y Kim, J H Lee, J B Lee, E K Kim

Institute of Vision Research, Department of Ophthalmology, Yonsei University College of Medicine, Seoul, Korea

Correspondence to: Eung Kweon Kim, MD, Institute of Vision Research, Department of Ophthalmology, Brain Korea 21 Project, Division of Medical Sciences, Yonsei University College of Medicine, 134 Shinchon-dong, Sodaeum-gu, Seoul, Korea, 120-752, eungkkim@yumc.yonsei.ac.kr

Accepted for publication 8 May 2002

**References**


**Traumatic enucleation with chiasmal damage: magnetic resonance image findings and response to steroids**

Gouging has been reported throughout history and this case demonstrates it is still encountered in modern times. Its impact on visual function extends beyond the loss of an eye because of concomitant psychological morbidity.

**Case report**

We present a case report of a patient who suffered a traumatic enucleation. Our patient is a 24 year old security manager who, while trying to prevent youths from harassing...
The incidence of traumatic enucleation reported by Erie and colleagues after performing a population based study in Minnesota from 1956–88 was 12 per 100 000 but only one of these was due to an assault.9

In our case the attacker aimed specifically for the victim's eyes. Bukhanovskiy and colleagues assessed the mental status of 10 eye gougers and found that four of these attackers were psychotic at the time of eye gouging.6 It is important to look not only at the psychology of the attacker but also the psychological impact on the victim. Our patient went to the psychiatry team weekly for counselling following the trauma.

Suzuki and colleagues reported that if the optic nerve transected is 4 cm or more in length than intracranial complications are likely. These include contralateral visual field loss (which was seen in our case) hypothalamic involvement, subarachnoid haemorrhage, and cerebrospinal fluid leakage.6

The use of steroids in traumatic optic neuropathy remains controversial.5 It is thought to restore altered vascular permeability and decreases the swelling of the optic nerve. We saw a marked improvement in the visual field which may be attributed to the steroids.

Comment

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B Parmar, B Edmunds, G Plant
Eye Department, St Thomas's Hospital, Lambeth Palace Road, London SE1 7NH, UK

Correspondence to: B Parmar; parmabina@hotmail.com

Accepted for publication 13 May 2002

References


Cyclodiode laser therapy to control intraocular pressure during pregnancy

Controlling intraocular pressure (IOP) during pregnancy can be problematic because currently available topical ocular hypotensive agents are contraindicated in pregnancy. We report the case of a patient with aphakic glaucoma controlled with topical agents, for whom we performed cyclodiode laser therapy, since she wanted to become pregnant. The use of cyclodiode laser therapy for this indication has not been described previously.

Case report

A 30 year old woman was referred to our specialist glaucoma clinic with a diagnosis of aphakic glaucoma in her right eye. She had a past ophthalmic history of chronic uveitis secondary to juvenile chronic arthritis, complicated right cataract surgery and subsequent aphakic glaucoma.

At the time of referral her visual acuities were 6/36 in her right aphakic eye and 6/5 in the left eye. She had an IOP of 19 mm Hg in her right eye and significant glaucomatous disc cupping (cup:disc ratio 0.85). Her right IOP was controlled with dorzolamide eye drops 2%, carteolol eye drops 2%, both twice a day, and latanoprost eye drops 0.005% at night. Our patient wanted to become pregnant but was justifiably concerned that the topical medication she was using might affect her pregnancy. It was decided that we would perform cyclodiode laser therapy on her right eye, under peribulbar anaesthesia, and stop her topical antiglaucomatous therapy. The first session of cyclodiode (1500 mW×1500 ms×40 shots over 360° with 5 and 9 o'clock sparing) was unsuccessful and 6 weeks later her IOP was 33 mm Hg on no ocular hypotensive therapy. The second session of cyclodiode therapy (2000 mW×2000 ms×40 shots) was partially successful, the IOP being 22 mm Hg 6 weeks later, although this had risen to 28 mm Hg by 4 months. By this time our patient had become pregnant and it was felt that the IOP was still too high. A third attempt at lowering IOP by cyclodiode (2000 mW×2000 ms×40 shots over 360°) was made, following which the IOP was persistently well controlled at 12–14 mm Hg.

Comment

The use of cyclodiode laser therapy to control IOP in women before and during pregnancy has not been described. It seems a rational choice in treating these patients since the use of topical antiglaucomatous therapy is either contraindicated or cautioned in pregnancy.1 β-blockers, sympathomimetics, carbonic anhydrase inhibitors, parasympathomimetics, apraclonidine, and prostaglandin analogues have all been shown to have adverse effects in animal fetal models.4–8 Understandably, there have been no equivalent studies in the human fetus and resulting teratogenesis in laboratory animals cannot be extrapolated to humans.9 However, some specific lower effects related to specific topical medications have been quoted in the literature.4–8 Topical timolol has been shown to cause fetal cardiac arrhythmia and bradycardia.4 These have been one reported case that correlates the use of acetazolamide in early pregnancy with sacrococcygeal teratoma in the neonate.7 Prostaglandins are involved in the physiology of pregnancy and thus the use of latanoprost is not advised during pregnancy.10

Figure 2 Arrow A shows high signal left hemichiasm and arrow B shows normal right hemichiasm.

Figure 3 A left temporal hemianopia after injury (left) and the repeat Goldmann perimetry following pulsed methylprednisolone (right).
Pregnancy is usually a period of lower IOP and less therapy may be required than before gestation, although that was not the case with our patient. It could be argued that optic nerve damage over a 9 month period would be minimal such that a mildly raised IOP could be tolerated for the length of pregnancy. However, when glaucomatous damage is severe, it is generally accepted that a low target IOP should be attained.

There is a paucity of literature describing the use of local anesthetics for ophthalmic procedures during pregnancy. However, in a large multicentre retrospective study, exposure to local anesthetic during early pregnancy was associated with no increased incidence of fetal malformation.3 One patient could have been offered augmented filtration or tube implantation surgery but cycloide was felt to be less invasive and, ideally, exposure to antimetabolites should be avoided in pregnancy.

Cycloide is a safe and effective treatment to control IOP before and during pregnancy. The risk of topical medication to the fetus is eliminated and more complex surgical interventions are avoided.

References


Choroidal ischaemic infarction following ocular contusion with small framed spectacles: Hutchinson-Siegrist-Neubauer syndrome

We read with interest the article by Clarke et al.1 The authors describe their findings in a 79 year old aphasic woman with a so called non-penetrating injury caused by compression with small framed spectacles. This type of injury is defined to be a contusion/closed globe injury.2 The authors presented a fundus photograph that they interpreted as a “choroidal rupture following blunt injury to the globe.” In the superior hemisphere, there was a large temporally localized area with hypopigmentation and malperfusion of the choroid, corresponding to the segmental structure of the choroidal vasculature.3 However, the findings shown by the authors are more indicative of a segmental choroidal infarction (Hutchinson-Siegrist-Neubauer syndrome).4 A choroidal rupture type presentation as an arched choroidal defect at the posterior pole following indirect trauma or with other well defined defects in case of direct trauma. We found in all of our patients (7% of all severe contusions) with choroidal rupture a circumscripted and/or sicken shaped configuration.

By contrast, this clinical picture of a segmental traumatic choroidal infarction was first described by Hutchinson in 1889.5 Siegrist confirmed this new disease in 1895.6 Neubauer observed in three patients the typical segmental choroidal atrophy with corresponding visual field defects.7 Lang and associates explained this severe complication following ocular contusion as follows: as a result of Bell’s phenomenon and excessive eye squeezing, the globe is rotated upwards and moves backwards into the orbit between 0.7 to 1.7 mm. An occlusion or rupture of short ciliary arteries may result caused by sudden retroplacement of the globe into the orbit, which leads to the typical segmental choroidal infarction of the corresponding vascular choroidal segments.8 We found Hutchinson-Siegrist-Neubauer syndrome in 1% of all contusions of the Erlangen Ocular Contusion Registry.9

Our theory is supported with the recommendation of Vinger, who discussed the need for appropriate protective eyewear for all patients who had undergone intraocular surgery, especially in high risk patients—that is, after ICCE surgery.

A Viestenz, M Küche
Department of Ophthalmology, University Erlangen-Nürnberg, Schwabachanlage 6, D91054 Erlangen, Germany
Correspondence to: Arne Viestenz, MD, Department of Ophthalmology, University Erlangen-Nürnberg, Schwabachanlage 6, D91054 Erlangen, Germany, Arne.Viestenz@online.de

References


Anti-Acanthamoeba efficacy in contact lens disinfecting systems

Hiti et al produced an interesting study investigating the susceptibility of three strains of Acanthamoeba to a PHMB (polychlorhexamethylen biguanide) based multipurpose solution (MPS), a one step peroxide system (3% H2O2), and a two step peroxide system (0.6% H2O2). However, no comment was made regarding...
the choice of strains tested, only one of which
(A castellanii 4CL) was typical of those that
cause keratitis in humans. This isolate belongs
genotype T4, the predominant genotype in
Acanthamoeba to genotype T4, the predominant genotype in
cause keratitis in humans. This isolate belongs
genotype T5, A lenticulata
tions is rapidly neutralised.
shown to be less effective than two step
reduced effect occurs with 0.0001% PHMB.
A hatchetti
dilution (0.3% or 3000 µg/ml) had lost its
trophicidal at 8 hours exposure, but not as a 1:2
dilution (0.3% or 3000 µg/ml) had lost its
cysticidal effect.
The MPS tested was designed for use with
rigid gas permeable lenses and contained
PHMB at a concentration of 0.0005% (5 ppm
or µg/ml). This is higher than that found in
the majority of soft lens MPS, which contain
0.0001% PHMB. As the minimum tropho-
cidal concentration (MTC) of PHMB is 1 µg/ml and the minimum cysticidal
mCC (3 µg/ml), the solution has performed as expected against the cli-
cial strain. Also, a recent study by
our group testing the efficacy of a variety of
MPS for soft lenses, including one containing
0.0005% PHMB, found this concentration to be
cysticidal after the manufacturer’s recom-
manded disinfection time of 4 hours. A reduced effect occurs with 0.0001% PHMB.
One step peroxide systems have been shown to be less effective than two step
systems, as the peroxide in the one step solu-
tion was rapidly neutralised. Therefore, again,
the results for the one step peroxide system
were as expected. In addition, a recent cohort
study of CL wearers in Hong Kong has shown
that lens storage cases were more likely to
yield “no bacterial growth” and less likely to
yield “heavy bacterial growth” if MPS were
used in preference to one step peroxide
solutions.
The results for all three solutions when
tested against clinically relevant strains of
Acanthamoeba were as expected. The authors
could have recommended the two step peroxide system (0.6% H₂O₂) for overnight
storage or contact lenses as an effective dis-
infactant against Acanthamoeba. However, anti-
microbial efficacy is not the only factor that
should be considered when advising a CL
 wearer about a disinfecting solution. Thought
must be given to ease of use and personal
compliance. It has been suggested that
the introduction of MPS has led to a reduced
incidence of the infection since 1995. Although
may not be as cysticidal as a two step
peroxide system, compliance by the CL wearer
is higher. In addition, the MPS kills bacteria and fungi within lens cases which reduces
growth factors for Acanthamoeba.

Technology for Vision 2000
The latest issue of Community Eye Health (No 42) focuses on technology and training, with
an editorial by Catherine Cross, chairperson of the International Agency for the Prevention of
Blindness (IAPB). For further information please contact: Journal of Community Eye Health, International Centre for Eye Health, Institute of Ophthalmology, 11–43 Bath Street, London EC1V 9EL, UK (tel: +44 (0)20 7608 6910; fax: +44 (0)20 7250 3207; email: eyeresource@ucl.ac.uk; website: www.jcch.co.uk), Annual subscription (4 is-
issues) £525/$640. Free to workers in developing
countries.

International Centre for Eye Health
The International Centre for Eye Health has published a new edition of the Standard List of
Medicines, Equipment, Instruments and Optical Supplies (2001) for eye care services
in developing countries. It is compiled by the Task Force of the International Agency for
the Prevention of Blindness. Further details: Sue Stevens, International Centre for Eye
Health, 11–43 Bath Street, London EC1V 9EL, UK (tel: +44 (0)20 7608 6910; email:
eyeresource@ucl.ac.uk).

Specific Eye Conditions (SPECS)
Specific Eye Conditions (SPECS) is a not for
profit organisation which acts as an umbrella
organisation for support groups of any condi-
tions or syndrome with an integral eye disor-
der. SPECS represents over fifty different
organisations related to eye disorders ranging
from conditions that are relatively common to
very rare syndromes. We also include groups
who offer support of a more general nature to
visually impaired and blind people. Support
groups meet regularly in the Boardroom at
Moorfields Eye Hospital to offer support to
each other, share experiences and explore new
ways of working together. The web site
www.secondsight.org.uk acts as a portal
providing direct access to support groups own
sites. The SPECS web page is a valuable
resource for professionals and may also be of
terest to people with a visual impairment or
who are blind. For further details about
SPECS contact: Kay Parkinson, SPECS Devel-
opment Officer (tel: +44 (0)1803 524238;
email: k@endowconitions.org.uk or
by contacting Dr Lucy Mathen
(lucymathen@yahoo.com).

The British Retinitis Pigmentosa Society
The British Retinitis Pigmentosa Society (BRPS) was formed in 1975 for visually impaired
people with retinitis pigmentosa and their
families. The principle aims of BRPS are
to raise funds to support the programme of
medical research into an eventual cure for this
hereditary disease, and through the BRPS
welfare service, help members and their
families copy with the everyday concerns caused
by retinitis pigmentosa. Part of the welfare
service is the telephone helpline (+44 (0)1280 860 363), which is a useful resource
for any queries or worries relating to the problems retinitis pigmentosa can bring. This service is especially valuable for those recently diagnosed with retinitis pigmentosa, and all calls are taken in the strictest confidence. Many people with retinitis pigmentosa have found the Society helpful, providing encouragement, and support through the Helpline, the welfare network and the BRPS branches throughout the UK. (tel: +44 (0)1280 821 334; email: lynda@brps.demon.co.uk; website: www.brps.demon.co.uk.)

Introductory Course in Osteo-odontokeratoprosthesis (OOKP)
The University of Brighton Postgraduate Medical School is holding an introductory course in osteo-odontokeratoprosthesis (OOKP) 20–21 November 2002 in the New Seminar Room, Sussex House, Brighton & Sussex University Hospitals Trust in Brighton. The course will comprise of a variety of lectures with live surgery, two way audio and video links (Stage 1 and Stage 2 OOKP surgery), and examination of patients. Further details: Mrs Erica Strange, University of Brighton, Postgraduate Medical School, Falmer Campus, Brighton, East Sussex BN1 9PH, UK (tel: +44 (0)1273 644 005; fax: +44 (0)1273 644 002; email: e.strange@brighton.ac.uk).

23rd Annual Conference and Dinner Glaucoma Society (UK & EIRE)
The 23rd Annual Conference and Dinner of the Glaucoma Society will be held on Thursday 21 November 2002, 8.30am to 5.00pm at The Royal College of Surgeons, London. The Annual Dinner is from 6.30pm to 10.00pm at The Annual Dinner. (Maximum number of places 250—apply now to secure your place). Further details: Janet Flowers, Administrator, 29 Quarry Hill, Grays, Essex, RM17 5BT, UK (tel/fax: 01375 383172; email: glausoc@ukeire.freeserve.co.uk).

Retinal Detachment Course with international faculty and case presentations preceding Vitrektomie-Kurs—Wetlab
The Retinal Detachment Course with international faculty and case presentations and Vitrektomie-Kurs—Wetlab will be held 13 February 2003 (in English) and 14–15 February 2003 (in German) respectively, at Verwal­tungsga­be­ae­ule der KA Rudolf­stiftung, 1030 Vienna, Boehr­han­ge­v­as­sa­eg 8a, Austria. Further details and registration: Firma Askin & Co, Albert-Schweritzer-Gasse 6, A-1140 Vienna, Austria (tel: +43 (1) 979 88 44; fax: +43 (1) 979 88 46).

Detachment Course with international faculty on: Retinal and Vitreous Surgery with Case Presentations preceding Retina Meeting
The detachment course with international faculty on: Retinal and Vitreous Surgery with Case Presentations and the Retina Meeting will be held 14–15 March 2003 and 16 March 2003 respectively, in Mexico City, Mexico. Further details: Scientific programme: Prof Ingrid Kreissig, University of Tuebingen, Schleichstr. 12, Breuningerbau, 72076 Tuebingen, Germany (tel: +49 7071 295209; email: ingrid.kreissig@med.uni-tuebingen.de). Local organisation: Dr Arman Masheyekhi, Dr Sia­mack Moradian, Dept of Ophthalmology, Lab­banlinejad Medical Center, Pasdaran Ave, Boodan 9, Tehran, 16666, Iran (fax: +98 21 254 9039; email: labbali@hotmail.com).

16th Annual Meeting of German Ophthalmic Surgeons
The 16th Annual Meeting of German Ophthalmic Surgeons will be held 8–11 May 2003 in Nürnberg, Germany, Messe­zentrum. Or­ga­nised by the Professional Association of German Ophthalmologists Ophthalmic Surgery Group the conference will cover cataract surgery, refractive surgery, glaucoma surgery, vitreoretinal surgery, corneal surgery, eye surgery in developing countries, and orbita, lacrimal and lid surgery. Further details: MCN Medizinische Congress organisation Nürnberg AG, Zerzabelhofstr 29, 90478 Nürnberg, Germany (tel: +49 911 3931621; fax: +49 911 3931620; email: doc@mcnag.info; website: www.doc-nuernberg.de).

Vitrektomie-Kurs—Wetlab
The Retinal Detachment Course with international faculty and case presentations and Vitrektomie-Kurs—Wetlab will be held 13 February 2003 (in English) and 14–15 February 2003 (in German) respectively, at Verwal­tungsga­be­ae­ule der KA Rudolf­stiftung, 1030 Vienna, Boehr­han­ge­v­as­sa­eg 8a, Austria. Further details and registration: Firma Askin & Co, Albert-Schweritzer-Gasse 6, A-1140 Vienna, Austria (tel: +43 (1) 979 88 44; fax: +43 (1) 979 88 46).
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T K Beattie, A Tomlinson and D V Seal

Br J Ophthalmol 2002 86: 1319-1320
doi: 10.1136/bjo.86.11.1319-b

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