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-anesthetic 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 predisposed individuals. 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 psychoactive 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 corneal anaesthetic. 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-anesthetic nausea. 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.

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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 leucocytes, a modification of the complement pathways, an inhibition of the polymorphonuclear leucocyte chemoattractant 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 eosinophilia 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 days 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 intraconjunctival 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 corneal 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 with mononuclear lymphocytes and eosinophils. May-Grunwald-Giemsa staining confirmed eosinophilic polynuclear 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 limbic keratoconjunctivitis-type reaction. Neither the transient nature of asthma and eosinophilia, nor the biopsy specimen was suggestive of Churg-Strauss disease in our patient. Different ocular manifestations, such as splinter haemorrhages and arterial emboli have been reported in the hypereosinophilic syndrome (eosinophilic counts greater than 1.5 × 10^9/l), linked to chorioretinal 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.

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Figure 1 Slit lamp view of the conjunctival nodules with injection of the superficial and deep episcleral vessels.

Figure 2 (A) Histopathological examination showed a normal conjunctival epithelium with oedema of the substantial propria. Dilated capillaries were surrounded by mononucleated inflammatory cells (haematoxylin and eosin, original magnification ×10). (B) Histopathological examination showed mononucleated inflammatory cells surrounding a vessel in the substantial propria and eosinophilic infiltrates (haematoxylin and eosin, original magnification ×40).

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**Important 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 percieved 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).

**Figure 2** Family pedigree showing the dominant pattern of inheritance. *Members who underwent OPA1 screening. Results of SSCP analysis of exon 27 are shown. Individuals with the 2708delT (TTAG) mutation show a different banding pattern compared to the wild type sequence.

**Table 1** Data showing the clinical results of visual acuity, colour vision, field analysis, and funduscoppy from selected family members. The colour vision was tested for monocular and binocular tritan (blue-yellow), deutan (green), and protan (red) defects which were scored out of 10 plates.

<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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<td>5.5</td>
<td>6/12 6/9</td>
<td>N</td>
<td>N</td>
<td>Generalised</td>
</tr>
</tbody>
</table>

Y = Yes; N = No. *Classified as affected from clinical tests alone.
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References

Vanishing corneal epithelial crystals following thalidomide induced resolution of myeloma related paraproteinemia

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 (6/5 both eyes) and an otherwise unremarkable ocular examination (anterior chamber, lens, vitreous, and fundus). The crystalline keratopathy stretched from limbus to limbus and consisted of a homogeneous spread of tiny yellow crystals located in the epithelium and to a lesser extent the anterior corneal stroma. Two months later, following investigations for breathlessness he was diagnosed as suffering from multiple myeloma. Investigations revealed a raised plasma viscosity (3.28 cp), an IgG-κ paraprotein (55.9 g/l), free immunoglobulin light chains in the urine (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 VIII) and was randomised to receive ABCM (adriamycin, BCNU, cyclophosphamide, melphalan). However, his renal function rapidly deteriorated (creatinine 714 µmol/l) requiring plasma exchange. Seven courses of ABCM were completed before the myeloma reached plateau phase (paraprotein 23.0 g/l) at which point interferon alfa was begun as maintenance therapy. Seven months later, following episodes of bony pain requiring radiotherapy the myeloma relapsed (paraprotein 38.3 g/l) and CIDEK (CCNU, idarubicin, dexamethasone) therapy was initiated. Six courses of CIDEK were completed until his paraprotein again plateaued (paraprotein 23.0 g/l).

Over the 6 month period following diagnosis visual acuity deteriorated to 6/9 as the crystalline keratopathy worsened (Fig 1A, B). Epithelial debridement, followed by a bandage contact lens and topical antibiotic (while the epithelium healed) improved corneal clarity, visual acuity, and reduced glare for several weeks before deterioration occurred once more. Over the next 2 years the epithelium was debrided alternately from one eye and then the other every 2–3 months and visual acuity was maintained at 6/6 to 6/9 Snellen.

Two months after completing CIDEK the myeloma relapsed again (paraprotein 38.7 g/l) and despite the use of weekly cyclophosphamide his renal function deteriorated and required haemodialysis. A month later a decision was made to try oral thalidomide therapy as the disease was progressing (paraprotein 52.8 g/l) and no longer responding to treatment. Initially he was started upon 50 mg daily, however, a peripheral neuropathy developed when the dose was increased to 100 mg daily and treatment was stopped for 3 weeks. The thalidomide was re-introduced in combination with dexamethasone, 4 days on 4 days off. The dexamethasone was slowly tailed off 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 1C, 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 gammopathies, infective crystalline keratopathy, Schyder’s central crystalline dystrophy, Bietti’s marginal crystalline dystrophy, gout, chronic renal disease, lipid keratopathy, and chrysiasis.1

Crystalline keratopathy occurs only rarely in association with multiple myeloma and monoclonal gammopathies.2,3 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,4,5 and also within the cells of the corneal stroma,6,7 conjunctiva, and lens.8,9

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,7,9 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 chemotherapy10 and plasma exchange9 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 angiogulation. 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, Figure 1 Slit lamp photographs of the left cornea. (A) Before thalidomide, low power. (B) Before thalidomide, high power. (C) After thalidomide, low power (pupil dilated). (D) After thalidomide, high power (pupil dilated).
like our patient, achieving complete remission. Although the anti-angiogenic effects of thalidomide are generally considered a key factor in the efficacy of the drug in multiple myeloma, the poor response of extramedullary plasmacytomas and the lack of correlation between bone marrow microvascular density and clinical response suggest that other actions of the drug may also be important.2

In summary, we report a case of a refractory multiple myeloma with associated corneal crystalline keratopathy in which resolution of the paraproteinaemia and corneal crystalline deposits occurred concurrently and following the initiation of thalidomide therapy. The report provides further support for the use of thalidomide in refractory multiple myeloma.

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References


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.2 Microscopic chromosome microdeletions 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, palatal, thymic hypoplasia, and hypothyroidism. The severity of the condition can be 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).3 The “velo” in VCFS refers to the palatal abnormalities such as cleft palate that are sometimes present. The characteristic facies appearance of del(22)(q11.22) is a prominent nose, broad nasal root, narrow palpebral fissures, and retrognathia.4 Recognised ocular findings include retinal vascular tortuosity, small optic nerves, and narrow palpebral fissures.5 An iris coloboma has been described in one case of VCFS and coloboma was listed as a rare feature in a series of cases of DGS.6

Case report

As part of a population based study of children with congenital anophthalmos, clinical microphthalmos, or iris coloboma (with or without fundus coloboma), we identified a teenage girl with bilateral iris coloboma, right fundus coloboma, left cataract, and clinical microphthalmos (Fig 1). On further examination she was found to have upslanting palpebral fissures, prominent nose, small mouth, dental crowding, unilateral deafness, arachnodactyly, second and third toe syndactyly (Fig 2), a ventriculoseptal defect (VSD) requiring surgical correction, and developmental delay. Both parents had no eye abnormalities. A clinical diagnosis of VCFS was made and microsatellite analysis of this girl’s DNA along with that of her parents confirmed a de novo deletion of the paternal 22q11.22 region. Two microsatellite markers (D22S944 and D22S264) within the common three megabase deletion interval demonstrated non-transmission of the paternal allele. In view of this unexpected diagnosis we analysed a further 21 study cases with structural eye malformations (mainly iris coloboma) where DNA was available from both parents. No additional del(22)(q11.22) cases were identified.

Comment

Here we report a girl with ocular coloboma and VCFS with a molecularly defined deletion of 22q11.22. Iris coloboma occurring in VCFS has been described once, but with no confirmation of a microdeletion.7 The cat-eye syndrome (CES), a disorder with a variable pattern of multiple congenital anomalies of which iris coloboma and anal atresia are the best known, is characterised cytogenetically by an extra ishalled marker chromosome.8 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 22q 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.

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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.9,10 Atropine is used widely as a form of eye drops for mydriasis, cyclopiaegia, 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 synechiae 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 synechiae. 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 materials were 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, the underlying sclera in the inferotemporal bulbar conjunctiva showed a considerable area of melting (2 x 3 mm).

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 eyedrops were tapered to four times daily 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 inflammation and proteinase 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.

**References**


**Trumatic 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.1

**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...
passengers on a bus, was assaulted by one of them. The attacker grabbed the patient from behind and attempted to insert his index finger into each orbit, successfully on the right and the right eye was gouged out (Fig 1). He also sustained a minor head injury following some blows to the head but was not knocked out.

On examination the only remaining attachment to the globe were the lateral rectus and inferior oblique. His optic nerve was completely avulsed. Examination of his left eye revealed a temporal hemianopia. His vision was 6/6. He was admitted and taken to theatre for a complete enucleation. He had a Goldmann field performed which confirmed a temporal hemianopia. An magnetic resonance imaging (MRI) scan was carried out which showed swelling of the left side of the chiasm (Fig 2) He had a 3 day course of intravenous methylprednisolone after MRI findings. There was an interval of 12 days between the day of injury and commencing methylprednisolone.

His visual field test done at the end of the 3 day course of methylprednisolone showed a marked improvement in the intertemporal quadrant (Fig 3). We presume the mechanism for chiasmal damage is traction on the chiasm. The MRI suggested oedema of hemichiasm perhaps interfering with function of the crossing fibres. Horton’s recent findings suggest that Willebrand’s knee of fibres may be an artefact and if so damage to crossing fibres in the stump of the avulsed nerve is unlikely to be an explanation for the field defect.

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

Comment

The incidence of traumatic enucleation reported by Eric 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.

In our case the attacker aimed specifically for the victim’s eyes. Bukhanovskykh 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. 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 leak.

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

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References


Cycloidiode 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 or cautioned in pregnancy. We report the case of a pregnant patient with aphakic glaucoma controlled with topical agents, for whom we performed cycloidiode laser therapy, since she wanted to become pregnant. The use of cycloidiode 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 6/6 in the left eye. She had an IOP of 19 mm Hg in her right eye and significant glaucomatous disc cupping (cup/disk 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 cycloidiode laser therapy on her right eye, under peribulbar anaesthesia, and stop her topical antiglaucomatous therapy. The first session of cycloidiode therapy (1500 mW × 1500 ms × 40 shots over 360° with 3 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 cycloidiode 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 cycloidiode (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 cycloidiode 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. β Blockers, sympathomimetics, carbonic anhydrase inhibitors, parasympathomimetics, apraclonidine, and prostaglandin analogues have all been shown to have adverse effects in animal fetal models. Understandably, there have been no equivalent studies in the human fetus and resulting teratogenesis in laboratory animals cannot be extrapolated to humans. However, some specific examples of adverse effects related to specific topical medications have been quoted in the literature. Topical timolol has been shown to cause fetal cardiac arrhythmia and bradycardia. There has been one reported case that correlates the use of acetazolamide in early pregnancy with sacrococcygeal teratoma in the neonate. Prostaglandins are involved in the physiology of pregnancy and thus the use of latanoprost is not advised during pregnancy.
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 anaesthetics for ophthalmic procedures during pregnancy. However, in a large multicentre retrospective study, exposure to local anaesthetic during early pregnancy resulted in no increased incidence of fetal malformation. Our patient could have been offered augmented filtration or tube implantation surgery but cycloplegia was felt to be less invasive and, ideally, exposure to antimetabolites should be avoided in pregnancy.1

Cycloplegia 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 intervention with general anaesthesia is avoided.

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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 aphagic 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.1

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 localised area with hypopigmentation and malperfusion of the choroid, corresponding to the segmental structure of the choroidal vasculature. However, the findings shown by the authors are more indicative of a segmental choroidal infarction (Hutchinson-Siegrist-Neubauer-syndrome).1 A choroidal rupture typically presents as an archetypal choroidal defect with a posterior pole follow- ing 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 circumscribed and/or sickle shaped configuration.1

By contrast, this clinical picture of a segmental traumatic choroidal infarction was first described by Hutchinson in 1889.2 Siegrist confirmed this new disease in 1895.3 Neubauer observed in three patients the typical segmental choroidal atrophy with corresponding visual field defects.4 Lang and associates explained this severe complication following ocular contusion as follows: as a result of Bell’s phenomenon and excessive eyelid squeezing, the globe is rotated upwards and moves backwards into the orbit between 0.7 to 1.7 mm.4 An occlusion or rupture of short ciliary arteries may result caused by sudden retroposition of the globe into the orbit,2 which leads to the typical choroidal infarction of the entire choroidal vascular segment.5 We found Hutchinson-Siegrist-Neubauer syndrome in 1% of all contusions of the Erlangen Ocular Contusion Registry.6

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.1

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References

Anti-Acanthamoeba efficacy in contact lens disinfecting systems

Hiti et al.1 produced an interesting study investigating the susceptibility of three strains of Acanthamoeba to a PHMB (polyhexamethylene 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 11DS) is typical of those known to cause keratitis in humans. This isolate belongs to genotype T4, the predominant genotype in Acanthamoeba keratitis.11,12 Their second strain (A. hatchetti 111DS) is of T6 genotype, which, with the exception of one incident,13 has not previously been reported as causing keratitis. The third strain (A. lenticalata 72:2) belongs to genotype T5,14 and as the authors themselves state, is not reported to cause Acanthamoeba keratitis.15 Although testing of all three strains was of interest to the microbiologist, the only result that can be extrapolated to clinical practice and be of clinical relevance to the contact lens wearer is that for A. castella-
ni (4CL). The majority of two step peroxide systems currently available contain 3% hydrogen peroxide. The authors tested a 1:1:10 peroxide solution (0.6% or 6000 µg/ml), but gave no comment as to why they chose a system with such a low active concentration. The two step system was, however, found to be effective next after an 8 hour exposure, but not at a 1:2 dilution (3000 µg/ml). This result was to be expected as Zanetti et al, in 1995, demonstrated that a 1:2 dilution of a 3% hydrogel peroxide solution killed cysts of A. castellanii after 30 minutes, but that a 1:10 dilution (0.3% or 3000 µg/ml) had lost its cy-
tical effect. The MPS was tested for use with rigid gas permeable lenses and contained 8% 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 0.001% PHMB. As the minimum trophozoite amoebicidal concentration (MTAC) of PHMB is 1 µg/ml and the minimum cysticidal concentration (MCC) is 3 µg/ml, the solution has performed as expected against the clini-
cally relevant 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 by the manufacturer's recom-
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concluded by recommending the two step tested against clinically relevant strains of cause keratitis in humans. This isolate belongs to genotype T4, the predominant genotype in Acanthamoeba keratitis.11 Their second strain (A. hatchetti 111DS) is of T6 genotype, which, with the exception of one incident, has not previously been reported as causing keratitis. The third strain (A. lenticalata 72:2) belongs to genotype T5, and as the authors themselves state, is not reported to cause Acanthamoeba keratitis. Although testing of all three strains was of interest to the microbiologist, the only result that can be extrapolated to clinical practice and be of clinical relevance to the contact lens wearer is that for A. castellanii (4CL). The majority of two step peroxide systems currently available contain 3% hydrogen peroxide. The authors tested a 1:1:10 peroxide solution (0.6% or 6000 µg/ml), but gave no comment as to why they chose a system with such a low active concentration. The two step system was, however, found to be effective next after an 8 hour exposure, but not at a 1:2 dilution (3000 µg/ml). This result was to be expected as Zanetti et al, in 1995, demonstrated that a 1:2 dilution of a 3% hydrogel peroxide solution killed cysts of A. castellanii after 30 minutes, but that a 1:10 dilution (0.3% or 3000 µg/ml) had lost its cyntical effect. The MPS was tested for use with rigid gas permeable lenses and contained 8% 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 0.001% PHMB. As the minimum trophozoite amoebicidal concentration (MTAC) of PHMB is 1 µg/ml and the minimum cysticidal concentration (MCC) is 3 µg/ml, the solution has performed as expected against the clinically relevant 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 by the manufacturer's recommended disinfection time of 4 hours. A be cysticidal after the manufacturer's recommended disinfection time of 4 hours. A be cysticidal after the manufacturer's recommended disinfection time of 4 hours. A be cysticidal after the manufacturer's recommended disinfection time of 4 hours.

References

10 Stevenson RWW, Seal DV. Has the application of a multipurpose disinfecting solution contributed to a reduced incidence of Acanthamoeba keratitis in contact lens wearers? Contact Lens Anterior Eye 1999; 21: 89–92.

Second Sight
Second Sight, a UK based charity whose aims are to eliminate the backlog of cataract blind in India by the year 2020 and to establish strong links between Indian and British oph-
thalmologists, is regularly sending volunteer surgeons to India. Details can be found at the charity website (www.secondsight.org.uk) or by contacting Dr Lucy Mathen (lucymathen@yahoo.com).

Specific Eye Conditions (SPECs)
Specific Eye Condition’s (SPECs) is a not for profit organisation which acts as an umbrella organisation for support groups for any of 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.eyeconditions.org.uk acts as a portal giving direct access to support groups own sites. The SPECs web page is a valuable resource for professionals and may also be of interest to people with a visual impairment or who are blind. For further details about SPECs contact: Kay Parkinson, SPECs Develop-
ment Officer (tel: +44 (0)1803 524238; email: k@endications.org.uk; website: www.eyecondi-
tions.org.uk).

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tions.org.uk).
for any queries or worries relating to the problems of 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)1228 821 334; email: lynda@brps.demon.co.uk; web site: www.brps.demon.co.uk)

Introductory Course in Osteo-odonto-keratoprosthesis (OOKP)
The University of Brighton Postgraduate Medical School is holding an introductory course in osteo-odonto-keratoprosthesis (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. Conference charges: £60 members; £80 non-members. Price entitles delegates to refreshments, lunch, abstract book, programme, and 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 Verwaltungsgebäude der KA Rudolfstiftung, 1030 Vienna, Boehrnhavegasse 4a, Austria. Further details and registration: Firma Askin & Co, Albert-Schweitzer-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, Breuningerbaur, 72076 Tuebingen, Germany (tel: +49 7071 295209; email: ingrid.kreissig@med.uni-tuebingen.de). Local organisation: Prof. Quiroz-Mercado, Prof. Munoz, and Prof Gonzalez “Hospital Luis Sanchex Bulnes”, Asociacion para Evitar la Ceguera en Mexico Vicente Garcia Torres #46, Coyoacan, Mexico DF 04330 (fax: +5255 5659 5928; email: retinamex@yahoo.com).

16th Annual Meeting of German Ophthalmic Surgeons
The 16th Annual Meeting of German Ophthalmic Surgeons will be held 8–11 May 2003 in Nuernberg, Germany, Messegzenrum. Organised by the Professional Association of German Ophthalmologists Ophthalmic Surgery Group the conference will cover cataract surgery, refractive surgery, glaucoma surgery, vitrectominal surgery, corneal surgery, eye surgery in developing countries, and orbita, lacrimal and lid surgery. Further details: MCN Medizinische Congress organisation Nurnberg AG, Zerabelshofstr 29, 90398 Nurnberg, Germany (tel: +49 911 3931621; fax: +49 911 3931620; email: doc@mcnag.info; web site: www.doc-nuernberg.de).

Detachment Course with international faculty on: Retinal and Vitreous Surgery with Case Presentations preceding the Annual Meeting of Iranian Society of Ophthalmology
The detachment course with international faculty on: Retinal and Vitreous Surgery with Case Presentations preceding Annual Meeting of Iranian Society of Ophthalmology will be held on 29–30 November 2003 and 1–4 December 2003 respectively, at the Razi Conference Center, Hemmat Hyv, Tehran, Iran. Further details: Scientific programme: Prof Ingrid Kreissig, University of Tuebingen, Schleichstr. 12, Breuningerbaur, 72076 Tuebingen, Germany (tel:+49 7071 295209; email: ingrid.kreissig@med.uni-tuebingen.de). Local organisation: Dr Arman Masheyekhi, Dr Siavash Moradian, Dept of Ophthalmology, Labbafinejad Medical Center, Pasdaran Ave, Boosstan 9, Tehran, 16666, Iran (fax: +98 21 254 9039; email: labbafi@hotmail.com).