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Primary Angle Closure Glaucoma

Acute Primary Angle Closure in Thailand

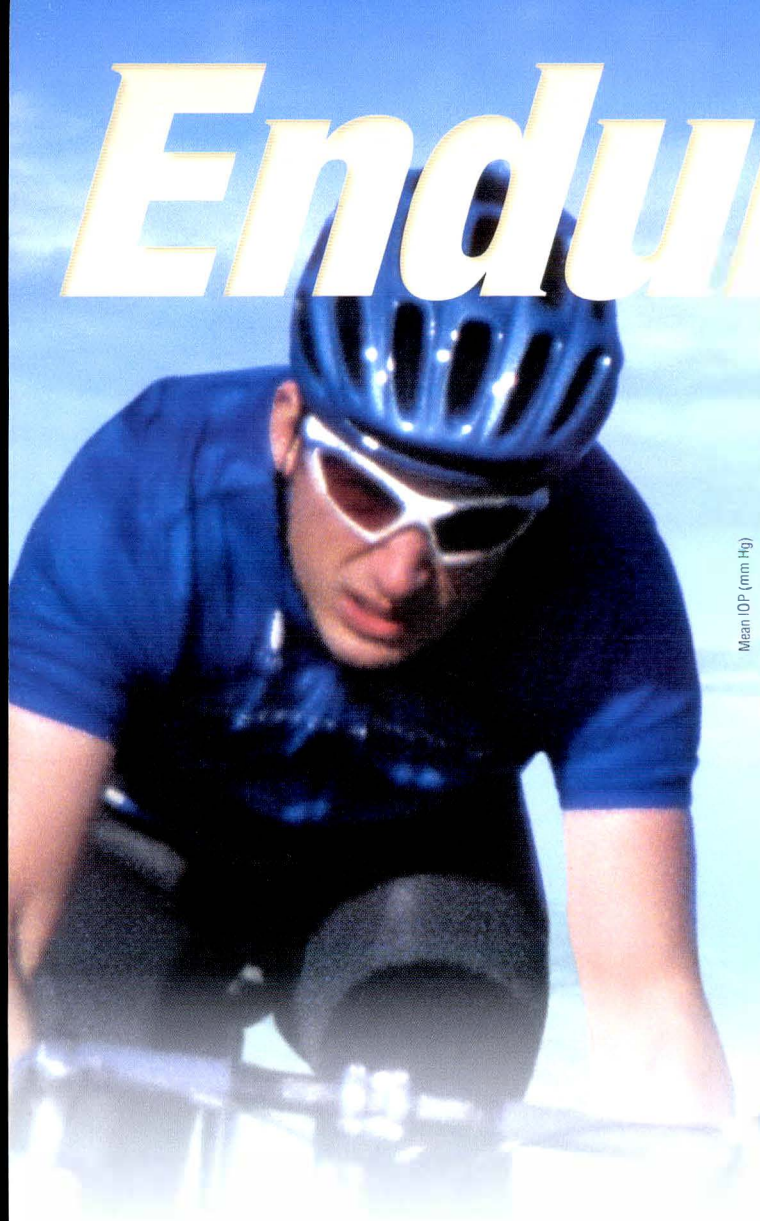
***Helicobacter pylori* and Open Angle Glaucoma**

Contact Lens-associated Infectious Keratitis

Asian Journal of OPHTHALMOLOGY

Scientific Communications

Endurance



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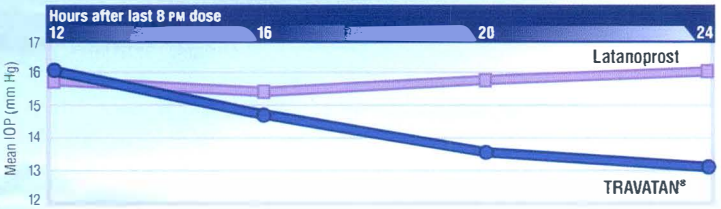

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TRAVATAN® (travoprost 0.004%) Ophthalmic Solution Sterile **DESCRIPTION** Travoprost is a highly selective, potent agonist for the FP prostanoid receptor. Its chemical name is isopropyl (Z)-7-[(1*R*,2*R*,3*R*,5*S*)-3,5-dihydroxy-2-[(1*E*,3*R*)-3-hydroxy-4-[(α,α,α-trifluoro-*m*-tolyl)oxy]-1-butenyl]cyclopentyl]-5-heptenoate. Its molecular formula is $C_{26}H_{35}F_3O_6$. Travoprost is a clear, colorless to pale yellow oil, which is very soluble in acetonitrile, methanol, octanol, and chloroform. It is practically insoluble in water. TRAVATAN® 0.004% Ophthalmic Solution is supplied as a sterile, buffered aqueous solution of travoprost with a pH of approximately 6.0 and an osmolality of approximately 290 mOsmol/kg. Each mL of TRAVATAN® 0.004% contains 40 µg travoprost. Preservative: benzalkonium chloride 0.015%. Inactive ingredients: polyoxyl 40 hydrogenated castor oil, tromethamine, boric acid, mannitol, edetate disodium, sodium hydroxide and/or hydrochloric acid (to adjust pH) and purified water. **CLINICAL PHARMACOLOGY** Mechanism of Action Travoprost free acid is a highly selective, potent agonist for the FP prostanoid receptor. FP receptor agonists are reported to reduce intraocular pressure by increasing uveoscleral outflow. Pharmacokinetics/Pharmacodynamics Absorption: Travoprost is absorbed through the cornea. Studies in rabbits have shown peak concentrations in aqueous humor were reached one to two hours following topical administration. In humans, peak plasma concentrations of travoprost free acid were low (25 pg/mL or less) and occurred within 30 minutes following topical administration. Elimination from plasma was rapid resulting in concentrations below the limit of quantitation (< 10 pg/mL) by one hour. Metabolism: Travoprost, an isopropyl ester prodrug, is rapidly hydrolyzed by esterases in the cornea to the biologically active free acid. Systemically, travoprost free acid is rapidly and extensively metabolized to inactive metabolites. Biotransformations include beta-oxidation of the α(carboxylic acid) chain to give the 1,2-dinor and 1,2,3,4-tetranor analogs, oxidation of the 15-hydroxyl moiety, as well as reduction of the 13,14 double bond. Excretion: In rats, 95% of a subcutaneous radiolabeled dose was eliminated within 24 hours. The major route of elimination was via the bile (61%) with the remainder excreted by the kidneys. **INDICATIONS AND USAGE** TRAVATAN® Ophthalmic Solution is indicated for the reduction of intraocular pressure in patients with open-angle glaucoma or ocular hypertension. **CLINICAL STUDIES** TRAVATAN® 0.004% Ophthalmic Solution dosed once-daily in patients with open-angle glaucoma or ocular hypertension produced significant reductions in intraocular pressure (IOP) when used either as primary therapy or adjunctively to TIMOPTIC* (timolol maleate ophthalmic solution) 0.5% BID. As primary therapy, TRAVATAN® 0.004%, dosed QD, reduced IOP 7 to 9 mmHg. Stable diurnal IOP reductions were achieved as early as 2 weeks after initiation of therapy and were maintained over the 6 to 12 month treatment periods in three (3) well-controlled studies. The IOP reductions with TRAVATAN® (travoprost 0.004%) Ophthalmic Solution were superior to those obtained with TIMOPTIC* and equal or better than those obtained with XALATAN* (latanoprost ophthalmic solution) 0.005% QD. TRAVATAN® 0.004% demonstrated an earlier stabilization of IOP reduction and better IOP control throughout the day compared to XALATAN* 0.005%. TRAVATAN® 0.004% was significantly more effective (up to 1.4 mmHg) than XALATAN* 0.005% in reducing IOP in black patients. A responder analysis (IOP reduction ≥30% or mean IOP ≤17 mmHg) demonstrated that TRAVATAN® 0.004% had a significantly higher responder rate (56%) compared to XALATAN* 0.005% (50%) and which were both significantly greater than TIMOPTIC* (40%). In a 6-month well-controlled study, TRAVATAN® 0.004% dosed QD adjunctively to TIMOPTIC* 0.5% BID provided additional clinically significant IOP reductions (6 to 7 mmHg). **CONTRAINDICATIONS** Known hypersensitivity to travoprost, benzalkonium chloride or any other ingredients in this product. TRAVATAN® may interfere with the maintenance of pregnancy and should not be used by women during pregnancy or by women attempting to become pregnant. **WARNINGS** TRAVATAN® may gradually change eye color, increasing the amount of brown pigmentation in the iris by increasing the number of melanosomes (pigment granules) in melanocytes. The long term effects on the melanocytes and any consequences thereof are currently unknown. The change in iris color occurs slowly and may not be noticeable for months to years. These changes may be permanent. Periorbital and/or eyelid skin darkening has been reported in association with the use of TRAVATAN®. TRAVATAN® may gradually change eyelashes in the treated eye; these changes include: increased length, thickness, pigmentation, and/or number of lashes. Patients who receive treatment in only one eye may experience increased brown pigmentation of the iris, periorbital and/or eyelid tissue, and eyelashes in the treated eye. They may also experience disparity between the eyes in length, thickness, and/or number of eyelashes. These changes in pigmentation and lash growth may be permanent. **PRECAUTIONS** General There have been reports of bacterial keratitis associated with the use of multiple-dose containers of topical ophthalmic products. These containers had been inadvertently contaminated by patients who, in most cases, had a concurrent corneal disease or a disruption of the epithelial surface (see Information for Patients). Patients may slowly develop increased brown pigmentation of the iris. This change may not be noticeable for months to years (see Warnings). This change in eye color has predominantly been seen in patients with mixed colored irides, i.e., blue-brown, grey-brown, yellow-brown and green-brown; however, it has also been observed in patients with brown eyes. Based upon information from the literature, the color change is believed to be due to increased melanin content in the stromal melanocytes of the iris. Typically the brown pigmentation around the pupil spreads concentrically towards the periphery in affected eyes, but the entire iris or parts of it may become more brownish. No further increase in brown iris pigment has been observed after discontinuation of treatment, but the resultant color change may be permanent. TRAVATAN® should be used with caution in patients with active intraocular inflammation (iritis/uveitis). Macular edema, including cystoid macular edema, has been reported during treatment with prostaglandin $F_{2\alpha}$ analogues. These reports have mainly occurred in aphakic patients, pseudophakic patients with a torn posterior lens capsule, or in patients with known risk factors for macular edema. TRAVATAN® (travoprost 0.004%) Ophthalmic Solution should be used with caution in these patients. Patients should remove contact lenses prior to administration of the solution. Lenses may be reinserted 15 minutes following administration of TRAVATAN®. Information for Patients Patients should be advised concerning all the information contained in the Warnings and Precautions sections. Patients should also be instructed to avoid allowing the tip of the dispensing container to contact the eye or surrounding structures because this could cause the tip to become contaminated by common bacteria known to cause ocular infections. Serious damage to the eye and subsequent loss of vision may result from using contaminated solutions. Patients also should be advised that if they develop an intercurrent ocular condition (e.g., trauma, or infections) or have ocular surgery, they should immediately seek their physician's advice concerning the continued use of the multi-dose container. Patients should be advised that if they develop any ocular reactions, particularly conjunctivitis and lid reactions, they should immediately seek their physician's advice. Patients should also be advised that TRAVATAN® contains benzalkonium chloride which may be absorbed by contact lenses. Contact lenses should be removed prior to administration of the solution. Lenses may be reinserted 15 minutes following administration of TRAVATAN®. If more than one topical ophthalmic drug is being used, the drugs should be administered at least five (5) minutes apart. Carcinogenesis, Mutagenesis, Impairment of Fertility Travoprost was not mutagenic in bacteria, in one mouse lymphoma assay, in the mouse micronucleus tests and in the rat chromosome aberration assay. In another mouse lymphoma assay, higher concentrations of travoprost were slightly mutagenic only in the presence of activation enzymes. In life and early post-mortem evaluations of carinogenicity studies in rats and mice suggest no evidence of a carcinogenic potential. Travoprost did not affect mating or fertility indices in male or female rats at subcutaneous doses up to 10 µg/kg/day (250 times the recommended human dose). The mean number of corpora lutea was slightly reduced at that dose, and the post-implantation losses were increased, but was not affected at 3 µg/kg/day (75 times the maximum recommended human dose). Pregnancy: Teratogenic Effects Pregnancy Category: C In reproduction studies conducted in pregnant rats and mice, travoprost reduced fetal viability when administered during gestation at doses as low as 1.0 µg/kg/day (25 times the maximum recommended human dose) with the lowest no effect level at 0.3 µg/kg/day (7.5 times the maximum recommended human dose). The incidence of skeletal malformations was increased in fetuses of rat dams receiving travoprost by subcutaneous injection at 10 µg/kg/day (250 times the maximum recommended human dose), but not at 3 µg/kg/day (75 times the maximum recommended human dose). No fetal abnormalities were observed in mice at 1.0 µg/kg/day (25 times the maximum recommended human dose). No adequate and well-controlled studies have been performed in pregnant women. TRAVATAN® may interfere with the maintenance of pregnancy and should not be used by women during pregnancy or by women attempting to become pregnant. Nursing Mothers A study in lactating rats demonstrated that radiolabeled travoprost and/or its metabolites were excreted in milk. It is not known whether this drug or its metabolites are excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when TRAVATAN® is administered to a nursing woman. Pediatric Use Safety and effectiveness in pediatric patients have not been established. Geriatric Use No overall differences in safety or effectiveness have been observed between elderly and other adult patients. **ADVERSE REACTIONS** (see Warnings and Precautions) The most common ocular adverse event observed in controlled clinical studies with TRAVATAN® (travoprost 0.004%) Ophthalmic Solution was ocular hyperemia which was reported in 35 to 50% of patients. 95% of the ocular hyperemia observed with TRAVATAN® (travoprost 0.004%) Ophthalmic Solution was mild in intensity and subsided over time without treatment. Approximately 3% of patients discontinued therapy due to conjunctival hyperemia. Ocular adverse events reported at an incidence of 5 to 10% included decreased visual acuity, eye discomfort, foreign body sensation, pain, and pruritus. Ocular adverse events reported at an incidence of 1 to 4% included, abnormal vision, blepharitis, blurred vision, cataract, cells, conjunctivitis, dry eye, eye disorder, flare, iris discoloration, keratitis, lid margin crusting, photophobia, subconjunctival hemorrhage, and tearing. Nonocular adverse events reported at a rate of 1 to 5% were accidental injury, angina pectoris, anxiety, arthritis, back pain, bradycardia, bronchitis, chest pain, cold syndrome, depression, dyspepsia, gastrointestinal disorder, headache, hypercholesterolemia, hypertension, hypotension, infection, pain, prostate disorder, sinusitis, urinary incontinence, and urinary tract infection. **OVERDOSAGE** A single-dose intravenous study in rats was conducted to elucidate maximal acute hazard. The dose employed was 250,000-times the proposed daily clinical exposure and over 5000-times the possible exposure from the entire contents of one product container. No treatment related pharmacotoxic signs were present in the animals receiving Travoprost. If overdosage with TRAVATAN® occurs, treatment should be symptomatic. **DOSAGE AND ADMINISTRATION** The recommended dosage is one drop in the affected eye(s) once-daily in the evening. The dosage of TRAVATAN® should not exceed once-daily since it has been shown that more frequent administration may decrease the intraocular pressure lowering effect. Reduction of intraocular pressure starts approximately 2 hours after administration and the maximum effect is reached after 12 hours. TRAVATAN® may be used concomitantly with other topical ophthalmic drug products to lower intraocular pressure. If more than one topical ophthalmic drug is being used, the drugs should be administered at least five (5) minutes apart. **HOW SUPPLIED** TRAVATAN® (travoprost 0.004%) Ophthalmic Solution is a sterile, isotonic, buffered, preserved, aqueous solution supplied in Alcon's oval DROP-TAINER® package system inside a sealed foil pouch. This package system is comprised of a plastic oval shaped dispenser bottle, a dropper tip and tamper evident neck-band which shrinks to conform around the closure and neck area of the package. 0.004%: 2.5 mL fill Storage Store between 2° to 25°C (36° to 77°F). Refrigeration is not required. Rx Only (USA) **CAUTION:** Federal (USA) law prohibits dispensing without prescription.

*TIMOPTIC is a registered trademark of Merck & Co.Inc. †XALATAN is a registered trademark of Pharmacia Corp. U.S. Patent Nos. 5,631,287; 5,849,792; 5,889,052; 6,011,062 and 6,235,781

* A washout period of 4 weeks was followed by 2 weeks of TRAVATAN® Solution (n=16) or latanoprost monotherapy (n=18). At day 14, the final dose was administered at 8 pm and IOP measurements were taken. Baseline values for the two treatment groups were not significantly different. The standard deviations for the TRAVATAN® group were 3.9 mm Hg (12 hours), 2.9 mm Hg (16 hours), 3.2 mm Hg (20 hours), and 2.1 mm Hg (24 hours). For the latanoprost group, the standard deviations were 3.8 mm Hg (12 hours), 3.0 mm Hg (16 hours), 3.2 mm Hg (20 hours), and 3.1 mm Hg (24 hours). The difference between the two groups at 24 hours post dose was statistically significant (p=0.017)

Reference 1. Dubiner HB, SircyMD, Landry T, et al. Comparison of the diurnal ocular hypotensive efficacy of travoprost and latanoprost over a 44-hour period in patients with elevated intraocular pressure. Clin Ther 2004;26:84-91

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SEAGIG
South East Asia Glaucoma Interest Group

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As new technologies and therapeutic interventions are continually being developed, ophthalmology has become a field of rapid change, particularly in the Asia-Pacific region, where disease patterns and health care delivery differ greatly from those seen in the West. *Asian Journal of OPHTHALMOLOGY* was established in 1998 and became the official journal of SEAGIG in 2003, with the aim of disseminating information relevant to ophthalmology and glaucoma throughout Asia and to interested groups worldwide. The objectives of *Asian Journal of OPHTHALMOLOGY* are as follows:

- to provide a platform for the publication of information with a focus on ophthalmology in Asia
- to disseminate information that will improve the care of patients with all types of ophthalmological disorders, with a special focus on glaucoma
- to increase the understanding of such disorders through reporting of educational activities
- to publish the results of research programmes to expand knowledge about the causes, prevention, and treatment of ophthalmological disorders
- to work closely with Asian and international researchers to achieve these aims
- to provide a forum for young and relatively inexperienced researchers to present their research results as Original Articles via an international platform
- to maintain and promote relationships with any organisation with similar goals.

Although the focus of *Asian Journal of OPHTHALMOLOGY* is on glaucoma, other topics relevant to the region will not be ignored, and submissions on all aspects of ophthalmology are welcome.

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The most frequently reported adverse events occurring in approximately 15% to 45% of patients dosed once daily, in descending order of incidence, were conjunctival hyperemia, growth of eyelashes, and ocular pruritus.

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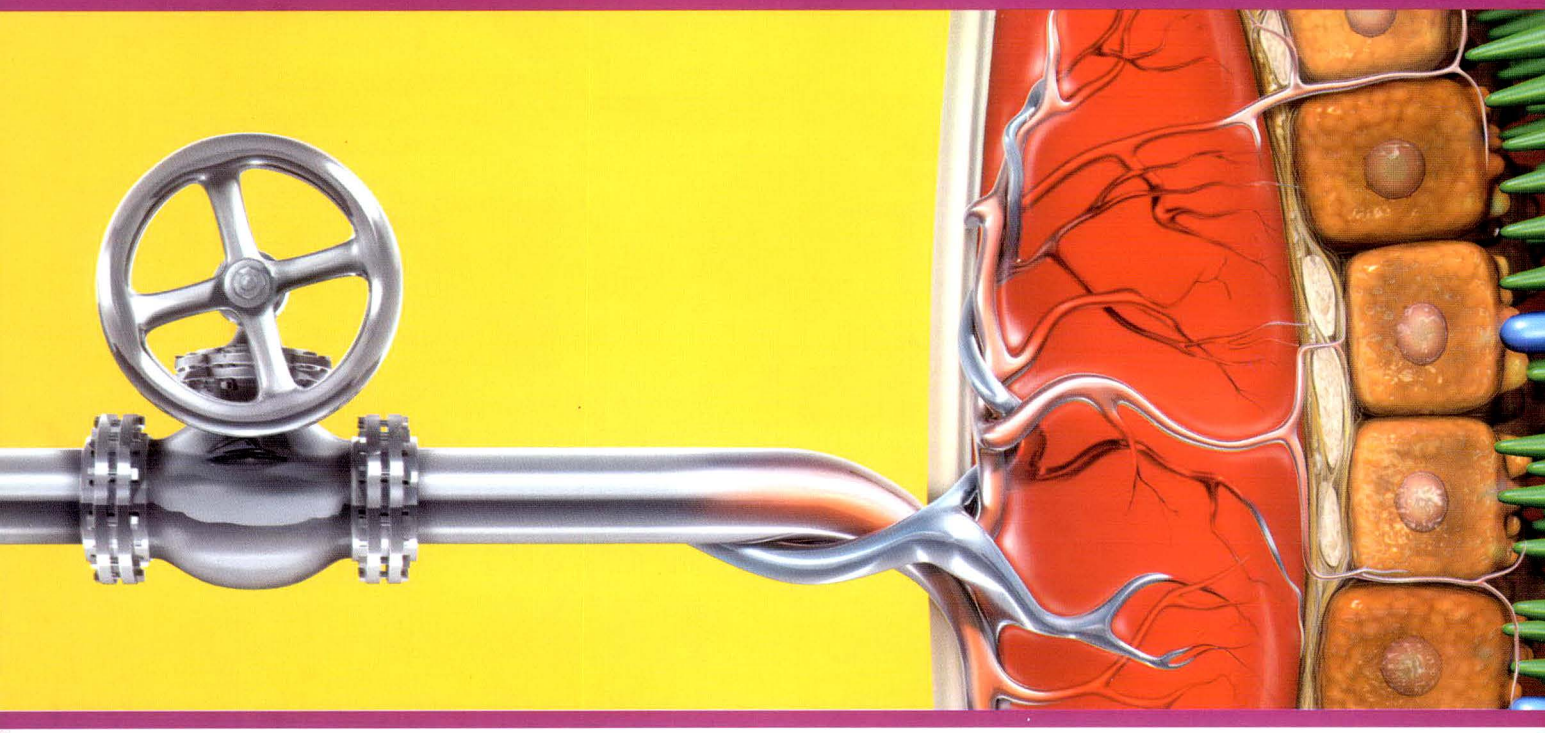

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* Age-related macular degeneration. † Vascular endothelial growth factor ‡ Defined as <15 letters lost over 2 years.

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MACUGEN ABBREVIATED PACKAGE INSERT TRADE NAME: Macugen **PRESENTATION:** Macugen injection is supplied in a single use 1 mL glass syringe containing pegaptanib sodium 0.3 mg in a 90 µL deliverable volume. **INDICATIONS:** Treatment of neovascular (wet) age-related macular degeneration. **DOSAGE:** 0.3 mg administered once every six weeks by intravitreous injection into the eye to be treated. Macugen should be inspected visually for particulate matter and discoloration prior to administration. The injection procedure should be carried out under controlled aseptic conditions. Adequate anesthesia and a broad-spectrum microbicide should be given prior to the injection. **CONTRAINDICATIONS:** Patients with ocular or periocular infections. **WARNINGS & PRECAUTIONS:** FOR OPHTHALMIC INTRAVITREAL INJECTION ONLY. Intravitreous injections have been associated with endophthalmitis. Proper aseptic injection technique should always be utilized when administering Macugen. Patients should be monitored during the week following the injection to permit early treatment, should an infection occur. Intraocular pressure as well as the perfusion of the optic nerve head should be monitored and managed appropriately. **INTERACTIONS:** Pegaptanib is metabolized by nucleases and is generally not affected by the cytochrome P450 system. **PREGNANCY AND LACTATION:** Pregnancy Category B. Pegaptanib crosses the placenta in mice. There are no studies in pregnant women. The potential risk to humans is unknown. Macugen should be used during pregnancy only if the potential benefit to the mother justifies the potential risk to the fetus. It is not known whether pegaptanib is excreted in human milk; caution should be exercised when Macugen is administered to a nursing woman. **SIDE EFFECTS:** Anterior chamber inflammation, blurred vision, cataract, conjunctival hemorrhage, corneal edema, eye discharge, eye irritation, eye pain, hypertension, increased intraocular pressure (IOP), ocular discomfort, punctate keratitis, reduced visual acuity, visual disturbance, vitreous floaters, and vitreous opacities. Injection procedure related side effects include endophthalmitis, retinal detachment, iatrogenic traumatic cataract. **STORAGE:** Store in refrigerator at 2°C to 8°C. Do not freeze or shake vigorously. Reference: USPI (Dec 2004). **FULL PRESCRIBING INFORMATION IS AVAILABLE UPON REQUEST.**

MAC06003F

Primary Angle Closure Glaucoma: Improved Public Awareness and Prompt Access to Health Care can Reduce Visual Morbidity

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Glaucoma Services, National University Hospital, Singapore

It is estimated that primary angle closure glaucoma (PACG) will cause bilateral blindness in 3.9 million people worldwide by 2010. This number is estimated to increase to 5.3 million by 2020. Due to its greater morbidity, the number of people blinded by ACG is likely to be nearly equal to the estimated 5.9 million who will be blind from primary open angle glaucoma by 2020. Eighty six percent of people affected by ACG will be in Asia, with approximately 48.0% in China, 23.9% in India, and 14.1% in Southeast Asia.[1] These estimations highlight the importance of understanding the disease, its natural history, and its underlying pathophysiology, so that we may try to establish effective methods of treatment and preventative measures to delay, or even arrest, disease progression, thereby reducing visual morbidity. More research needs to be done, especially in certain Southeast Asian populations for whom there are still a relative paucity of useful data.

The management of acute primary angle closure (APAC) has conventionally been with topical and systemic intraocular pressure (IOP)–lowering medications, followed by laser peripheral iridotomy. While this suffices to control the IOP in some patients, others require long-term antiglaucoma medication. Yet others continue to have uncontrolled IOP and require filtration surgery. The reasons underlying these differences in outcomes require investigation. We know from experience and logical deduction that the duration of symptoms prior to presentation, the level of presenting IOP, the duration of significantly raised IOP during the acute attack, and the amount of ischaemic sequelae are some of the important factors that must be considered. However, is the duration of symptoms prior to presentation more important or is the level of IOP at presentation the principal factor? Are there any symptoms that are more ominous and associated with a worse prognosis?

In this issue of *Asian Journal of Ophthalmology*, Kitnarong et al present their findings in a retrospective review of 68 eyes presenting with APAC, particularly looking at predictive factors for the need for filtration surgery.[2] The authors reported that 66.2% of eyes had successfully controlled IOPs with laser/surgical peripheral iridotomy with or without medications, while 33.8% of eyes required additional filtration surgery. The authors analysed the differences between the non-surgery and surgery groups and found that the duration of certain symptoms, including ocular pain, red eye, decreased vision, and halos, were significantly longer in the surgery group compared with the non-surgery group, with no statistical difference in other factors, including the level of IOP at presentation. The mean IOP in the surgery group was also found to be significantly higher 24 and 48 hours after the initiation of IOP-lowering treatment. The mean pupil size was also noted to be significantly larger at all time points in the surgery group. This study confirms that a longer duration of symptoms prior to presentation is an important predictive factor for the need for filtration surgery. The longer duration of raised IOP during APAC is likely to lead to more severe ischaemia, as manifested by the larger pupil size, although chronic PACG presenting as APAC may also be the case in some of these patients. However, being a retrospective review, without clear and consistent documentation of the presence or absence of glaucomatous optic neuropathy at presentation, standardised gonioscopy, and visual field perimetry for all patients, it is not possible to stratify the need for filtration surgery according to whether the eyes had PAC or established PACG.

The natural progression of ACG is currently believed to be from the status of PAC suspect (PACS; defined as an eye in which appositional contact between the peripheral iris and posterior trabecular meshwork is considered possible) to PAC (an eye with an occludable drainage angle and features indicating that trabecular obstruction by the peripheral iris such as peripheral anterior synechiae [PAS], elevated IOP, iris whorling, glaucomflecken, or excessive pigment deposition on the trabecular surface has occurred) and then to PACG (PAC together with evidence of glaucoma), as defined at the congress of the International Society for Geographical and Epidemiological Ophthalmology in 1998.[3]

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Early screening at the stage of PACS should allow at-risk individuals to be treated with prophylactic laser iridotomy or iridoplasty to prevent the establishment of PAS and progression to PAC. In turn, this should reduce the risk for further progression to PACG. However, this simplistic way of managing ACG is fraught with problems. At present, we still do not have an ideal screening tool or method that is cost-effective and reliable across various populations. Much research is being done to find an effective screening method on a large scale for patients who are at high risk for angle closure.[4-7] Furthermore, while laser peripheral iridotomy has been shown to be effective in widening the anterior chamber angle in Mongolian eyes[8] and maintaining IOP control in the long term after APAC in Caucasian eyes,[9,10] various authors have suggested differing mechanisms of angle closure other than pupil block in different populations, so laser iridotomy may not be equally effective in all populations.[11,12]

It remains imperative to continue to develop and evaluate methods to effectively manage APAC to halt progression to PACG, with its attendant visual morbidity. Various approaches such as early laser iridoplasty, anterior chamber paracentesis, and lens extraction by phacoemulsification in the acute setting have been suggested and prospective randomised controlled trials are underway to evaluate them.[13]

Kitnarong et al have clearly shown that late presentation, with more severe ischaemic sequelae, leading to delayed treatment results in a poorer outcome, with more patients going on to require filtration surgery for IOP control.[2] We therefore must not forget that efforts should also be targeted at improving public awareness of the symptoms and signs of ACG, educating primary health care providers to enable their prompt recognition of the disease, and improving access to higher levels of health care for appropriate treatment, as these measures can favourably affect the final visual prognosis and reduce visual morbidity.

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Acute Primary Angle Closure in Thailand

Naris Kitnarong, Surasa Libratanasakul, Ankana Metheetrairut, Ngamkae Ruangvaravate
Department of Ophthalmology, Faculty of Medicine, Siriraj Hospital, Mahidol University, Bangkok, Thailand

Aim: *To evaluate the clinical presentation, progression, and treatment outcome after acute primary angle closure in Thai patients.*
Methods: *This was a retrospective study of 68 eyes of 66 consecutive patients with acute primary angle closure presenting to the Faculty of Medicine, Siriraj Hospital, Bangkok, Thailand, from 2000 to 2002. Acute angle closure from secondary causes was excluded. The predictive factors for the need for filtration surgery were studied.*
Results*: There were 15 men and 51 women with a mean age of 60.5 years (range, 35 to 81 years). The mean intraocular pressure at presentation was 58.2 mm Hg (SD, 14.3 mm Hg). After initial treatment with antiglaucoma medications, all eyes underwent laser peripheral iridotomy. Three eyes needed additional surgical peripheral iridotomy. The intraocular pressure of 45 eyes (66.2%) was successfully controlled at <21 mm Hg without further intervention. Of the 45 eyes, only 7 (15.5%) required no antiglaucoma medication at discharge. The remaining 23 eyes (33.83%) underwent filtration surgery. The filtration surgery group had a mean duration of symptoms significantly longer than that of the non-filtration surgery group ($p < 0.05$) and a significantly greater mean pupil size within 24 hours of the initial treatments ($p < 0.05$).*
Conclusion*: Prompt diagnosis and early treatment for acute primary angle closure is important to prevent subsequent morbidity. Laser peripheral iridotomy and medications can control acute episodes of angle closure and its sequelae for most patients, with one-third requiring surgical intervention for control of intraocular pressure.*

Key words*: Filtering surgery, Glaucoma, angle closure, Laser surgery*

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Introduction

Primary angle closure glaucoma (PACG) has been reported to have a higher prevalence in Asian populations than in Caucasian populations.[1,2] In China, it is estimated that PACG affects 3.5 million people, and a further 2 million people have occludable angles.[3] The reported prevalence rate indicates that angle closure is at least as common as open angle glaucoma in South and East Asia.[2] The high prevalence of angle closure is an important factor leading to the high incidence of acute primary angle closure (APAC). Singapore has the highest reported incidence of APAC, with an annual incidence of 12.2/100,000 population aged 30 years and older.[4]

After an episode of APAC, these eyes have wide range of morbidity, including peripheral anterior synechiae (PAS) and blindness. Clinical recognition and early treatment are necessary to prevent further morbidity. Peripheral iridotomy (PI) has been proven to be an effective means of treatment and prophylaxis for APAC.[5-8] Laser peripheral iridotomy (LPI) is now more commonly performed than surgical PI because LPI is non-invasive and can be performed quickly in an outpatient setting. Although treatment with antiglaucoma medications and PI are implemented in the acute phase to break the acute attack, more than half the eyes with APAC develop ACG or need further interventions.[9,10] There is a paucity of literature discussing the factors that influence treatment outcome after APAC. This study was designed to assess the prognoses and treatment outcomes after APAC.

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Methods

Patients

This was a retrospective chart review of patients who presented to the Faculty of Medicine, Siriraj Hospital, Mahidol University, Bangkok, Thailand, from October 2000 to September 2002, with signs and symptoms of APAC. The following criteria were used for diagnosis:

- the presenting intraocular pressure (IOP) was >21 mm Hg
- the anterior chamber angle was grade 0 to 1 by the Shaffer grading system for at least 180° by gonioscopy
- at least one of the following symptoms was present: ocular pain, red eye, decreased vision, headache, nausea/vomiting, or halos
- one of the following signs was evident at ocular examination: ciliary conjunctival injection, corneal oedema, corneal epithelial microcyst, or semi-dilated pupil (measured by slit-lamp biomicroscopy).

Design

Initial treatment included a topical β-blocker, topical pilocarpine 2% 4 times every 15 minutes, and oral acetazolamide 500 to 1000 mg. An oral or intravenous hyperosmotic agent was administered to patients with severe symptoms, IOP >45 mm Hg, or an advanced glaucomatous cup and no contraindications. After the initial treatment, all patients were admitted to hospital and underwent LPI once the cornea allowed good visualisation. The laser settings were as follows: Argon laser of 200 to 500 mW, 50 to 200 micron spot size, 0.1-second exposure, followed by Nd:YAG laser of 1.5 to 5.0 mJ. An Abraham iridotomy contact lens was used. The common sites were the superonasal and superotemporal areas. If the laser treatment did not provide a patent iridotomy, surgical iridotomy was considered. If medical treatment failed to control the IOP, then trabeculectomy was indicated. Trabeculectomy was done by, or under the supervision of, 1 of 4 surgeons. The surgical technique was similar for all surgeons, and involved a limbal-based conjunctival flap, with or without intraoperative mitomycin C (MMC) application. The use and timing of MMC was at the discretion of the surgeon.

The data collected included demographic characteristics, presenting signs and symptoms, duration of each symptom, pre- and post-treatment IOP, visual acuity, gonioscopic findings, surgical interventions, and treatment outcome. Humphrey automated perimetry was used to obtain the visual field within 3 months of the acute attack. The statistical analyses included the Student *t* test for comparisons of demographic data, duration of symptoms, pre- and post-treatment IOP, and difference in pupil size between the filtration surgery and non-filtration surgery groups. The Mann-Whitney *U* test was used for non-parametic data analysis and included comparisons of visual field defect pattern and mean deviation between groups. The risk assessment for surgical intervention was analysed using logistic regression analysis.

Results

Sixty eight eyes of 66 patients diagnosed with APAC were included in the study. Two patients had bilateral symptoms. There were 15 men and 51 women with a mean age of 60.5 years (range, 35 to 81 years). Sixty two patients were Thai and the remaining 4 patients were Chinese. The right eye (40 eyes) was affected more often than the left eye (28 eyes). The mean IOP at presentation was 58.2 mm Hg (SD, 14.3 mm Hg). Twenty five eyes (36.8%) had initial best-corrected visual acuity (BCVA) better than 6/60. Mean duration of ocular pain was 6.6 days (SD, 10.0 days), of decreased vision was 6.4 days (SD, 10.3 days), of red eye was 5.8 days (SD, 7.5 days), of headache was 4.3 days (SD, 12.8 days), of nausea and/or vomiting was 6.0 days (SD, 9.8 days), and of halos was 6.4 days (SD, 9.2 days). Within 48 hours of initial treatment with medication, all patients received LPI in the affected eye. Three eyes underwent additional surgical iridotomy because of severe corneal oedema, subsequent to a non-patent iridotomy. Seventeen eyes (23.9%) revealed a vertical cup-disc ratio of >0.4. Six of 11 eyes had widening of the anterior chamber angle (increase at least 1 grade by the Shaffer grading system) for more than 180° after LPI. Prophylactic LPI was successfully done in the contralateral eye of all patients and no episodes of APAC occurred.

Forty five eyes (66.2%) had the IOP controlled to <21 mm Hg after LPI without any further surgical intervention. In this group, 37 patients (38 eyes; 84.5%) were discharged from hospital with topical medication and the remaining 7 patients (7 eyes; 15.5%) needed no antiglaucoma medication. Twenty three eyes (33.8%) had uncontrolled IOP and filtration surgery was indicated. Comparisons of demographic data and duration of each presenting symptom between the filtration surgery and non-filtration surgery groups are presented in Table 1. Duration of ocular pain, red eye, decreased vision, and halos were significantly longer in the filtration surgery group than in the non-filtration surgery group. There were no statistically significant differences in age, sex, headache duration, nausea/vomiting duration, or presenting IOP. Figures 1 and 2 demonstrate the sequential changes of mean IOP and pupil size after initial treatment, respectively, of the filtration surgery group and the non-filtration surgery group. The mean IOP for the non-filtration surgery patients was significantly lower than for the filtration surgery patients at 24 and 48 hours ($p = 0.001$ and $p < 0.001$, respectively), but not after 48 hours or at discharge from hospital. Forty eyes (88.8%) in the non-filtration surgery group

Table 1. Comparison of demographic data and duration of each presenting sign and symptom between the filtration surgery and non-filtration surgery groups.

	Non-filtration surgery group	Filtration surgery group	p Value
Number of patients	43	23	
Number of eyes	45	23	
Age (years) [mean (SD)]	60.1 (10.4)	61.1 (9.8)	0.696*
Sex			0.690*
Male	9	6	
Female	34	17	
Initial intraocular pressure (mm Hg) [mean (SD)]	58.5 (14.4)	57.6 (14.3)	0.820*
Duration of symptoms (days)			
Ocular pain	4.6 (6.9)	8.0 (8.5)	0.008*
Decreased vision	4.6 (7.1)	7.5 (8.7)	0.019*
Red eye	3.8 (5.5)	7.2 (5.2)	0.003†
Headache	2.9 (4.5)	5.9 (4.1)	0.126*
Nausea/vomiting	1.3 (0.9)	2.4 (2.6)	0.083*
Halos	2.9 (2.6)	7.0 (2.8)	0.006*

* Student *t* test.
† Mann-Whitney *U* test.

had IOP <20 mm Hg within 24 hours, whereas in the filtration group, the IOP could not be controlled despite a patent LPI and medical treatment 48 hours after admission. The mean pupil size was significantly smaller in the non-filtration surgery group at 24, 48, and after 48 hours and at discharge ($p < 0.001$ at each time point). Seventeen eyes (37.8%) in the non-filtration surgery group and 8 eyes (34.8%) in the filtration surgery group had initial BCVA better than 6/60. After initial treatment, 32 eyes (72.7%) in the non-filtration surgery group had BCVA better than 6/60 at 24 hours, and this increased to 34 eyes (77.3%) at 48 hours, compared with 11 eyes (55.0%) and 12 eyes (60.0%), respectively, in the filtration surgery group. Change in BCVA in the non-filtration surgery eyes is shown in Figure 3. At discharge, patients in the non-filtration surgery group had a mean IOP of 13.7 mm Hg (SD, 4.5 mm Hg), mean pupil size of 3.6 mm, and BCVA better than 6/60 in 39 eyes (86.7%). In the filtration surgery group, the mean IOP was 11.5 mm Hg (SD, 3.9 mm Hg), the mean pupil size was 5.3 mm, and 15 eyes (65.2%) had BCVA better than 6/60. Risk assessment analysis showed that the duration of ocular pain ($p = 0.033$), decreased vision ($p = 0.034$), red eye ($p = 0.029$), and pupil size at 24 hours ($p = 0.011$), 48 hours ($p = 0.002$), and at discharge ($p = 0.004$) were significant risks for surgical intervention.

Table 2 demonstrates the automated Humphrey visual field obtained for 19 patients (20 affected eyes) within 3 months of discharge from hospital. All affected eyes showed a visual field defect, with mean deviation (MD) of -14.6 (range, -29.4 to -1.2), whereas the contralateral eyes had a normal visual field in 4 and 5 eyes in the non-filtration surgery and filtration surgery groups, respectively. There were no statistically significant differences between the non-filtration surgery and filtration surgery groups for pattern of visual field defect and MD in the affected eyes.

Figure 1. Sequential changes of mean intraocular pressure for the filtration surgery and non-filtration surgery groups.

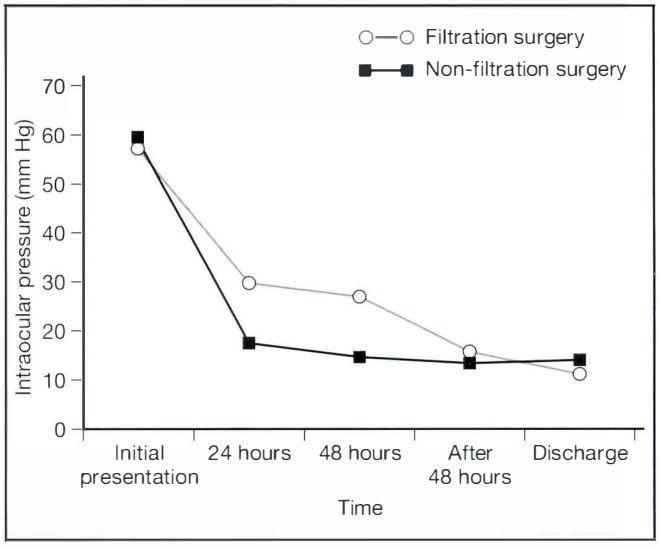

Figure 2. Change in pupil size for the filtration surgery and non-filtration surgery groups.

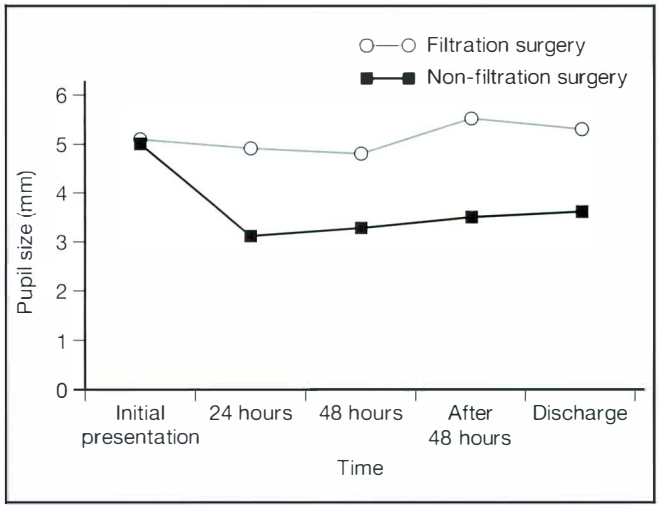

Figure 3. Change in best-corrected visual acuity for the non-filtration surgery group before and after laser peripheral iridotomy and medications.

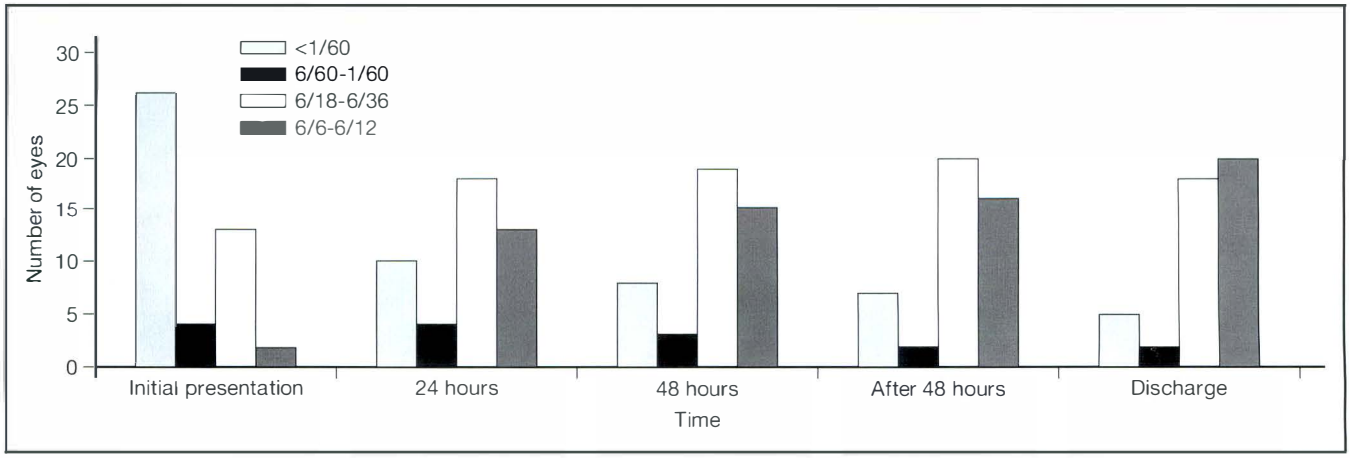

Table 2. Visual field defect patterns in 20 affected and fellow eyes of 18 patients with acute primary angle closure.*

	Visual field defect	Number of eyes
Affected eyes		
Non-filtration group	Generalised depression	2
	Tubular field	3
	Nasal step	2
	Generalised depression with arcuate scotoma	4
	Temporal wedge	1
	Superior altitudinal defect	1
Filtration group	Generalised depression	3
	Tubular field	1
	Arcuate scotoma	2
	Paracentral scotoma	1
Fellow eyes		
Non-filtration group	Generalised depression	5
	Tubular field	3
	Normal	4
Filtration group	Generalised depression	1
	Normal	5

* One patient had bilateral acute primary angle closure.

Discussion

Acute angle closure (AAC) is a common ophthalmologic emergency in Thailand. The clinical recognition and prompt diagnosis of AAC by general practitioners and ophthalmologists is the most important means of establishing early treatment, which can prevent long-term morbidity. The clinical criteria for APAC diagnoses vary and have long been used inconsistently; indeed, no standard criteria have been established. Recently, Saw et al published the findings of evidence-based research of interventions for ACG.[11] The definitions for angle closure diagnosis were classified into AAC, acute angle closure glaucoma (AACG), primary angle closure (PAC), and PACG.[11] The most widely adopted system for diagnosis of APAC is the presence of at least 2 of the following symptoms: ocular or periocular pain, nausea and/or vomiting, and a history of intermittent blurring of vision with halos; and at least 3 of the following signs: IOP >21 mm Hg, conjunctival injection, corneal epithelial oedema, mid-dilated non-reactive pupil, and shallow anterior chamber in the presence of an occludable angle.[9,11] This study focused on the clinical presentations as well as the course of APAC after initial treatment.

The demographic data in this study are similar to those reported in previous studies, except that Thais were the predominant race in this study.[9-11] Women were affected more often than men by a ratio of 3:1. This confirms several studies that found that elderly women are at increased risk for developing APAC.[4,10] This study also found that the right eye was predominately affected by a ratio of 4:3.

The treatment regimen used for this study underscores the effectiveness of both medical and surgical intervention for IOP control. LPI is an effective treatment for APAC or PAC and is effective prophylaxis for the contralateral eye.[5-8] The reported success rate after LPI is higher in Caucasian than in Asian individuals.[6,9,10,12,13] In eyes of Caucasian individuals with APAC, the IOP can be controlled with iridectomy alone in 65% to 76% of patients, and only 1% to 13% require filtration surgery.[6,12,13] In Asian individuals, more than half the eyes develop chronic ACG after APAC, despite a patent LPI, and approximately one-third require filtration surgery.[9,10] In this study, 33.8% of eyes needed filtration surgery in the acute phase, and the remaining 66.2% had controlled IOP after iridectomy, of which more than 80% needed topical medication. This finding supports the fact that eyes in Asian individuals may require filtration surgery more often than those of Caucasians.[6,9,13-15]

In this study, risk assessment analysis showed that the duration of presenting symptoms and pupil size after presentation were indications for surgical intervention. If the patient had a long duration of symptoms, especially longer than 1 week, or the pupil could not be constricted within 24 hours of presentation, the

patient had a tendency to have uncontrolled IOP requiring surgery. The possible mechanisms for this include trabecular damage, inflammation, the apposition of the iris to the trabecular meshwork, and the development of PAS.[16] Saunders concluded that the duration of symptoms prior to presentation was a significant factor in distinguishing between patients who could be treated successfully with iridotomy and those who would need additional medication or surgery.[17] The reported possible risk factors for an increase in IOP after APAC include age younger than 60 years, any cardiovascular risk factors, and delayed presentation of more than 3 days.[9]

Pilocarpine was included in the medical treatment regimen in this study. Pilocarpine has been shown to be an effective treatment for APAC, as has been found in several studies.[18-20] Pilocarpine can change the anterior chamber angle, break pupillary block, and pull the iris root away from the trabecular meshwork, resulting in acute phase resolution.[21-23] On the other hand, pilocarpine can cause forward movement of the lens, aggravating any shallowness of the anterior chamber.[23,24] LPI and medical treatment are still the main treatments, although new modalities to treat the acute phase of APAC have been developed, including Argon laser peripheral iridoplasty,[25-28] primary lens extraction,[29,30] and anterior chamber paracentesis.[31]

Aung et al reported a failure rate of 33.4% after trabeculectomy for patients for whom medical treatment for APAC had failed (mean follow-up, 22 months, and no antimetabolite application).[15] These authors concluded that trabeculectomy is not recommended as a first-line treatment for patients with medically unresponsive APAC because of a high risk of surgical failure and complications after filtration surgery.[15] Surgical outcome after filtration surgery to control IOP in the patients in this study was effective. All patients in the filtration surgery group had IOP <21 mm Hg at discharge. Sixty five percent of patients had BCVA better than 6/60 compared to only 35% preoperatively. Long-term follow-up is needed to ascertain the long-term outcome of surgical treatment for APAC.

After initial treatment, only 17 eyes (23.9%) had vertical cup-disc ratios >0.4. This implies that most of the patients might have ocular anatomical predisposing factors rather than pre-existing PACG. After APAC, all eyes had visual field defects, which may be a result of APAC. Preventive ophthalmology therefore has an important role for early identification of high-risk patients, as LPI has been proven to be efficient prophylaxis for APAC.[6,32]

This was a retrospective study with several limitations. Multiple ophthalmologists were involved, leading to a variety of diagnostic criteria, treatments, and the inconsistency of IOP measurement taken by Goldman applanation or Schiotz tonometry. The gonioscopy and visual field details were often incomplete. A prospective study should be conducted to determine the long-term outcome after APAC as well as factors affecting the treatment.

The predictive factors for the need for surgical treatment were long duration of symptoms (longer than 1 week) and non-constricted pupil within 24 hours of initial treatment. LPI and medication can control an APAC and its sequelae for most patients, with one-third of patients requiring surgical intervention for IOP control.

Acknowledgements

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Relationship between *Helicobacter pylori* Infection and Open Angle Glaucoma in China

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Aim: *To determine the prevalence of* Helicobacter pylori *infection in patients with open angle glaucoma and control participants.*
Methods: *Twenty four Chinese patients with glaucoma were investigated, including 18 patients with primary open angle glaucoma, 4 with normal tension glaucoma, 1 with ocular hypertension, and 1 with pigmentary glaucoma. Twenty four age-matched control participants from the general ophthalmology clinic were also enrolled.* ^{13}*C-urea breath test was performed to detect* Helicobacter pylori *infection.*
Results: *Positivity of* Helicobacter pylori *detected by* ^{13}*C-urea breath test was significantly higher in patients with glaucoma (54.2%) than in control participants (20.8%) [p = 0.017]. The odds ratio for association between* Helicobacter pylori *and primary open angle glaucoma was 4.49, and the 95% confidence interval ranged from 1.26-16.01. The mean visual field defect and cup-disc ratio of patients with glaucoma showed no significant differences between patients who were* Helicobacter pylori*-positive or* Helicobacter pylori*-negative.*
Conclusion: *This study suggests that* Helicobacter pylori *infection might be associated with open angle glaucoma in Chinese patients.*

Key words: *Glaucoma, open-angle,* Helicobacter pylori

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Introduction

Open angle glaucoma (OAG) is one of the most common causes of blindness in the world, but the mechanism remains unknown. According to recent evidence, this disease may be associated with changes in endothelium-dependent vascular regulation[1,2] and impaired ocular blood flow.[3] Moreover, accumulating evidence suggests that autoimmune mechanisms may be responsible for progressive glaucomatous optic neuropathy in some patients with glaucoma.[4,5]

Helicobacter pylori infection is one of the most common infections, although the infection rate varies significantly among different countries.[6] This infection has been linked conclusively with gastric ulceration[7,8] and gastric carcinoma.[9] In addition, *H pylori* has been implicated in numerous extra-digestive conditions, including cerebrovascular disorders,[10] vascular disorders,[3] coronary heart disease,[11] and some autoimmune conditions such as Sjögren's syndrome[12] and immune thrombocytopenic purpura.[13] A significantly higher prevalence of *H pylori* in patients with glaucoma than in those without glaucoma in Greece has been reported,[14] suggesting a potential pathogenetic association between *H pylori* infection and glaucoma. Furthermore, the same study group documented that *H pylori* eradication may be beneficial in the management of chronic OAG.[15] This study aimed to investigate the prevalence of *H pylori* infection in Chinese patients with different stages of OAG compared with healthy control individuals.

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Methods

This was a case-control study performed at the Peking University Eye Center, Beijing, China, from June 2004 to June 2006.

Patients

Twenty four consecutive Chinese patients with glaucoma were enrolled in the study. Eighteen patients had primary OAG (POAG), 4 had normal tension glaucoma (NTG), 1 had ocular hypertension (OHT), and 1 had pigmentary glaucoma (PG). All patients met the inclusion criteria of:

- intraocular pressure (IOP) ≥21 mm Hg (<21 mm Hg for patients with NTG and >21 mm Hg on 2 or more occasions without abnormal optic disc or visual field changes for patients with OHT)
- glaucomatous optic nerve head changes, including rim thinning, notching in the inferior or superior temporal area of the optic nerve head, or total glaucomatous cupping
- visual field loss such as a scotoma or a nasal step.

PG was considered a type of POAG, caused by occlusion of the trabecular meshwork by pigment deposited in the anterior chamber.

The control group consisted of 24 consecutive age-matched participants selected from the general ophthalmology clinic at the same hospital. Control participants underwent slit-lamp examination, direct ophthalmoscopy, IOP measurement, and visual field examination. No control participants had glaucomatous optic nerve head changes or visual field changes and their IOP was <21 mm Hg.

All patients and control participants underwent automated perimetry examination with the Octopus program TG-2 (Octopus 101 automated perimetry; Interzeag AG, Switzerland). IOP was measured using a calibrated Goldmann applanation tonometer and visual acuity was measured using the Snellen eye chart.

Exclusion criteria for both groups included diabetes mellitus, severe systemic diseases or neoplasms, myopic refractive error exceeding –10 D, and serious eye disease. Furthermore, participants were also excluded if they had taken H_2-receptor antagonists, proton pump inhibitors, antibiotics, bismuth compounds, or non-steroidal anti-inflammatory drugs in the previous 4 weeks.

All patients and control participants were fully informed and signed consent forms. The local ethics committee approved the study protocol.

Design

^{13}C-urea breath test (^{13}C-UBT; Automated Breath ^{13}Carbon Analyser; Europa Scientific Limited Co, Crewe, UK) was performed after overnight fasting. Breath samples were collected from each participant in plastic tubes before ingestion of ^{13}C-urea 45 mg dissolved in 50 mL water and 20 and 30 minutes afterwards. A value higher than 0.4 was considered positive.

Results

The mean age and sex ratios were not significantly different between patients with glaucoma and the control participants. The demographic and clinical characteristics of the study participants are shown in Table 1.

For patients with glaucoma, the mean visual acuity was 0.8 (range, 0.2 to 1.0), the mean IOP was 15 mm Hg (range, 6 to 22 mm Hg), the mean deviation (MD) was 7.13 dB, and the mean cup-disc ratio was 0.67. Table 2 shows the antiglaucoma therapy used by the patients.

Table 1. Demographic and clinical characteristics of the study participants.

Characteristic	Patients	Controls	p Value
Age (years)			
Mean (SD)	63.9 (14.4)	60.8 (14.6)	0.464
Range	22-81	23-80	
Sex			
Male/female	18/6	13/11	0.227
^{13}C-urea breath test-positive (%)	13 (54.2)	5 (20.8)	0.017

Table 2. Antiglaucoma therapy for patients with glaucoma.

Treatment	Number of eyes
Medication	
β-Blocker	18
α-Agonist	4
Latanoprost	2
β-Blocker plus α-agonist	3
β-Blocker plus latanoprost	1
α-Agonist plus latanoprost	1
Surgery	
Trabeculectomy	9
Non-penetrating deep sclerectomy	4
Medicine and surgery	
Trabeculectomy plus β-blocker	2
Trabeculectomy plus β-blocker plus α-agonist	2
Non-penetrating deep sclerectomy plus β-blocker	1
None*	1

* This eye was the fellow eye of the patient with ocular hypertension; the intraocular pressure and visual field were normal.

Table 3. Mean visual field defect and cup-disc ratio of patients with glaucoma and their *Helicobacter pylori* status by ^{13}C-urea breath test (^{13}C-UBT).

	^{13}C-UBT positive	^{13}C-UBT negative	p Value
Number of patients	13	11	
Mean deviation (SD) [dB]	6.40 (5.32)	8.04 (8.22)	0.411
Mean cup-disc ratio (SD)	0.64 (0.20)	0.70 (0.18)	0.272

The prevalence of *H pylori* infection determined by ^{13}C-UBT was 54.2% (13 of 24) in patients with glaucoma and 20.8% (5 of 24) in the control group. The odds ratio for association between *H pylori* and POAG was 4.49 and the 95% confidence interval ranged from 1.26 to 16.01. Table 3 shows that the mean visual field defect and cup-disc ratio did not achieve statistical significance for *H pylori* positivity.

Discussion

In this study, 24 patients with glaucoma were investigated and the results compared with 24 age-matched control participants. ^{13}C-UBT was used to detect *H pylori* infection. *H pylori* positivity detected by ^{13}C-UBT was significantly higher in patients with glaucoma (54.2%) than in the control participants (20.8%) [p = 0.017]. These results show a high prevalence of *H pylori*

infection associated with open angle glaucoma. There was no association between *H pylori* infection and stage of glaucoma.

^{13}C-UBT was used to detect *H pylori* infection because it is the best non-invasive method for detecting current *H pylori* infection, and is preferred to an invasive method for screening purposes. ^{13}C-UBT is an increasingly popular method for screening for *H pylori,* as it employs an innocuous non-radioactive isotope that can be safely used, and the sensitivity and specificity are high.[16] The test exploits the hydrolysis of orally administered urea by the enzyme urease, which *H pylori* produces in large quantities. Urea is hydrolysed to ammonia and carbon dioxide, which diffuses into the blood and is excreted by the lungs. Isotopically-labelled carbon dioxide can be detected in the breath.

It was noted that *H pylori* infection was strongly associated with age and socioeconomic conditions. Most of the participants came from Beijing. *H pylori* infection is endemic in China,[6] but the infection rate is significantly different between urban and rural locations. The infection rate for the control participants in this study was 20.8%, which is similar to the infection rate in cities, in which the socioeconomic conditions are similar to those of Beijing.

These authors suggest that *H pylori* infection could affect the pathophysiology of glaucoma as follows:

- promoting platelet and leukocyte aggregation
- releasing proinflammatory and vasoactive substances such as cytokines (interleukins 1, 6, 8, 10, 12, tumour necrosis factor-α, and interferon-γ, eicosanoids (leukotrienes and prostaglandins), and acute phase proteins (fibrinogen and C-reactive protein) involved in various vascular disorders[17] (migraine, systemic hypertension, Raynaud's phenomenon, cardiovascular disease, and possibly glaucoma)[18]
- stimulating mononuclear cells to induce a tissue factor–like procoagulant activity that converts fibrinogen into fibrin
- causing mimicry between endothelial and *H pylori* antigens
- producing oxidative stress and circulating lipid peroxides
- influencing the apoptotic process.

H pylori infection and glaucoma share the Fas/FasL and the mitochondria-mediated apoptotic pathways.[19] These variables might also exert their own effects in the induction or progression of glaucomatous optic neuropathy and other neurodegenerative disorders.[18,20]

Another comparative study has identified a possible association between *H pylori* and glaucoma,[14] suggesting a common factor that predisposes patients to both *H pylori* infection and glaucoma. The same study group also determined that *H pylori* eradication may positively influence the features of glaucoma, suggesting a possible causal link between *H pylori* and glaucoma.[15] However, another prospective case-control study was unable to provide additional confirmation of a link between *H pylori* and glaucoma.[21] The studies mentioned above all used invasive testing methods, relying on histologic analysis or serologic detection of antibodies.

The gold standard for establishing a diagnosis of current *H pylori* infection is endoscopic biopsy,[22] but the procedure is costly and uncomfortable for patients. In addition, it is not justified for healthy individuals or patients who are frail or have systemic disease. Furthermore, biopsy-based tests may have sampling errors and are related only to local infection in the stomach. Serologic testing by enzyme-linked immunosorbent assay requires invasive sampling of blood[23] and does not discriminate between current and old infections.[18]

^{13}C-UBT is a good screening method for current *H pylori* infection, although it is expensive. However, this test requires fasting, false-negative results may occur if antibiotics have been used within the previous 4 weeks, and false-positive results can occur from urease present in the mouth.[24] Some patients with glaucoma in this study were using topical antiglaucoma medicine. Whether these drugs have an effect on infectious manifestations needs further research.

The significance of these findings is limited by the small number of patients. Unlike Kountouras et al's report,[25] this study found no association between *H pylori* infection and the stage of the glaucoma, because the mean visual field defect and cup-disc ratio of patients with glaucoma showed no significant differences between patients who were *H pylori*-positive or *H pylori*-negative.

These data suggest that *H pylori* infection is associated with glaucoma, but there is no association between *H pylori* infection and the stage of glaucoma. The possibility that patients with glaucoma could be more susceptible to infectious diseases may be explained by the existence of a common genetic factor that predisposes to both *H pylori* and glaucoma[14] or the *H pylori* infection may be a causal factor for OAG.

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Contact Lens–associated Infectious Keratitis in Thailand

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Aim: *To evaluate the clinical characteristics, risk factors, management, and outcome of infectious keratitis associated with contact lens wear.*
Methods: *The study comprised all consecutive patients presenting with contact lens–related presumed microbial keratitis during a 5-year period. Detailed demographic data, type of contact lens, risk factors, clinical findings, microscopic profile, treatment, and final visual outcome were evaluated.*
Results: *Thirty six patients had contact lens-associated bacterial keratitis; 30 patients used daily-wear soft lenses, 4 used extended-wear soft lenses, and 2 used hard lenses.* Pseudomonas aeruginosa *was isolated in 38.8% of patients and* Staphylococcus aureus *in 13.8%;* β*-haemolytic* Streptococcus, Serratia marcescens, *and* Staphylococcus epidermidis *were also common pathogens. The risk for keratitis due to overnight contact lens wear was 33.3%. Twenty six patients (72.2%) presented with initial visual acuity of 6/36 or worse. Visual acuity following treatment improved for 13 patients (36.1%).*
Conclusion*: Contact lens-associated keratitis was seen most frequently in patients using daily-wear soft contact lenses.* Pseudomonas aeruginosa *was the most commonly encountered causative agent. Overnight contact lens wear is a major risk factor for keratitis among contact lens wearers. Therefore, overnight wear should be avoided.*

Key words: *Contact lenses, Keratitis,* Pseudomonas aeruginosa

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Introduction

Many patients wear contact lenses for cosmetic, visual, or therapeutic purposes. The number of patients wearing contact lenses for visual purposes has increased during the past few decades. Contact lens wear has become a major predisposing factor for microbial keratitis.[1] Infectious keratitis is the most devastating complication of contact lens use and may result in permanent visual loss from corneal scar or perforation.[2] There have been a number of case reports of severe microbial keratitis caused by contact lenses.[3-5]

The objective of this study was to investigate the clinical findings, risk factors, management, and outcome of infectious keratitis associated with contact lens wear.

Methods

Patients

The records of 310 patients admitted to the Department of Ophthalmology, Chiang Mai University Hospital, Chiang Mai, Thailand, with infectious keratitis between 1 August 2000 and 1 August 2005 were reviewed retrospectively. Patients with keratitis not associated with contact lens use were excluded. The remaining 36 patients (11.6%) were assessed with regard to demographic data, type of contact lens, risk factors, duration of symptoms before admission, initial visual acuity, clinical finding of keratitis, underlying systemic and ocular disease, microscopic examination and sensitivity testing, treatment, and final visual outcome. All 36 patients were strongly suspected to have contact lens–related infectious keratitis.

Procedure

After slit-lamp examination of the affected eye, the topical anaesthetic benoxinate hydrochloride (Novesin 0.4%; Novartis Ophthalmics, Hettlingen, Switzerland) was instilled prior to corneal scraping. Specimens were obtained by using a Kimura spatula or sterile 25-G needle. Laboratory investigations included gram stain, 10% potassium hydroxide wet mount, and inoculation on blood agar, chocolate agar, and Sabouraud dextrose agar. Specimens from the contact lenses, contact lens care solutions, and contact lens cases were also cultured when they were available. Selected media were used for the patient who was suspected of having *Acanthamoeba* infection. In vitro disc sensitivity tests were performed on positive cultures.

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All patients were initially treated with topical fortified cefazolin sodium 33 mg/mL and gentamicin sulphate 14 mg/mL every hour for the first 48 hours to provide broad-spectrum activity against gram-positive and gram-negative bacteria while awaiting culture and sensitivity results. Topical antibiotic agents were progressively tapered and/or modified according to the clinical responses.

Descriptive statistical analysis was performed. The research was approved by the Research Ethics Committee, Faculty of Medicine, Chiang Mai University.

Results

There were 36 patients (10 men and 26 women) with bacterial keratitis following contact lens use. The mean age was 25.8 years (SD, 6.6 years; range, 17 to 41 years). All patients wore contact lenses for refractive correction. The study population consisted predominantly of young adults. Most of the patients were women (Table 1).

The clinical data are summarised in Table 2, according the type of contact lens worn: hard contact lens (2 eyes), daily-wear soft contact lens (30 eyes), and extended-wear soft contact lens (4 eyes). The mean duration of symptoms before admission was 5.9 days (SD, 4.6 days; range, 1 to 21 days). Most patients (72.2%) presented with initial visual acuity of 6/36 or worse.

Twenty two patients (61.1%) had prior treatment with antibiotics obtained from a pharmacy. Five patients (13.9%) had been treated by ophthalmologists or general practitioners. Nine patients (25.0%) had not been previously treated. Most patients (97.2%) wore fitted contact lenses from optical shops. One patient had contact lenses fitted by an ophthalmologist. Eighteen patients used disposable soft contact lenses.

Nineteen patients had a greatest ulcer diameter of ≥2.5 mm. Only 4 patients (patients 6, 16, 19, and 22) had hypopyon. No patients had corneal perforation, although 3 patients with *Pseudomonas aeruginosa* infection (patients 6, 16, and 19) demonstrated marked stromal thinning with approximately 80% loss of normal thickness. Cultures isolated from corneal scraping were positive in 25 patients (69.4%). The bacterial cultures isolated from contact lenses, contact lens cases, and contact lens solutions were similar to the bacteria isolated in the corneal specimens in 3 patients (8.3%; patients 7, 8, and 16). Thirteen patients (36.1%) achieved final best-corrected visual acuity of 6/36 or better.

Table 1. Demographic data of patients with contact lens–associated infectious keratitis.

	Number of patients (%)
Mean age (years)	25.8
Range (SD)	17-41 (6.6)
Sex	
Male	6 (16.7)
Female	30 (83.3)
Affected eye	
Right	17 (47.2)
Left	19 (52.8)

Discussion

Contact lenses are a successful method of visual correction. However, under certain circumstances, inflammatory adverse responses can occur during contact lens wear and the most severe of these is contact lens–related infectious keratitis, which has the potential to cause visual loss.[6] Estimates for the incidence of contact lens–related infectious keratitis depend on the type of contact lens being worn.[2,7] Two types of contact lens are commonly used throughout the world. These are rigid gas permeable lenses and soft hydrogel lenses. In this study, contact lens–related infectious keratitis accounted for 11.6% of all patients who were admitted with infectious keratitis and most cases (83.3%) were associated with daily-wear soft contact lenses. The potential limitation of this study was that only inpatient records were assessed, so the number of infections may be underestimated. Half of the patients with infectious keratitis wore disposable contact lenses. Theoretically, disposable contact lenses are discarded daily and the frequent use of new contact lenses should reduce the risk for infection. Therefore, the risk attributed to contamination of contact lens–associated equipment also needs to be considered.

Various factors that occur during contact lens wear can affect the risk for developing infectious keratitis. This study showed that overnight wear is a major risk factor, which accounted for 33.3% of infections. Overnight wear of contact lenses has been found to be the principal risk factor associated with infectious keratitis in many studies.[7-10] Interestingly, the risk for corneal infection with soft contact lens overnight wear was 10 to 20 times that for no overnight wear.[8] Levy et al showed that patients who wear contact lenses while sleeping may experience hypoxia, epithelial oedema, and superficial punctate keratitis, which may predispose to corneal infection.[11] Some hypotheses such as large numbers of microbes, low tear secretion, and increased bacterial adhesion with biofilm have been demonstrated.[12] Consequently, Schein et al have shown that 49% to 74% of cases of contact lens–associated ulcerative keratitis could be prevented by eliminating overnight wear.[9] Other risk factors in the study presented here included corneal abrasion, topical steroid use, dry eye, human immunodeficiency virus infection, frequent overwear, diabetes, and chronic blepharitis.

Previous reports have shown culture-positive rates ranging from 43% to 76%.[2,13,14] Corneal scrapings from 69.4% of patients yielded positive cultures in the present study. Prior antibiotic treatment and pre-scraping anaesthetics containing preservatives may

Table 2. Characteristics of patients with contact lens–associated infectious keratitis according to type of contact lens.

Patient number	Duration of symptoms (days)	Risk factors	Corneal culture results	Initial visual acuity	Ulcer size (mm)	Ulcer location	Final visual acuity
Hard contact lens							
1	3	None reported	Negative	6/60	2.0	Centre third	2/60
2	5	Topical steroid use	*Pseudomonas aeruginosa*	CF	4.5	Centre third	CF
Daily-wear soft contact lens							
3	6	None reported	Negative	3/60	2.0	Centre third	CF
4	4	Overnight wearing	β-Haemolytic *Streptococcus*	6/36	2.0	Superior third	6/36
5	7	Dry eye	*Staphylococcus aureus*	CF	6.0	Centre third	CF
6	12	Corneal abrasion	*Pseudomonas aeruginosa*	CF	5.5	Centre third	CF
7	5	Overnight wear	*Pseudomonas aeruginosa*	6/60	2.0	Centre third	1/60
8	2	Overnight wear	*Pseudomonas aeruginosa*	6/36	2.5	Centre third	6/24
9	1	None reported	*Staphylococcus epidermidis*	6/12	2.0	Peripheral third	6/12
10	20	Topical steroid use	*Pseudomonas aeruginosa*	CF	8.0	Centre third	CF
11	14	Corneal abrasion	Negative	6/60	1.5	Centre third	3/60
12	2	None reported	Negative	6/12	1.0	Peripheral third	6/9
13	9	Overnight wear	Negative	CF	2.5	Centre third	CF
14	7	Overnight wear	*Pseudomonas aeruginosa*	6/60	5.0	Inferior third	CF
15	4	None reported	*Staphylococcus aureus*	6/36	1.5	Centre third	6/36
16	3	Overnight wear	*Pseudomonas aeruginosa*	6/60	5.5	Centre third	CF
17	21	None reported	Negative	CF	2.0	Centre third	No result
18	5	Chronic blepharitis	*Staphylococcus aureus*	6/24	3.0	Centre third	6/12
19	10	Overnight wear	*Pseudomonas aeruginosa*	CF	4.5	Centre third	6/60
20	2	Water pistol shot in eye	*Pseudomonas aeruginosa*	6/36	3.0	Centre third	6/60
21	7	Dry eye	Negative	1/60	1.5	Centre third	4/60
22	9	Overnight wear	β-Haemolytic *Streptococcus*	CF	7.0	Inferior third	CF
23	2	Corneal abrasion	*Staphylococcus epidermidis*	6/24	2.0	Centre third	6/12
24	6	HIV infection	*Pseudomonas aeruginosa*	6/24	2.0	Peripheral third	6/9
25	6	Overnight wear	*Pseudomonas aeruginosa*	6/36	2.5	Centre third	3/60
26	7	None reported	Negative	4/60	2.5	Centre third	6/60
27	3	HIV infection	*Serratia* species	CF	5.5	Centre third	HM
28	4	Overnight wear	*Pseudomonas aeruginosa*	6/12	2.0	Centre third	6/60
29	2	Overnight wear	Negative	6/9	1.0	Centre third	6/6
30	1	Overnight wear	*Staphylococcus epidermidis*	6/12	1.5	Centre third	6/9
31	4	Chronic blepharitis	*Staphylococcus aureus*	6/36	2.5	Peripheral third	6/24
32	4	None reported	Negative	6/60	2.0	Centre third	6/36
Extended-wear soft contact lens							
33	3	Frequent overwear	*Staphylococcus aureus*	6/60	3.0	Centre third	6/36
34	5	None reported	Negative	6/24	2.5	Centre third	6/60
35	3	Diabetes	*Pseudomonas aeruginosa*	CF	6.0	Centre third	CF
36	7	Frequent overwear	*Pseudomonas aeruginosa*	6/24	2.0	Centre third	6/60

Abbreviations: CF = counting fingers; HM = hand movement; HIV = human immunodeficiency virus.

have decreased the positive-culture result. Bennett et al found a relationship between lesion size and positive culture when the lesion was ≥4.0 mm².[15] Not surprisingly, the negative culture results occurred in patients with a greatest ulcer length of ≤2.5 mm in this study.

These findings and those of other reports confirm that *P aeruginosa* is the most frequently isolated pathogen in contact lens–related keratitis. A corneal epithelial defect and inoculation with *Pseudomonas* microorganisms have been implicated in the pathogenesis of *Pseudomonas* corneal infection.[16] Contact lenses may compromise the ocular surface by interfering with the corneal epithelium of normal tear flushing and from the non-specific humoral immune mechanisms.[17] Superficial punctuate keratitis caused by microtrauma may lead to adhesion of the bacterial surface to the cornea, establishing a corneal ulcer.[12] A *Pseudomonas* corneal ulcer is usually located centrally,[18] which was found in this series. Recent studies have reported a high incidence of gram-positive organisms from contact lens–related infectious keratitis,[19,20] although up to 47.3% of patients have gram-negative bacteria. The bacteria recovered from the corneal scrapings in this study were identical to the bacteria isolated from the contact lenses and contact lens care equipment for 3 patients. These items can therefore be presumed to be the source of contamination. *Pseudomonas* has a high degree of survivability in water and moist products. Improper care of contact lenses predisposes a patient to use of contact lenses contaminated with *Pseudomonas*.

Interestingly, *Acanthamoeba* and fungal organisms were not found in this study. The probable explanation for this result lies in the urbanisation of Chiang Mai, where the study was conducted. Compared with rural agricultural areas, it is a rapidly growing

area of concrete construction, which does not produce the same high count of airborne fungal spores produced from vegetable and soil matter.

Despite early diagnosis and prompt aggressive treatment, the most severe case of keratitis (due to *Pseudomonas*) resulted in poor visual outcome. Therefore, prevention of infection is the most important concern. Careful patient selection and lens fitting, long-term medical supervision, and patient compliance with instructions will enable contact lenses to be used safely. When corneal infection does occur, it is almost certainly because at least one of these variables has been neglected. In this study, the main risk factor for corneal infection was overnight wear of contact lenses not designed for this purpose, which should be avoided.

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Acetazolamide-induced Glaucoma

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Two patients presented with bilateral flat anterior chamber and high intraocular pressure several hours after uneventful cataract surgery in the fellow eye. The affected eye had undergone uneventful cataract surgery 6 weeks earlier. A single dose of oral acetazolamide 500 mg had been given as a routine preoperative measure prior to each surgery. Bilateral malignant glaucoma was suspected and intravenous mannitol and oral acetazolamide 250 mg 4 times daily were administered, along with topical steroids, β-blockers, and atropine. Ultrasound biomicroscopy showed choroidal effusion in both patients. Acetazolamide was withdrawn and complete resolution of choroidal effusion occurred rapidly. Bilateral secondary angle closure glaucoma can masquerade as bilateral malignant glaucoma and is usually a result of choroidal effusion secondary to an idiosyncratic response to a drug. In these patients, the instigating agent was acetazolamide. A non-invasive treatment approach of drug withdrawal and conservative management is usually effective.

Key words: *Acetazolamide, Glaucoma*

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Introduction

Acetazolamide is a sulpha derivative that rarely produces an allergic reaction, although an allergic reaction may present as ciliary body oedema, uveal effusion, forward pushing of the lens iris diaphragm, and secondary angle closure glaucoma.[1]

This report is of 2 patients in whom an allergic reaction induced by acetazolamide was noted.

Case Report

Two patients presented with bilateral raised intraocular pressure (IOP) and shallow anterior chamber after cataract surgery to the second eye. The initial cataract surgery had been uneventful 4 to 6 weeks previously. Both patients had received oral acetazolamide during the initial and subsequent cataract surgeries. Ultrasound biomicroscopy (UBM) showed choroidal effusion. Both patients improved dramatically after withdrawal of the drug.

Patient 1

A 55-year-old woman presented to the Medical and Vision Research Foundation, Sankara Nethralaya, Chennai, India, in March 2005 with bilateral cataract. She was being treated for diabetes and hypertension. Best-corrected visual acuity (BCVA) was hand movements in the right eye and counting fingers at 2 m in the left eye. Slit-lamp examination was unremarkable except for brunescent cataracts in both eyes. IOP measured by applanation tonometry was 14 mm Hg in both eyes. The left eye appeared normal on indirect ophthalmoscopy. Ultrasound evaluation of the right eye was normal.

The patient underwent extracapsular cataract surgery with posterior chamber intraocular lens (IOL) implantation in her right eye. Her BCVA at 3 weeks was 6/6, N6. She had been given a single dose of oral acetazolamide 500 mg as a routine preoperative measure.

She underwent cataract surgery in her left eye 3 weeks later and had an uneventful intraoperative period. As per the protocol, she was given oral acetazolamide 500 mg. She presented to the emergency department 5 hours later with vomiting, reduced vision, pain, and watering in the fellow eye. Her visual acuity was counting fingers at 2 m (2/60). Slit-lamp examination revealed conjunctival chemosis, a hazy cornea, flat anterior chamber (van Herrick grade 0) [Figure 1], mid-dilated pupil, patent peripheral iridectomy, and a stable IOL in both eyes. Her IOP was 53 mm Hg in both eyes and posterior pole examination showed a hyperaemic disc with a normal macula.

Bilateral malignant glaucoma was suspected and intravenous mannitol and oral acetazolamide 250 mg 4 times daily were administered along with topical steroids, β-blockers, and atropine.

On the next day, both eyes showed a similar picture with flat anterior chambers and IOPs of 48 mm Hg in both eyes. Nd:YAG

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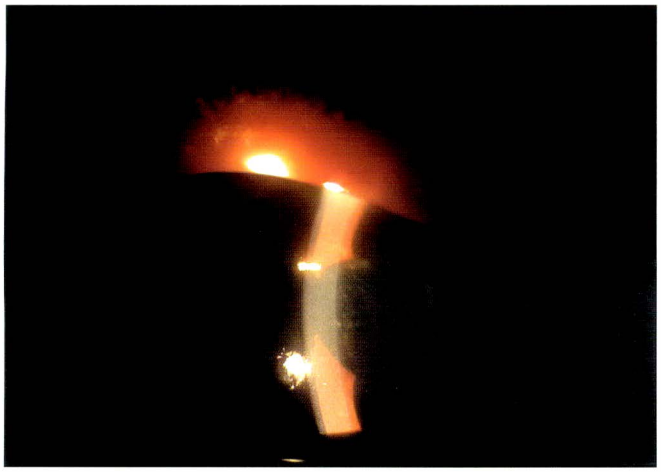
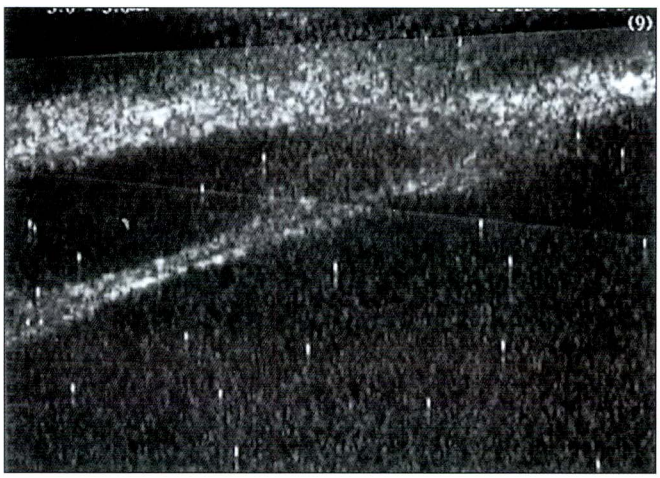
Figure 1. Slit-lamp photograph showing a shallow anterior chamber in the right eye of patient 1.



laser hyaloidotomy was done in the right eye with no improvement. An acetazolamide-related choroidal effusion was suspected and this was confirmed by UBM analysis (Figure 2). Acetazolamide was withdrawn, and topical medications were continued in addition to an α-adrenergic agonist. After 24 hours, the patient was comfortable with subjective improvement in vision. Examination revealed a clear cornea and a deep anterior chamber; the IOP was 30 mm Hg in both eyes. Progressive improvement was noted and antiglaucoma medications were withdrawn after 1 week and the steroid dose was tapered. After 1 month, her BCVA was 6/6, N6 in both eyes with normal slit-lamp findings. Her IOPs were 12 mm Hg and 14 mm Hg in the right and left eyes, respectively. Fundus examination revealed a healthy optic disc in both eyes and was unremarkable. Gonioscopy at 6 weeks revealed 4 clock hours of superior peripheral anterior synechiae (PAS) in the right eye, while the left eye had broad PAS in all quadrants with open areas in between. The patient attends the glaucoma clinic for periodic review.

Figure 2. Ultrasound biomicroscopy of the right eye of patient 1 showing choroidal effusion.



Patient 2

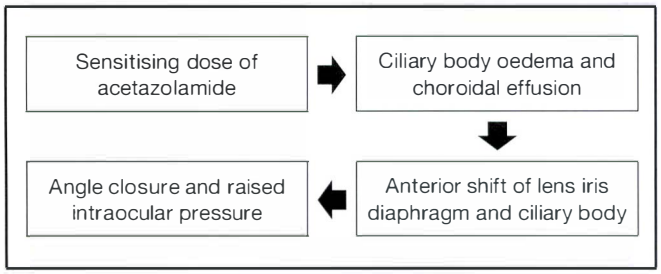
A 52-year-old woman underwent extracapsular cataract extraction with IOL implantation in her left eye. Oral acetazolamide 500 mg was given preoperatively. Her recovery was uneventful. After 6 weeks, her BCVA was counting fingers 3 m in the right eye and 6/9, N6 in the left eye. Her IOPs were 15 mm Hg and 10 mm Hg in the right and left eyes, respectively. She underwent extracapsular cataract surgery with IOL implantation in the left eye in May 2005 and was given preoperative oral acetazolamide 500 mg. Surgery was uneventful. On the first postoperative day, she developed shallow anterior chambers in both eyes and her IOPs were 26 mm Hg and 32 mm Hg in the right and left eyes, respectively. She had patent iridectomies in both eyes. Bilateral malignant glaucoma was suspected and she was prescribed oral acetazolamide 250 mg three times daily and topical timolol maleate eye drops twice daily. However, her IOPs remained high the following day and there was no change in the anterior chamber depth. UBM of the left eye showed a 360° choroidal effusion. An idiosyncratic reaction to acetazolamide was suspected and the drug was stopped; topical antiglaucoma medications were continued. On the third postoperative day, the anterior chamber was formed in both eyes and her IOPs were 18 mm Hg and 10 mm Hg in the right and left eyes, respectively. After 6 weeks, the BCVA was 6/9, N6 in both eyes, she had fully formed anterior chambers, and the IOP was 12 mm Hg in both eyes without antiglaucoma medications.

Discussion

Acetazolamide is no longer indicated when phacoemulsification is the primary mode of cataract surgery because of the closed chamber dynamics, although preoperative acetazolamide is necessary for extracapsular cataract surgery to counter positive pressure in an open globe. The clinical scenario described above can occur in a susceptible individual where there is a need for acetazolamide therapy for extracapsular cataract surgery or for other reasons.

The rare allergic reaction of bilateral choroidal effusion masquerading as malignant glaucoma and presenting in a postoperative scenario is a diagnostic challenge. Both the patients described in this report presented after the second cataract surgery. Pupillary block glaucoma was ruled out as the eyes had undergone conventional extracapsular cataract surgery and had a patent peripheral iridectomy. All features were typically suggestive of bilateral malignant glaucoma. However, there was no predisposing risk factor such as chronic angle closure or nanophthalmos. Rare instances of bilateral malignant glaucoma with spontaneous onset have been reported.[2,3] Failure of anterior hyaloidotomy and the authors' experience with another sulpha agent (topiramate)[4] raised

Figure 3. Mechanism of secondary glaucoma due to choroidal effusion.



the index of suspicion for glaucoma induced by choroidal effusion. UBM confirmed the diagnosis. Both patients presented after receiving the second dose of acetazolamide, suggesting a sensitisation mechanism to the reaction.[5] The time interval between the 2 doses was approximately 6 to 8 weeks for both patients.

It is known that sulphonamide drugs cause transient myopia and angle closure associated with supraciliary effusion. The exact reason for effusion following sulphonamide usage is still unknown, although it has been speculated that drug-induced elevated prostaglandins contribute to oedema in the ciliary body without evidence of a systemic allergic response.[6] The mechanism by which the effusion leads to angle closure and raised IOP is depicted in Figure 3.

It is essential to differentiate the scenario described in this report from malignant glaucoma, which requires an aggressive approach often culminating in surgery. An alternative diagnosis should be considered when the presentation is bilateral. A conservative approach of withdrawal of the instigating agent was effective for these 2 patients with drug-related ciliochoroidal effusion. Awareness about other sulpha derivative–induced reactions with a similar presentation[7-10] is important.

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Esthesioneuroblastoma: an Unusual Cause of Acute Visual Loss

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Esthesioneuroblastoma (olfactory neuroblastoma) is an uncommon malignant neoplasm of the nasal cavity and paranasal sinus region. Close proximity to the brain and visual apparatus can lead to significant morbidity. Ophthalmic manifestations are not common. This report is of an unusual presentation of this tumour with rapid locoregional spread.

Key words: *Esthesioneuroblastoma, olfactory, Eye manifestations*

Asian J Ophthalmol. 2007;9:216-8

Introduction

Esthesioneuroblastoma was first described in 1924. Esthesioneuroblastoma is a rare tumour, so visual manifestations are uncommon. The most common visual manifestations are proptosis, extraocular motility dysfunction, and intraorbital optic nerve involvement. This report is of a patient with esthesioneuroblastoma with profound acute visual loss.

Case Report

A 32-year-old woman was referred to the Department of Ophthalmology, Warrington Hospital, Warrington, UK, in April 2004 with acute profound visual loss in the left eye for 30 minutes. She was diagnosed with right-sided esthesioneuroblastoma of the ethmoidal sinus, confirmed by biopsy. At the time, she was 20 weeks pregnant, so waited for surgical resection of the tumour at 32 weeks' gestation with a planned caesarean section delivery. Her past ophthalmic history revealed poor vision in the right eye since childhood. Examination of her left eye 2 weeks earlier, following a routine referral, was unremarkable with visual acuity of 6/6, full colour vision, and no clinical evidence of optic neuropathy in the left eye. The right eye showed a dense cataract and visual acuity of counting fingers at 0.5 m.

Examination revealed visual acuity of hand movement in the right eye and counting fingers in the left eye. She was able to identify only the first plate on Ishihara testing with each eye. Pupillary examination revealed a fixed dilated pupil on the right side and sluggish reaction in the left eye. Conjunctival chemosis with limited ocular movements on the right side were noted but the left eye ductions were of full range. Her intraocular pressures were 20 mm Hg and 18 mm Hg in the right and left eye, respectively. Fundal examination revealed bilateral swollen optic nerve heads.

Neuroimaging showed an extensive spread of tumour involving the ethmoidal sinuses on both sides, middle meati, and frontal lobes (Figure 1). These findings were not reported on scans taken 2 weeks previously (Figure 2). The cavernous sinuses, pituitary fossa, and clivus also showed involvement. A large mass was seen in the right orbit, with optic nerve compression, but the intraorbital portion of the left optic nerve was spared. Intracanalicular and chiasmal involvement was seen on both sides.

The tumour was deemed 'inoperable' and the patient was offered combination chemotherapy. The patient refused further treatment after the first chemotherapy cycle and died 2 days after delivering a healthy baby at 32 weeks' gestation.

Discussion

Although an uncommon condition, several case series of esthesioneuroblastoma have now been reported, reflecting greater awareness of the disease.[1-3] However, visual manifestations are uncommon due to the rarity of the tumour.[4] Rakes et al have reported the largest series of patients with esthesioneuroblastoma and ophthalmic manifestations.[5] Proptosis, extraocular motility dysfunction, and intraorbital optic nerve involvement are the most common ophthalmic sequelae. To date, no patient with profound acute visual loss resulting from intracanalicular/chiasmal involvement with rapid spread has been reported to our knowledge. There is no definite treatment consensus; combined surgery and radiotherapy with or without adjuvant chemotherapy are considered the initial treatment.

This patient highlights the challenge for the treating physician when no clear-cut management guidelines are available. Rapid

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Figure 1. Contrast-enhanced coronal computed tomography showing (a and b) a large mass in the anterior ethmoidal sinuses, extending into the right orbit, up to the optic nerve (asterisk); (c) the mass extending into the right optic canal; and (d) the mass around the optic chiasma (arrow).

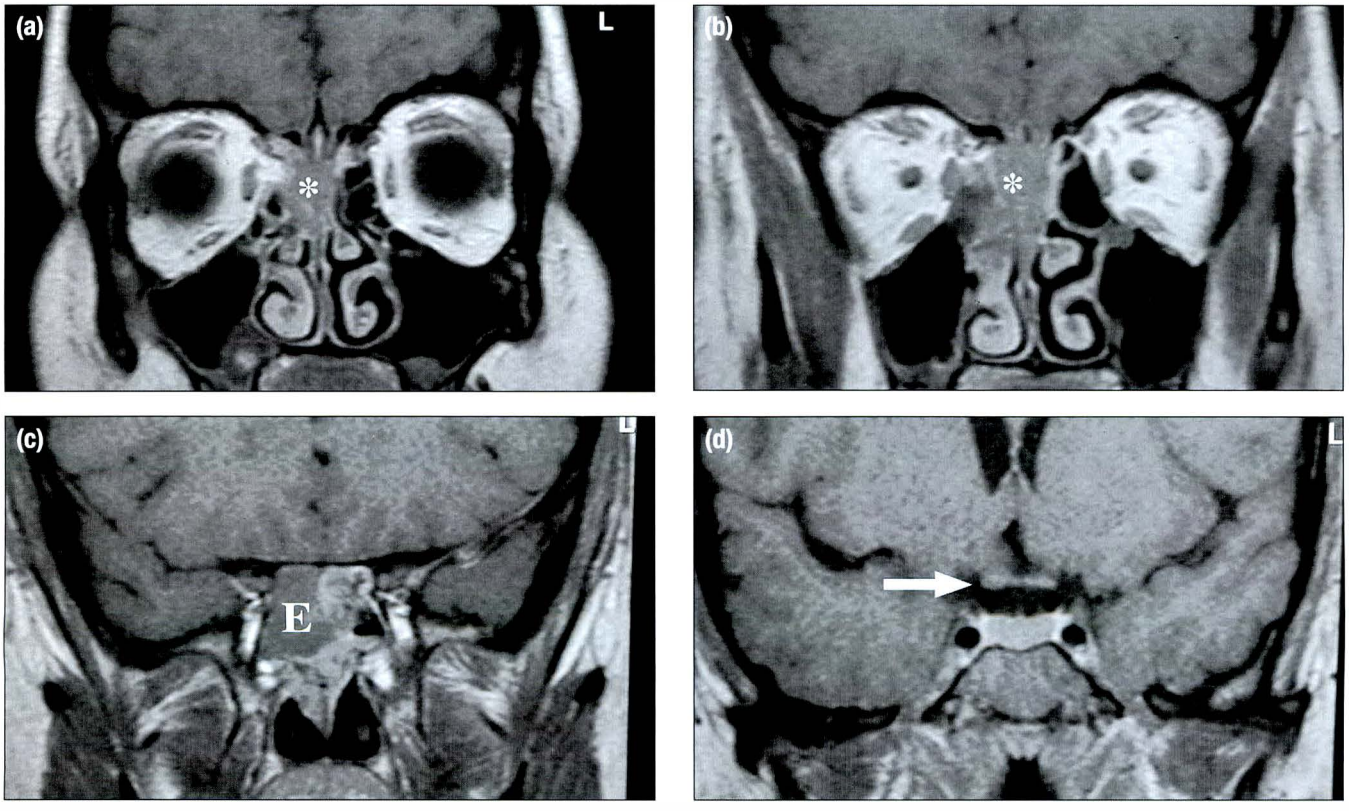
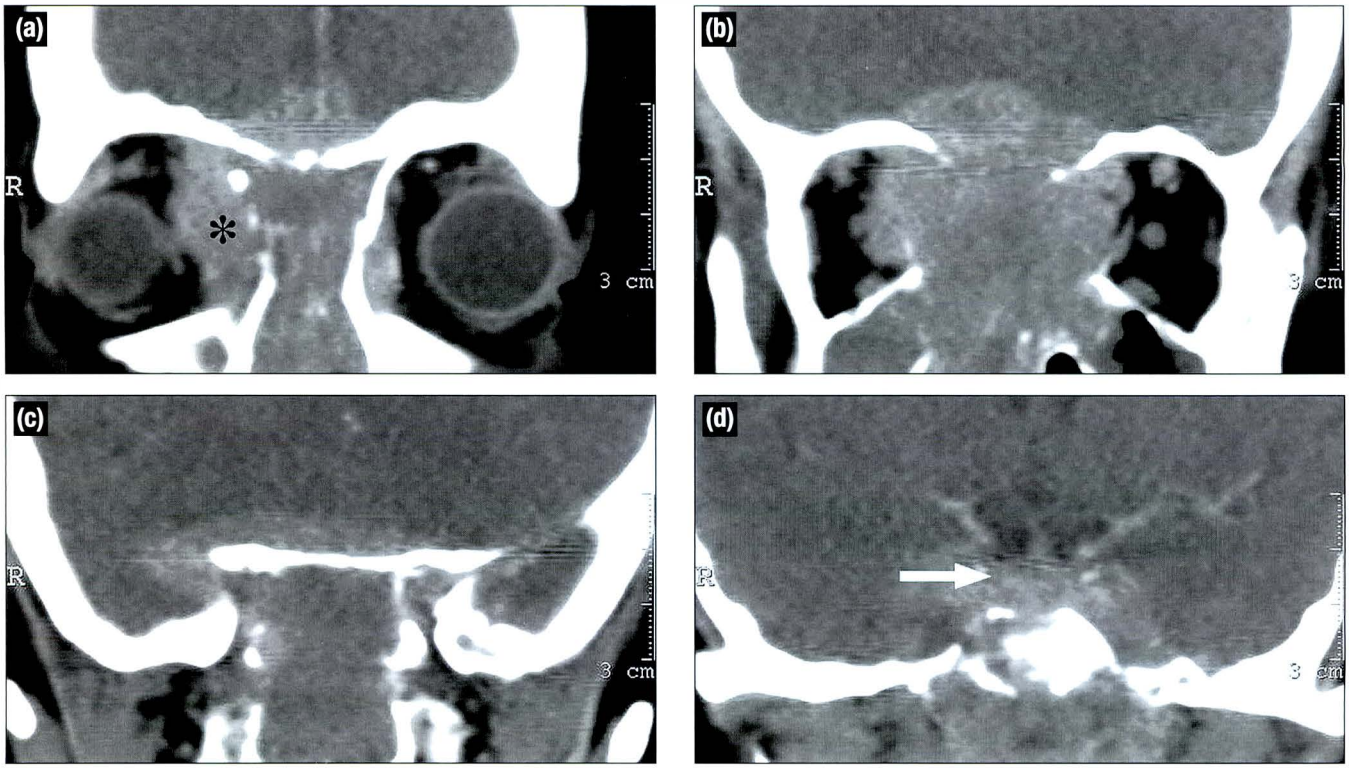


Figure 2. T1-weighted gadolinium-enhanced magnetic resonance image at presentation showing (a and b) the mass in the right anterior ethmoidal sinus (asterisk), extending to touch the right medial rectus muscle; (c) mass and fluid in the right posterior ethmoid sinus; and (d) the optic chiasma (arrow) with no disease around it.



loco-regional spread of tumour is not well recognised with esthesioneuroblastoma, and individual tailored treatment appears to be the best management strategy. It is hoped that, as the clinical experience of esthesioneuroblastoma management increases, definitive therapeutic guidelines will emerge.

Acknowledgement

With thanks to Dr C Yeoung, Consultant Radiologist, Warrington Hospital, Warrington, UK.

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Silicone Oil Granuloma Masquerading as a Subconjunctival Mass

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This report is of a patient with subconjunctival silicone oil granuloma that was masquerading as a chronic subconjunctival cystic lesion after removal of intravitreal silicone oil.

Key words: Granuloma, Silicone oils

Asian J Ophthalmol. 2007;9:219-20

Introduction

Silicone oil in the subconjunctival space is an uncommon complication of surgery. This report is of a 50-year-old man with a subconjunctival silicone oil granuloma appearing as a chronic subconjunctival cystic lesion.

Case Report

A 50-year-old man presented in 2003 with acute diminution of vision in his right eye. He had undergone cataract surgery in both eyes for congenital cataract at the age of 10 years. His best-corrected visual acuity with +9.0 DS was 2/60 in the right eye and 3/60 in the left eye. He was evaluated and diagnosed to have bilateral aphakia with nebulomacular corneal opacities with retinal detachment and divergent squint in his right eye and a macular scar in his left eye. He underwent vitreoretinal surgery with silicone oil injection in his right eye, after which he developed glaucoma, for which silicone oil removal was done. Following this, he was given topical brimonidine 0.12% twice daily and timolol 0.5% twice daily for intraocular pressure control.

Two years later, he developed a mass in his right eye. An elevated cystic vascularised non-tender lesion, 6 x 5 mm, was noted in the superior bulbar conjunctiva in his right eye (Figure 1). Multiple small shiny translucent nodules were seen through the cyst wall. At gonioscopy, the angle in his right eye was completely closed superiorly with silicone oil bubbles, and showed extensive peripheral anterior synechiae inferiorly with only 40° of the angle open inferonasally.

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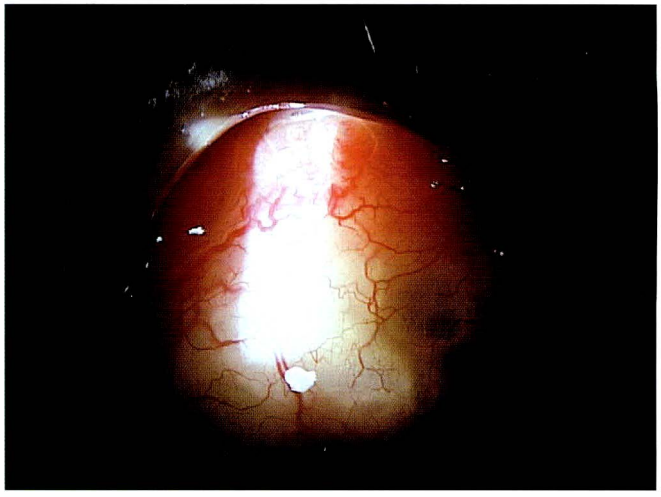
Figure 1. Subconjunctival cyst showing the translucent vacuoles.



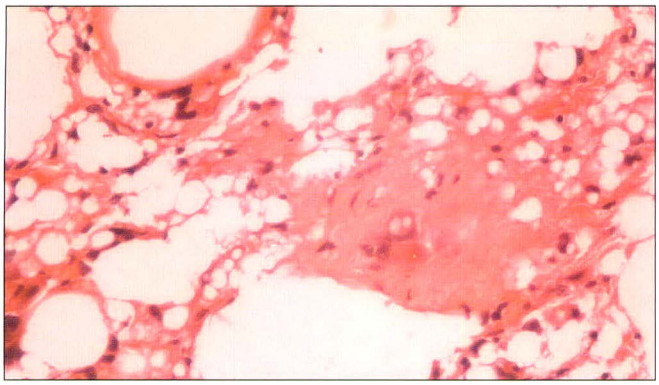
The patient underwent conjunctival mass excision. Histological analysis of the lesion revealed chronic granulomatous inflammatory reaction, predominantly with histiocytes and occasional multinucleated giant cells to extracellular lipid vacuoles, which were of varying sizes (Figure 2).

Discussion

Silicone oil leakage has been implicated as a cause of episcleral nodules adjacent to vitrectomy entry sites,[1] and in the bleb,[2] orbit,[3] or subconjunctival space.[4] Silicone oil in the subconjunctival space is uncommon, although it can occur as a complication either intraoperatively during injection of silicone oil or after surgery. This subconjunctival migration of silicone oil can be avoided by copious irrigation with saline solution.[5]

As well as mechanically blocking the trabecular meshwork, silicone oil from the vitreous cavity can migrate and lodge in the bleb area and cause granulomatous reactions, and subsequent

Figure 2. Haematoxylin and eosin stain showing chronic granulomatous inflammatory reaction predominantly with histiocytes and occasional multinucleated giant cells surrounding lipid droplets (original magnification, x 200).



scarring and failure of the trabeculectomy surgery.[2] In this patient, emulsified silicone oil was masquerading as a chronic subconjunctival cystic lesion that had incited fibrosis and a granulomatous reaction within the lesion.

While silicone oil has been reported as a cause of chronic inflammation, silicone oil migration occurred after removal in this patient. It is therefore important to consider migration of the oil as a cause of a subconjunctival mass even after removal of silicone oil.

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Advanced Primary Myoepithelial Carcinoma of the Lacrimal Gland Treated by Palliative Radiotherapy

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Myoepithelial carcinoma is a rare malignant epithelial tumour and there is a relative lack of understanding about its clinical behaviour. Approximately 120 cases have been reported in the literature, most of which were located in the salivary glands, with a few exceptions occurring in unusual locations such as the breast, nose, paranasal sinus, trachea, bronchus, and lung. Only 3 cases of myoepithelial carcinoma of the lacrimal gland have been reported. This report is of a patient who presented with advanced disease with intracranial extension and was treated by palliative radiotherapy of 20 Gy in 5 fractions over 1 week. The patient achieved excellent palliation and now has stable disease.

Key words: *Carcinoma, Lacrimal apparatus, Palliative care, Radiotherapy*

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Introduction

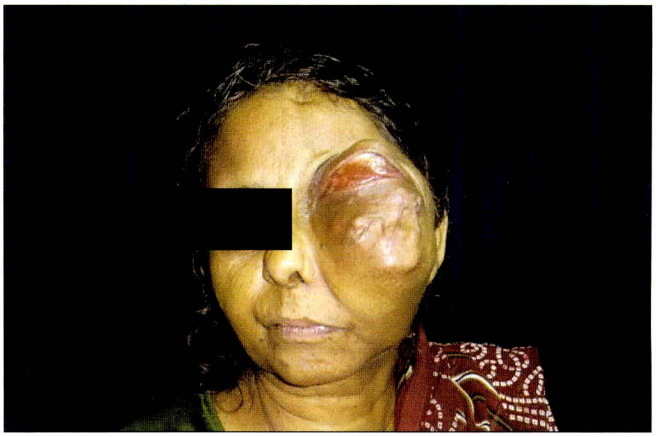
Myoepithelial carcinoma is a rare malignant epithelial tumour. Due to the rarity of this tumour, there is a relative lack of understanding of its clinical behaviour. Myoepithelial carcinoma is a low-grade malignant neoplasm, composed of variable proportions of ductular cells and large clear-staining myoepithelial cells arranged around the periphery of the ducts. Approximately 120 cases have been reported in the literature, most of which were located in the salivary glands, with a few exceptions occurring in unusual locations such as the breast, nose, paranasal sinus, trachea, bronchus, and lung.

It is difficult to state the prevalence of lacrimal gland tumours, as these tumours are rare. Malignant epithelial neoplasm of the lacrimal gland accounts for approximately 2% of all orbital neoplasms.[1] This report describes a patient with advanced myoepithelial carcinoma of the lacrimal gland treated by palliative radiotherapy.

Case Report

A 55-year-old woman presented to the Department of Radiotherapy, Institute Rotary Cancer Hospital, All India Institute of Medical Sciences, New Delhi, India, in October 2004 with a history of gradual progressive prominence of the left eye for 1 year. This symptom was associated with pain and loss of vision in the left eye. She had no previous relevant history of fever, trauma, or swelling. General physical examination revealed a well-nourished woman with no evidence of pallor or jaundice. Local examination showed a large mass on the left side of the face with dilated veins. The mass arose from the left orbit, displacing the globe (Figure 1). Visual examination showed that there was no perception of light in the left eye. Examination of the right eye was normal. There was no evidence of any pre-auricular or cervical lymphadenopathy. An ear, nose, and throat examination did not reveal any significant findings. Systemic examination was within normal limits.

Figure 1. Large mass over the left side of the face with dilated veins arising from the left orbit.



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Complete blood counts, erythrocyte sedimentation rate, C-reactive protein, and liver and kidney function tests were normal. Computed tomography scan of the face showed a large irregular mass filling the left orbit with bony destruction, intracranial extension, and extension to the ethmoid and sphenoid sinuses (Figure 2). Based on the clinical and radiological findings, a diagnosis of malignant tumour of the lacrimal gland was considered. An incisional biopsy showed a nested arrangement of cells with ductular cells in the centre and large clear-staining myoepithelial cells arranged around the periphery of the ducts, typical of myoepithelial carcinoma (Figure 3). Immunohistochemistry showed strong cytokeratin positivity in the central ductal cells (Figure 4) and S-100 positivity in the outer myoepithelial cells (Figure 5). Chest X-ray, ultrasound of the abdomen, and bone scan showed no evidence of any distant metastasis.

In view of the extensive local disease with intracranial extension, the tumour was considered inoperable and the patient was given palliative radiotherapy of 20 Gy in 5 fractions over 1 week in November 2004 by direct anterior and left anterior oblique wedge fields (3D plan) [Figure 6]. Radiotherapy halted the progression of the disease and, in August 2006, the patient had stable disease. She is currently in the care of the pain and palliative care clinic.

Figure 2. (a) Axial and (b) coronal computed tomography of the head showing an irregular mass filling the left orbit with bony destruction and intracranial extension, extending to the ethmoid and sphenoid sinuses

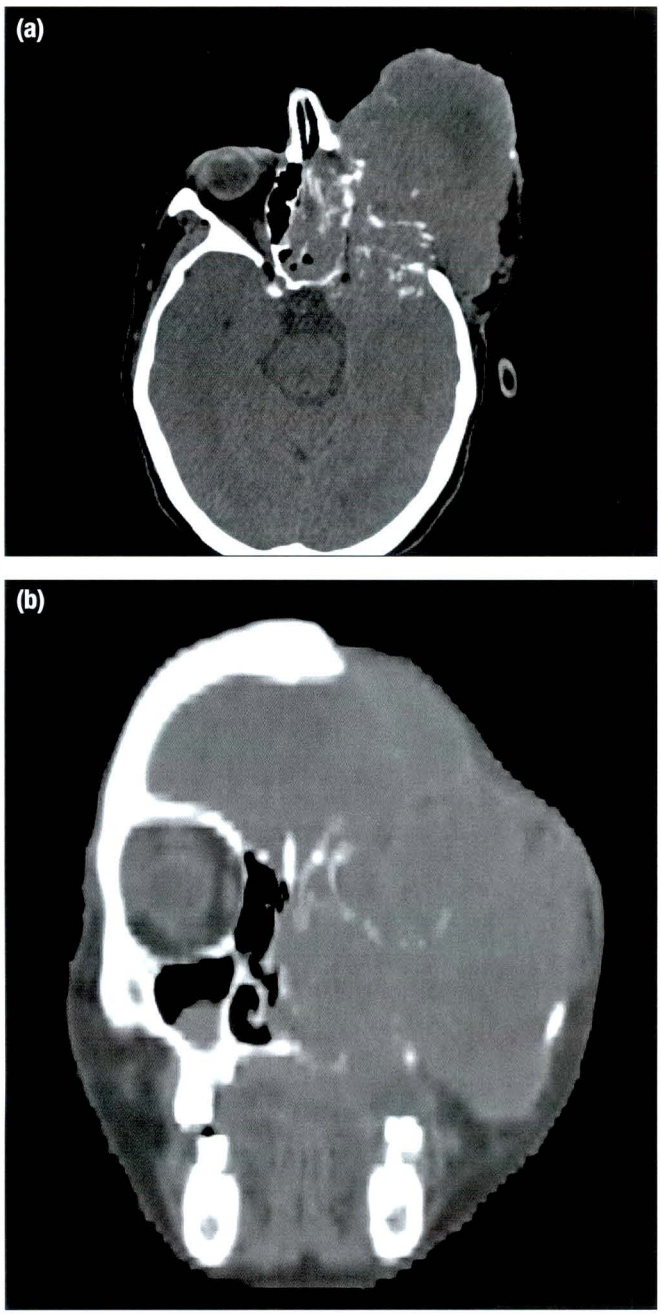


Figure 3. Photomicrograph of a biopsy from the orbital mass showing a nested arrangement of cells typical of myoepithelial carcinoma (haematoxylin and eosin stain; original magnification, x 400).

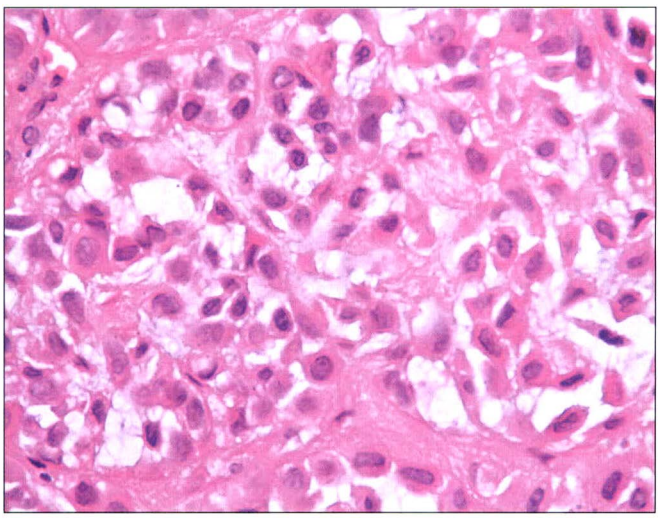


Figure 4. Immunohistochemistry showing strong cytokeratin positivity in the ductal cells (Avidin-Biotin stain; original magnification, x 400).

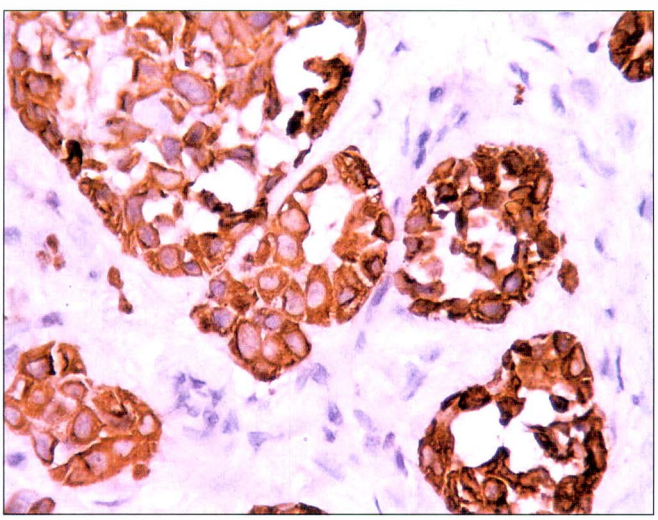


Figure 5. Immunohistochemistry showing S-100 positivity in the outer myoepithelial cells (Avidin-Biotin stain; original magnification, x 400).

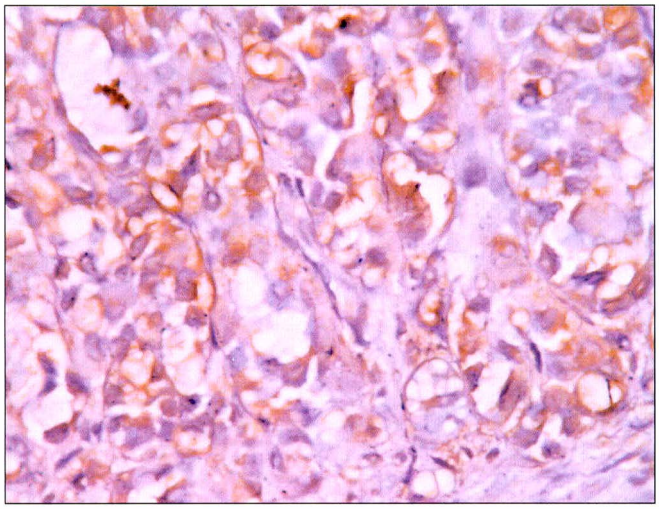
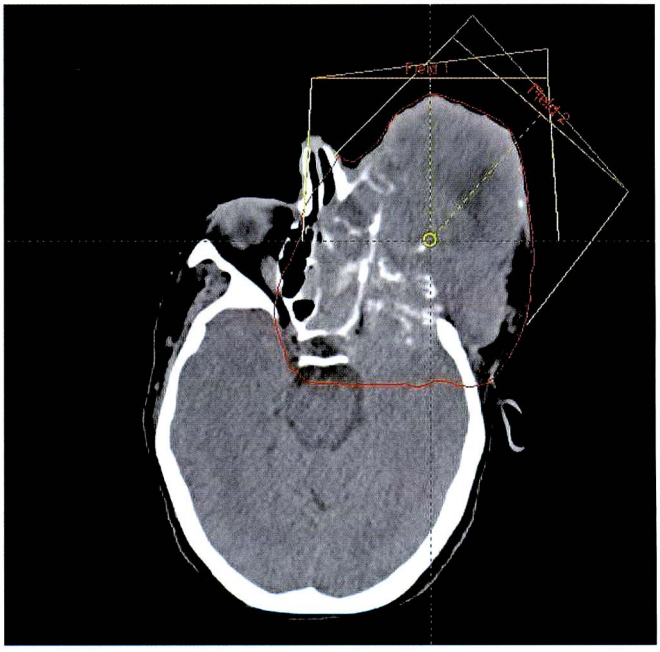


Figure 6. Radiotherapy field — 20 Gy over 5 fractions was given by direct anterior and left anterior oblique wedge fields.



Discussion

Mass lesions of the lacrimal gland can be classified broadly into inflammatory and neoplastic subtypes. Inflammatory aetiologies include dacryoadenitis, sarcoidosis, and orbital inflammatory pseudotumour. Fifty percent of neoplastic lesions in the lacrimal gland are benign and 50% are malignant. Benign lesions include pleomorphic adenomas, benign reactive lymphoid hyperplasia, and oncocytomas. Malignant tumours of the lacrimal gland include adenoid cystic carcinoma, adenocarcinoma, squamous cell carcinoma, mucoepidermoid carcinoma, and malignant lymphomas.[2,3] Adenoid cystic carcinoma is the most common malignant lacrimal gland tumour, comprising 50% of malignant tumours of the lacrimal gland and 25% of all lacrimal gland tumours.[3]

Myoepithelial carcinoma is a rare group of malignant tumours. This tumour started gaining wider recognition after its inclusion in the World Health Organization histological classification of salivary gland tumours in 1990. More than two-thirds of these tumours arise in the parotid gland,[4-6] but they can also originate elsewhere, in the major or minor salivary glands or breast.[7] Reports of the occurrence of myoepithelial carcinoma in sites such as the lacrimal gland, nose, paranasal sinus, trachea, bronchus, and lung are presently limited to case reports.[8-12] The peak age of incidence of myoepithelial carcinoma is in the sixth decade, with no predilection for either sex.[13]

Histological features that are considered helpful in discriminating benign and malignant myoepithelial tumours include cytological atypia, tumour infiltration, mitotic rate, and necrosis.[14] Nagao et al suggested that assessment of cell proliferative activity may be helpful for the differential diagnosis between benign and malignant myoepithelial tumours, and that >7 mitoses/10 high-power fields or a Ki-67–labelling index of >10% is diagnostic of a malignant tumour.[5] Tumour cells show variable frequencies of immunoreactivity for cytokeratin (90%), cytokeratin 14 (100%), actin (70-80%), calponin (100%), S-100 protein (100%), glial fibrillary acidic protein (50%), epithelial membrane antigen (100%), carcinoembryonic antigen (0%), and human melanoma black 45 (0%). The characteristic biphasic cell arrangement and immunostaining features help to distinguish this tumour from common tumours such as adenoid cystic carcinoma with an infiltrative cribriform growth pattern and pleomorphic adenoma with melting of the epithelial cells in myxoid or chondroid stroma.[15,16]

To the best of the author's knowledge, only 3 cases of myoepithelial carcinoma have been reported in the lacrimal gland. One of these was a hybrid carcinoma,[9] the second was an epithelial-myoepithelial carcinoma with background pleomorphic adenoma,[8] and the third was the only de novo myoepithelial carcinoma of the lacrimal gland.[10] Computed tomography scan of the orbit shows an irregular mass, with possible bony erosion and occasional calcification. Biopsy and immunohistochemical studies are mandatory for the diagnosis.

Due to its rarity, there have been no randomised treatment trials for this tumour. The therapeutic approach to these tumours remains a challenge. Patients with early disease should be considered for surgery, either exenteration or wide local excision. If wide excision is done, the patient should be considered for postoperative radiotherapy. For advanced disease, craniorbital exenteration by a multidisciplinary approach followed by radiotherapy should be considered. This patient was not considered

for radical surgery because of extensive local disease, and was treated by palliative radiotherapy.[17]

Recurrence and metastasis rates for epithelial-myoepithelial carcinoma of the salivary gland have been reported to range from 35% to 50% and 8.1% to 25%, respectively.[11] This rare tumour may behave as a low-grade locally aggressive malignant neoplasm, in contrast to other malignant epithelial tumours of the lacrimal gland such as adenoid cystic carcinoma, which usually has a worse prognosis.

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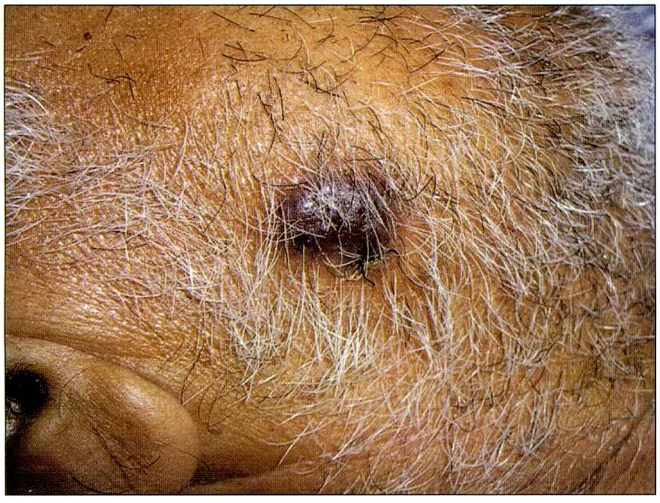
Human Immunodeficiency Virus Type I, Viscerocutaneous Leishmaniasis, and Cytomegalovirus Retinitis

Dear Editor,

The incidence of leishmaniasis as an opportunistic infection in patients with acquired immunodeficiency syndrome (AIDS) has been increasing since the first case of human immunodeficiency virus (HIV)-*Leishmania* coinfection.[1-2] *Leishmania* parasites and HIV destroy the same cells, thus producing cumulative immunosuppression, resulting in an exponential increase in disease severity, sequelae, and occurrence of opportunistic infections.[1-5] We recently treated a patient with HIV type I infection who also had evidence of viscerocutaneous leishmaniasis and cytomegalovirus (CMV) retinitis.

A 48-year-old cachetic and emaciated patient with a 6- to 7-month history of diarrhoea, chronic progressive weight loss, non-tender hepatosplenomegaly, and multiple firm pigmented non-tender skin nodules of approximately 2 x 3 cm on his right cheek, neck, and scalp (Figure 1) presented with sudden loss of vision in his right eye. The best-corrected visual acuity in his right eye was finger counting close to his face; his left eye had no light perception. Slit-lamp evaluation and intraocular pressure were normal. Pupillary reaction in the right eye was sluggish to direct light stimulation. Fundus evaluation revealed pale discs in both eyes. In his right eye, the posterior pole showed yellowish infiltrates with fluffy margins and superficial haemorrhages close to the vessel arcades, indicating retinal inflammation, with multiple sclerosed vessels. There was no vitreous infiltration. The left eye showed scattered pigmented scars in the retina with markedly sclerosed vessels and consecutive optic atrophy (Figure 2). Neurological evaluation, computed tomography of the brain, chest X-ray, and liver function tests were normal.

Figure 1 Pigmented skin lesion on scalp.



Laboratory investigation revealed bicytopenia and lymphopenia with anaemia. Giemsa stain of biopsy material from the skin granulomas revealed amastigote forms of *Leishmania* in macrophages, which were confirmed in biopsy material from the gastrointestinal tract (Figure 3). The patient was found to be positive for HIV-1 and cytomegalovirus viraemia. The response to treatment with intravenous gancyclovir and liposomal amphotericin was slow (Figure 4).

CMV retinitis commonly occurs in immunocompromised individuals. A decline in the incidence of CMV retinitis has been

Figure 2. Fundus picture of (a) the right eye showing occlusive vasculitis with retinitis; and (b) the left eye showing a pale disc with pigmented scars on the retina.

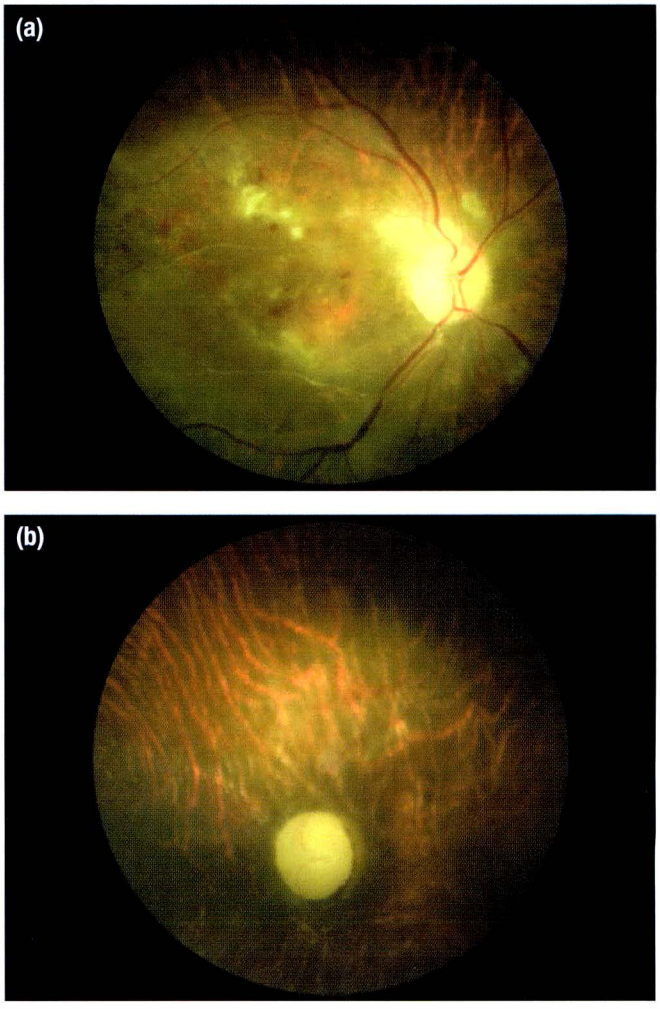


Figure 3. Amastigote form of *Leishmania* in (a) macrophages from the skin lesions; and (b) gastrointestinal tract biopsy.

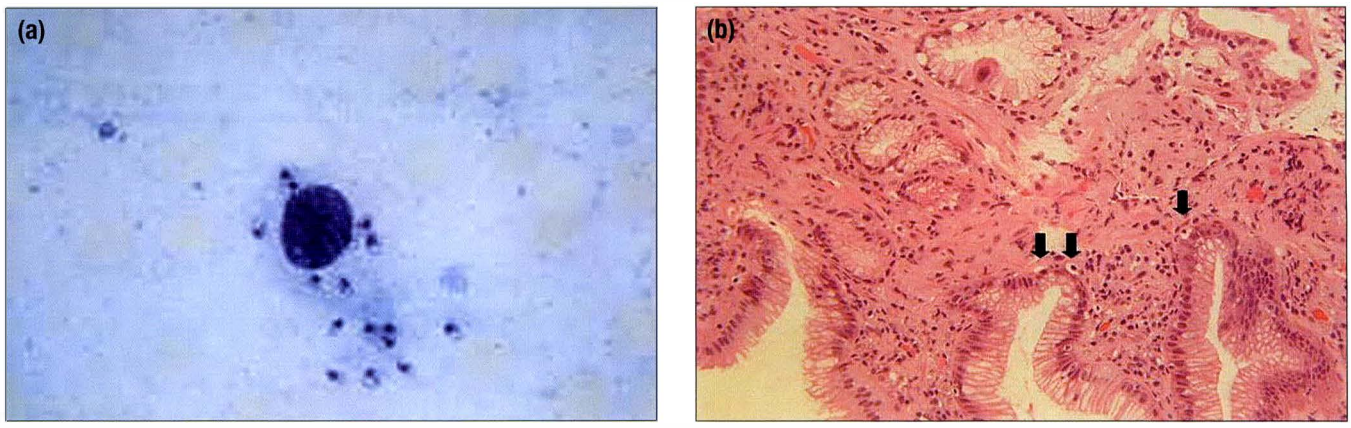
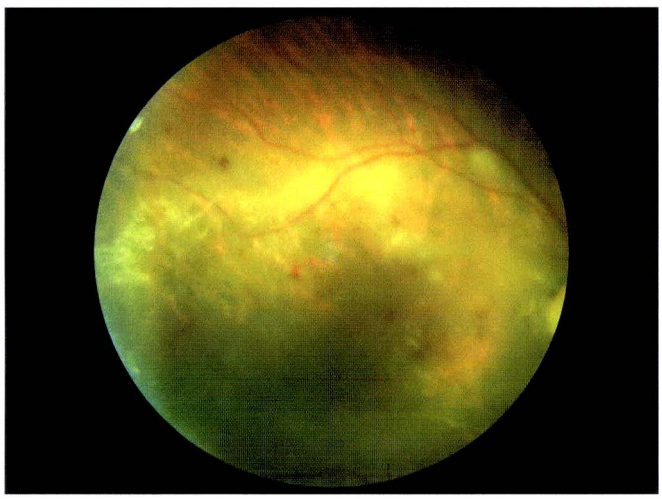


Figure 4. Fundus picture of the right eye showing response to treatment 10 days after starting therapy.



reported following the introduction of highly active retroviral therapy (HAART) for the treatment of HIV. However, the concurrent reduction in AIDS-related mortality associated with HAART, together with the cumulative immunosuppression produced by opportunistic coinfections such as *Leishmania*,[1,2] has possibly led to resurgence in the number of patients with CMV disease.

Treatment of the underlying conditions can prevent development of the immune environment required for CMV retinitis, thereby reducing ocular morbidity and blindness. Prevention practices to reduce the transmission of HIV will concomitantly reduce the incidence of CMV retinitis.[3-5] Awareness of rare coinfections occurring in patients with immunosuppression is important, as early recognition and appropriate intervention can help to reduce the resultant morbidity and mortality.[1-5]

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Recent Developments with SLT

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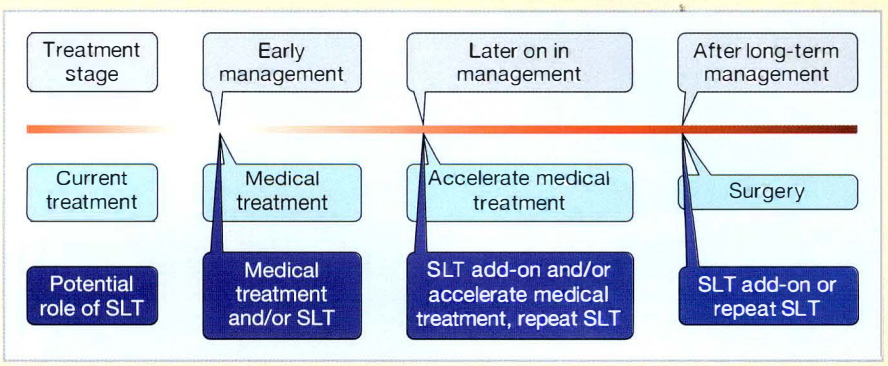
The conventional glaucoma treatment paradigm involves medical therapy, followed by laser trabeculoplasty, and finally, incisional surgery. However, there are potential problems with all 3 treatments. Medical treatment is associated with problems of compliance, persistence, effectiveness, ongoing costs, and side effects. Argon laser therapy (ALT) has limited efficacy at re-treatment; may cause peripheral anterior synechiae and tissue damage; is contraindicated for narrow angles and angle closure (AC); may cause coagulative damage to the trabecular meshwork (TM); and may be associated with a post-treatment increase in intraocular pressure (IOP). The problems of surgery include failure to control IOP and short- and long-term postoperative complications.

Selective laser trabeculoplasty (SLT) was initially designed as an alternative to ALT for the management of glaucoma. However, these techniques do have significant differences. SLT is as effective as ALT, but is titratable, potentially repeatable, and applicable to many eyes with AC. SLT broadens the treatment choices for clinicians and patients, both in terms of the timing during the course of the disease and for the range of glaucomas that may be treated by this technique. SLT can be incorporated into a wide range of management decisions, for example, as a replacement for ALT, at initiation of antihypertensive therapy, or to accelerate treatment (Figure 1). Given the evidence for its high therapeutic index, SLT may be incorporated as an alternative to medical treatment for first-line therapy, or as a substitute for polypharmacy when escalating treatment. In the future, laser therapy may overlap increasingly with medical therapy as the cornerstone of presurgical antiglaucoma treatment.

Figure 1. Potential roles of selective laser trabeculoplasty (SLT) in glaucoma management.



Mechanism of Action of SLT

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The probable mechanism of action of SLT is to increase trabecular outflow. According to Goldman's equation, the aqueous inflow must be equal to the trabecular and uveoscleral outflow. Antiglaucoma drugs such as pilocarpine reduce IOP by increasing the outflow facility. If SLT acts in a similar way, the outflow facility would be expected to increase causing a decrease in IOP.

A study has been performed at St Thomas' Hospital, London, UK, to assess the changes in outflow facility and IOP in 69 previously untreated eyes of 60 patients with ocular hypertension (OHT) or primary open angle glaucoma (POAG). All eyes had an IOP between 21 mm Hg and 35 mm Hg. The laser power was 0.6 mJ and increased in 0.1 mJ steps until small champagne-like bubbles appeared at the treated area. The primary outcome measures were IOP level and Schiotz outflow facility. Data from 45 eyes were analysed at 1 month and from 41 eyes at 3 months.

Table 1. Change in intraocular pressure and outflow facility 3 months after selective laser trabeculoplasty (n = 41).

Time	Intraocular pressure (% change) [mm Hg]	p Value	Outflow facility (% change)	p Value
Baseline	25.4		0.104	
1 month	17.7 (-30)	<0.0001	0.134 (+29)	0.025
3 months	17.4 (-31)	<0.0001	0.135 (+25)	0.025

Preliminary results show that, after 1 month, there was an overall 29% decrease in IOP from 25.3 mm Hg to 18.0 mm Hg ($p < 0.0001$) and the outflow facility had increased by 30% from 0.10 to 0.13 ($p = 0.025$). For the 41 responders, there was a decrease in IOP of 30% and 31% at 1 and 3 months, respectively, and an increase in outflow facility of 29% and 25%, respectively (Table 1).

This study found that SLT could provide a 30% reduction in IOP and a 30% increase in outflow facility. However, according to Goldman's equation, the outflow facility must increase by 100% to achieve a 30% decrease in IOP, suggesting another possible mechanism of action for this procedure.

In conclusion, primary SLT for OHT or POAG decreases IOP by 30% and increases the outflow facility by 30%, with a non-responder rate of 10%. The effect on trabecular outflow is likely to be a key mechanism of action, but SLT may also affect other aqueous dynamic parameters.

SLT in Clinical Practice

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Clayton Eye Centre
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Key to the successful management of glaucoma is early diagnosis and treatment. The ideal treatment for glaucoma should offer sufficient reduction in IOP; provide long-term IOP reduction; be associated with minimal IOP fluctuation; be independent of the compliance factor; be devoid of, or offer tolerable, systemic and local side effects; and be economical. While there is no ideal treatment, SLT has the best risk-benefit ratio for glaucoma treatment.

SLT was first introduced in 1995, but it remains uncertain how the procedure fits into the treatment paradigm — as first-line, adjunctive, or replacement therapy. A retrospective analysis of the case notes of all patients with glaucoma treated with SLT at Clayton Eye Centre, Wakefield, UK, from January 2000 to December 2005 has been performed to ascertain the long-term effect and the efficacy of re-treatment with SLT. SLT was performed as either primary or secondary treatment.

In the primary treatment group (n = 229), the IOP decreased by 32% from 27.8 mm Hg to 19.0 mm Hg ($p < 0.001$). In the secondary treatment group (n = 198), the IOP decreased by 33% from 26.0 mm Hg to 17.2 mm Hg ($p < 0.001$).

In a secondary objective to assess the survival time of SLT, 50% of eyes reached the 5-year follow-up successfully. Although there was an initial steep attrition rate, this stabilised after 3 years to <5%. Enhancement resulted in a 26.4% reduction in IOP from 26.4 mm Hg to 19.7 mm Hg ($p < 0.001$), while repeat treatment resulted in a 23.7% reduction from 25.2 mm Hg to 20.1 mm Hg ($p < 0.001$).

SLT has the best benefit-risk ratio of all glaucoma treatments. While the effect of SLT wears off over time, the procedure is repeatable, unlike ALT. SLT not only reduces IOP, but also improves quality of life, as fewer medications, with their concomitant side effects and compliance issues, are required. The introduction of SLT may alter the current treatment paradigm for glaucoma, offering an option for first-line therapy.

SLT for Primary Angle Closure Glaucoma

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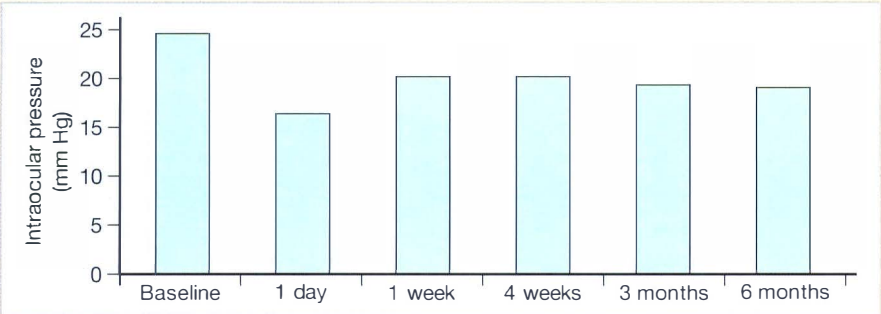
Although primary angle closure glaucoma (PACG) is more common in Asia than in the West, it has been found to comprise more than one-quater of all glaucomas in a western study population. As changes in the TM may be present in PACG, even without the presence of visible peripheral anterior synechiae, the challenge for the treatment of PACG using SLT is the limited amounted of treatable area and the unhealthy condition of the remaining TM. A multicentre, multinational prospective interventional study was performed in Southeast Asia to determine whether SLT could lower the IOP in 67 eyes with PACG. All eyes had a patent peripheral iridotomy and a degree of visible trabecular meshwork.

The results were significant ($p < 0.01$) at all follow-up visits, with the IOP reduction from baseline ranging from 4.4 mm Hg (17.9%) to 8.2 mm Hg (33.5%) [Figure 2]. Seventy nine percent of patients achieved an IOP reduction of ≥3 mm Hg, 54% achieved a reduction of ≥20%, and 23% achieved a reduction ≥30%. There were no significant complications. SLT therefore appears to be a safe, effective, and simple method of reducing IOP in eyes with PACG with a patent iridotomy and a sufficient extent of visible angle.

Figure 2. Intraocular pressure after selective laser trabeculoplasty.



A retrospective comparative case series was performed in Thailand to compare the efficacy of SLT on IOP-lowering for patients with OAG and ACG. Sixty one patients with visible trabecular meshwork >180° who underwent SLT were enrolled. Thirty four patients had OAG (mean baseline IOP, 18.6 mm Hg) and 13 had ACG patients (mean baseline IOP, 18.3 mm Hg).

The main outcome measure was a >20% IOP reduction from baseline at each follow-up visit. After 12 weeks, 14 patients with OAG (41%) and 2 patients with ACG (15%) achieved a >20% IOP reduction. The conclusion from this study was that SLT can reduce IOP in eyes with OAG and ACG.

In conclusion, SLT appears to be a good alternative therapy for treatment of PACG. The efficacy is acceptable, with more than 50% of treated eyes achieving ≥20% IOP reduction. SLT has an excellent safety profile and is a simple and quick procedure to perform.

From the Ellex satellite symposium Beyond Convention — Recent Developments in SLT *held at the World Glaucoma Congress, Singapore, 20 July 2007.*

Asian Oceanic Glaucoma Society 2007

AOGS 2007 BANGKOK

Bangkok, Thailand, 2-4 December 2007

The Asian Oceanic Glaucoma Society meeting will take place in Bangkok, Thailand, from 2 to 4 December 2007. The venue is the Central World Convention Center.

The main topics to be covered include:

- Angle closure glaucoma
- Glaucoma drainage devices
- Imaging in glaucoma
- Laser trabeculoplasty
- Neuroprotection
- Nanotechnology
- Normal tension glaucoma
- Congenital glaucoma
- Antivascular endothelial growth factor
- Glaucoma and associated diseases

Important dates

Abstract submission deadline	1 September 2007
Notification of abstract acceptance	15 October 2007
Early-bird registration deadline	30 September 2007
Pre-congress registration deadline	31 October 2007

For further details, contact the website at: **www.aogs2007.org**

2008 SEAGIG/ AACGC Joint Congress

Seoul, Korea, 25-27 September 2008

SEAGIG 2008 Seoul, Korea

AACGC

The 5th Congress of the South East Asia Glaucoma Interest Group (SEAGIG 2008) and the 6th Meeting of the Asian Angle-Closure Glaucoma Club (AACGC) will take place in Seoul, Korea, from 25-27 September 2008. SEAGIG was established to facilitate contact between glaucoma specialists in the region, to encourage collaborative research and service projects, to increase the opportunities for exchange of skills and knowledge in this rapidly advancing field, and to assist comprehensive ophthalmological colleagues and other eye care workers (whether medically trained or not) to keep up to date with advances in all aspects of glaucoma diagnosis and management.

The conference organising committee plans to introduce an educational and scientific programme that will cover cutting-edge basic and clinical research topics in the field of glaucoma. You are invited to make the scientific programme more dynamic and stimulating by submitting abstracts and registering for the conference.

Important Dates

Abstract submission deadline	6 June 2008
Early registration	30 June 2008
Abstract acceptance notice	11 July 2008

For further details, contact the website at: **www.seagig2008seoul.org**

IMAGE Modules Released

The South East Asia Glaucoma Interest Group (SEAGIG) is pleased to announce the release of the final 2 modules of the educational resource from the Initiative for Management, Awareness and Glaucoma Education (IMAGE) project. Intended for use by ophthalmologists for their own educational advancement, as well as to facilitate educational programmes, the slides have been prepared by SEAGIG/IMAGE members to be clinically relevant to glaucoma care in the region and to have educational value relevant to the region.

The *Gonioscopy* module discusses the aims, principles and methods of gonioscopy. Gonioscopy is an important clinical skill required to diagnose and monitor various eye conditions associated with glaucoma. However, difficulties in technique and interpretation may detract from its usefulness as a diagnostic tool. This workshop provides an overview of the different gonioscopic techniques used as part of a comprehensive ophthalmological examination to detect and assess glaucoma. Instruction aids include photographs (Figure 1), diagrams and video clips showing various gonioscopic procedures and equipment.

The aims, principles, and methods of gonioscopy will be discussed, followed by separate sections dedicated to other key clinical aspects of gonioscopy. The final slide will list important take-home messages from the presentation.

At the end of the presentation, participating clinicians should be able to:

- list the reasons for performing gonioscopic assessment
- understand the principles behind gonioscopy
- recognise and be familiar with the different types of gonioscopic equipment and procedures
- gain confidence in conducting a gonioscopic examination on a patient
- identify different anatomical structures that may be seen during gonioscopy
- describe methods of grading angle width in a patient with glaucoma and develop an awareness of developmental abnormalities that may be seen during gonioscopy.

Gonioscopy is a demanding skill and an essential part of the examination of a patient with glaucoma. To gain familiarity with the different gonioscopic techniques, their advantages and limitations, and the various clinical instances in which they are most useful, clinician's must practice them diligently. It is only by undertaking gonioscopy on every patient with glaucoma that clinicians will become familiar with the variety of normal and abnormal findings that may be present.

Figure 1.

Figure 2.

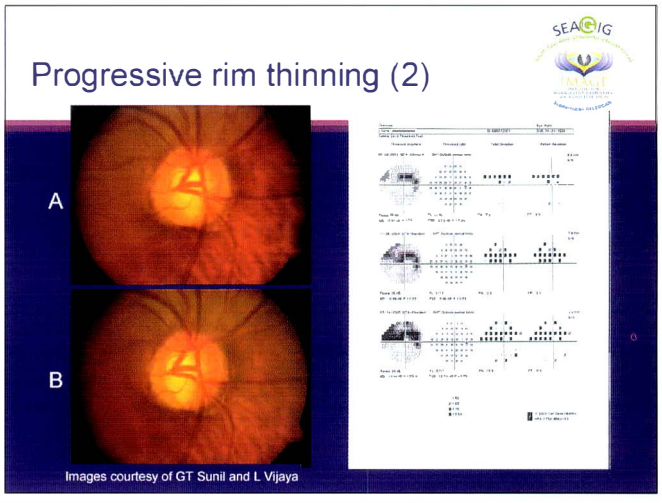
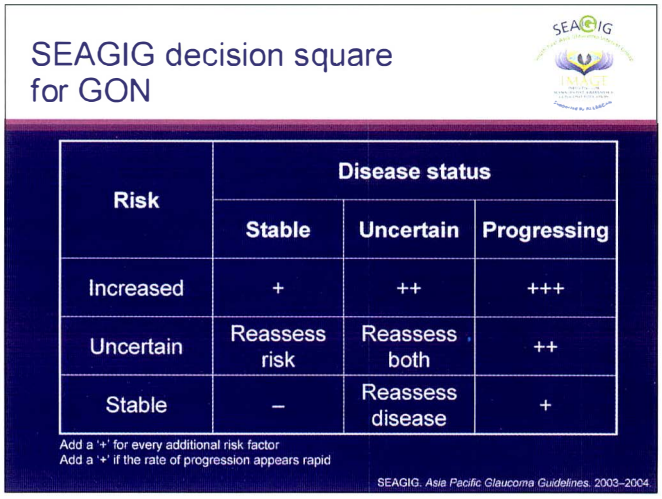


Figure 3.



The *Follow-up* module is intended to aid clinicians in the long-term management of patients with glaucoma, including how to assess the effects of treatment on the patient's overall well-being, identify features indicating optic disc changes, and evaluate disease progression (Figures 2 and 3). The strategies outlined in this module are not intended as a guide for care immediately following a surgical or laser procedure; this is covered in more detail in previous modules.

The follow-up process starts with the management plan made at the initiation of therapy. Educating patients about the benefits and risks of medication, as well the seriousness of glaucoma and the importance of adhering to the treatment regimen, is a key part of effective long-term management. Upon completion of this module, clinicians should have a good understanding of how to assess the effects of treatment on the patient's overall wellbeing, identify features indicating optic disc changes, and evaluate disease progression.

SEAGIG

2008 SEAGIG/AACGC Joint Congress

Seoul, Korea, 25-27 September 2008

AACGC

The South East Asia Glaucoma Interest Group (SEAGIG) and the Asian Angle-Closure Glaucoma Club (AACGC) have joined forces to hold the 2008 SEAGIG/AACGC Joint Congress in Seoul, Korea, on 25-27 September 2008.

The aim of the AACGC is to establish a scientific network for Asian glaucomatologists who are interested in exchange of knowledge about angle closure glaucoma. For further information about the AACGC, visit the website at: **www.aacgc.org/**

For full conference details, visit the website at: **www.seagig2008seoul.org**

Ophthalmo 2007

7-9 December 2007, Lahore, Pakistan

Ophthalmo 2007, the 27th meeting of the Ophthalmological Society of Pakistan, Lahore branch, will take place on 7-9 December 2007. The meeting will include symposia, instructional courses, named lectures, and free papers. The venue is the Pearl Continental, Lahore, Pakistan.

The last date of submission of abstracts is 31 October. Please submit your abstracts electronically to: ospllhr@gmail.com. Come and share your clinical experience with us and meet old friends.

For further information, e-mail: **ospllhr@gmail.com**

International Symposium on Retinoblastoma and Pediatric Ophthalmic Tumors

21-22 December 2007, Singapore

The International Symposium on Retinoblastoma and Pediatric Ophthalmic Tumors will be held in Singapore on 21-22 December 2007. Jointly organised by the National University Hospital Eye Surgery Center and the Department of Pediatrics, the symposium venue is the Yong Loo Lin School of Medicine at the National University of Singapore.

The meeting highlights include:

- Overview of retinoblastoma — diagnosis, management, challenges
- Regional perspectives
- Pediatric ophthalmic tumors
- Expert panel discussion
- Poster presentations

There is an international faculty from Malaysia, India, The Philippines, Sri Lanka, and Indonesia, as well as local experts from Singapore.

For further information, e-mail: **Ai_Meei_Ee@nuh.com.sg**

November 2007

10-13
2007 Annual Meeting of the American Academy of Ophthalmology (AAO)
New Orleans, USA
Contact: American Academy of Ophthalmology
Tel: (1 415) 561 8500
Fax: (1 415) 561 8533
E-mail: aaoe@aao.org
Website: www.aao.org/annual_meeting/2006.cfm

24-28
2007 National Congress of the Royal Australian & New Zealand College of Ophthalmologists
Perth, Australia
Contact: Congress West
Tel: (61 89) 389 6906
E-mail: conwes@congresswest.com.au
Website: www.congresswest.com.au/RANZCO2007?Ophthalmology

December 2007

2-4
Asian Oceanic Glaucoma Society 2007
Bangkok, Thailand
Contact: Secretariat
E-mail: tenkn@mahidol.ac.th

7-8
Retinal and Glaucoma Imaging 2008: Ocular Coherence Tomography (OCT) Applications and Future Technology
Palm Beach, FL, USA
Contact: Department of CME, Bascom Palmer Eye Institute Dept. of CME
Tel: (1 305) 326 6110
E-mail: bpeicme@med.miami.edu
Website: www.bascompalmer.org

February 2008

20-24
Scientific Meeting of the Glaucoma Research Society
Queenstown, New Zealand
E-Mail: glaucoma2008@tourhosts.com.au
Website: http://www.glaucomasociety.org/

22-24
30th Annual Congress of the Ophthalmological Society of Pakistan and 4th Khyber Eye Symposium
Peshawar, Pakistan
Contact: Tariq Farooq Babar
Tel: (92 91) 5825 087
E-mail: osp_nwfp@hotmail.com

28-2 March
7th International Symposium on Ocular Pharmacology and Therapeutics
Budapest, Hungary
Contact: Robert Nesbitt
Tel: (44 229) 080 488
Fax: (44 227) 322 850
E-mail: isopt@kenes.com

March 2008

7-11 March
European Congress of Radiology
Vienna, Austria
Contact: ESR Office, Neutorgasse 9/2AA-1010, Vienna
Tel: (43 1) 5334 0640
Fax: (43 1) 5334 064448
E-mail: communications@myESR.org

30-3 April
7th International Diabetes Federation Western Pacific Region Congress, Diabetes Asia Pacific, Working for Solutions
Wellington, New Zealand
Contact: Russ Finnerty
Tel: (64 44) 738 442
E-mail: congress@diabetes.org.nz

April 2008

4-9
American Society of Cataract and Refractive Surgeons Annual Meeting
Chicago, IL, USA
Contact: 4000 Legato Rd. Suite 700, Fairfax, VA 22033, USA
Tel: (1 703) 591 2220
Fax: (1 703) 591 0614
Website: www.ascrs.org/

Note to Readers
This section is intended to highlight activities of interest to glaucoma specialists and ophthalmologists in Asia. Please let us know of any forthcoming activities that you may be organising or wish to feature on this section.

May 2008

21-24
18th International Visual Field & Imaging Symposium (IPS2008)
Nara, Japan
Contact: Chota Matsumoto
Tel: (81 72) 366 0221
Fax: (81 72) 368 2559
E-mail: ips2008@med.kindai.ac.jp

June 2008

1-6
European Glaucoma Society Quadriennial Meeting
Berlin, Germany
Website: www.eugs.org/

28-2 July
World Ophthalmology Congress
Hong Kong
Contact: Ms Angela Cho
Tel: (852) 2762 3128
Fax: (852) 2194 0695
E-mail: angelacho@woc2008hongkong.org
Website: www.woc2008hongkong.org/

October 2008

1-4
European Association for Vision and Eye Research
Portoroz, Slovenia
Contact: Kapucijnenvoer 33, B-3000 Leuven, Belgium
Tel: (32 16) 233 849
Fax: (32 16) 234 097
E-mail: ever@ever.be

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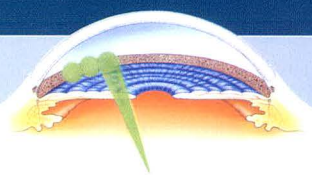

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References:
1. Higginbotham EJ, Feldman R, Stiles M, Dubiner H, for the Fixed Combination Investigative Group. Latanoprost and timolol combination therapy vs monotherapy: one-year randomized trial. *Arch Ophthalmol.* 2002;120:915-922. 2. Data on file. Pfizer Inc, New York, NY.

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