Journal of the South East Asia Glaucoma Interest Group

Volume 9 Number 6 December 2007

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World Glaucoma Day

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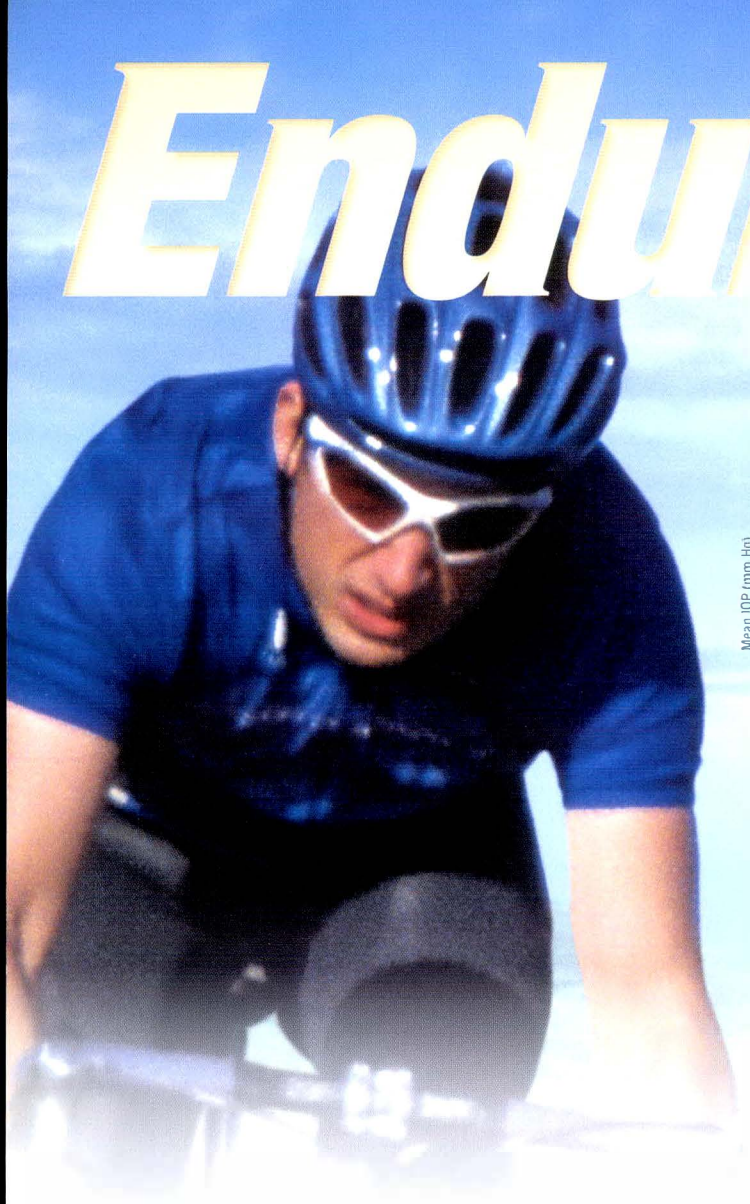
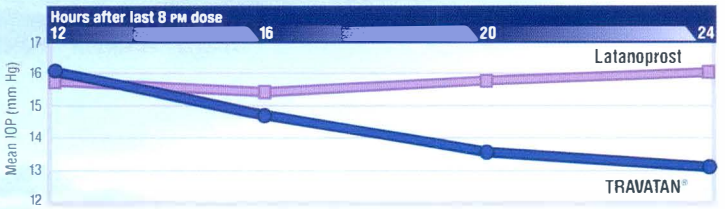
Endurance



TRAVATAN® solution is still going strong 24 hours post dose.[1*]



TRAVATAN®

(travoprost eye drops solution 0.004%)

Control That Lasts.

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 ω-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 test, 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 carcinogenicity 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 ($p=0.0117$).

Reference 1. Dubiner HB, Sircy MD, 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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Scientific Communications

LUMIGAN® (bimatoprost ophthalmic solution) 0.03% is indicated for the reduction of elevated intraocular pressure (IOP) in patients with open-angle glaucoma or ocular hypertension who are intolerant of other IOP-lowering medications or insufficiently responsive (failed to achieve target IOP determined after multiple measurements over time) to another IOP-lowering medication.

IMPORTANT SAFETY INFORMATION

LUMIGAN® (bimatoprost ophthalmic solution) 0.03% has been reported to cause changes to pigmented tissues. These reports include increased pigmentation and growth of eyelashes and increased pigmentation of the iris and periorbital tissue (eyelid). These changes may be permanent.

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.

See LUMIGAN® Prescribing Information.

Visit us at www.lumigan.com

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LUMIGAN®
(bimatoprost ophthalmic solution) 0.03%

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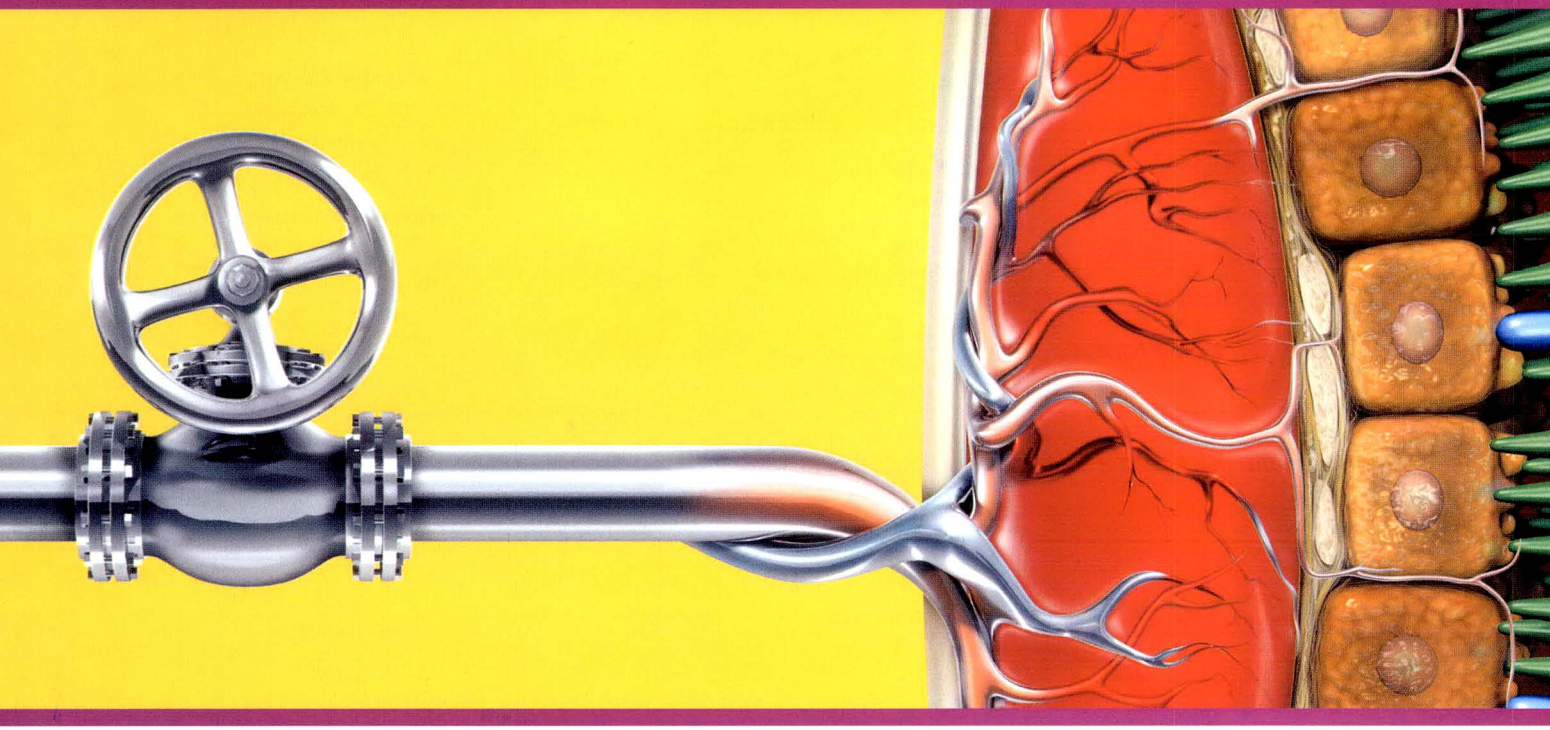

MACUGEN is the first selective VEGF$_{165}$† inhibitor with clinical efficacy to preserve‡ visual acuity in all neovascular AMD

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

References: 1. Data on file. Pfizer Inc, New York, NY. 2. Gragoudas ES, Adamis AP, Cunningham ET Jr, Feinsod M, Guyer DR, for the VEGF Inhibition Study in Ocular Neovascularization Clinical Trial Group. Pegaptanib for neovascular age-related macular degeneration. *N Engl J Med*. 2004;351:2805-2816.

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.**

MAC060003P

World Glaucoma Day, 6 March 2008: Tackling Glaucoma Internationally

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We have all heard many times that "Glaucoma is the second most common treatable cause of blindness worldwide". Glaucoma is a significant public health concern, being the leading cause of irreversible blindness and consistently ranking among the leading causes of blindness in virtually every nation.[1] In developing countries, cataract is the leading cause of blindness. In developed countries, the leading cause of blindness is age-related macular degeneration (AMD). However, there is a fundamental difference between these 2 diseases and glaucoma; their high rank as causes of blindness is due to structural reasons that are hard to address (limited access to surgical infrastructure for cataract and lack of an effective preventive treatment for AMD), whereas in the case of glaucoma, the main reason is low awareness of the disease and its implications. Indeed, it is estimated that only 50% of those affected with glaucoma in developed nations are aware that they have the disease,[2] while as many as 90% or more of people with glaucoma in underdeveloped countries are unaware of having the disease or have even heard of glaucoma. Despite our better understanding of risk factors for glaucoma, we have yet to see an improvement in these numbers. Worse, although glaucoma occurs in all age groups, it is more common in older adults, and with an ageing population, estimates of glaucoma prevalence are increasing. It has been predicted that by 2020, 79.6 million people worldwide will have glaucoma, 11.2 million of whom will be bilaterally blind,[1] up from the current 4.5 million.[3]

Recent years have seen considerable progress in the diagnosis and treatment of glaucoma. Technological advances in optic nerve and retinal nerve fibre layer imaging and visual field testing make it possible to diagnose glaucoma at earlier stages, when treatment has a better prognosis. Medical treatment is available and effective for controlling glaucoma for most patients,[4] while for those who have uncontrolled disease, laser and surgical interventions are often successful.

Optic nerve and visual field damage are irreversible. As damage progresses gradually, often unnoticed by the patient, early detection and treatment are of paramount importance to prevent blindness. For individuals with known risk factors for glaucoma, particularly elevated intraocular pressure, increasing age, African descent, family history of glaucoma, vasospasm, low blood pressure, and high myopia, the importance of routine examinations cannot be understated. Also, despite strong evidence that lowering intraocular pressure can delay the onset and progression of glaucoma,[5-7] reported rates of non-compliance with glaucoma therapy range from 5% to as high as 80%.[8] This high variability results from different definitions for non-compliance and the way it is measured.

World Glaucoma Day

The year 2008 is a pivotal one for glaucoma awareness. To combat the ignorance that leads to so much loss of vision, the World Glaucoma Association and the World Glaucoma Patient Association have joined forces to launch a global initiative aimed at raising awareness of glaucoma through an annual World Glaucoma Day. 6 March 2008 has been selected for the first World Glaucoma Day, as a way to jumpstart awareness activities and to enlist the active support of governments, eye care professionals, and patient support groups in many nations. Rather than have a single large event held in conjunction with a major conference, World Glaucoma Day will consist of the sum total of local, regional, and national initiatives and events organised by willing groups and individuals around the world.

These activities will include media campaigns; official recognition (3 countries will issue commemorative stamps and a delegation from the American Glaucoma Society will visit the US Congress); public-oriented events (eg, screenings in public places); and institutional events (open-door days at eye clinics and universities, with lectures addressed to professional and lay

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audiences). All these events, together with supporting material, general information on the disease, and useful links, will be listed at www.wgday.net.

The public needs to know that there are means for detecting and treating glaucoma and that sight can be saved. People should be made aware of the insidious nature of glaucoma and the importance of compliance with treatment and follow-up to minimise their risk of visual loss. People with glaucoma need to inform family members, so that they too can be screened. Health care providers, together with their government, should promote public awareness campaigns encouraging regular eye examinations, especially for those with known risk factors. Ophthalmologists should take the lead by writing articles for local newspapers, contacting radio stations, or organising public meetings on glaucoma. Earlier diagnosis and treatment will reduce visual disability, improve patients' quality of life, and decrease the overall costs of treatment. The World Glaucoma Association has set a goal of reducing the undiagnosed rate of glaucoma from 50% to "No more than 20% by 2020". This goal is attainable if all parties work together to increase awareness of glaucoma, both among the public as well as among health care providers, and if we all ensure quality eye examinations are available worldwide.

Your Participation

Each of us can help to make this a day to remember. The possibilities abound. Groups and individuals around the world have volunteered to organise events of the types described. Others have come up with original ideas: a national ophthalmology journal will publish a special glaucoma issue; other scientific journals will publish editorials on glaucoma; and a group of volunteers will run in the Geneva annual marathon under the WGD colours. In Latin America, a tongue-in-cheek movie has been produced to remind patients humorously that compliance with therapy is the best way to keep the "sneak thief of sight" at bay.

The success of World Glaucoma Day also depends on ophthalmologists actively participating at a national level, and by developing local strategies to achieve this goal. Every journey begins with a small step, but it is the cumulative number of steps that will determine how far we will go. As World Glaucoma Day approaches, take that first step: ask yourself "What can I do?" and make this campaign a success.

The possibilities are endless, but the success of the day will depend not only on the number and attendance of events organised globally, but also on their visibility. So, to maximise the impact of this initiative, please take a moment to visit either www.wgday.org or www.wgday.net and fill out the Intent Form in the section 'Be a Part of It', and tell the world how you will contribute to the success of the first-ever World Glaucoma Day.

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Comparison of the Efficacy and Safety of Bimatoprost and Timolol for Treatment of Chronic Angle Closure Glaucoma

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Aim: *To compare the efficacy, safety, and tolerability of bimatoprost 0.03% once daily with timolol 0.5% twice daily for patients with chronic angle closure glaucoma.*
Methods: *In this multicentre double-masked randomised comparative trial conducted in Thailand, India, and The Philippines, patients with chronic angle closure glaucoma who had previously undergone laser peripheral iridotomy were treated with bimatoprost (n = 107) or timolol (n = 105) for 3 months. Patients were assessed at baseline, 2 weeks, 6 weeks, and 3 months. The primary outcome measures were the mean percentage change in intraocular pressure from baseline (efficacy) and the number and type of adverse events (safety and tolerability).*
Results: *The mean total percentage decrease in intraocular pressure was significantly greater for the bimatoprost group than the timolol group at 2 weeks (30.6% vs 19.2%; p < 0.001), 6 weeks (29.7% vs 18.8%; p < 0.001), and 3 months (28.3% vs 18.4%; p < 0.001). The reduction in mean intraocular pressure 3 months from baseline was greater for the bimatoprost group than the timolol group (mean difference, -2.49 mm Hg; 95%confidence interval, -3.79 to -1.19 mm Hg). Conjunctival hyperaemia was the most frequently reported adverse event. There was no significant difference in the number of patients in each treatment group who withdrew because of an adverse event.*
Conclusions: *Bimatoprost once daily was more effective than timolol for lowering intraocular pressure in patients with chronic angle closure glaucoma. Both bimatoprost and timolol were well tolerated, with few systemic adverse events reported.*

Key words: *Bimatoprost, Clinical trial, Glaucoma, angle-closure, Timolol*

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Introduction

Chronic angle closure glaucoma (CACG) is of great concern in Asian populations because of the increasing number of patients affected, and because the condition is often asymptomatic and associated with significant visual morbidity.[1-4] By 2020, it is estimated that 87% of angle closure glaucoma will occur in people of Asian origin.[5] Despite the emergence of CACG as a significant public health issue in Asia, limited information is available on the most appropriate intervention for this disease.

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Currently, the preferred treatment for CACG is laser iridotomy to remove pupillary block and prevent further synechial closure, in conjunction with drug therapy to lower elevated intraocular pressure (IOP).[6] This combination of laser and drug therapy is not always successful for treating CACG in Asian patients, the majority of whom eventually require further surgery to alleviate the condition.[7,8] Several randomised controlled comparative clinical trials have established that the synthetic prostaglandin analogue, latanoprost, is associated with fewer systemic side effects and is more effective than timolol or timolol/dorzolamide for lowering IOP for patients with CACG.[9-11] Bimatoprost is a synthetic prostaglandin and prostamide analogue that is reported to enhance outflow of the aqueous humour through the uveoscleral pathway as well as the trabecular meshwork.[12,13] While the efficacy and tolerability of bimatoprost

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for reducing IOP in patients with open angle glaucoma has been established by large double-masked randomised comparative clinical trials enrolling predominantly Caucasian patients,[14-18] to the authors' knowledge, there is limited information on the efficacy and safety of bimatoprost for Asian patients with CACG.

The objectives of this study were to compare the efficacy, safety, and tolerability of bimatoprost monotherapy to timolol monotherapy given for 3 months to a large cohort of Asian patients with CACG.

Methods

Study Design and Setting

This prospective randomised multicentre double-masked parallel-group 3-month clinical trial was conducted in Thailand (4 centres), India (5 centres), and The Philippines (4 centres) between June 2002 and September 2004. The study was conducted in accordance with the International Conference on Harmonisation Guidelines for Good Clinical Practice, and local regulatory requirements. All investigators obtained appropriate institutional ethics committee approval before the study commenced. The study was conducted in compliance with the ethical principles of the Declaration of Helsinki and all patients gave voluntary signed informed consent before any study-related procedures or changes in treatment.

Patients

The primary inclusion criteria for enrolment were: age ≥18 years, good general health, unilateral or bilateral CACG confirmed by indentation gonioscopy, patent iridotomy/iridectomy 4 or more weeks before the baseline visit (or any other glaucoma surgery 12 or more weeks before baseline visit), IOP ≥21 mm Hg and <30 mm Hg (study LUM-CACG-001) or <34 mm Hg (study LUM-CACG-002) in one or both eyes at baseline after washout of any previous antiglaucoma medication, best-corrected visual acuity 20/100 or better in each eye, and a negative urine pregnancy test for women of childbearing age at the baseline visit. The definition of CACG in this study was appositional or synechial primary angle closure of at least 180° with at least half the trabecular meshwork blocked, some peripheral anterior synechiae or synechial angle closure present with glaucomatous optic neuropathy, and/or visual field defect.

Primary exclusion criteria were: uncontrolled systemic disease or active ocular disease other than glaucoma or ocular hypertension, significant ocular irritation at baseline, any known allergy to the study medications or formulation components, anticipated alteration of ongoing therapy with agents that could have a substantial effect on IOP or interact with the study medications or outcomes, chronic use of ocular medications other than the study medications, clinically relevant low or high heart rate or blood pressure for age, any contraindications to β-blocker therapy, any corneal abnormalities preventing accurate measurement of IOP with an applanation tonometer, refractive surgery, laser trabeculoplasty, or other intraocular or antiglaucoma surgery within the previous 3 months, pregnancy, lactation, or no reliable birth control for women of childbearing age.

Intervention and Randomisation

The study comprised a screening visit and 4 study visits (baseline, week 2, week 6, and month 3). Eligible patients being treated with ocular hypotensive medications began an appropriate washout period (4 weeks for topical β-blockers or prostaglandins, 2 weeks for α-agonists or sympathomimetics, 4 days for carbonic anhydrase inhibitors or parasympathomimetics) before commencing treatment at the baseline visit.

At the baseline visit, patients were randomised in a 1:1 ratio to receive bimatoprost 0.03% (Lumigan®; Allergan Inc, Irvine, USA) once daily (administered at night) and a placebo vehicle solution once daily (administered in the morning) or timolol maleate 0.5% twice daily (administered in the morning and at night). A randomisation list was generated using Statistical Analysis Systems (SAS) software (Version 8) [SAS Institute, Inc, Cary, USA] and code break envelopes were printed for each patient. Investigators and study coordinators were masked to the treatment each patient received.

All study medications were packaged in identical bottles labelled 'night only' and 'day only' as appropriate. Study medications were self-administered by instillation of 1 drop per eye, between 7 am and 9 am and between 7 pm and 9 pm. Treatment began in the evening of the baseline visit. At study visits, investigators administered the masked study medications in the morning, immediately after IOP was measured and after the patients' eyes were examined.

Procedures

Data from the worst eye were used for all analyses. IOP was measured at each study visit, at approximately 8 am, 10 am, and 4 pm using an applanation tonometer. The primary outcome measure of efficacy was the mean percentage change in total IOP at each time point (8 am + 10 am + 4 pm IOP/3) at each time point from baseline at 3 months. Secondary outcome measures were the mean change in IOP from baseline at 8 am (trough timolol effect), 10 am (peak timolol effect), and 4 pm at 3 months.

Severity of glaucoma (mild, moderate, or severe) and relationship of adverse events to study medication, slit-lamp biomicroscopy (5-point scale, none to severe), best-corrected visual acuity (Snellen equivalent units), sitting blood pressure (mm Hg) and pulse rate (bpm) were evaluated at each study visit. Visual fields (normal or abnormal; mean loss in dB) were examined by standard automated

perimetry within 60 days before the baseline visit and at month 3. Ophthalmoscopic examinations and measurement of cup/disc ratios were conducted using standard methods at the baseline and month 3 visits.

Statistical Analysis

All statistical analyses were completed using SAS software. Analyses of efficacy and safety were conducted on the intention-to-treat population (all patients who received study medication and had a valid assessment at the baseline visit and at least 1 follow-up visit), with the last observation carried forward. Ordinal categorical variables were analysed using the Wilcoxon rank-sum test. Nominal categorical variables were analysed using Fisher's exact test, Pearson's chi-squared test or Cochran-Mantel-Haenszel methods, where appropriate. Continuous variables were analysed using the Wilcoxon 2-sample test. Differences were considered significant at $p < 0.05$. Post-hoc analysis indicated that a sample size of 63 patients per group was needed to achieve 80% power with a difference of 1.5 mm Hg in mean IOP and a standard deviation of 3 mm Hg.

Results

Patient Demographics

212 patients were enrolled in the study; 83 from Thailand, 97 from India, and 32 from The Philippines. 107 patients were randomised to the bimatoprost treatment group and 105 to the timolol treatment group. The intention-to-treat population comprised all 212 patients.

There were no statistically significant differences in age, male:female ratio, or eye colour between the 2 groups at baseline (Table 1). There were no statistically significant differences in mean baseline IOP between groups at 8 am or 10 am, but the mean baseline IOP was 1.9 mm Hg higher in the bimatoprost group than in the timolol group at 4 pm ($p = 0.03$).

Twenty six patients withdrew from the study, 14 (13%) from the bimatoprost group and 12 (11%) from the timolol group (Table 2). The main reasons for withdrawal from the bimatoprost and timolol groups were loss to follow-up and adverse events, respectively.

Efficacy

Bimatoprost once daily was more effective than timolol twice daily in lowering IOP. IOP decreased in both treatment groups within 2 weeks and remained low until the end of the study. The mean percentage decrease in IOP from baseline was greater in the bimatoprost group than in the timolol group at each visit and all differences between treatment groups were statistically significant (Figure 1). At month 3, IOP in the bimatoprost and timolol groups were 17.8 mm Hg (SD, 6.7 mm Hg) and 19.0 mm Hg (SD, 4.9 mm Hg), respectively. The difference between the bimatoprost

Table 1. Patient demographics and baseline characteristics.

Characteristic	Bimatoprost group (n = 107)	Timolol group (n = 105)	p Value
Age (years)			
Mean (SD)	61 (8)	60 (10)	0.51
Range	41-86	32-83	
Sex			
Male (%)	42 (39)	37 (35)	0.55
Female (%)	65 (61)	68 (65)	
Baseline intraocular pressure (SD) [mm Hg]			
8 am	25.0 (5.7)	24.6 (4.0)	0.96
10 am	25.1 (5.1)	23.8 (4.5)	0.08
4 pm	24.5 (5.8)	22.6 (4.8)	0.03
Eye colour			
Brown (%)	58 (54)	54 (51)	
Dark brown (%)	49 (46)	49 (47)	0.35
Other (%)	0 (0)	2 (2)	

Table 2. Reasons for withdrawal from the study.

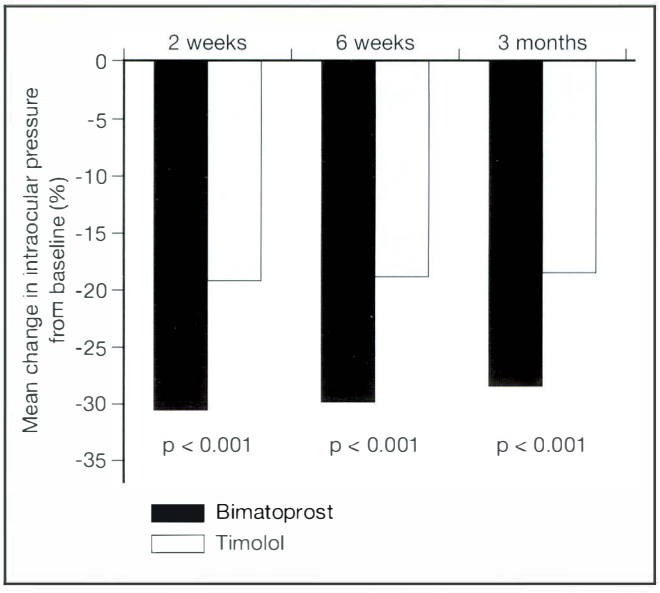
Reason for withdrawal	Bimatoprost group (n = 107)	Timolol group (n = 105)	All patients (n = 212)
Adverse event	3	3	6
Loss to follow-up	6	2	8
Other	2	5	7
Protocol violation	3	2	5
All reasons (%)	14 (13)	12 (11)	26 (12)

Table 3. Most frequent adverse events.*

Adverse event	Bimatoprost group (%) [n = 107]	Timolol group (%) [n = 105]
Conjunctival hyperaemia	28 (26)	9 (9)
Pruritus	16 (15)	—
Conjunctival congestion	6 (6)	—
Eyelash growth	5 (5)	—
Punctate keratitis	—	4 (4)
Eye irritation	4 (4)	4 (4)
Eye abnormality	3 (3)	—
Headache	3 (3)	—
Vertigo	—	3 (3)

* For events where frequency was >2.5%.

Figure 1. Mean change in total intraocular pressure from baseline at each follow-up visit. p Values indicate statistically significant differences between treatment groups.



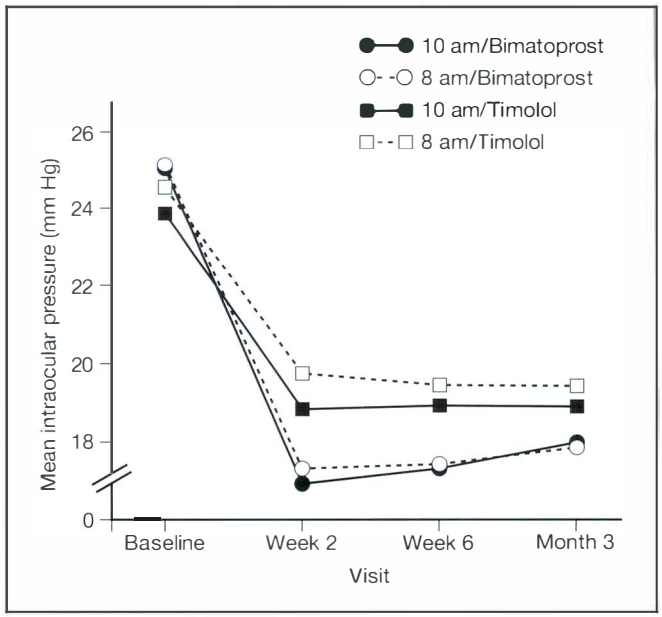
and timolol groups in IOP reduction from baseline at month 3 was -2.49 mm Hg (95% confidence interval [CI], -3.79 to -1.19 mm Hg).

The ability of bimatoprost once daily to lower IOP was consistent throughout the day for all follow-up visits (Figure 2). The reductions in mean IOP at month 3 from the baseline visit were significantly greater in the bimatoprost group than in the timolol group at the 8 am (28.0% vs 20.2%; $p < 0.001$), 10 am (28.1% vs 18.2%; $p = 0.001$), and 4 pm (26.6% vs. 13.4%; $p < 0.001$) time points. The mean differences in the reduction in IOP at month 3 from baseline at the 8 am, 10 am, and 4 pm time points between the bimatoprost and timolol groups were -2.0 mm Hg (95%CI, -3.5 to -0.6 mm Hg), -2.3 mm Hg (95%CI, -3.8 to -0.7 mm Hg), and -3.0 mm Hg (95%CI, -4.5 to -1.5 mm Hg), respectively.

Safety and Tolerability

Bimatoprost and timolol had favourable safety and tolerability profiles. The majority of the adverse events in the bimatoprost

Figure 2. Mean intraocular pressure at 8 am (trough timolol effect) and 10 am (peak timolol effect).



(n = 90) and timolol (n = 44) groups were mild (78% vs 65%, respectively). Approximately half of the adverse events in each group were considered to be related to the study medication (58% vs 44%, respectively). Three patients withdrew from each treatment group because of an adverse event. The reasons for each patient withdrawing from the bimatoprost group were face and body swelling, periocular pigmentation, or red eyes. The reasons for patients withdrawing from the timolol group were breathlessness and bronchospasm, anxiety and palpitations, or conjunctival hyperaemia. In both treatment groups, the most frequently reported adverse event was conjunctival hyperaemia (Table 3).

There were no significant differences between treatment groups in cup-disc ratio, best-corrected visual acuity, visual fields, biomicroscopic signs, blood pressure, or pulse rate at month 3 compared with baseline (data not shown).

Discussion

This study provides new data on the efficacy, safety, and tolerability of bimatoprost for Asian patients with CACG. In this cohort of patients, bimatoprost was well-tolerated and was more effective than timolol for lowering IOP and providing diurnal IOP control.

To date, most studies examining the efficacy and tolerability of bimatoprost have been conducted in patients with open angle glaucoma from predominantly Caucasian populations.[14-18] To the authors' knowledge, only one small, preliminary study has examined the ocular hypotensive efficacy of bimatoprost in patients with angle closure glaucoma.[19] In this non-randomised uncontrolled study of 32 patients with CACG from India, Agarwal et al found a

31% reduction in IOP in patients who had switched from timolol/pilocarpine combination therapy to bimatoprost monotherapy after 3 months of treatment.[19] In this larger randomised comparative study, mean IOP decreased by 28% after 3 months of treatment for patients who were randomised to receive bimatoprost and by 18% for those who received timolol. The reductions in IOP with bimatoprost in this study confirm the preliminary findings of Agarwal et al and are equivalent to the reductions achieved in similar comparative studies of CACG in Asian patients with latanoprost and timolol[9,10] and latanoprost and travoprost.[20] Together, these data confirm that synthetic prostaglandins are more effective than timolol for lowering IOP in patients with CACG.

Although the findings of this study represent mean responses from groups of patients and do not reflect the range of individual responses that may occur, significantly greater reductions in IOP were achieved with bimatoprost than with timolol at each time point throughout the day, with the mean IOP in the bimatoprost group decreasing from 25 mm Hg at baseline to 18 mm Hg after 3 months. These results, if sustained in the long term, suggest that clinically significant reductions in loss of visual field can be achieved.[21] Overall, the results from this study should reassure practitioners that bimatoprost is an effective therapy that can be used as an alternative to timolol for patients with CACG. Moreover, as bimatoprost is a prostamide analogue that is reported to lower IOP through 2 different mechanisms,[12,13] bimatoprost may also be a useful alternative for patients who are unresponsive to the hypotensive effects of other synthetic prostaglandin analogues.[22]

The favourable safety and tolerability profiles of bimatoprost and timolol in this Asian cohort are similar to the findings from other studies of bimatoprost conducted in Asian patients with CACG[19] and open angle glaucoma,[23,24] and are consistent with the findings of trials enrolling predominantly Caucasian patients with open angle glaucoma.[14-18] Overall, bimatoprost and timolol were well-tolerated with few systemic adverse events reported, most of which were mild. The patient who withdrew from the timolol group because of breathlessness and bronchospasm highlights the potential for timolol to cause respiratory side effects in patients with undiagnosed respiratory impairment,[25-27] and supports the need for an assessment of respiratory function in patients, particularly elderly people, who are treated with topical timolol.[26] While localised adverse events were reported more frequently in the bimatoprost group than in the timolol group, few events in either group resulted in patients discontinuing treatment. Therefore, despite the greater frequency of local adverse events with bimatoprost, the benefits of a greater reduction in IOP with this drug can be considered to outweigh most local adverse events that may arise.

In conclusion, this randomised multicentre trial conducted in 3 Asian countries confirms that bimatoprost is an effective and well-tolerated alternative to timolol for lowering IOP in Asian patients with CACG.

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Effect of Diabetes on Visual Acuity following Phacoemulsification Cataract Surgery

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Aim: *To assess the effect of diabetes on visual acuity following phacoemulsification cataract surgery.*
Methods: *This was a comparative cross-sectional study of 51 eyes of patients with diabetes with no diabetic retinopathy and 51 eyes of patients without diabetes who underwent phacoemulsification cataract surgery. Visual acuity was assessed with the logMAR visual acuity chart for best-corrected near and distance vision at 1, 6, and 12 weeks postoperatively. Fundus photographs were taken at each follow-up visit for assessment and documentation of diabetic retinopathy. Glycosylated haemoglobin was determined for the patients with diabetes at the start (glycosylated haemoglobin$_1$) and end (glycosylated haemoglobin$_2$) of the study. The anterior chamber reaction was assessed on the first postoperative day for all patients. The follow-up period was 12 weeks.*
Results: *The eyes of patients with diabetes were significantly associated with poorer best-corrected near visual acuity ($p = 0.027$). These eyes also had a slower rate of best-corrected near and distance visual acuity improvement. The glycosylated haemoglobin$_1$ and glycosylated haemoglobin$_2$ were correlated with the best-corrected near visual acuity ($r = 0.282$ and $p = 0.04$; and $r = 0.355$, $p = 0.01$, respectively). A similar correlation was found for glycosylated haemoglobin$_1$ and glycosylated haemoglobin$_2$ for best-corrected distance visual acuity ($r = 0.287$, $p = 0.04$; and $r = 0.393$, $p = 0.004$, respectively). The anterior chamber reaction was generally more severe in the patients with diabetes. Five of 51 eyes of patients with diabetes (9.8%) developed mild non-proliferative diabetic retinopathy.*
Conclusions: *The patients with diabetes had significantly poorer best-corrected near visual acuity postoperatively, although there was no clinical evidence of diabetic retinopathy at the start of the study. The postoperative visual acuity was correlated with diabetic control.*

Key words: *Diabetic retinopathy, Phacoemulsification, Visual acuity*

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Introduction

Cataract and diabetic retinopathy are 2 major causes of blindness in many countries. These 2 ocular pathologies often coexist in older populations. In 1995, the prevalence of diabetes among adults was estimated to be 4.0% (135 million), and was estimated to rise to 5.4% (300 million) by the year 2025.[1] With the increase in the number of people with cataracts as populations age, these pathologies will have a health and economic impact, particularly in the developing countries of Asia.

The Beaver Dam Eye Study[2] and the National Health and Nutrition Examination Survey[3] had shown that cataract occurred more commonly in patients with diabetes. Cataract surgery in patients with diabetes has been associated with a high incidence of post-operative complications such as fibrinous uveitis, posterior capsule opacification,[4] anterior segment revascularisation, accelerated progression of diabetic retinopathy,[5] and macular oedema.[6]

In patients with diabetes, excess glucose is converted to sorbitol, which accumulates in the lens and leads to cataract formation. In addition, non-enzymatic glycation of the soluble protein of the crystalline lens is reported to be higher in diabetic lenses than in non-diabetic lenses.[7]

Trauma during cataract surgery may contribute to the break-down of the blood-retina barrier, which is already impaired in patients with diabetes. The underlying pathogenic process is inflammatory in origin, with the retinal capillaries manifesting a pathological response to cataract surgery, resulting in disruption

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of the blood-retina barrier.[8] This process may explain the increased incidence of retinal haemorrhage, exudates, and macular oedema following cataract surgery.

During cataract surgery, the opening of the limbal or corneal wound leads to a sudden drop in intraocular pressure (IOP) to almost 0 mm Hg. By Starling's law, the balance of hydrostatic pressure and the oncotic pressure at the vessel wall determines the passage of fluid across the capillary wall. Hypotony would induce movement of fluid from the vascular lumen to the extravascular space, thus aggravating macular oedema.[9] Further, human lens extracts can inhibit vascular endothelial proliferation.[10]

Many studies have shown evidence of progression of diabetic retinopathy following cataract surgery.[5,11,12] However, less is known about the visual outcome and the risk of developing diabetic retinopathy for patients with diabetes without clinically detectable diabetic retinopathy. The current inability to manage diabetic retinopathy effectively, coupled with the potential public health impact of diabetic retinopathy, are compelling reasons for continuing to investigate this condition, when damage limitation is still possible. Therefore, this study was designed to evaluate the outcomes and the factors contributing to the visual acuity of patients with diabetes with no retinopathy after cataract surgery, and to compare these factors with patients with no diabetes. Phacoemulsification cataract surgery was preferred to extracapsular cataract extraction for this study because of the reduced early postoperative inflammation, wound-related complications, astigmatism, and posterior capsule opacity.[13]

Methods

The study was a comparative cross-sectional study.

Patients

All patients undergoing planned Kelmann-phacoemulsification cataract surgery in the Ophthalmology Department, Pulau Pinang Hospital, Pulau Pinang, Malaysia, from 1 June 2004 to 31 August 2005 who fulfilled the selection criteria were included. The study group consisted of 51 patients with diabetes mellitus type 2 with no pre-existing diabetic retinopathy. The patients were matched for age, sex, and race with 51 patients without diabetes (fasting plasma glucose level of <7 mmol/L [normal range, 3.9-6.1 mmol/L] and absence of diabetic symptoms) as a control group. All patients with other types of diabetes, pre-existing ocular disease, previous ocular surgery, astigmatism >1 D, or myopia >6 D were excluded.

Design

Each patient underwent phacoemulsification cataract surgery performed by a single surgeon. The surgeon was masked to the diabetic status of the patient. Only one type of intraocular lens (IOL) — a hydrophobic acrylic single-piece IOL — was implanted. The selected IOL power aimed for postoperative emmetropia for all patients.

Patient selection was done after fundus examination to exclude those with pre-existing diabetic retinopathy. Glycosylated haemoglobin (HbA_{1c}) was tested once the patients had fulfilled the criteria for the study group (HbA_{1c1}). All patients were treated with Maxitrol (dexamethasone 0.1%, neomycin sulfate, polymyxin B sulfate) 2-hourly after surgery. After 1 week, Maxitrol was reduced to 4 times daily, and treatment was stopped after 6 weeks, depending on the anterior chamber reaction. All postoperative complications were treated accordingly.

Eye examination included visual acuity assessment using the logMAR visual acuity chart for near and distance vision, and slit-lamp examination and fundoscopy with a 90 D lens was performed for all patients after 1 day, and 1, 6, and 12 weeks. The anterior chamber reaction was assessed by the surgeon using slit-lamp microscopy and was graded based on the Hogan-Kimura-Thygeson system. Fundus photographs were taken using a Nikon NF-505 AF fundus camera (Nikon, Tokyo, Japan). The severity of diabetic retinopathy and clinically significant macular oedema were defined according to the Early Treatment Diabetic Retinopathy Study grading system and criteria.[14] A second HbA_{1c} test was done at week 12 (HbA_{1c2}). Defaulters during the study period were excluded from the study and replaced with the next patient who fulfilled the selection criteria.

Statistical Analysis

The sample size was calculated based on a study power of 0.80, with an α error of 0.05 and standard deviation of 0.18. The data were tabulated by using the Statistical Package for the Social Sciences (SPSS) for Windows version 13.0. Statistical analysis was done using SPSS for Windows version 13.0 and Statisca Version 5A.

Results

The demographic characteristics of the study participants are summarised in Table 1. The best-corrected near visual acuity (BCNVA) and best-corrected distance visual acuity (BCDVA) of patients with diabetes and the control patients at the end of the study are shown in Table 2. There was a statistically significant association between BCNVA and the diabetic status of the patients ($p = 0.027$); patients with diabetes had poorer BCNVA than the control patients. However, no significant differences were found for BCDVA.

Both groups had significant improvement in BCNVA and BCDVA postoperatively. Figure 1 shows the BCNVA at week 12.

Table 1. Demographic characteristics of patients with and without diabetes undergoing cataract surgery.

	Patients with diabetes	Patients without diabetes
Number of patients	51	51
Age at surgery (years)		
Mean (SD)	64.22 (7.89)	64.16 (7.48)
Range	47-75	42-75
Sex		
Male	27	27
Female	24	24
Ethnicity		
Chinese	36	36
Malay	8	8
Indian	7	7
Duration of diabetes (years)		
Mean (SD)	7.10 (7.96)	
Range	1-30	

Table 2. Best-corrected near and distance visual acuity 12 weeks after cataract surgery.

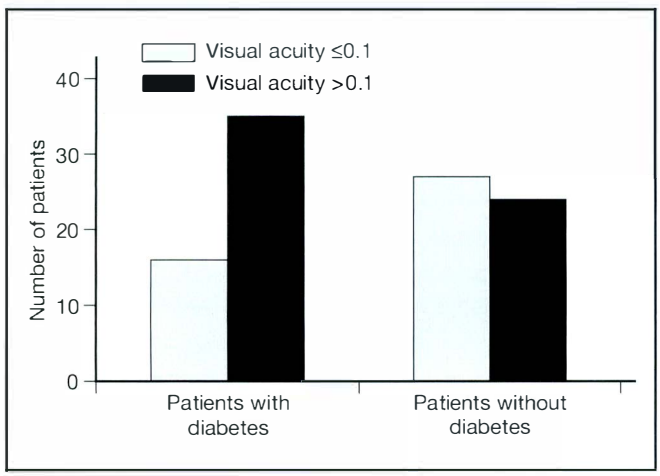
	Patients with diabetes Number (%)	Patients without diabetes Number (%)
Best-corrected near visual acuity		
≤0.10*	16 (31.37)	27 (52.94)
>0.10	35 (68.63)	24 (47.06)
Best-corrected distance visual acuity		
≤0.10	33 (64.71)	38 (74.51)
>0.10	18 (35.29)	13 (25.49)

* 0.10 logMAR is equivalent to 6/7.5 Snellen visual acuity.

The rates of improvement for both BCNVA and BCDVA were faster for the control group than for the study group, although the difference was not statistically significant (Figure 2).

There was a correlation between the HbA_{1c} levels (HbA_{1c1} and HbA_{1c2}) and BCNVA at week 12 ($r = 0.282$, $p = 0.04$; and $r = 0.355$, $p = 0.01$, respectively) [Figure 3]. A similar pattern was also found for BCDVA ($r = 0.287$, $p = 0.04$; and $r = 0.393$, $p = 0.004$, respectively).

Figure 1. Best-corrected near visual acuity of patients with and without diabetes 12 weeks after cataract surgery.



The anterior chamber cell activity was generally more severe in patients with diabetes than in those without diabetes, but this was not statistically significant (Figure 4).

Five of 51 patients with diabetes (9.80%) developed mild non-proliferative diabetic retinopathy with no maculopathy. However, there was no significant difference in duration of diabetes between the patients who developed diabetic retinopathy and those who did not. There was no significant difference in the HbA_{1c} levels at the beginning and end of the study. The patients with diabetes who developed diabetic retinopathy had a mean HbA_{1c1} of 7.10% (SD, 2.08%), which was lower that that of patients with diabetes who did not develop diabetic retinopathy (mean HbA_{1c1}, 7.36%; SD, 1.35%). However, patients with diabetes who developed diabetic retinopathy had a higher mean HbA_{1c2} of 7.66% (SD, 2.01%) than that of patients with diabetes who did not develop diabetic retinopathy (mean HbA_{1c2}, 7.40%; SD, 1.54%). All patients who developed diabetic retinopathy were men, but no significant differences for age and race were found.

The postoperative complications are summarised in Table 3. Patients with diabetes generally had more corneal complications. One patient with diabetes developed a corneal ulcer, which significantly impaired the final BCNVA and BCDVA. Three patients with diabetes had severe punctate epithelial erosion, which required intensive lubrication and more frequent follow-up. One patient in the control group developed clinical cystoid macular oedema (CMO) but fundus fluorescein angiography did not support the diagnosis.

Figure 2. Comparison of best-corrected near and distance visual acuity of patients with and without diabetes.

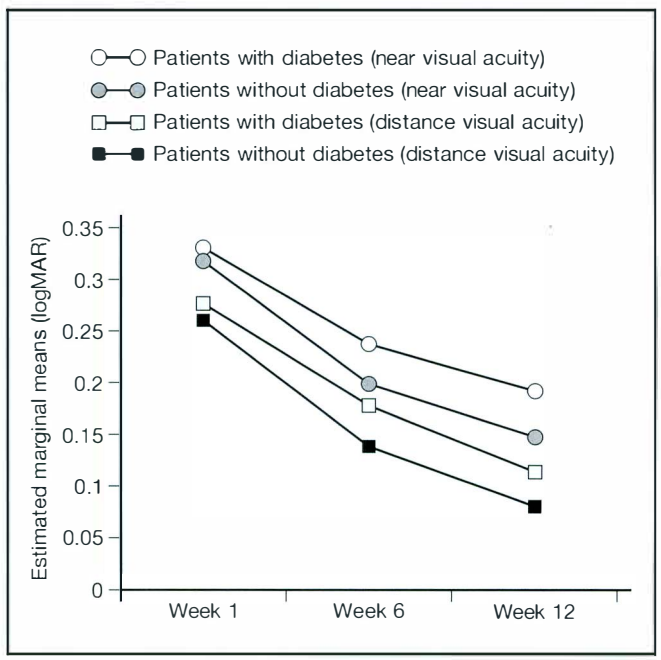


Figure 3. Correlation of glycosylated haemoglobin at the start and end of the study with best-corrected near visual acuity.

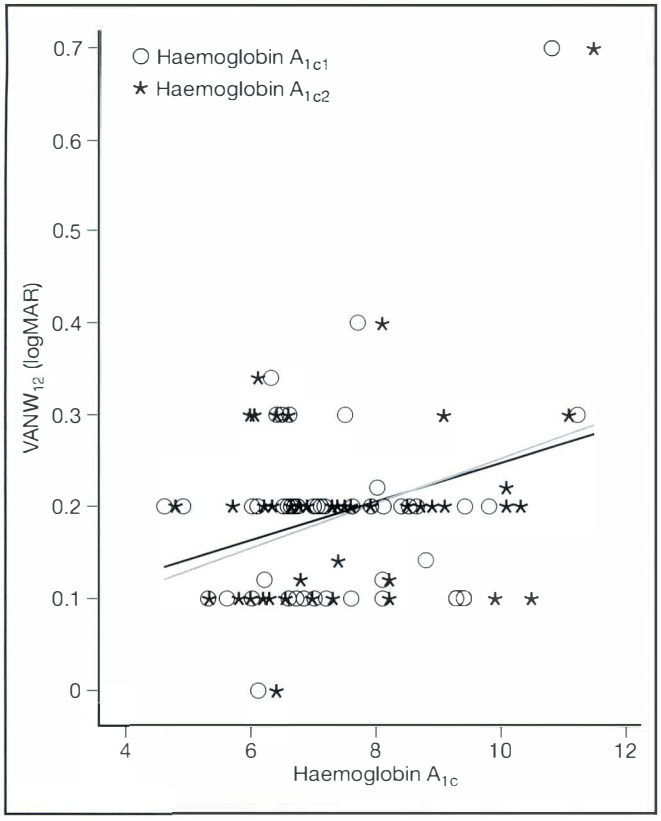


Figure 4. Comparison of the anterior chamber cell activity between patients with and without diabetes on postoperative day 1.

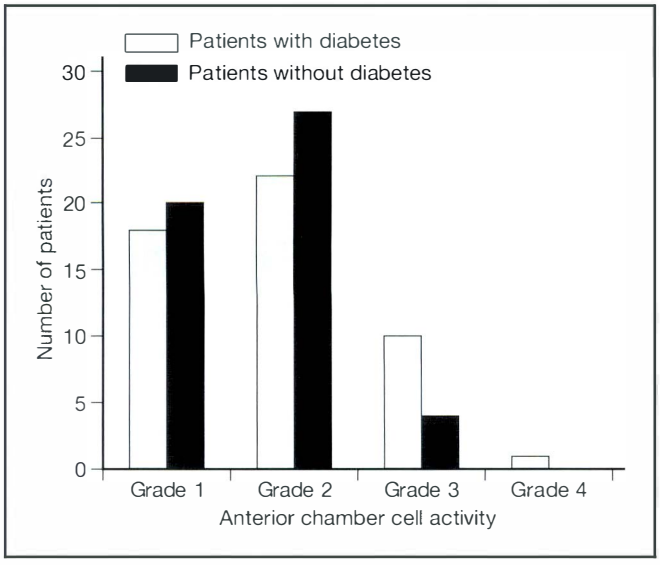


Table 3. Postoperative complications of patients with and without diabetes undergoing cataract surgery.

	Patients with diabetes	Patients without diabetes
Wound leak	3	2
Iris prolapse	1	1
Suture infiltrate	3	6
Corneal ulcer	1	0
Severe punctate epithelial erosion	3	1
Capsular opacity	5	3
Intraocular lens malposition	1	0
Significant iris trauma with updrawn pupil	1	0

Discussion

The study results showed that the patients with diabetes had poorer BCNVA at week 12 than the control group, but the difference between the 2 groups for BCDVA was not statistically significant. Patients with diabetes also had slower improvement of visual acuity during the postoperative period than the control group. One possible explanation for these findings may be that there is a high incidence of increased retinal thickness after cataract surgery in eyes of patients with diabetes, which has been demonstrated by optical coherence tomography. It has been shown that eyes with diabetic retinopathy developed thickening of 18 μm and 14 μm 1 and 3 months after cataract surgery, respectively.[15,16] Eyes with diabetic retinopathy had more severe thickening and were associated with poorer visual acuity. In addition, the macular is more sensitive to detailed near vision. Cusick et al demonstrated that central visual function had a statistically significant relationship with the National Eye Institute 25-Item Visual Function Questionnaire Near Activities subscale scores but not with the Distance Activities subscale scores.[17]

The study reported here also concluded that an important predictor of postoperative visual acuity is HbA_{1c} level. The pre- and postoperative HbA_{1c} levels were correlated with the final BCNVA and BCDVA. The higher HbA_{1c} level during the visual recovery period (HbA_{1c2}) might have contributed to the poorer BCNVA in patients with diabetes.

Several large prospective clinical studies have shown a strong relationship between HbA_{1c} level and diabetic complications for both type 1 and type 2 diabetes.[18-20] Klein et al have extensively studied the relationship of glycaemic control and diabetic complications, and estimated that 1% increase in HbA_{1c} at baseline was associated with nearly 60% increase in the incidence of diabetic retinopathy and nearly 100% increase in the rate of progression to proliferative diabetic retinopathy.[21-23]

The possible pathogenic process of chronic hyperglycaemia causing microvascular complications is through the formation of advanced glycation endproducts, which initiates a series of damaging processes, including protein kinase C (PKC) activation, oxidative stress, and increased vascular endothelial growth factor (VEGF) expression.[24-26] Activation of specific PKC isoforms appears to play a role in reduced retinal blood flow, extracellular matrix deposition, basement membrane thickening, increased permeability, and neovascularisation.[25-27]

Hyperglycaemia is also thought to increase the expression or action of vasoconstrictors such as endothelin-1 and angiotensin. The overexpression of vasoconstrictors results in increased vascular resistance and decreased retinal blood flow.[28,29] Overproduction of the reactive free radical molecule superoxide in diabetic eyes induces VEGF expression in the retina and amplifies the damaging effect of hyperglycaemia.[30]

Patients with diabetes with no clinical evidence of diabetic retinopathy may experience a subclinical pathological process that involves hyperglycaemia-induced reactive free radical molecule superoxide and PKC activation, causing vascular damage. Amin et al demonstrated VEGF expression in diabetic eyes with little or no diabetic retinopathy.[31] The initial metabolic lesions that eventually produce diabetic retinopathy may develop some time before the disease becomes evident clinically.

In this study, there was no significant difference in the HbA_{1c} level between patients with diabetes who developed diabetic retinopathy and those who did not. This finding does not support some important clinical trials,[18,19] but it is consistent with the results of a study by Suto et al[32] of the effect of perioperative glycaemic control in progression of diabetic retinopathy and maculopathy. These authors found no significant differences in the rate of progression of diabetic retinopathy and maculopathy between the poor glycaemic control group and the well-controlled group.

It is well known that eyes of patients with diabetes have more pronounced postoperative inflammation.[33,34] Diabetic eyes also have difficulty with pupil dilation, large lenses, and steep anterior lens curvature, which may make surgery more traumatic, as well as alterations in the blood-retina barrier, which contributes to the severity of the anterior chamber reaction. This study found that the anterior chamber cell activity was consistently more severe in the group of patients with diabetes than in the control group. However, there was no significant effect on the final visual acuity at week 12. The clinical implications of the findings are that patients with diabetes require frequent review and intensive topical anti-inflammatory agents postoperatively.

In this study, 5 of 51 patients with diabetes (9.8%) developed diabetic retinopathy at week 12. In a 6-month study of 223 patients by Wagner et al, 18.4% of eyes without preoperative diabetic retinopathy developed background diabetic retinopathy.[35]

No significant association was found between the duration of diabetes and the development of diabetic retinopathy. A possible explanation for this observation may be that the time of diagnosis of type 2 diabetes does not reflect the real duration of the disease, which may have been undetected for several years. This is especially likely in the developing countries of Asia. It is important to note that duration of disease does not always have a consistent linear effect on the rate of development of diabetic retinopathy. Other factors such as genetic predisposition may explain why some patients are more resistant to developing diabetic retinopathy.

Postoperative onset of CMO after cataract surgery with IOL implantation is a frequent complication for patients with diabetes, but this did not occur in this study. Angiographic CMO may have been present, but this was not clinically evident. In addition, the follow-up period of this study was short, and post–cataract surgery CMO may occur months or years after the operation. Improvements in surgical technique and the exclusion of cases complicated by posterior capsule rupture or vitreous loss intraoperatively may explain the absence of CMO found in this study. Pollack et al reported that CMO occurred postoperatively in 32% of eyes without pre-existing diabetic retinopathy, and that angiographic CMO was more common than clinical CMO.[6]

In conclusion, patients with diabetes had significantly poorer BCNVA than patients without diabetes at the end of the study. Pre- and postoperative HbA_{1c} was correlated with BCNVA and BCDVA. Thus, HbA_{1c} can be a useful predictor of postoperative visual acuity. The anterior chamber cell activity following cataract surgery was generally more severe in patients with diabetes.

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Temporalis Muscle Transfer for the Treatment of Lagophthalmos

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Lagophthalmos is one of the ocular conditions associated with leprosy and seventh cranial nerve palsy due to other causes. Both medical and surgical treatments are available for this condition. Medical treatment is generally symptomatic, comprising lubricating eye drops or gel. There are several surgical techniques available, one of which is temporalis muscle transfer to provide static support with dynamic function. This review presents the results for 6 patients with lagophthalmos secondary to Hansen's disease who underwent temporalis muscle transfer.

Key words: *Bell palsy, Cranial nerve diseases, Leprosy, Paralysis*

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Introduction

Lagophthalmos is the inability to close the upper eyelid. The condition is a form of facial palsy affecting the orbicularis oculi muscle (paralysis of the mandibular division of the facial nerve). There are many causes of lagophthalmos, and the condition may be congenital or acquired (associated with Bell's palsy, Hansen's disease, trauma, and tumours).

The eyelid plays an important role in protecting and nourishing the eye. When blinking or eyelid closure function is lost, the health of the eye is at risk. Complications associated with lagophthalmos include severe dry eye and discomfort, exposure keratitis, corneal ulceration and perforation, decreased vision, and cosmetic disfigurement.

Treatment Options for Lagophthalmos

Both medical and surgical treatment options are available for lagophthalmos. If the paralysis is expected to be short term (<6 months), medical treatment with lubricating eye drops or gel used throughout the day will help to maintain the health of the ocular surface. If the paralysis is likely to persist for the long term (>6 months) or there is no improvement with medical therapy, then surgery may be performed to improve the function of the eyelid.

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There are several surgical procedures for the correction of lagophthalmos,[1] including:

- lateral tarsorrhaphy
- lid loading procedure
- stainless steel weight implant[2]
- gold weight implant
- Morel-Fatio spring[3]
- silicone sling
- lid magnets
- Edgerton-Montandon procedure[4]
- temporalis muscle transfer surgery and its modifications[5,6]
- free muscle graft
- cross seventh cranial nerve transfer
- temporalis muscle re-innervation with sural nerve graft.

A modified method of temporalis muscle transfer using fascia lata from the thigh has been successfully performed at the Shree Sharadadevi Eye Hospital and Research Centre, Swami Vivekananda Integrated Rural Health Centre, Pavagada, India. This review describes the technique and outlines the results of this procedure for 6 patients with lagophthalmos secondary to Hansen's disease.

Background

In 1991, the Swami Vivekananda Integrated Rural Health Centre incorporated leprosy control and prevention of disability programmes. The Government of India has pledged to conduct reconstructive surgeries in 5 districts of Karnataka (Tumkur, Chiradurga, Chikkamagalur, Davanagere, and Kolar). Six patients

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with lagophthalmos secondary to Hansen's disease[7] underwent reconstructive surgery, in the form of temporalis muscle transfer using fascia lata from the lateral aspect of the thigh.

All 6 patients were men (age range, 20 to 45 years). Five patients had had lagophthalmos for 2 years and 1 for 1 year. One patient had poor Bell's phenomenon leading to altered ocular surface integrity. All patients had intact corneal sensation.

Technique

After performing a femoral nerve block, the lateral part of the thigh was cleaned and covered. An incision measuring approximately 4 cm was made over the lateral aspect of the thigh, approximately 10 cm above the head of the fibula. The tissue was dissected to identify the fascia lata, which was freed from its attachments. A second incision of approximately 1.5 cm was made over the lateral aspect of the thigh approximately 10 cm above the first incision. The fascia lata was harvested from the lateral aspect of the thigh with a fascia lata stripper. A 15-cm strip of fascia lata, with a width of 1.0 to 1.5 cm, was isolated and stripped in a proximal direction and removed via the stab incision. The fascia lata was placed in normal saline and both incisions were closed with sutures.

After local infiltration of adrenaline, the operation site was cleaned and covered. A posteriorly curved incision of 4 to 5 cm was made above the zygoma, at the superior limit of the external border of the pinna, within the hairline. The incision extended through the levator pinnae to the temporalis fascia. A 2- to 3-cm window was cut in the temporalis fascia to expose the fibres of the temporalis muscle.[6] Using blunt-nosed dissection scissors, a fascicle of muscle, 1 cm thick and 2 to 3 cm wide, was isolated and freed (Figure 1).

The fascia lata was cleaned and any attached tissues were removed. The fascia lata was tightly attached to the free end of the isolated muscle belly. The free end of the fascia lata was passed beneath the full thickness of the temporal skin to a radial incision over the lateral orbital margin, where it was divided into 2 slips, one for each eyelid (Figure 2). A curved incision was made over the medial palpebral ligament and the ligament was exposed and cleaned. Two small incisions were made parallel to and close to the ciliary margin in the middle of the upper and lower eyelids. A fine supramuscular tunnel was made beneath the full thickness of the eyelid skin near the lid margin in both eyelids. The slips of the fascia lata were passed from the lateral orbital incision, through the lid tunnels, to the medial incision using the middle incision as a step (Figure 3). Both the slips were attached to the medial palpebral ligament[6] under sufficient tension to close the eyes with a small amount of overlap of the upper over the lower lids; the overlap disappears after a few days. Antibiotic ointment was placed in the conjunctival sac. The incisions were closed with fine non-absorbable sutures and the wounds were dressed and bandaged.

Figure 1. Dissection of the temporalis muscle and tying of fascia lata.

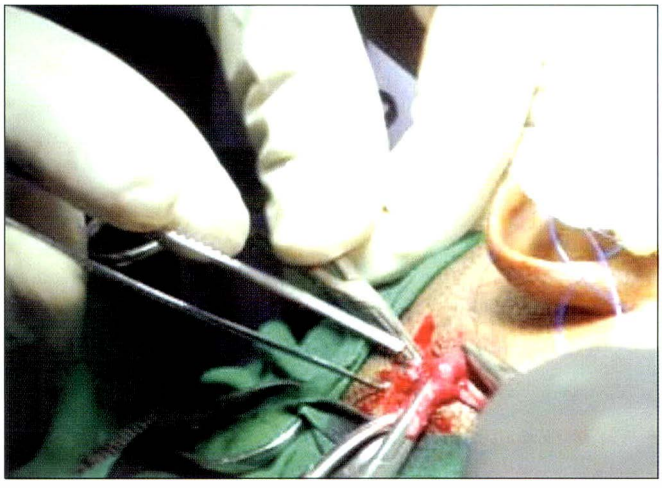


Figure 2. Dividing the fascia lata into 2 parts for each eyelid.

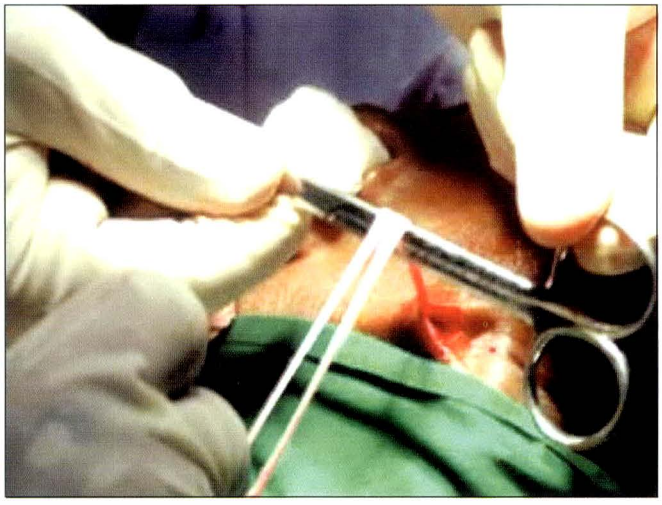
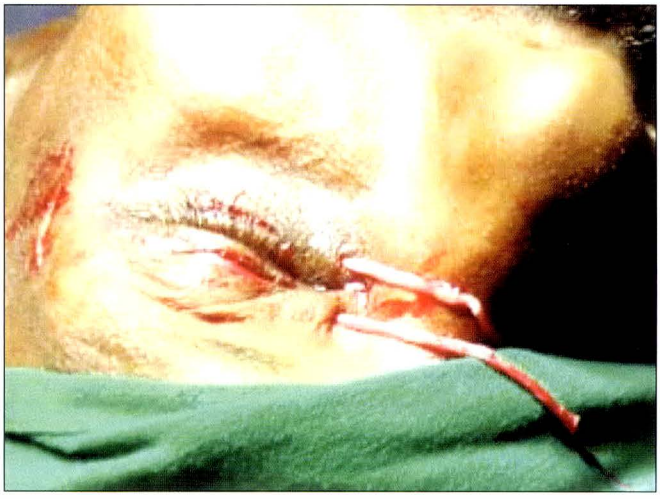


Figure 3. Fascia lata after passing through the eyelids.



Postoperatively, patients were required to eat only soft food for 2 weeks. After 2 weeks, patients were encouraged to gently chew on chewing gum and to observe the effect in a mirror. Patients were instructed to frequently close their eyes using the muscle slip. For the first 2 to 3 months, patients tended to clench their teeth for eye closure, although they soon dissociated clenching of teeth and were able to close their eyelids effortlessly. Follow-up was done after 1, 3, and 6 months, and yearly up to 3 years.

Outcomes

The results were based on palpebral fissure height during light eyelid closure and tight eyelid closure, and ocular surface integrity such as tear film deficiency, persistent epithelial defect, tear film stability, and symptoms of redness and tearing. The ocular surface integrity, symptoms of redness and tearing, and palpebral fissure height improved over time (Table 1). Figures 4 and 5 show the eyelid closure of one patient pre- and postoperatively.

Conclusion

Dynamic reconstruction of eyelid closure by muscle transposition or by free muscle transplantation offers an effective solution for regaining near-normal eye protection without the need for implants. One of the surgical techniques for lagophthalmos is temporalis muscle transfer by Gillie's method.[5] Many ophthalmic surgeons have tried this procedure using temporalis fascia,[6] and have achieved good results. However, the long-term function has not been promising.

Temporalis muscle transfer is the procedure of choice for adults,[7] but free muscle transplantation is preferred for children. This is so as not to interfere with the growth of the face by transposition of a masticatory muscle.

Table 1. Outcomes after temporalis muscle transfer using fascia lata from the thigh to treat lagophthalmos.

	Preoperative	Postoperative		
		1 month	3 months	6 months
Symptoms of tearing and redness	Present	Improved	Improved	Asymptomatic
Palpebral fissure height* at rest (mm)	8-10	8-9	8-9	8-9
Palpebral fissure height during light eyelid closure (mm)	7-9	5-6	2-3	1-2
Palpebral fissure height during tight eyelid closure (mm)	5-7	2-3	0-1	0
Ocular surface integrity† and tear film stability	Present in 5 patients Not present in 1 patient	Disappearance of persistent epithelial defect	Disappearance of persistent epithelial defect	Disappearance of persistent epithelial defect

* Palpebral fissure height = measurement between the upper and lower lid margins.
† Persistent epithelial defect and tear film deficiency.

Figure 4. Preoperative eyelid closure. (a) Eyelid open at rest; (b) light eyelid closure; and (c) tight eyelid closure.

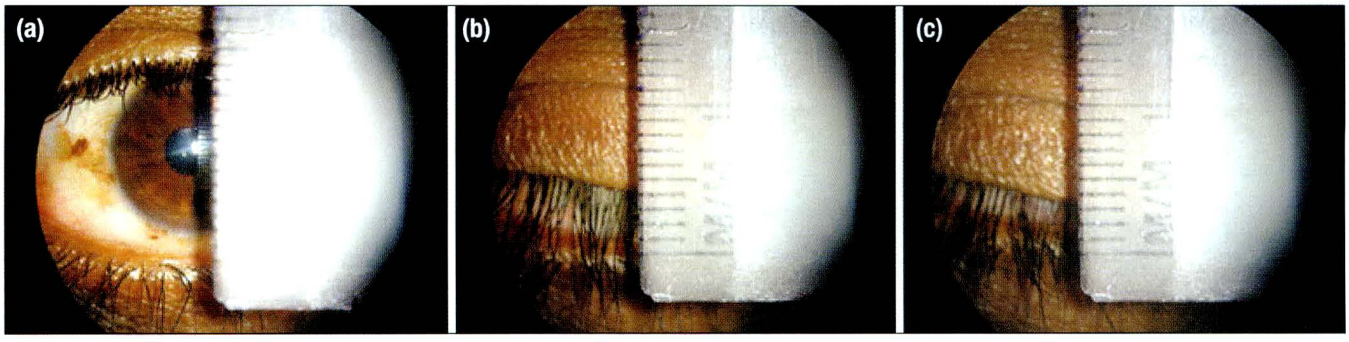
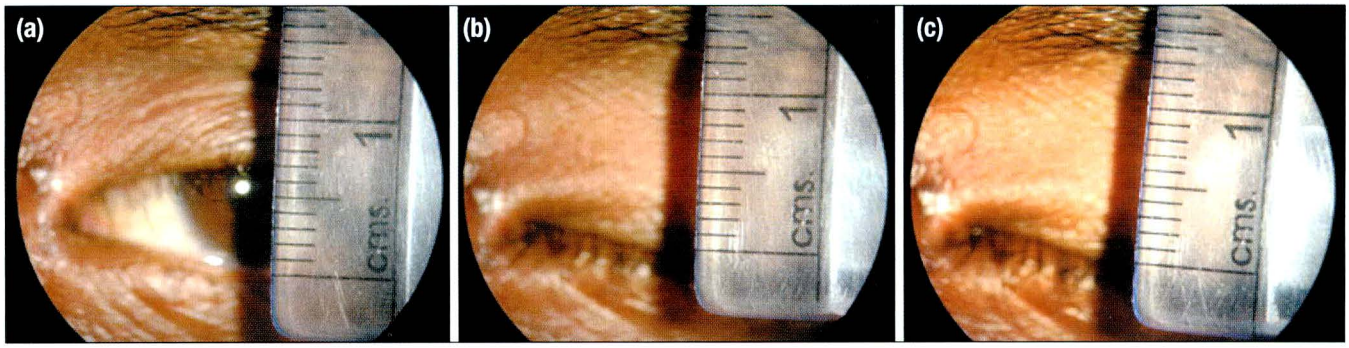


Figure 5. Postoperative eyelid closure. (a) Eyelid open at rest; (b) light eyelid closure; and (c) tight eyelid closure.



Temporalis muscle transfer using fascia lata from the lateral aspect of the thigh is one of most effective and promising modalities for the treatment of lagophthalmos. The procedure may be performed by ophthalmologists, but the long-term results still need to be evaluated.

Acknowledgements

The authors are grateful to Swami Japanandaji, Chairman, Shree Sharadadevi Eye Hospital and Research Centre, Pavagada, India, for enabling this study. The authors are also grateful to Dr P Krishna Murthy, Secretary, Damien Foundation, for supporting the leprosy reconstructive surgery at Shree Sharadadevi Eye Hospital and Research Centre, Pavagada, India.

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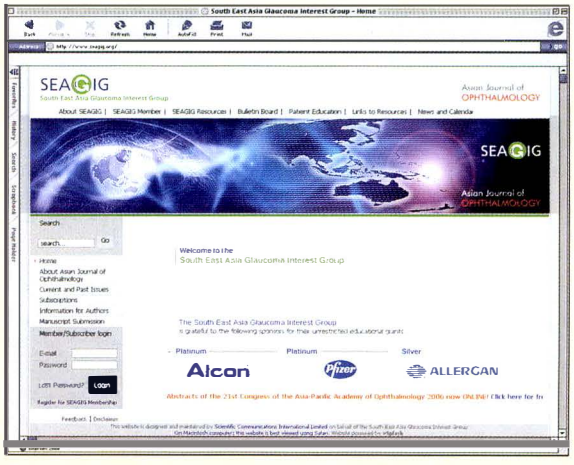

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Cluster Endophthalmitis Caused by *Pseudomonas aeruginosa*

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Seven patients developed endophthalmitis 1 day after phacoemulsification and intraocular lens implantation. All patients underwent vitrectomy, and were given intraocular antibiotics and dexamethasone injection. Postoperatively, antibiotics and corticosteroids were administered topically and systemically. Initial visual acuity was hand motion or less for all patients, but this increased to ≥20/50 for 5 patients after 6 weeks. Pseudomonas aeruginosa *isolated from the vitreous and the internal tubing of the phacomachine had similar antibiotic susceptibility. This report describes the outcome for the 7 patients.*

Key words: *Endophthalmitis, Phacoemulsification,* Pseudomonas aeruginosa, *Vitrectomy*

Asian J Ophthalmol. 2007;9:255-7

Introduction

The incidence of postoperative endophthalmitis has decreased to 0.06% owing to improvements in sterilisation procedures and understanding of the mechanisms of post-surgical infections.[1] The sources of infection in endophthalmitis following cataract surgery have been traced to patients' conjunctival flora, contaminated irrigating fluids, intraocular lenses, and phacoemulsifiers.[1] This report is of the clinical outcome of 7 patients with *Pseudomonas aeruginosa* endophthalmitis caused by contamination of the internal tubing of the phacomachine.

Case Report

Three patients with nuclear sclerosis 3+ and 4 patients with nuclear sclerosis 2+ with cortical opacity 3+ underwent uneventful phacoemulsification with posterior chamber intraocular lens implantation via a self-sealing scleral incision using the divide and conquer technique at the LV Prasad Eye Institute, Hyderabad, India, in 2003. On the first postoperative day, all 7 patients were diagnosed to have endophthalmitis (Table 1).

All the patients were operated on by a single surgeon, using the same phacomachine with a peristaltic pump. No prophylactic antibiotics were given to any of the patients. Severe pain with decrease in vision occurred in all patients within 24 hours of surgery.

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At examination, visual acuity was hand motion or less for all patients. All patients had oedematous lids, congested conjunctiva, oedematous cornea with hypopyon, and exudative membrane on the intraocular lens. Mean applanation intraocular pressure was 35.5 mm Hg (range, 28-44 mm Hg). The retina could not be visualised due to the exudate in the vitreous. Ultrasound B-scan showed multiple dots and membranous echoes in the vitreous cavity with an attached retina.

All the patients underwent vitrectomy with intravitreal injections of vancomycin 1 mg/0.1 mL, amikacin 400 µg/0.1 mL, and dexamethasone 400 µg/0.1 mL. Vitreous microscopy showed gram-negative bacilli. All patients were treated with topical ciprofloxacin 0.3% and prednisolone acetate 1% hourly and cyclopentolate 1% 3 times a day. Oral ciprofloxacin 750 mg was administered twice a day for 7 days. Prednisone was administered at 1 mg/kg body weight, tapering by 10 mg/kg every 5 days. Vitreous culture on blood agar, chocolate agar, and brain-heart infusion broth showed significant growth of *P aeruginosa* identified by API 20 NE (bioMèrieux, Marcy-l'Etoile, France). Isolated *P aeruginosa* was resistant to vancomycin and cefazolin and sensitive to amikacin, ceftazidime, chloramphenicol, ciprofloxacin, and gentamicin.

The infection control committee at the LV Prasad Eye Institute was informed about the cluster of postoperative endophthalmitis. The committee inspected the operating room, sterilisation room, and the preoperative and postoperative areas. Samples collected included swabs from the phacoprobe, the internal tubing of the phacomachine, ringer lactate that was used as the irrigating solution, and the viscoelastic. *P aeruginosa* was isolated from the

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Table 1. Clinical characteristics of patients with *Pseudomonas aeruginosa* endophthalmitis and treatment outcomes.

Patient number	Age (years)/ sex	Eye	Intraocular pressure (mm Hg)	Initial visual acuity	Initial treatment	Visual acuity at 6 weeks	Comments
1	55/M	Right eye	35	Hand movements close to face	Pars plana vitrectomy/ intraocular antibiotics/ dexamethasone	20/20	
2	75/M	Right eye	28	Light perception	Pars plana vitrectomy/ intraocular antibiotics/ dexamethasone	No light perception	Corneal necrosis with phthisis
3	70/M	Right eye	40	Hand movements close to face	Pars plana vitrectomy/ intraocular antibiotics/ dexamethasone	20/20	
4	62/F	Left eye	44	Light perception	Pars plana vitrectomy/ intraocular antibiotics/ dexamethasone	No light perception	Corneal necrosis with phthisis
5	62/M	Right eye	36	Hand movements close to face	Pars plana vitrectomy/ intraocular antibiotics/ dexamethasone	20/30	
6	60/M	Left eye	30	Hand movements close to face	Pars plana vitrectomy/ intraocular antibiotics/ dexamethasone	20/30	
7	69/M	Left eye	28	Hand movements close to face	Pars plana vitrectomy/ intraocular antibiotics/ dexamethasone	20/50	

internal tubing of the phacomachine. Organisms isolated from the vitreous and the internal tubing had similar antibiotic susceptibility patterns and genotyping.

Postoperative progressive decrease in inflammation with improvement in visual acuity ≥20/50 occurred in 5 patients after 6 weeks. Two patients who lost light perception developed corneal necrosis and became phthisical.

Discussion

Postoperative endophthalmitis outbreaks have been reported following contaminated internal tubing of the phacomachine.[2-4] *P aeruginosa* has been associated with all cases of contamination of internal tubing.[2-4] The prevalence of endophthalmitis at the LV Prasad Eye Institute calculated from 6919 phacoemulsification surgeries performed during the same year was 0.001%.

The increased intraocular pressure in all 7 patients could have been due to rapid onset of severe inflammation. Endophthalmitis associated with *P aeruginosa* is associated with poor visual outcome.[5] However, good visual recovery has been noted in one of the previous outbreaks.[2-4] In the series reported here, 5 patients regained good vision. This could be attributed to prompt intervention and administration of active intravitreal drugs.[6] The poor visual outcome of 2 patients could be attributed to the corneal involvement.[7]

Since the advent of modern sterilisation techniques, the bacterial flora of patients' eyes are considered to be the source of intraoperative infections, rather than contaminated surgical equipment. However, internal tubing of automated surgical equipment may act as a reservoir for the source of infection and may serve as a source of infection if the fluid in the internal tubing is contaminated. Reflux from the internal tubing during surgery has been considered as a potential source of endophthalmitis.[8] Hoffmann et al demonstrated the possibility of retrograde flow from the internal tubing of the phacoemulsifier using methylene blue.[4] De Kaspar et al modified the automated evacuation system of the phacoemulsification machine and created an external vacuum control manifold (VCM) that can be disinfected.[9] These authors showed that, in comparison to machines that have an internal VCM, the aspiration fluid specimens were sterile, and they recommended changing to more advanced systems equipped with an external VCM, which can either be sterilised or disinfected.[9]

It is imperative to find the reservoir source of the infecting agent in cluster endophthalmitis to prevent further infection. Ophthalmic surgeons should be aware of the possibility of surgical equipment as a potential risk for infection. Although *P aeruginosa* endophthalmitis is associated with poor visual outcome, early diagnosis and prompt treatment could result in good visual outcome in some, but not all, patients.

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Isolated Orbital Cysticercosis Causing Painless Proptosis

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Ocular cysticercus can involve the anterior segment, posterior segment, or adnexa. The most common sites of infestation in the eye are subretinal and intravitreal. Orbital involvement, in which the parasite localises within the extraocular muscles or lies subconjunctivally, is rare. Orbital cysticercosis commonly presents with signs of inflammation, restricted extraocular motility and proptosis. This report is of a 14-year-old girl with orbital cysticercus lying in the retrobulbar space, presenting as a painless proptosis of sudden onset.

Key words: *Cysticercus, Exophthalmos, Orbit*

Asian J Ophthalmol. 2007;9:258-60

Introduction

Cysticercosis is a cause of serious ocular morbidity. The cysticercus parasite has a predilection for subcutaneous tissue, muscle, the central nervous system, and the eye.[1] Ocular involvement occurs in 13% to 46% of patients with cysticercosis, with the posterior segment being the most common location.[2] Although ocular cysticercosis is not uncommon, orbital involvement is rare.[3,4] Cysticercosis can masquerade as almost any disease of the orbit or adnexa, especially in children and young adults.[5-7] Orbital cysticercosis commonly presents with signs of acute inflammation and muscle dysfunction. This report is of a patient with cysticercosis in the retrobulbar space presenting as asymptomatic proptosis of sudden onset.

Case Report

A 14-year-old girl presented with sudden protrusion of the right eye associated with mild discomfort for the previous 2 to 3 days in 2002. She had no previous history of pain, redness, or swelling in the eye and no relevant medical history. At examination, her visual acuity was 6/6, N/6 in both eyes. There was axial proptosis of the right eye; the adnexa were quiet with no signs of infection or inflammation. At palpation, a firm mass was felt in the lateral orbit. There was no tenderness or lymphadenopathy. Ocular movements were painless and there was minimal restriction of abduction of the right eye.

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Orbital sonography showed an ill-defined heterogeneous mass measuring 8.8 mm, with moderate to high internal reflectivity, lying in the lateral aspect of the orbit close to the lateral and inferior rectus muscle. There were areas of hyperechoic and anechoic areas suggestive of a cystic lesion (Figure 1). Computed tomography scan showed a lobulated soft tissue density mass intraconally, close to the lateral rectus muscle, measuring 2 x 1.3 x 1.8 cm and involving the adjacent orbital fat. The mass showed inhomogeneous enhancement on contrast study and had interspersed low-density non-enhancing regions. There was no bony erosion (Figure 2).

The patient underwent lateral orbitotomy under general anaesthesia. Intraoperatively, part of the lateral wall was removed and, as the periorbita was opened, a translucent pearly white cyst popped out of the retro-orbital region. At gross examination, the

Figure 1. B-scan orbital sonography showing a mass of moderate internal reflectivity, with a high reflective spike within the lesion.

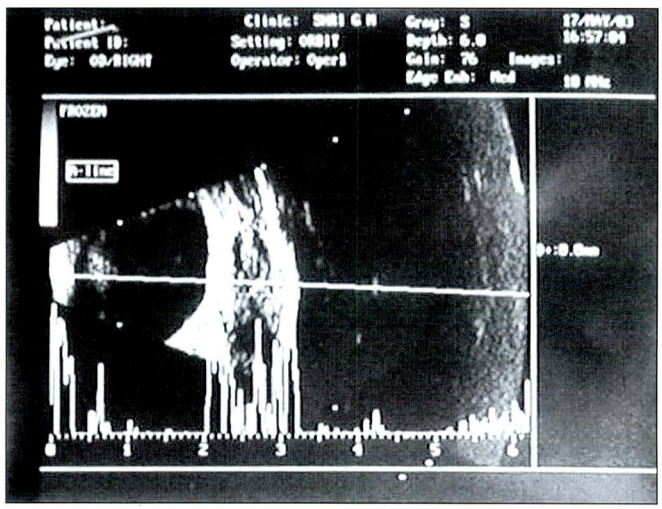

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Figure 2. Computed tomography scan showing a lobulated soft-density mass intraconally, close to the lateral rectus muscle (black arrow).

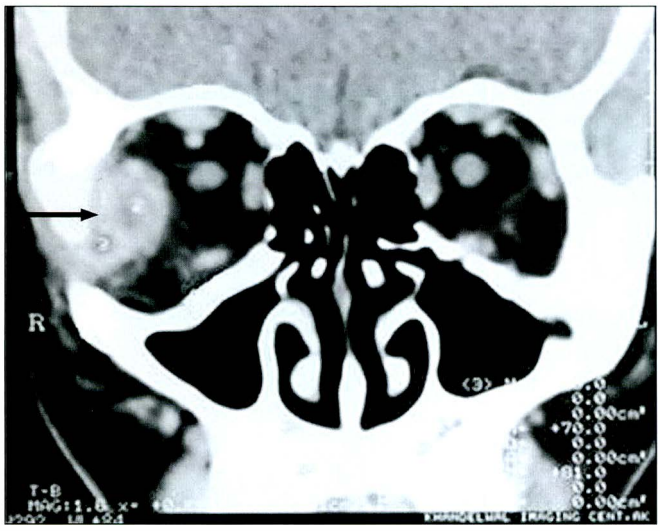
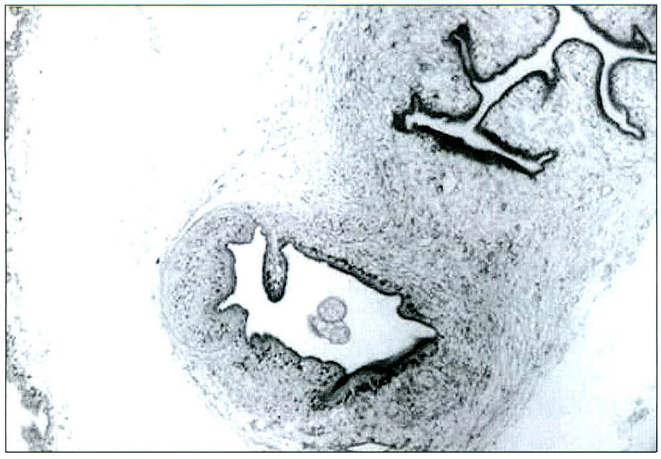


Figure 3. Microscopy of the cyst showing scolex with hooklets (haematoxylin and eosin stain; original magnification, x 40).



cyst measured 6 mm and there was a dense white structure within the cyst suggestive of cysticercus. Histopathological examination of the cyst confirmed the diagnosis of cysticercosis (Figure 3). The postoperative period was uneventful and the patient retained normal ocular functions.

Discussion

Cysticercosis is caused by the encystment of the cestode larva, *Cysticercus cellulosae*, of the tapeworm *Taenia solium*. The parasite causes 2 types of disease in humans. Eating undercooked pork containing cysticercus can lead to intestinal taeniasis. However, cysticercosis is caused by ingestion of the parasite's eggs via food, water, or other material contaminated with human faeces.[1,2,4] Ocular involvement occurs in 13% to 46% of patients with cysticercosis.[2] Of more then 500 reported cases of ocular cysticercosis, approximately 4% involve the eyelid or orbit, 8% involve the anterior segment of the eye, 20% are located subconjunctivally, and 68% involve the posterior segment of the eye.[5] Cysticercus within the orbit is rare.[3,4] The condition occurs more commonly under the conjunctiva, extraocular muscles, or in the globe; in rare cases where an orbital infection occurs, the site is usually near the orbital margin. The cyst does not reach a large size and, owing to the anterior position, proptosis is unusual, although displacement and diplopia is common. A literature review concluded that of the 520 cases of ocular cysticercus reported worldwide, only 3 involved the orbit.[6] In a recent report of 20 patients with orbital cysticercosis by Sekhar and Lemke, the cyst was intramuscular in 11 (55%) and subconjunctival in 9 (45%).[7] These authors reported a close association of the cyst with extraocular muscle and pointed out that subconjunctival presentation could be a secondary stage in those patients in whom the cyst may have extruded from the primary extraocular muscle site. These cysts were not within the orbit, but within the extraocular muscle or subconjunctival area as reported by earlier authors.[8-10]

This patient presented with signs of painless proptosis of sudden onset. Orbital cysticercosis usually presents with signs of recurrent inflammation, restricted ocular movements, diplopia, and globe displacement.[5,7,11] Fusiform enlargement of the affected muscle and a well-defined cystic lesion with scolex on imaging study are characteristics of cysticercosis.[7,12] Extraocular muscle cysticercosis has been treated successfully with oral albendazole and systemic steroids.[7,8,12] Some surgeons have performed surgical removal of the cysts for cysticercosis in extraocular muscle.[7,9,10] This patient had no signs of inflammation or diplopia; she also had no prior history of such episodes. Due to the silent presentation, a cysticercosis was not considered and a decision for surgical removal of the lesion was made.

Walrath et al published a report of a 55-year-old woman with isolated orbital cysticercus located in the orbital fat who was considering orbital fat excision for cosmesis.[6] Isolated orbital cysticercus has not often been reported in the literature, but it should be considered in the differential diagnosis of asymptomatic proptosis of sudden onset, especially in patients from an endemic region.

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Interocular Autologous Sclerocorneal Transplantation from a Phthisical to a Perforated Globe

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This report is of a 60-year-old woman with globe perforation at the site of staphyloma following minor blunt trauma to the left eye with her thumb. The globe perforation was repaired with autologous sclerocorneal and conjunctival tissue harvested from the fellow phthisical blind eye. Postoperatively, she was treated with topical and oral antibiotics and steroids. Her visual acuity returned to 6/18 — her best-corrected vision. Tectonic scleral graft was later performed as a planned procedure.

Key words: *Eye, Rupture, Transplantation, autologous*

Asian J Ophthalmol. 2007;9:261-3

Introduction

Use of autologous sclerocorneal and conjunctival graft from the fellow eye to repair globe rupture is uncommon. This report is of a patient with corneoscleral ectasia associated with globe rupture, repaired by sclerocorneal and conjunctival graft harvested from the blind phthisical eye as a primary procedure.

Case Report

A 60-year-old woman presented with a globe rupture of the left eye due to minor blunt trauma with her thumb to the closed eye in January 2006. Her visual acuity was hand movements, and she had a shallow anterior chamber and choroidal effusion due to hypotony.

The patient first presented in 1969 with uveitis in the left eye. The right eye was phthisical and blind due to previous uncontrolled uveitis. She was diagnosed to have idiopathic chronic uveitis, as all other investigations had negative results. The inflammation in the left eye was treated with long-term topical and systemic steroids. However, this treatment resulted in secondary glaucoma and cataract, for which the patient underwent Scheie's procedure in 1973 and cataract extraction in 1976, respectively.

Since then, she had had recurrent episodes of anterior and posterior uveitis in the left eye, which was treated with systemic and topical steroids. Her intraocular pressure was maintained within normal limits and she had a stable best-corrected visual acuity of 6/18. As the patient had prolonged periods of steroid-free stability, further immunosuppression was not commenced.

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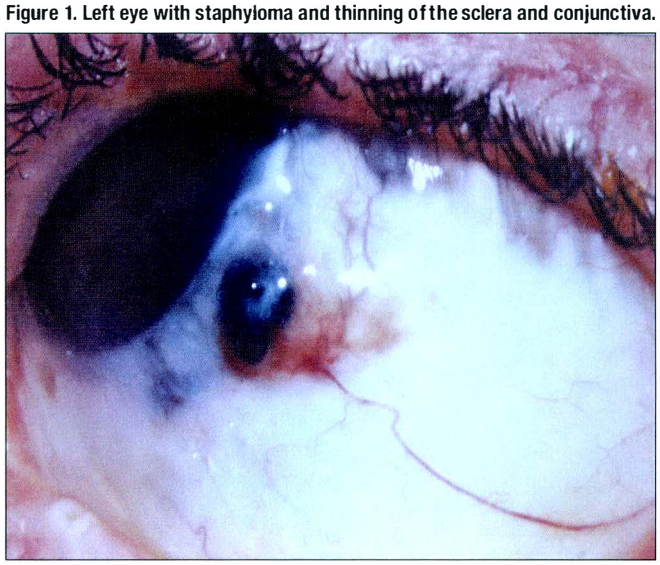
In 1994, she was noted to have conjunctival and scleral thinning with development of staphyloma away from the site of Scheie's procedure (Figure 1). The ocular condition remained stable with best-corrected visual acuity of 6/18.

The globe rupture of the left eye occurred at the site of the staphyloma. Due to the absence of viable conjunctival and healthy sclerocorneal tissue, direct closure was not possible and the globe was repaired with a partial thickness scleral flap harvested with adjacent limbal tissue, including cornea and a free graft of the overlying conjunctiva from the right phthisical eye, as an urgent procedure (Figure 2a). During the harvesting, balanced salt solution was injected into the pars plana to increase the intraocular pressure in the phthisical eye, to expand the tissue and facilitate lamellar dissection. Sclerocorneal and conjunctival grafts were taken as

Figure 1. Left eye with staphyloma and thinning of the sclera and conjunctiva.



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free grafts and sutured as separate layers in the affected eye (Figure 2b). Postoperatively, she was fitted with a bandage contact lens and commenced oral antibiotics plus topical steroids and antibiotics for 8 weeks. Tapering of the steroids was titrated to the inflammatory response at the graft site and control of intraocular inflammation. The conjunctival defect was closed in the phthisical eye, which remained comfortable after treatment with Maxitrol ointment twice a day for 1 month.

The visual acuity was 6/24 within 10 days of the surgery. Three months after the repair, thinning of the graft was noted (Figure 3a), with normal intraocular pressure and a visual acuity of 6/18. Due to thinning after the primary procedure, a tectonic scleral graft from donor sclera was performed as a planned second procedure after 6 months (Figure 3b) and she again commenced oral and topical antibiotics and steroids. The oral steroid dose was initially 1 mg/kg and was tapered according to the response.

At her latest follow-up visit, in July 2007, the graft was secure and she had a best-corrected visual acuity of 6/18. She administers topical steroids once a day and does not require oral steroids.

Discussion

Thinning of the sclera has several causes, including antiglaucoma filtering procedures and procedures using implants.[1] Other causes include pterygium excision,[2] retinal detachment surgery,[2] cataract extraction,[2] blunt or perforating trauma leading to staphyloma formation,[3] scleromalacia perforans,[1,2] spontaneous perforation of retinochoroidal coloboma,[4,5] and underlying connective tissue disorders such as Marfan's syndrome.[6] The patient in this report developed staphyloma due to the combination of underlying connective tissue disease and chronic steroid use.

Small scleral defects may be closed by simple surgical procedures such as direct suturing and conjunctival advancement.[3] Larger defects usually require patch grafts such as cadaveric dura mater,[1] dermal grafts,[2] fascia lata,[3] corneal grafts,[3] scleral grafts,[3] and amniotic membrane.[6]

Good clinical results have been obtained with the use of donor sclera, which can be either autologous or homologous. Autologous scleral grafts can be taken from the same eye or the fellow eye as a partial-thickness scleral flap. As autologous grafting from the same eye is more difficult to perform than donor grafting,[3] especially

Figure 2. (a) Right phthisical eye after graft harvest; and (b) left eye after receiving graft from the right phthisical eye.

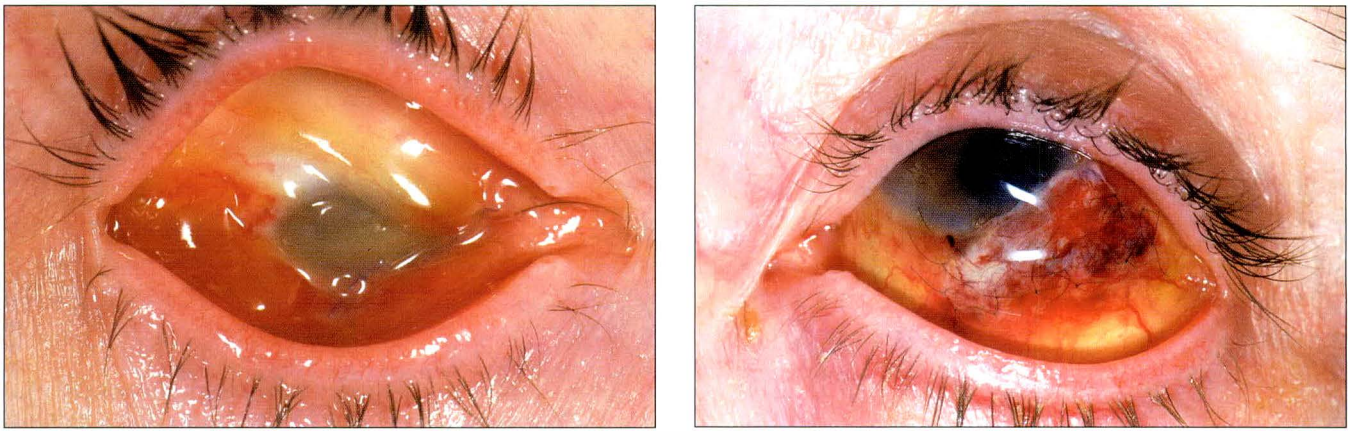
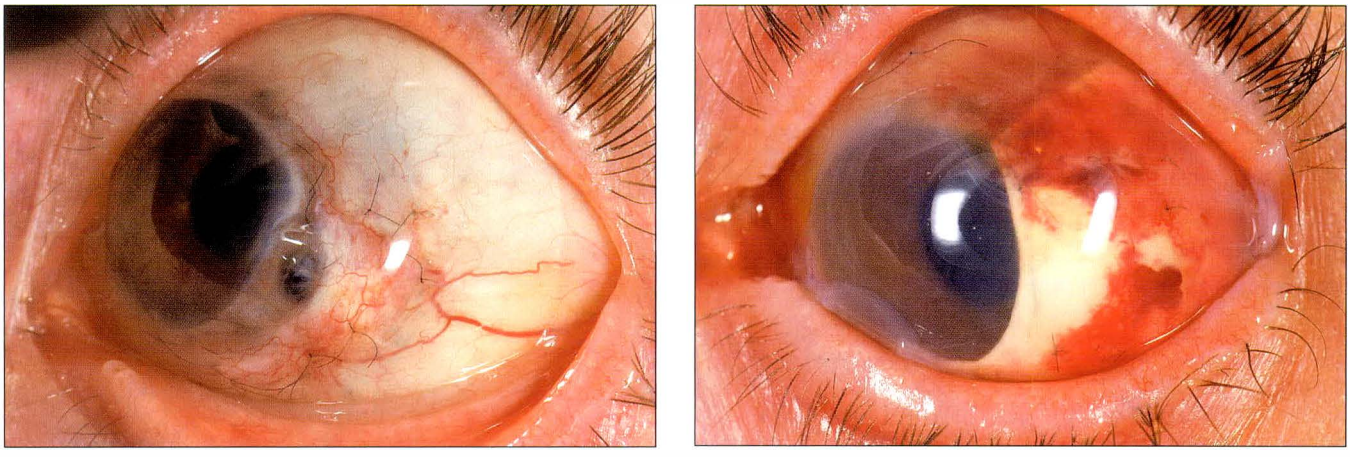


Figure 3. Left eye (a) after 3 months showing thinning of the graft; and (b) after secondary repair with tectonic graft with donor sclera.



when the sclera is friable because of previous surgery, injury, or any underlying connective tissue disorder, it may be possible to harvest tissue from the fellow eye, as was done for this patient. The potential difficulties of this approach include the technical challenge of lamellar dissection of a soft shrunken globe, the possibility of perforating the phthisical eye, and transfer of non-viable tissue to the perforated globe.

This approach was considered to incur less risk of immune rejection than allogenic grafting using fresh tissue and also avoided delays associated with receiving eye bank tissue. For patients with bilateral ocular disease affecting the corneosclera, with end-stage disease in one eye, preserving the globe should be considered prior to removal. While the cosmetic considerations might suggest evisceration and enucleation as possible options for an unsightly shrunken eye, the potential value of retaining outer tissue should be discussed with the individual. The results for this patient indicate that autologous sclerocorneal and conjunctival patch graft harvested from the phthisical blind eye is effective for the treatment of globe rupture when patch grafts are needed unexpectedly.

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Mirror Image Buphthalmos in Identical Twins

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This report is of identical female twins with bilateral buphthalmos, who were living apart. A 15-year-old girl presented with corneal opacity in both eyes, nystagmus, and diminution of vision. A detailed history revealed that she had a twin sister, living with her grandparents in another city, who also had the same problem, but in mirror image form. This report describes the features of the condition in twins.

Key words: *Corneal opacity, Hydrophthalmos, Twins, monozygotic*

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Introduction

The congenital glaucomas are a diverse group of usually inherited disorders, in which an ocular anomaly present at birth is responsible for increased intraocular pressure (IOP) and secondary visual loss. To date, 3 genes have been identified to be associated with congenital glaucoma, with locations at 2p21, 1p36, and 6p25.[1-3] Expression of these genes is of great interest in monozygotic twins as their presentation may be in mirror image or non-mirror image form. In the mirror image form, the features are present on the right side in one twin and on the left side in the other. The features may be generalised, as in the rare condition situs inversus,[4-6] or limited to only a few organs.[7-9]

Laterality is accomplished through a complex process of gene expression, which is turned on and off as the fertilised egg begins to divide and grow. The cells in the first few divisions are totipotent, in that they can develop independently into a foetus if separated. This splitting and separation of a single fertilised egg, occurring between 1 to 14 days post-conception, results in monozygotic twins (Figure 1). Identical twins develop if the separation is within the first week (Figure 1a). If the splitting occurs between 1 and 2 weeks, 'mirror image' twins are formed (Figure 1b). Siamese or conjoined twins are born if the separation takes place after 12 to 14 days (Figure 1c).

In hospitals where the facilities for genetic studies are not available, the twin study method can be used. In identical twins, the genetic structure is the same. If twins are living in different environments, factors responsible for a trait or disease may be determined. If both twins have the same pattern for disease, it is likely to be genetic. However, if the pattern for disease is not the

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Figure 1. Splitting of a fertilised ovum at different stages leading to development of (a) identical twins; (b) mirror image twins; and (c) conjoined twins.

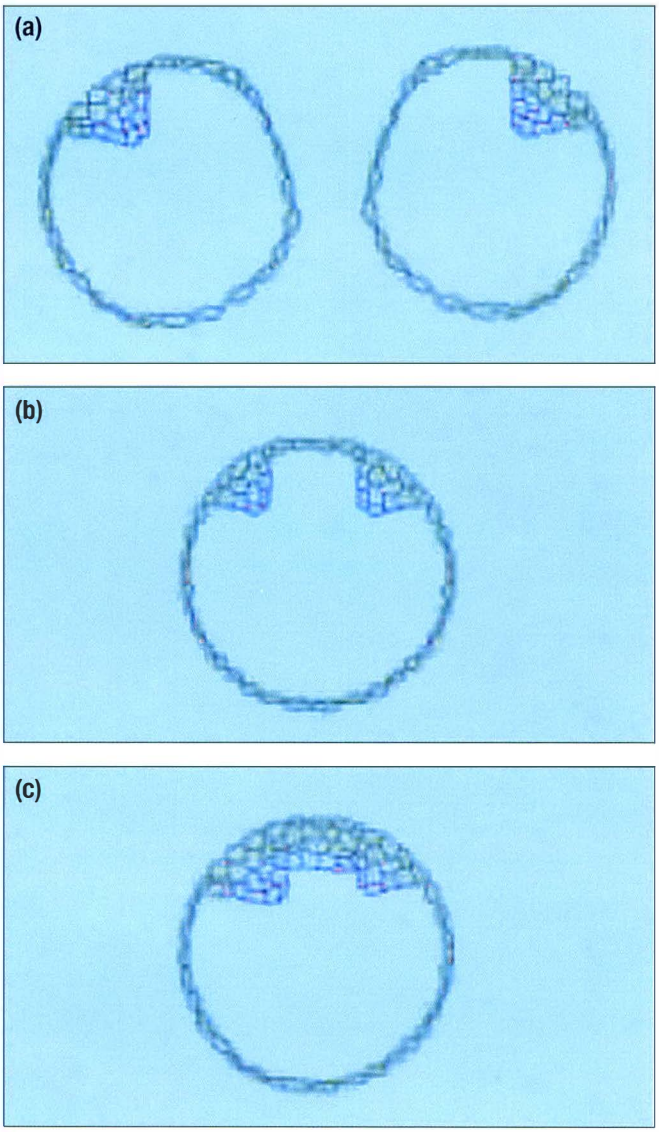

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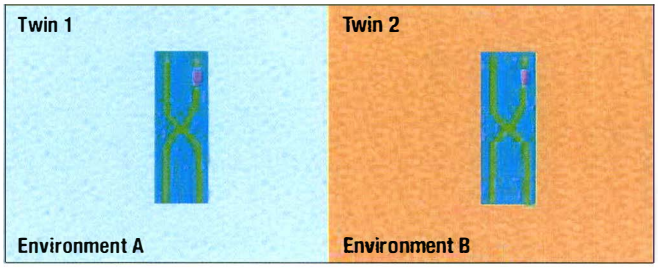
Figure 2. Environment or genes as a cause of a trait or disease in twins. For a genetic effect, the trait or disease is present in both twins, and for an environmental effect, the trait or disease is present only in one twin. Pink indicates that the genes for a trait or disease are the same.



same or is absent in one twin, the disease is likely to be due to environmental factors (Figure 2).

This report is of bilateral buphthalmos in identical female twins who were living apart, with mirror image presentation.

Case Report

Patient 1

A 15-year-old girl presented with corneal opacity in both eyes, nystagmus, and diminution of vision in 1995. A detailed history revealed that the patient had a twin sister, living with her grandparents in another city, who also had the same problem, but in mirror image form. The twin had not received treatment for economic reasons.

At examination, the patient had anterior staphyloma in the right eye, corneal opacity in the left eye, cataract in the left eye, and nystagmus in both eyes. Slit-lamp biomicroscopy of the right eye showed that the iris was in contact with the corneal endothelium in several places. Irregular corneal opacity was extending horizontally with vertical branching. Details of the anterior chamber could not be visualised, although the depth was normal when visualised through the cornea at the limbal area (van Herick grade 3 to 4) [Figure 3]. In left eye, there was a central corneal opacity involving approximately two-thirds of the cornea. Details of the anterior chamber and posterior segment could not be seen, although the depth appeared normal when visualised through the peripheral part of the cornea (van Herick grade 3).

The vision in the right eye was light perception and projection of rays, while in the left eye, only light perception was present and projection of rays was defective. The IOP was 40 mm Hg in the right eye and 36 mm Hg in the left eye by Goldman applanation tonometry. The corneal diameters were 14 mm in the right eye and 13 mm in the left eye. Axial length measured by B-scan ultrasonography was 24.24 mm in the right eye and 24.36 mm in the left eye. Gonioscopic examination was not possible due to the corneal opacity.

After controlling the IOP, initially with drugs and then by filtering surgery, penetrating keratoplasty was done in the right eye. The

Figure 3. Bilateral buphthalmos presenting with central corneal opacity in the left eye and bulging hazy cornea in the right eye.

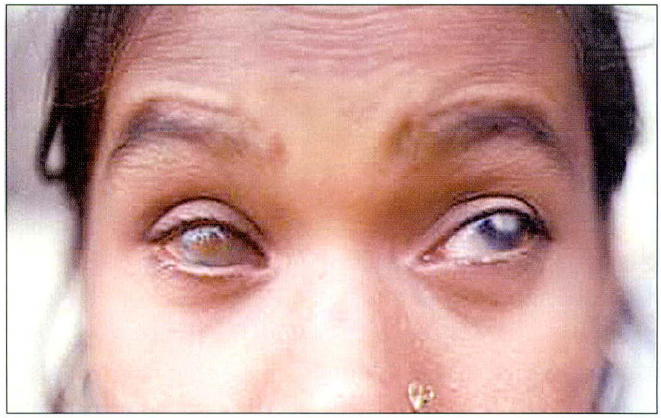
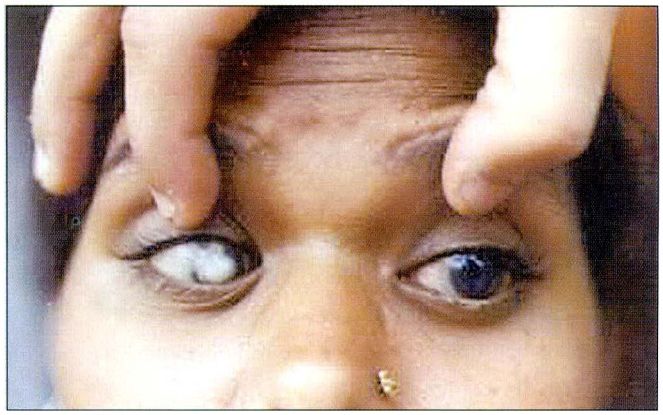


Figure 4. Bilateral buphthalmos presenting with central corneal opacity in the right eye and bulging hazy cornea in the left eye.



visual acuity improved to 2/60 and the patient's quality of life improved. She then defaulted follow-up.

Patient 2

The girl's twin subsequently presented with nystagmus with central corneal opacity in the right eye and immature cataract with corneal opacity in the left eye in 1995. Slit-lamp biomicroscopy revealed that the right eye had central corneal opacity covering three-quarters of the cornea (Figure 4). Details of the anterior chamber could not be visualised but the depth was normal (van Herick grade 3). In the left eye, the cornea was hazy. An irregular corneal opacity extending from 9 to 12 o'clock with branching to the centre of the cornea was noted. The anterior chamber was shallow and the lens was cataractous (van Herick grade 2 to 3).

The vision in right eye was light perception with defective projection of rays, while in the left eye both light perception and projection of rays were present. Goldman applanation tonometry showed an IOP of 42 mm Hg in the right eye and 34 mm Hg in the left eye. Axial length by B-scan ultrasonography was 24.40 mm in the right eye and 24.23 mm in the left eye. The diameter of the cornea was

12.5 mm in the right eye and 14.0 mm in the left eye. Gonioscopic examination was also not possible due to the corneal opacity.

The IOP was controlled medically, followed by filtering surgery in the left eye. The visual acuity improved to 3/60. The patient then defaulted follow-up.

Discussion

The twins were strikingly similar in general appearance and could be identified only by their corneas. The colour and texture of their hair was similar. They showed no marked difference in eyebrows, eyelashes, lips, ears, teeth, or height. The fingers and nails of their hands and feet were identical and mirror imaged. Their developmental milestones occurred at the same time. Both had menarche at the age of 13 years and their menstrual cycle was similar.

All these features fulfil Newman's postulates for monozygotic twins,[10] except that both the patients were right handed. The modern standard for evaluation of monozygotic twins is DNA markers. These authors had to depend on the clinical presentations of the patients to conclude that they are monozygotic twins, as there was no available facility for genetic evaluation by DNA markers.

The pathogenesis of buphthalmos may have started in utero because of expression of genes responsible for congenital glaucoma in both twins. In the case of these twins, the clinical presentation of IOP, corneal opacity, corneal diameter, axial length, and preoperative and postoperative visual acuity was present in mirror image form.

There have been only a few similar reports by ophthalmologists. Two patients with unilateral glaucoma in mirror image eyes were reported by Potts[11] and Rasmussen and Ellis.[12] Kiehle and Pugmire reported unilateral glaucoma in mirror image form.[13] Massimeo reported on a male twin who had ipsilateral involvement in the right eye.[14] Purtscher[15] and Scheie[16] reported on male twins with bilateral involvement, and Grebe[17] and Duncan and Maynard[18] reported on female twins with bilateral involvement.

Mirror image presentation of conditions other than glaucoma have also been reported, but these are rare. Dirani et al reported mirror image congenital esotropia in monozygotic twins.[7]

The patients in this report were living in different environments, so the authors conclude that environmental factors had very little or no effect on the pathogenesis of the disease. It is therefore likely to be the genetic make up of the patients that led to the development of congenital glaucoma.

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Isolated Traumatic Laceration of the Superior Oblique Muscle

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Traumatic laceration of extraocular muscles often occurs in the context of severe ocular and orbital injuries. Isolated lacerations are rare. This report describes the clinical and surgical findings and mechanisms of trauma of 2 patients with superior oblique muscle laceration caused by a carpet-weaving hook. There were no other significant ocular or orbital injuries. Surgical exploration revealed laceration of the superior oblique muscle proximal to the trochlea associated with avulsion of the trochlea.

Key words: *Eye, Lacerations, Oculomotor muscles, Wounds and injuries*

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Introduction

Traumatic laceration of extraocular muscles is uncommon, but usually occurs to the rectus muscles in association with globe rupture or other orbital injuries. Traumatic superior oblique muscle palsies are mostly due to severe orbital or head trauma (neurogenic superior oblique muscle palsy).[1-18] This report describes 2 patients with isolated traumatic superior oblique muscle laceration with trochlear avulsion without any other significant ocular or orbital injuries.

Case Report

Patient 1

An 18-year-old girl presented in 2000 with trauma to the right eye caused by a carpet weaving hook (Figures 1 and 2), which occurred 4 days previously. She had no relevant medical history and her responses to subjective tests were reliable.

At examination, the patient had abnormal head positioning with a head tilt to the left and her face was turned to the left. There was slight ptosis of the right upper lid. There was no proptosis. The visual acuity of both eyes was 20/20. The intraocular pressure (IOP) in both eyes was 10 mm Hg. The reaction of pupils to light and near reflexes were normal. No afferent pupillary defect was noted from the swinging flashlight test.

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Examination of the right eye revealed minimal oedema of the upper eyelid and 2 mm of ptosis. There was a small subconjunctival haemorrhage under the inferior bulbar conjunctiva. The upper nasal conjunctiva was hyperaemic. A muscular mass containing some

Figure 1. Carpet weaving hook.

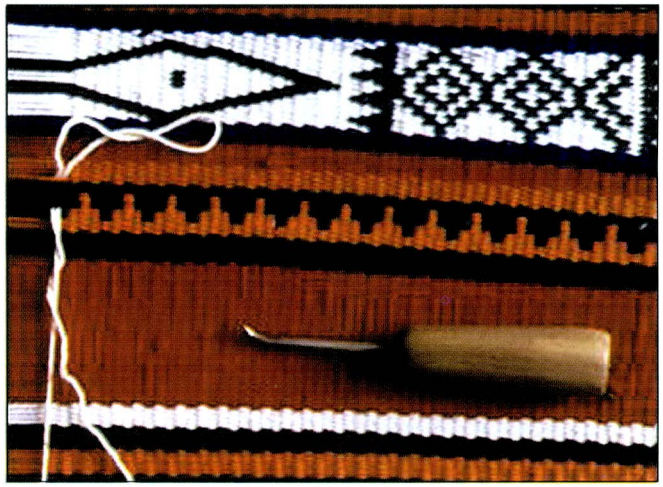
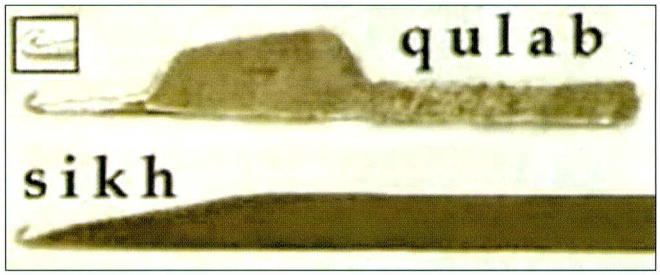


Figure 2. Two types of weaving hook.



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tendinous fibres extruded from a hole in the upper nasal forniceal conjunctiva and was hanging over the globe (Figure 3).

There were no other anterior segment abnormalities. Indirect funduscopy was unremarkable. The left eye was completely normal. There was no limitation of the ocular ductions of either eye. Cover-uncover test revealed right hypertropia, which measured 8 PD on alternate prism cover test in the primary position (Table 1 and Figure 4).

There was 5° of excyclodeviation of the right eye on double Maddox rod test. The hypertropia was 12 PD in right head tilt and zero in left head tilt. The preliminary diagnosis was traumatic laceration of the superior oblique muscle and the patient was admitted for exploration and primary repair, if needed. Exploration revealed that the superior oblique muscle was lacerated from 20 mm proximal to the trochlea (Figure 5). In addition, the trochlea was found to be avulsed. Exploration to find the proximal muscle stump was unsuccessful. As the laceration was 20 mm proximal to the trochlea, repair of the trochlea was not considered necessary. The distal stump in the extraconal space was released in its normal anatomic position. Other ocular and orbital structures were intact.

The abnormal head position remained for the first 3 days after the operation. Ptosis of the right lid had decreased by 1 mm. The patient defaulted follow-up thereafter.

Figure 3. A muscular mass containing some tendinous fibres was extruding from a hole in the upper nasal forniceal conjunctiva.

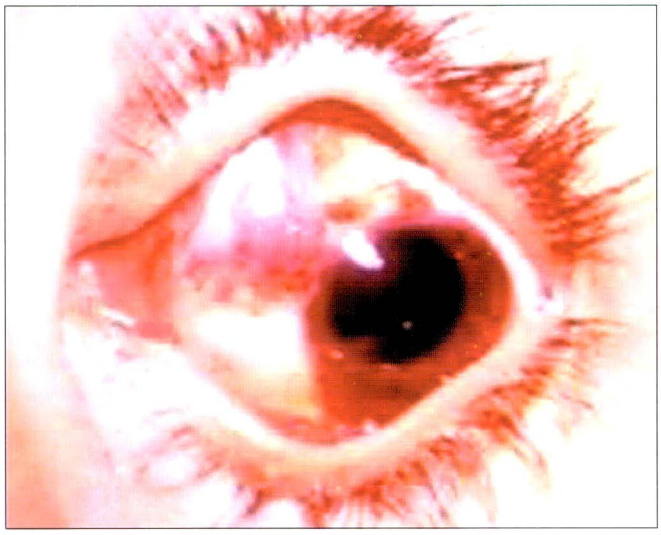
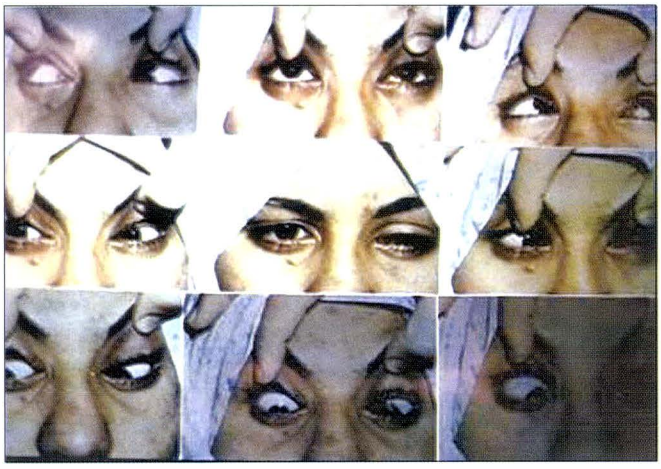


Table 1. Patient 1 — deviometry (sequential prism measurement).

Left gaze	Primary position	Right gaze
Right hypertropia = 10	Right hypertropia = 8	Orthotropia
Right hypertropia = 12	Right hypertropia = 8	Orthotropia
Right hypertropia = 16	Right hypertropia = 8	Orthotropia

Figure 4. Left hypertropia increasing in right gaze, with limitation of depression of the left eye at adduction.



Patient 2

A 15-year-old girl presented in 2005 with trauma to the left eye caused by a carpet-weaving hook while weaving a carpet. She sought medical consultation within 24 hours of injury. The patient had no relevant medical history.

She had acute diplopia in the primary position and down gaze. At examination, there was mild lid oedema, erythema, and a slight hymosis. No proptosis was detected. The visual acuity for both eyes was 20/20. The IOP in both eyes was within normal limits. The reaction of the pupils to light and near reflexes were normal.

At examination of ocular motility, there was restriction of passive duction of the left globe upward and inward; the globe moved freely in all other fields. There was mild superior oblique muscle under-action. Otherwise, ductions and versions were normal. Cover-uncover test revealed left hypertropia measuring 15 PD in the primary position by alternate prism cover test (Table 2 and Figure 6).

The left hypertropia increased to 25 PD when tilting the patient's head to the left, and decreased to >5 PD when tilting the head to the right. Slit-lamp examination of the left eye revealed mild oedema

Figure 5. Exploration revealed that the superior oblique muscle was lacerated 20 mm proximal to the trochlea.

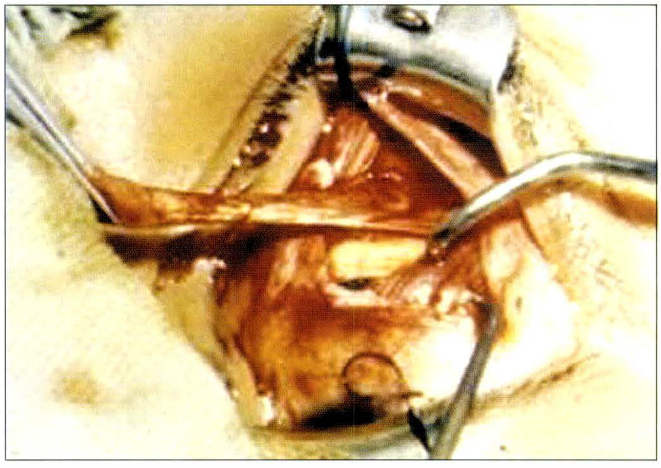
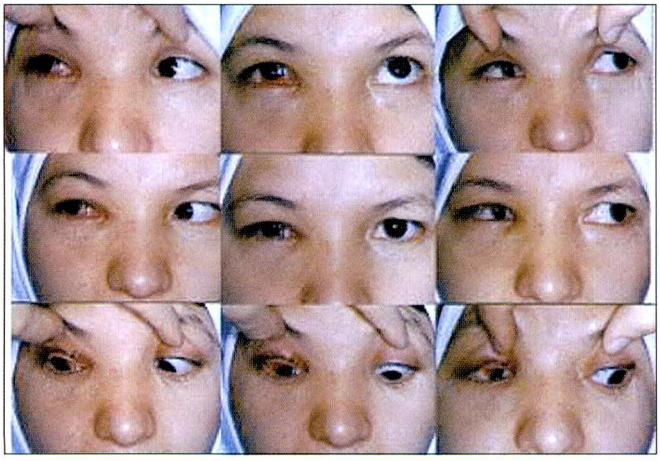


Table 2. Patient 2 — deviometry (sequential prism measurement).

Left gaze	Primary position	Right gaze
Orthotropia	Left hypertropia = 15	Left hypertropia = 20
Left hypertropia = 5	Left hypertropia = 15	Left hypertropia = 20
Orthotropia	Left hypertropia = 15	Left hypertropia = 25

Figure 6. Right hypertropia increasing at left gaze with limitation of right eye depression at adduction.



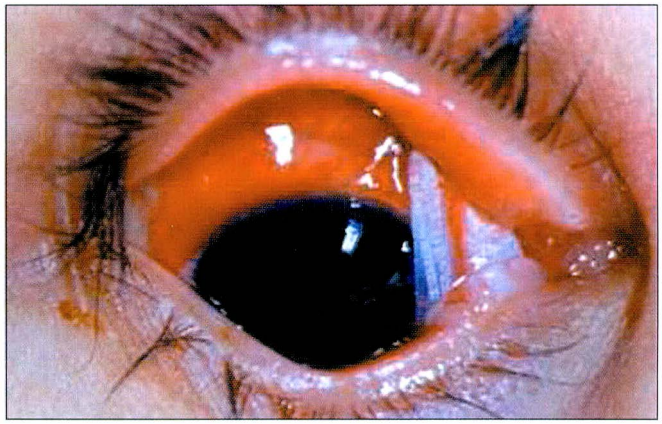
and erythema of the upper lid, especially on the nasal side. There was a small subconjunctival haemorrhage at the superonasal part of the bulbar conjunctiva. A small conjunctival laceration was seen at the site of the haemorrhage, but the globe was intact. There was redundant tendinous tissue within the laceration site (Figure 7). Indirect ophthalmoscopy was unremarkable, except for slight commotio retinae at the superonasal quadrant of the retina. There was 10° of left excyclotropia on double Maddox rod test.

The patient was admitted for exploration under local anaesthesia. Exploration revealed a superior oblique tendon laceration proximal to the muscle insertion. The trochlea was intact. The proximal end of the superior oblique muscle was not found. The cut end, which was left in the correct anatomical position, and the conjunctival laceration were repaired. The measured deviations did not change 1 week postoperatively. No steroid injection was attempted. The patient defaulted follow-up thereafter.

Discussion

Traumatic laceration of extraocular muscles usually occurs after severe orbital or ocular injury, and mostly affects the rectus muscles. It is unusual to encounter penetrating injuries that affect the superior oblique tendon in an isolated manner, without also damaging the bony orbit, the trochlear area, or both.[4] If modern shatterproof glass was not used in automobile windshields, this type of injury would occur as a result of motor vehicle accidents.[7,8] Other objects such as a ski pole,[13] tent pole, or metal wire[10] can also cause direct injury to the tendon.

Figure 7. Redundant tendinous tissue within the laceration.



In patient 1, it is likely that the superior oblique muscle was severed proximal to the trochlea when the patient withdrew the metal hook from the orbit. Dow reported a similar injury caused by a sharp piece of metal fence wire that was forcefully pulled out of the orbit.[10] Dow anchored the avulsed segment of tendon to the medial orbital wall, adjacent to the trochlea and obtained good results with full ocular motility.[10] Bachynski and Flynn reported two similar injuries caused by a spring-loaded hook used to balance window frames and the hook end of a brake tool.[4] The superior oblique tendon was sacrificed in these patients. Over time, the restriction in elevation and inflammatory response induced by the exposed tendon was relieved, but there was marked superior oblique muscle palsy. Subsequent contralateral inferior rectus recession combined with ipsilateral inferior oblique recession restored excellent ocular motility and a satisfactory field of binocular single vision.[4]

Extraocular muscle dysfunction is a rare surgical complication of sinus surgery. Rosenbaum and Astle described 5 patients with acquired strabismus and symptomatic vertical diplopia secondary to sinus surgery.[14] In all patients, the resultant diplopia was disabling. Four patients had frontal sinus window surgery performed, with incisions placed in the superonasal quadrant of the orbit, below the eyebrow (a modified Lynch incision). Three patients developed superior oblique paresis and the fourth developed Brown's syndrome. The location of the skin incision was critical to injury in the trochlear area. The fifth patient underwent a nasal polypectomy and antrostomy with secondary orbital haemorrhage and proptosis, which resulted in mild inferior rectus paresis. Lauer et al described a patient with acquired Brown's syndrome due to entrapment of the superior oblique muscle tendon in an orbital roof fracture.[15] Thacker et al described a patient with superior oblique palsy following endoscopic sinus surgery.[16] Legge et al described 3 patients with hypertropia following penetrating trauma to the trochlea.[17] Each patient underwent

computed tomography and/or magnetic resonance imaging to assist in determining the mechanism of superior oblique muscle dysfunction. Wise et al described 5 patients with dog-bite syndrome — palsy of the superior oblique muscle and a paradoxic inability to elevate the eye in adduction.[18] It has been suggested that no treatment should be given in the acute phase unless there are symptoms in the primary position, in which case, peritrochlear injection of a depot steroid administered within 2 weeks of the injury may be of benefit. If the ocular motility is still significantly restricted after several months, an attempt can be made to remove local scar tissue, with insertion of anchor sutures; recession of the inferior rectus of the fellow eye with adjustable sutures should correct residual symptomatic hypertropia.[18]

The interesting finding in the patients in this report was the absence of accompanying ocular or orbital injuries, except for a small hole in the conjunctiva from which the muscle was protruding and avulsion of the trochlea in patient 1 and a small conjunctival laceration and subconjunctival haemorrhage in patient 2.

Carpet weavers use a weaving hook to tighten the carpet knots in a pendular movement in the vertical plane. The injury to the superior oblique muscle in these patients occurred after the hook had passed under the upper lid and had perforated the conjunctiva, engaging the muscle's tendon and exerting a tractional force on the return. The withdrawal of the hook from the orbit probably produced more significant damage than its entry into the orbit. As the hook was withdrawn, the distal reflected portion of the superior oblique tendon was pulled forward into the conjunctival laceration. The later adherence of this part of the tendon to the lacerated conjunctiva produced more troublesome symptoms for the patients than the decreased field of single binocular vision.

These patients were interesting because of the mechanism and type of injury. These authors suggest that for patients with superior oblique muscle laceration in whom primary repair is difficult or impossible, releasing the muscle in its anatomic position is appropriate. Despite the fact that the surgery did not improve the field of single vision, the patients found that it made them more comfortable because it eliminated annoying eye movements. Patient 2 had an unusual form of 'canine tooth' or Knapp-type VII superior oblique palsy, in that restriction in elevation and adduction with paresis in the area of the superior oblique muscle was noted. This patient illustrates that, although such restrictions occur in the area of the trochlea, restriction may arise anywhere along the length of the superior oblique muscle.

Both of these patients demonstrate that penetrating injuries of the superonasal lid and orbit should trigger a high index of suspicion that the superior oblique muscle might be affected. The different angle of deviation in these patients may be related to the severity of the causative trauma. Extension of injury to the intermuscular septum and muscle pulleys may result in hypertropia. Surgical exploration of the area may be the only way to reliably detect foreign bodies, entrapment of the muscle in the bony orbit or lid, or partial transaction of the muscle. It may be helpful to treat the restrictive and paretic components separately. Early intervention to free entrapments or adhesions seems advisable, with later intervention to correct a stable superior oblique paresis if it is not possible to strengthen the damaged superior oblique at the time of the initial procedure. Bachynski and Flynn found recession of the contralateral inferior rectus muscle with an adjustable suture to be valuable for such patients. This procedure may be combined with other muscle repair procedures.[4]

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Bilateral Acute Myopia and Angle Closure Glaucoma following Topiramate Therapy

Dear Editor,

A 35-year-old woman presented with sudden onset of blurring of vision and mild ocular discomfort. At examination, she was found to have shallow anterior chambers with raised intraocular pressure (IOP) of 40 mm Hg in both eyes. Her uncorrected visual acuity (UCVA) was 6/36 in both eyes. Gonioscopy revealed closed angles with a flat iris configuration. A hazy view of the optic nerve revealed normal cup-disc ratios. Her initial medical history was unremarkable.

A laser peripheral iridotomy was performed, but the IOP and anterior chamber depth remained unchanged after 24 hours. A- and B-scan ultrasonography revealed relative lens thickening and 360° ciliochoroidal detachments in both eyes. Refraction revealed myopia of 8.00 D in both eyes. During a second medical history, the patient mentioned using topiramate as migraine prophylaxis for the previous 10 days, but had stopped taking the drug when admitted to hospital. The following day, repeat refraction revealed 3.00 D of myopia in both eyes with simultaneous deepening of the anterior chambers. After 4 days, she was completely symptom-free with a UCVA of 6/6, deep anterior chambers, and normal IOP in both eyes.

Topiramate, a sulfamate-substituted monosaccharide, is an antiseizure medication that is also used for the management of migraine, depression, and neuropathic pain. Topiramate may cause idiosyncratic ciliochoroidal detachments and ciliary body oedema leading to anterior displacement of the lens-iris diaphragm, lens thickening, and acute angle closure glaucoma.[1] These signs are reversible with immediate discontinuation of the drug.[2-4] Since visual blurring and ocular discomfort may be the initial symptoms, eye care providers are likely to be the first health care professionals to see patients with these conditions.[5] Importantly, topiramate should be suspected when patients present with bilateral myopia and angle closure, since an iridotomy does not improve the clinical course.

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Delayed Haemorrhage — a Potential Late Complication of Routine Chalazion Surgery

Dear Editor,

A 36-year-old man presented with acute non-traumatic profuse bleeding from his left eye 5 days after uncomplicated chalazion removal from under the left eyelid. The patient was otherwise fit and healthy. There was no prior history of coagulopathy or family history of bleeding disorder. The hospital record showed that no preoperative anticoagulants had been given.

Ophthalmic examination revealed normal visual acuity and an intact eye globe. Eversion of the tarsal plate revealed a single arterial vessel bleeding at the site of operation. The vessel was thermal cauterised and the patient's left eye was pressure

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patched. Ophthalmic examination the next day was unremarkable. Chalazion surgery is a common minor ophthalmic surgical procedure.[1] Complications such as postoperative bleeding usually occur within the first 24 hours. To date, there has been only one case report of delayed postoperative bleeding complicating chalazion surgery in an elderly lady with a history of hypertension.[2]

This patient illustrates that delayed (5 days) postoperative bleeding is possible in an otherwise healthy individual with no history of antecedent eyelid trauma or hypertension. The normal location of the peripheral vascular arcade of the lower lid is inferior to the lower tarsal border. Bleeding usually arises from one of the perforating branches of the marginal arterial arcade, which is small and lies on the tarsus just 3 mm from the lid margin.

The inferior palpebral arterial arches are attached to numerous vessels from both the medial and lateral sides.[3] However, anomalous vessels are known to exist and can pose problems in identifying the source of the bleeding. These branches may be inadvertently damaged in chalazion surgery. This patient's experience serves as a reminder that complications can occur after a simple chalazion excision and drainage, despite patients' good health and surgical haemostasis.

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Triamcinolone Intracameral Penetration following Peribulbar Injection Masquerading as Postoperative Endophthalmitis

Dear Editor,

A 60-year-old man underwent cataract surgery via a superior scleral tunnel small incision, bimanual nucleus delivery, and rigid posterior chamber intraocular lens implantation. Sphincterotomies were performed at 8 sites due to a miotic pupil (2.5 mm in diameter). The fellow eye had retinal geographic atrophy, so his visual prognosis was limited.

On the first postoperative day, a hypopyon of 2 mm was noted in the anterior chamber. There were no vitreous cells, external discharge, lid oedema, chemosis, conjunctival injection, corneal oedema, or pain. There was trace to mild cellular reaction in the anterior chamber and the red reflex was good. His vision was hand motion to counting fingers at 25 cm. His eyes were checked every 12 to 24 hours and a presumptive diagnosis of reactive endophthalmitis was made.

The hypopyon cleared by the fourth postoperative day, his vision improved to counting fingers at 1 m, and mild ocular hypertension resolved. At fundus examination, advanced age-related macular degeneration was noted. At the 3-month follow-up visit, his vision had improved to counting fingers at 2 m.

The surgical steps and the medical record were reviewed. At the end of the operation, a peribulbar injection of triamcinolone acetonide was given in the inferotemporal quadrant. The palpebral fissure was narrow and the access to the sub-Tenon's space was limited. Documentation showed that the medication dispersed subconjunctivally and leaked onto the external eye and the cornea. It was also documented that the scleral tunnel was bleeding; the bed was cauterised at the end of the operation to prevent intraocular blood penetration and postoperative hyphaema. The wound edge was retracted slightly at the temporal end and was not optimally approximated by the neighbouring sutures.

It is noteworthy that several of the conventional precautions for endophthalmitis prophylaxis had been taken, including perioperative topical quinolone antibiotics, 5% povidone-iodine forniceal and lid margin preparation, irrigation by vancomycin-supplemented balanced salt solution, and a subconjunctival antibiotic.

The differential diagnosis for a postoperative hypopyon should include infectious endophthalmitis, reactive endophthalmitis (including toxic anterior segment syndrome), and pseudoendophthalmitis (triamcinolone-induced pseudohypopyon in the relevant context).[1-3] In this patient, the symptomatology and the course were not consistent with true endophthalmitis; the acute onset of the condition, within the first 24 hours, should have been accompanied by more fulminant findings. However, the presentation was less severe than that of a reactive process and the anterior

chamber cellular reaction was disproportionately less than the hypopyon observed. Interestingly, there was lack of vitreous cellular reaction and pain at follow-up.[4]

Intravitreal triamcinolone is now recommended for persistent diabetic and cystoid macular oedema and exudative age-related macular degeneration, and is being considered for the management of retinal vein occlusions.[4-6] Intraocular injections may result in pseudohypopyon,[4-6] especially in an opened posterior capsule, and examination of anterior chamber aspirates has shown hypopyon of triamcinolone crystals.[5]

Routine peribulbar sub-Tenon's injection of triamcinolone has been shown to be as effective as postoperative topical steroids for controlling postoperative uveitis.[7] However, agents irrigated onto the external eye or injected peribulbarly can gain access into the eye.[8] In this patient, triamcinolone apparently gained access to the anterior chamber through the gap in the scleral wound.

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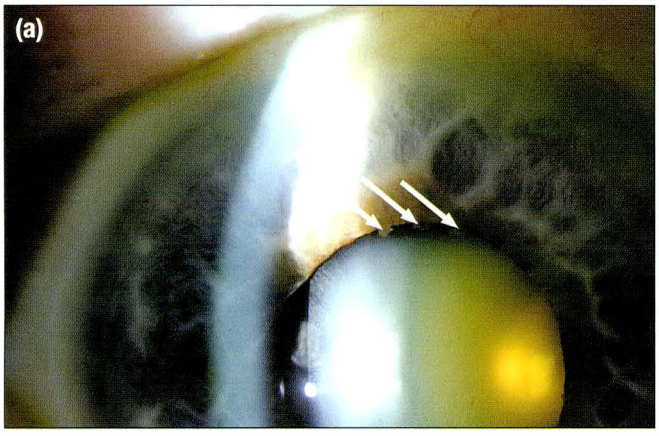
Spontaneous Hyphaema due to Iris Tufts

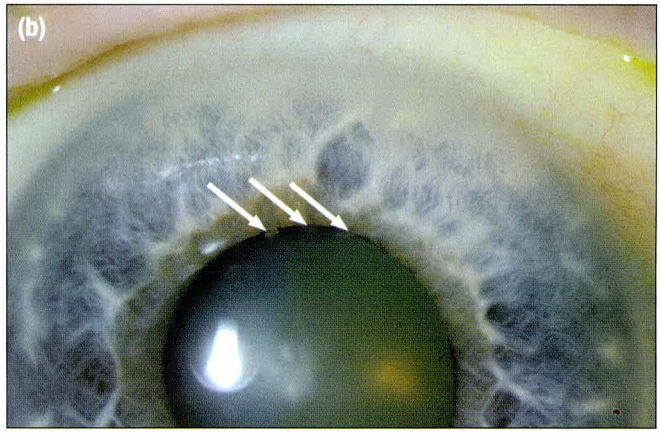
Dear Editor,

An 84-year-old man presented with reduced vision of his right eye on waking. He had no history of pain, photophobia, or ocular trauma. He had bilateral moderate cataracts and hypertension, for which he took medication. At examination, his visual acuity was 6/36 in the right eye and 6/12 in the left eye. A small blood clot was noted in the anterior chamber, attached to the iris, in his right eye. He had no rubeosis and his intraocular pressure was normal. Ocular examination was satisfactory.

The patient was treated with topical prednisolone and cyclopentolate eye drops and made a good visual recovery. At his second follow-up visit, when the blood clot had resolved, he was noted to have iris tufts (Figure 1). Iris neovascular tufts, also known

Figure 1. (a and b) Biomicroscopy photographs of iris tufts.





as capillary haemangioma or microhaemangioma of the iris, are biomicroscopic capillary outgrowths from the papillary margins. They were first described by Cobb in 1969 and are therefore also known as Cobb's tufts.[1] The vascular tufts may be single or multiple, are usually bilateral, and are 15 to 150 μm in size.[2] The tufts are separated from each other and do not form a vascular network. Fluorescein angiography demonstrates leakage from these lesions.[2]

Pupillary vascular tufts have been described in elderly people, in patients with diabetes mellitus, myotonic dystrophy, Sturge-Weber syndrome, or retinal venous occlusion, or in association with haemangioma of the orbit or eyelid. Although the lesions are benign and recurrent haemorrhages are unusual, serial argon laser photocoagulation of the iris vascular tufts can arrest further episodes of spontaneous hyphaema and facilitate uneventful cataract surgery.[3,4]

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World Glaucoma Day: A WGA/WGPA initiative

6 March 2008, Global

Fighting the glaucoma pandemic poses some complex challenges. Although this disease is one of the main causes of blindness globally, glaucoma doesn't elicit a 'sympathy factor' like other blinding diseases, mainly because of the wide geographical variation of required glaucoma prevention measures. More specifically, in developing countries where cataract or river blindness is endemic, there is little incentive to invest resources in diagnosing a disease that will require lifelong treatment and sophisticated follow-up. In developed countries, the pharmaceutical industry and health authorities consider that it is a relatively well-addressed disease, with limited unmet needs, especially when compared with conditions such as cancer, acquired immunodeficiency syndrome or, in the field of ophthalmology, retinal degeneration. Furthermore, the specific visual needs of patients with glaucoma are not well known by the authorities, doctors, and sometimes by the patients themselves.

Add to these considerations the incredible fact that globally, for every diagnosed patient with glaucoma there is an undiagnosed one, and the need for increased glaucoma awareness, advocacy, prevention, and education becomes obvious. To jumpstart a global and highly visible effort to coordinate activities in these areas, the World Glaucoma Association (WGA) and the World Glaucoma Patient Association (WGPA) have taken the initiative of organising the first World Glaucoma Day on 6 March 2008, hopefully to be established as an annual event. While significant efforts are directed at achieving recognition of World Glaucoma Day by national or international organisations (several countries are considering issuing World Glaucoma Day stamps, for instance), this will mostly be a day for local events to be simultaneously organised around the globe, in the form of newspaper articles, radio and television coverage, screening campaigns, 'open-door' days at glaucoma clinics, and so on. As these events will ultimately rely on voluntary work at every level, the WGA is calling upon all enthusiastic and resourceful volunteers to propose and implement new ideas for local and global events, to make the first World Glaucoma Day a resounding success.

Increasing Community Awareness

As glaucoma is a progressive disease causing irreversible visual loss, usually without warning until it is relatively advanced, and because 50% of affected people in the developed world, and 90% in developing countries, do not know they have the condition, and are not receiving treatment, we believe community awareness needs to be increased. This includes awareness of the disease, and of the need to have regular eye checks, thereby permitting earlier detection and avoidance of preventable visual disability.

We will work for recognition by national and international organisations and several governments in various ways, such as issuing commemorative stamps and media releases. WGA and WGPA will provide resources to assist local activities, including newspaper articles, radio and television coverage, public glaucoma 'screening' campaigns in public places and 'open-door' days at glaucoma clinics and ophthalmology departments.

WGA and WGPA ask you how you could contribute to the success of World Glaucoma Day. An Expression of Intent form listing various activities that could be organised on or around 6 March 2008 is printed on page 276. This is by no means exhaustive: all ideas would be most welcome, and would be shared globally. Please indicate to us what activities you would like to organise, or nominate someone who would work with you. Please send the completed form electronically to one of the Physician Liaison Committee Members listed below, or to the WGPA board member or liaison officer in your country (cc: George Lambrou).

- Ivan Goldberg, WGA President and WGPA Co-Chair, Physician Liaison Committee
  E-mail: eyegoldberg@gmail.com
- Robert Ritch, WGPA Co-Chair, Physician Liaison Committee
  E-mail: ritchmd@earthlink.net
- George Lambrou, WGPA EVP, Physician Liaison Committee; WGD Global Project Leader
  E-mail: gnlambrou@hotmail.com; george@lambrou.eu
- Scott Christensen, WGPA President
  E-mail: scott@glaucomafoundation.org

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세계 녹내장의 날
Hari Glaukoma Sedunia
NGÀY GLAUCOMA THẾ GIỚI
Dünya Glokom Günü
يوم الغلوكوما العالمي
ימלועה המוקואלגה סוי
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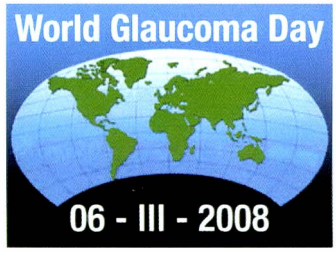

Международный День Глаукомы
Nemzetközi Zöldhályog Nap
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Diwrnod Byd Glaucoma
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Ihlabathi Glaucoma Imini
Wiase Nyinaa Glaucoma Da
Համաշխարհային Գլաուկոմայի Օր
Kelame Petee Glaucoma Liwe
Xexeame Kataa Glaucoma Nkeke
Dzen Fee Glaucoma Gbi
Ranar Tunawa Da Ciwon Ido na Gilakoma A Duniya
Ubochi uwa nile necheta Glacoma
Ayajo Agbaiye Arun Oju ti anpe nu Glokoma
Umsanzi Glaucoma Ilanga

WORLD GLAUCOMA DAY EXPRESSION OF INTENT FORM

Please, put an "X" in one of the 3 columns marked "yes", "no" or "maybe" and fill in the contact person's details, if not yourself (or write "myself"). If you cannot organize the activity, but you think that someone else could, please put an "X" in the "no" column and add that person's contact details – or at least their name – in the last column. Please add your own contact details at the bottom of the page.

ACTIVITIES	Yes	No	Maybe	Contact person details
Contact newspaper to publish an article				
Write newspaper article (you can also use the "contact person details" column to nominate suitable persons to do that)				
Contact radio station to cover the WGD				
Contact TV channel to cover the WGD				
Lobby my country's postal services to issue commemorative stamps				
Organize open-doors event at my institution				
Organize free screening day at my institution				
Organize free screening at public site (mall, market, place of worship, town hall, community center, etc)				
Organize patient-educational conference at my institution/ at a public site (please delete one)				
Organize a patient-run conference at my institution/at a public site (please delete one)				
Participate at a conference if someone else organizes it (you can also use the "contact person details" column to nominate suitable persons to speak at conferences)				
Post on my website, blog or newsletter/BBS relevant material advertising the World Glaucoma Day, and/or brochures, a patients' journal, patient interviews, etc (please delete as appropriate or add your ideas for an online event)				
Distribute at my practice/institution/at a public site printed material advertising the WGD, and/or brochures, a patient's journal, patient interviews, etc (please delete as appropriate)				
Other activity idea (please fill in)				
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Your name: _____

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Postal address: _____

IMAGE Modules — in Summary

The South East Asia Glaucoma Interest Group (SEAGIG) is proud to announce that all 9 modules from the educational resource that has emerged from the Initiative for Management, Awareness and Glaucoma Education (IMAGE) project have now been launched. Aiming to enhance glaucoma management throughout the region, this comprehensive slide and video kit is the result of contributions from more than 50 leading experts from 7 Asian countries. The resource covers the spectrum of glaucoma management, from diagnosis to follow-up, using the Asia Pacific Glaucoma Guidelines as its curriculum basis.

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 encompass topics that are clinically relevant to glaucoma care in the region and slides that have educational value relevant to the region. All modules are now available on the SEAGIG website, at www.seagig.org. Thumbnails will be freely visible by all visitors, with the full slide sets freely accessible by SEAGIG members.

Module 1: Glaucoma Assessment

This presentation aims to provide the practicing ophthalmologist with an updated understanding of the assessment of patients with glaucoma and those suspected of having glaucoma. Following the concepts outlined here can translate into improved detection and evaluation of glaucoma and, ultimately, more effective prevention of visual loss and blindness. This module provides an overview of objectives and components of an initial assessment, including patient histories, examination, and development of a management plan based on findings.

Module 2: Gonioscopy

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, diagrams, and video clips showing various gonioscopic procedures and equipment.

Module 3: Optic Disc Assessment/RNFL Overview

Recognising the characteristic features of glaucomatous optic neuropathy is a critical component in identifying and caring for patients with glaucoma. The 5 rules for assessing the optic disc and retinal nerve fibre layer (RNFL) in glaucoma will be introduced, followed by separate sections focused on detecting glaucoma and disease progression. With practice, the clinician can perceive glaucomatous changes easily and accurately using the information provided in these slides. Sample photographs are included for the purposes of discussion and testing participants' skills.

Module 4: Automated Perimetry

Automated perimetry is the gold standard for diagnosis and management of glaucoma. A careful appraisal of an automated perimetry printout will enable the practitioner to identify a visual field defect, determine whether the defect is due to glaucoma, and establish whether the lesion is progressing. However,

Figure 1.

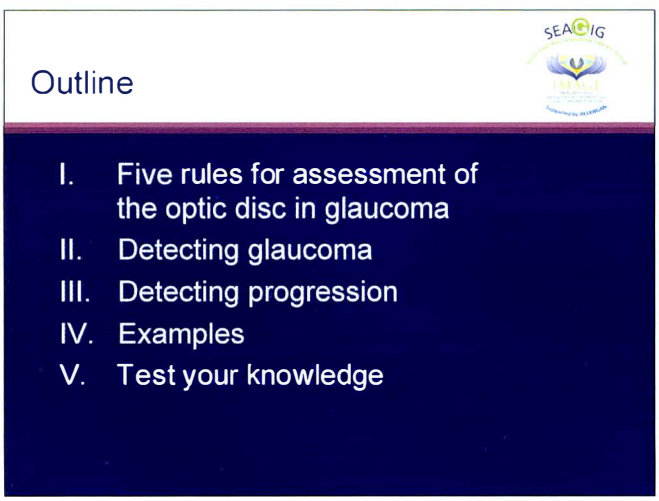
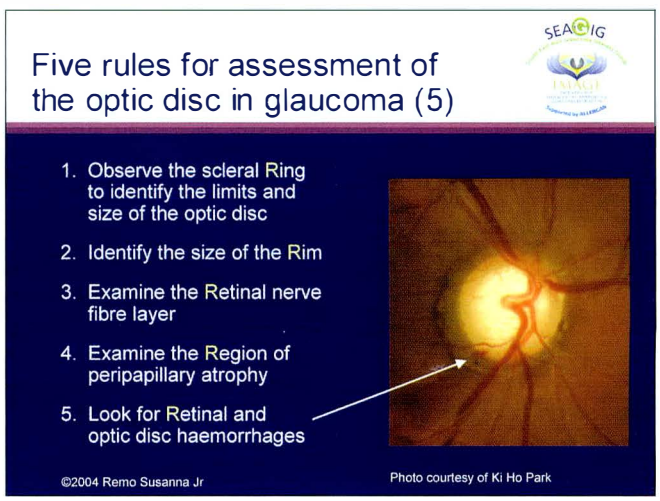


Figure 2.



interpretation requires both an understanding of the principles involved and practice. This presentation is intended to act as a practical guide to automated perimetry and its role in the diagnosis of glaucoma. Sample printouts are provided throughout the module to show clinicians how to interpret perimetry results in a systematic manner.

Module 5: Setting IOP Targets

Reducing the intraocular pressure (IOP) level is an important goal when treating patients with glaucoma, as several clinical trials have shown that most glaucoma-induced damage is pressure-dependent. Keeping IOP in check benefits patients by protecting the optic nerve from damage and preserving the visual field. However, determining the specific level to which IOP should be lowered to achieve optimal glaucoma management in every patient is a challenge. This module covers the rationale for setting target IOPs and outlines data from important glaucoma clinical trials with their implications for patient management.

Module 6: Medical Treatment

The *Medical Treatment* module provides an overview of the medical therapies available for the treatment of glaucoma. Clinical data on the efficacy and safety of these medicines is presented. Tips for optimising medical treatment, including how to maximise patient adherence, are covered in the first section of this module. A medical treatment algorithm is provided to guide clinicians in making treatment decisions, and the pros and cons of various drug classes are also discussed. The next portion of this module presents some of the clinical trial data available on the efficacy and safety of these medicines. Finally, the role of preservatives in ocular medications is explored.

Module 7: Laser Treatment

The *Laser Treatment* module covers some of the laser therapies available for open angle glaucoma and angle closure glaucoma. These guidelines are intended to help clinicians choose the most appropriate treatment for their patients and also provide useful tips on pre- and postoperative care. Each of the laser procedures (laser trabeculoplasty, iridotomy, iridoplasty, and cyclophotocoagulation) will be covered in detail, including an explanation of the procedure, when it is appropriate and why it is used, and general guidelines for managing the procedure.

Module 8: Surgery

The *Surgery* module outlines some of the common surgical options available to treat open angle, angle-closure, and childhood glaucomas. Upon finishing this module, clinicians should have a better understanding of when surgery is the most appropriate treatment choice and how to augment surgical procedures with pre- and postoperative care to optimise patient outcomes.

After beginning with an overview of when surgery is the most appropriate treatment for glaucoma and the indications for surgery, this module provides a more detailed look at some commonly performed surgical procedures, including trabeculectomy, bleb revision, and iridectomy. Surgical 'pearls' explaining each step of the procedure are included to give clinicians additional guidance on technique.

Module 9: Follow-up

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.

Acknowledgement

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Reference

South East Asia Glaucoma Interest Group. Asia Pacific Glaucoma Guidelines. Singapore: SEAGIG, 2003-2004. Available at: www.seagig.org/pdf/APGGuidelinesNMview.pdf.

Comments and Suggestions

The SEAGIG-IMAGE Project Working Group has endeavoured to make this educational resource as comprehensive as possible. It is intended to be user-friendly and responsive to the challenges faced by ophthalmologists and glaucomatologists today. Your feedback will be welcomed, including any suggestions for improvement. Please contact the SEAGIG-IMAGE Project Working Group, via *Asian Journal of Ophthalmology*, at: editor@seagig.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 aim of the AACGC is to establish a scientific network for Asian glaucomatologists who are interested in exchange of knowledge about angle closure glaucoma.

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.

Symposium Themes

- Normal-Tension Glaucoma
- Glaucoma Screening and Awareness in Asia
- Medical Treatment
- Surgical and Laser Treatment
- Imaging and Diagnosis
- Controversies/Future Trends
- Neuroprotection in Glaucoma
- Angle-Closure Glaucoma

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

Enquiries should be directed to:

info@seagig-aacgc.org

Win a Trip to Seoul for SEAGIG 2008 — the 2008 Writer's Award

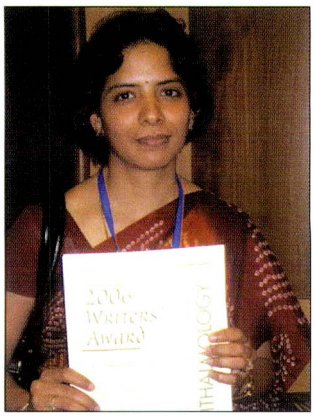
Do you have a scientific paper that you would like to have published in Asian Journal of OPHTHALMOLOGY? The South East Asia Glaucoma Interest Group (SEAGIG), in conjunction with Asian Journal of OPHTHALMOLOGY, is offering the opportunity to attend the SEAGIG 2008 meeting in Seoul, Korea, 25-27 September 2008 absolutely free to the first author of the best scientific paper submitted before **30 April 2008**.

The 2008 Writer's Award includes free transportation (economy air ticket), registration at the conference, and accommodation at the '**Lotte Hotel Seoul**' (conference hotel). The award-winning paper will be published in the **September 2008** issue of Asian Journal of OPHTHALMOLOGY, which will be distributed at the SEAGIG 2008 meeting. (Details of the meeting are available at **www.seagig2008seoul.org**)

Submitted papers must be Original Articles that have not been previously published in, and are not currently under consideration for, any other journal. Submitted manuscripts must follow the guidelines set out in the Information for Authors on the SEAGIG website (**www.seagig.org/authors.php**).

The papers will be judged on the basis of their scientific content, the clarity of writing, and manuscript preparation by a panel of SEAGIG members. The judges' decision will be final.

Dr Hemamalini Arvind, winner of the 2006 Writer's Award



ACS AND SNEC SCIENTIFIC MEETINGS

Singapore, 13-14 and 15-17 March 2008

The Council of the Asia Cornea Society (ACS) welcomes you to the Inaugural Asia Cornea Society Scientific Meeting, which will be held from 13 to 14 March 2008 at the Shangri-La's Rasa Sentosa Resort, Singapore.

True to the meeting's theme 'At the Forefront of the Eye!', this inaugural meeting brings together leading corneal clinicians and researchers from around the globe to share their expertise on pivotal medical and surgical developments at the cutting edge of the cornea and external eye, which are effecting paradigm shifts in ophthalmology today.

Delegates attending this landmark meeting can look forward to a stimulating scientific programme consisting of scientific symposia, plenary lectures, and poster sessions. The Singapore National Eye Center (SNEC) 18th Anniversary International Meeting immediately follows the ACS meeting. This meeting, to be held at the Suntec International Convention & Exhibition Centre from 15 to 17 March 2008, promises a comprehensive range of teaching courses for 'The Practical Ophthalmologist'.

For further information, visit the website at: **www.asiacorneasociety.org/IACS.asp**

ASIA CORNEA SOCIETY

At the Forefront of the Eye!

Inaugural Asia Cornea Society Scientific Meeting

13 to 14 March 2008 Shangri-La's Rasa Sentosa, Singapore

Organised by the recently formed Asia Cornea Society (ACS), this meeting will feature the latest, the most advanced and the cutting edge of management of corneal diseases, surgical and laser technologies true to its theme – ***At the Forefront of the Eye!***

Thematic Areas:

- Deep Anterior Lamellar Keratoplasty
- Endothelial Keratoplasty
- Femtosecond Laser-assisted Keratoplasty
- Keratoprosthesis surgery
- Ocular Surface Transplantation
- Ocular Surface Diseases
- Emerging Corneal Infections
- Keratoconus and Keratectasias
- Eye Banking
- Contact Lenses
- Corneal Refractive Surgery

Look forward to a stimulating scientific programme of symposia and plenary lectures participated by leading and prominent corneal clinicians and researchers from Asia, Europe and the USA conducted in the most relaxed and casual atmosphere.

Register for the meeting before 30 January 2008 for Early Bird Rates!

Ophthalmologist or Physician
US$ 200(S$ 320)

Resident / Trainee / Eye Care Professional
US$ 100(S$ 160)

Accompanying Person
US$ 75(S$ 120)

www.asiacorneasociety.org

*** Deadline for Poster Abstract Submission : 15 January 2008**

About the Asia Cornea Society

The formation of the Asia Cornea Society (ACS) is spurred by a common vision amongst corneal subspecialists and researchers throughout Asia to foster the exchange of knowledge and information on clinical, educational and research aspects of the corneal subspecialty with particular focus and relevance to Asian corneal diseases.

ACS members will enjoy full rebate of paid membership fees when they register for the Inaugural Asia Cornea Society Scientific Meeting!

Contact Information
Asia Cornea Society Secretariat 11 Third Hospital Avenue Singapore 168751
Fax : +65 6227 7291 Email : acs@snec.com.sg

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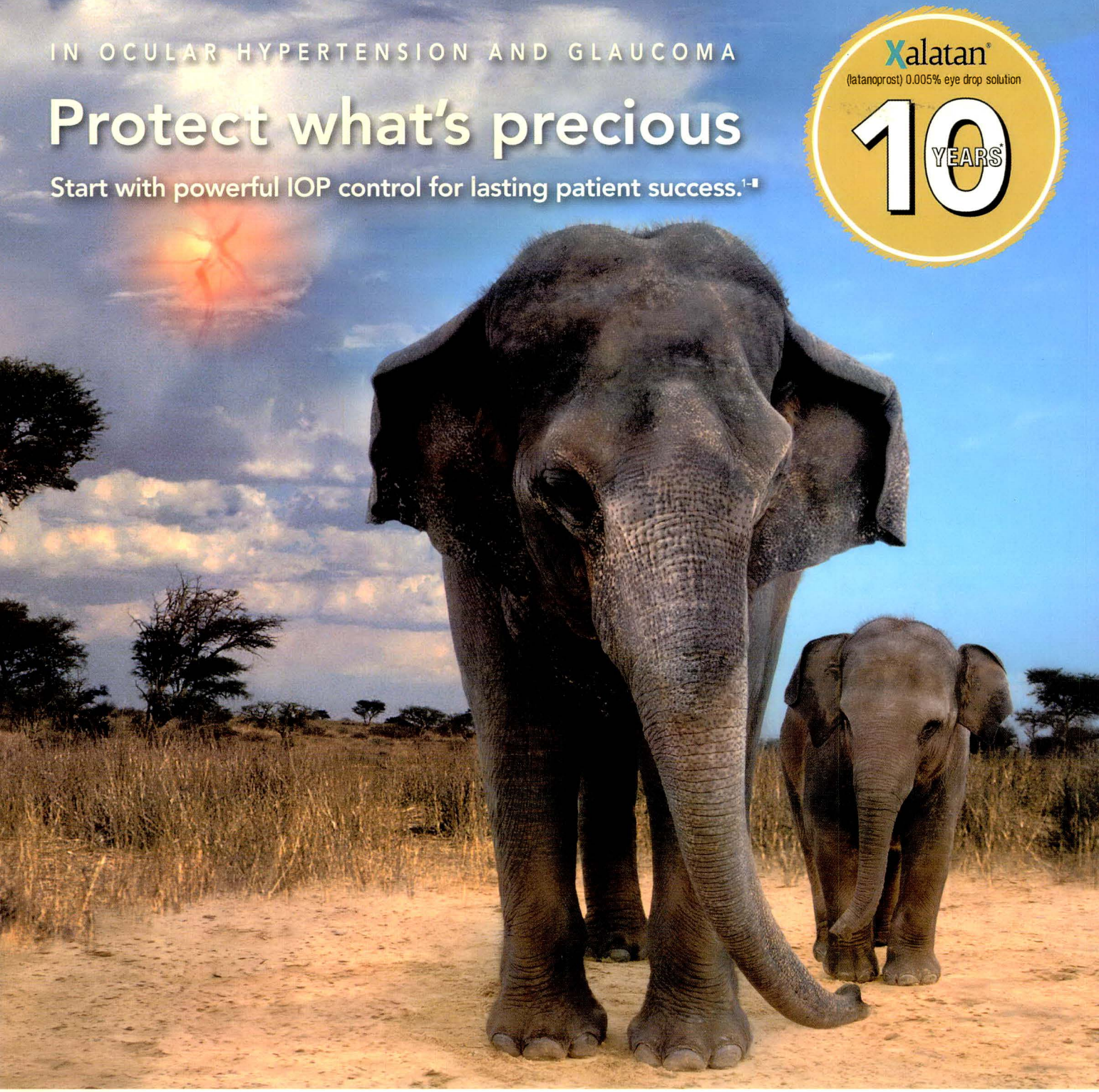

Xalatan® is indicated for the reduction of elevated intraocular pressure (IOP) in patients with open angle glaucoma (OAG) and ocular hypertension (OH). Prescribing Information. Please refer to the SmPC before prescribing Xalatan® 0.005% eye drops solution (latanoprost) **Presentation:** Plastic bottle containing 2.5ml eye drops. Each 1ml contains latanoprost 50 micrograms (0.005%) and benzalkonium chloride 0.20mg. Indication Reduction of elevated intraocular pressure in patients with OAG and ocular hypertension. **Dosage and Administration:** *Adults including the Elderly:* One eye drop into the affected eye(s) once daily in the evening. Contact lenses should be removed before instillation of the eye drops and may be reinserted after 15 minutes (see Precautions). *Children:* Not recommended. **Contra-indications:** Known hypersensitivity to any component. **Precautions:** Xalatan may increase brown pigment within the iris leading to a gradual change in eye colour usually within the first 8 months. This has predominantly been seen in patients with mixed coloured irides and may be permanent. Patients should be examined regularly and treatment discontinued if appropriate. Unilateral treatment can result in permanent heterochromia. Exercise caution in patients with severe or brittle asthma, inflammatory ocular conditions and other types of glaucoma, including OAG of pseudophakic patients, aphakic patients, pseudophakic patients with torn posterior lens capsule or anterior chamber lenses or patients with known risk factors for cystoid macular oedema. Xalatan contains the preservative benzalkonium chloride which has been reported to cause punctate keratopathy and/or toxic ulcerative keratopathy, may cause eye irritation and is known to discolour soft contact lenses. Close monitoring required with frequent or prolonged use of Xalatan in dry eye patients/conditions where the cornea is compromised. Contact lenses may absorb benzalkonium chloride. These should be removed before applying Xalatan but may be reinserted after 15 minutes (see Dosage and Administration). **Pregnancy:** Do not use. **Lactation:** Do not use or stop breast feeding. **Interactions:** Definitive data are not available. However, any other eye drops should be administered five minutes apart. **Side Effects:** Ocular side effects – Very common (>1/10): Increased iris pigmentation, eye irritation (including slight foreign body sensation), eyelash changes (darkening, thickening, lengthening, increased number); Common (>1/100 and <1/10): Mild to moderate conjunctival hyperaemia, transient punctate epithelial erosions (mostly without symptoms), blepharitis, eye pain. Please refer to SmPC for other ocular side-effects. Non-ocular side-effects – Uncommon (>1/1000 and <1/100): Skin rash; Rare (<1/1000): Asthma, asthma exacerbation and dyspnoea; Very rare (<1/10,000): Aggravation of angina in patients with pre-existing disease, chest pain. **Driving:** Vision may be blurred following eye drop instillation. **Overdosage:** Symptomatic treatment. **Pharmaceutical Precautions:** Store at +2°C – +8°C. Protect from light. Once opened, store at room temperature (≤25°C) and discard after 1 month. **Legal category:** POM. **Packaging Quantities and Basic NHS price:** 2.5 ml £13.14. **PL number:** PL 00032/0220. **PL Holder:** Pharmacia Limited, Ramsgate Road, Sandwich, Kent, CT13 9NJ, UK. **Date of preparation of PI:** September 2006. Further information is available on request from: Pfizer Limited, Walton Oaks, Dorking Road, Tadworth, Surrey, KT20 7NS, UK.

Adverse events should be reported to Pfizer Medical Information on 01304 616161 Information about adverse event reporting can also be found at www.yellowcard.gov.uk

REFERENCES: 1. European Glaucoma Prevention Study (EGPS) Group. Predictive factors for open-angle glaucoma among patients with ocular hypertension in the European Glaucoma Prevention Study. *Ophthalmology* 2007;114:3–9. 2. Gordon MO *et al.* The Ocular Hypertension Treatment Study. Baseline factors that predict the onset of primary open-angle glaucoma. *Arch Ophthalmol.* 2002;120:714–20. 3. Heijl A *et al.* Reduction of intraocular pressure and glaucoma progression: results from the Early Manifest Glaucoma Trial. *Arch Ophthalmol.* 2002 Oct;120(10):1268–79. 4. Advanced Glaucoma Intervention Study (AGIS) Investigators. AGIS. 7. The relationship between control of intraocular pressure and visual field deterioration. *Am J Ophthalmol.* 2000;130:429–40.

* registration in the UK 16/12/1996.

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