Journal of the South East Asia Glaucoma Interest Group

Volume 8 Number 5 October 2006

Amniotic Membrane – A Gift of Nature

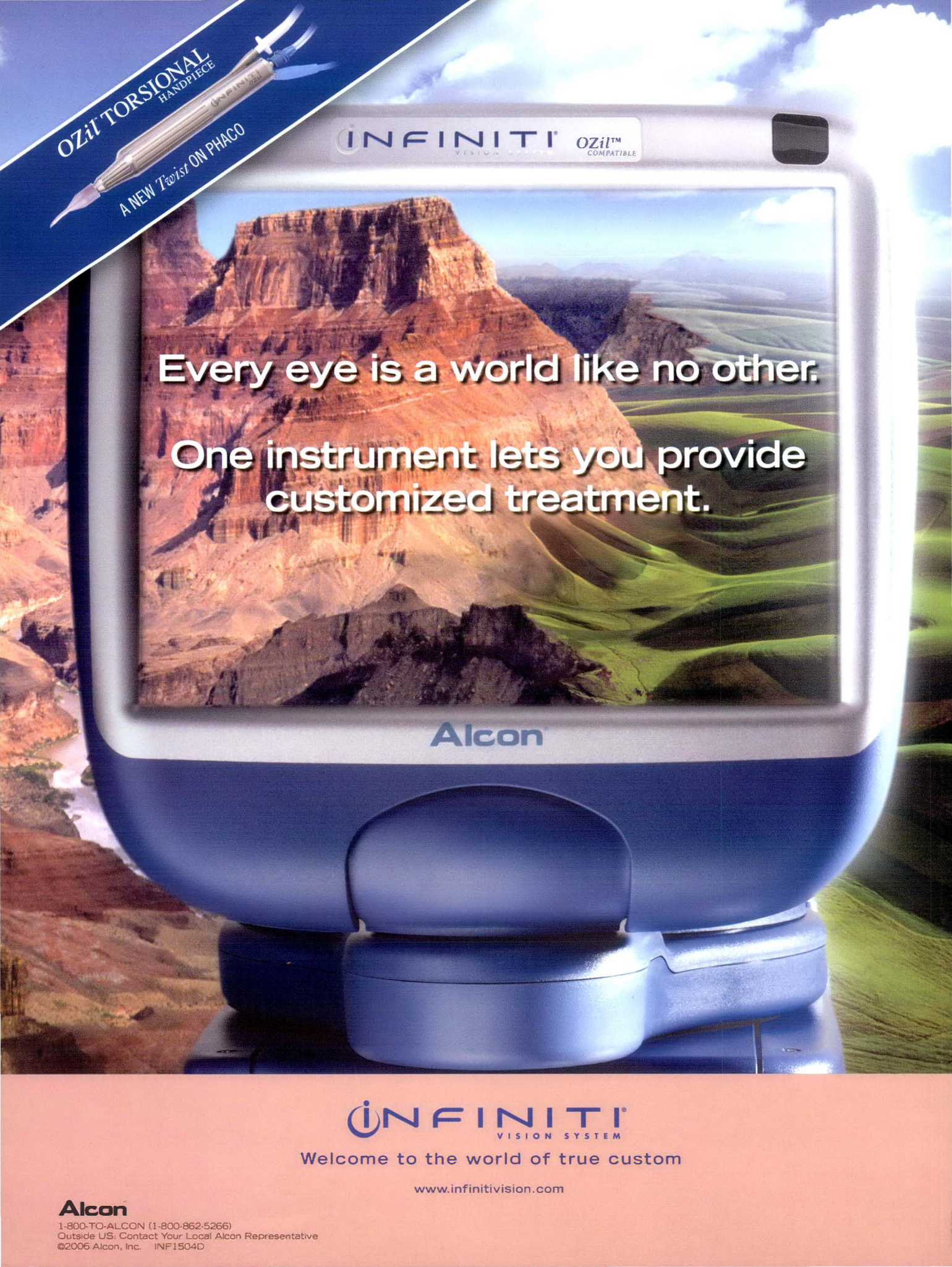
Amniotic Membrane Transplantation for Trachoma

Eye Diseases in School Children in Malaysia

Development of Myopia in Medical School

Asian Journal of OPHTHALMOLOGY

Scientific Communications



INFINITI VISION SYSTEM

Welcome to the world of true custom

www.infinitivision.com

Alcon

1-800-TO-ALCON (1-800-862-5266)
Outside US: Contact Your Local Alcon Representative
 INF1504D

Asian Journal of
OPHTHALMOLOGY
Volume 8 Number 5 October 2006

SEAGIG
South East Asia Glaucoma Interest Group

Asian Journal of OPHTHALMOLOGY is the official peer-reviewed journal of the South East Asia Glaucoma Interest Group (SEAGIG) and is indexed in EMBASE/Excerpta Medica. The website of *Asian Journal of OPHTHALMOLOGY* and SEAGIG membership details can be found at **www.seagig.org**.

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.

SEAGIG is grateful to the following sponsors:
Platinum: Alcon *Gold:* Pfizer *Silver:* ALLERGAN

Scientific Communications

ISSN 1560-2133

Editorial Office
Mary Smith
Scientific Communications International Limited
Suite C, 10/F, Wo On Building
10 Wo On Lane, Central, Hong Kong
Tel: (852) 2868 9171
Fax: (852) 2868 9269
E-mail: editor@seagig.org
Website: www.scientific-com.com

Manuscript Submissions
Information for Authors is available at the SEAGIG website (www.seagig.org), where manuscripts can be submitted online. Manuscripts can also be e-mailed to Kayoko Welsh at: manuscripts@seagig.org

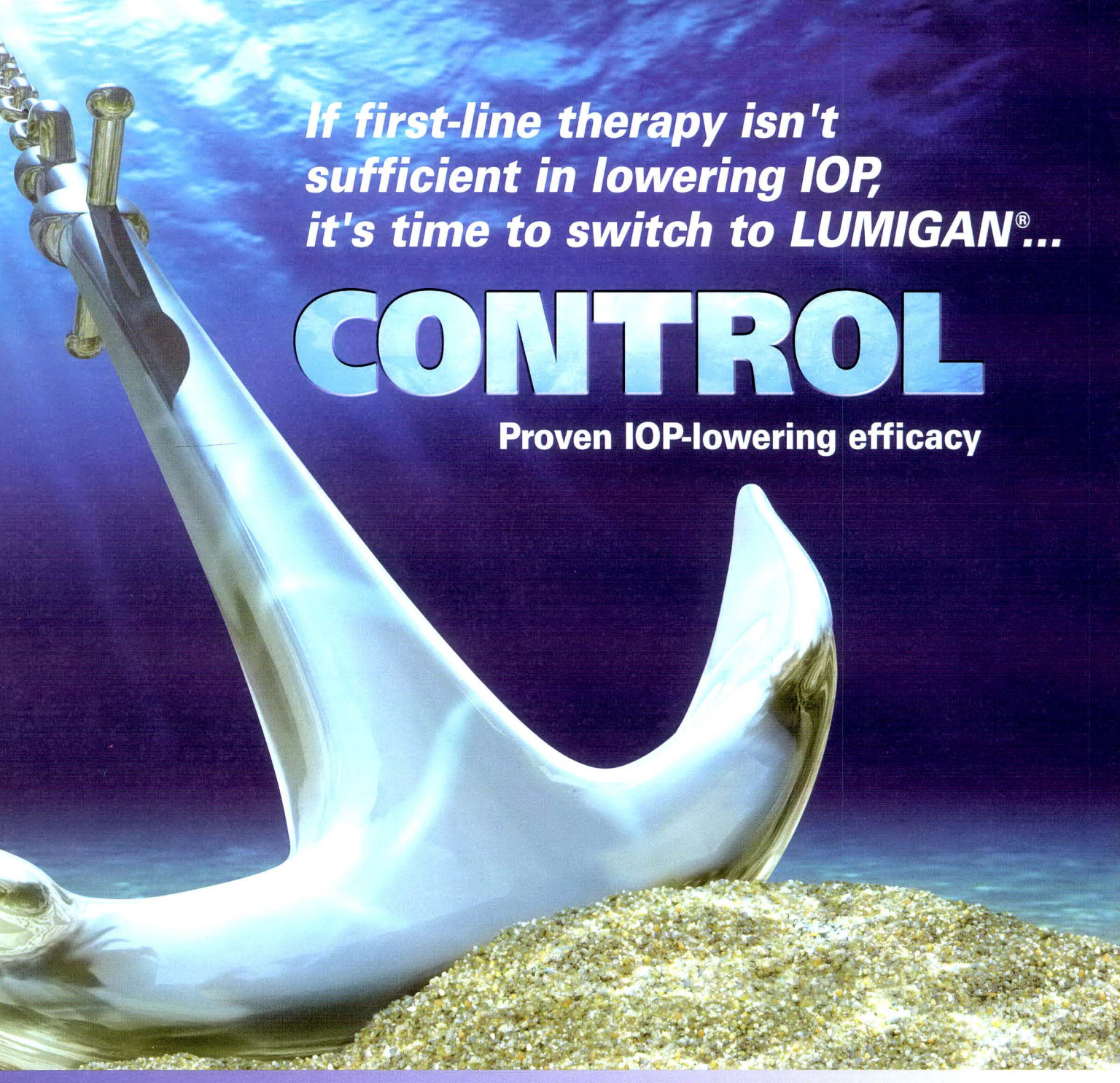
Subscriptions
Please visit www.seagig.org to subscribe online. For queries and address changes, e-mail: idachan@scientific-com.com

Advertising Enquiries
To advertise in the Journal or SEAGIG website, or to become a SEAGIG sponsor, please visit www.seagig.org to access the Media Kit. Please e-mail any advertising/sponsorship queries to: info@seagig.org

Back Issues and Reprints
A limited number of back issues are available from the publisher. Reprints in large quantities for commercial or academic use may be purchased from the publisher. For information and prices, e-mail: idachan@scientific-com.com

Asian Journal of OPHTHALMOLOGY is distributed to 3500 ophthalmologists throughout Asia, with bonus distribution of up to 4000 copies at international conferences.

SEAGIG is a member of the International Federation of Ophthalmological Societies.



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.

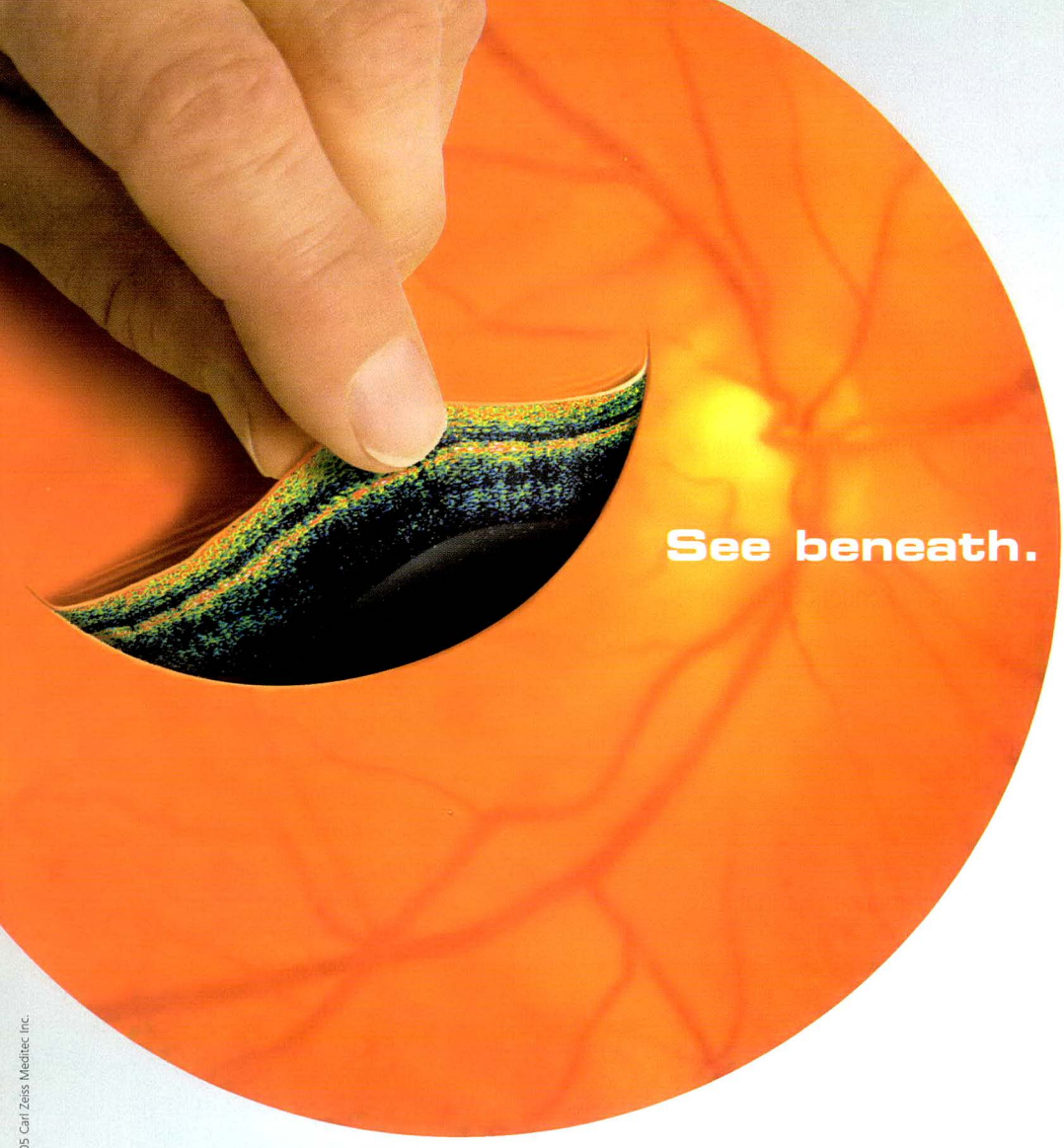
See LUMIGAN® Prescribing Information.

LUMIGAN®
(bimatoprost ophthalmic solution) 0.03%

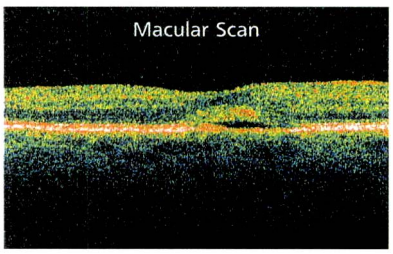
Visit us at www.lumigan.com

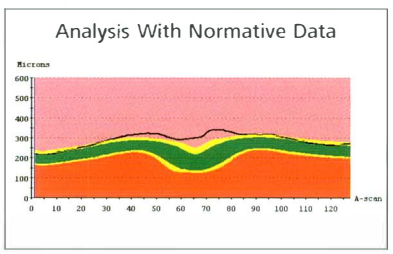
Asian Journal of
OPHTHALMOLOGY
Volume 8 Number 5 October 2006

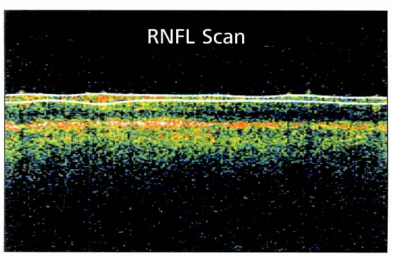
Table of Contents

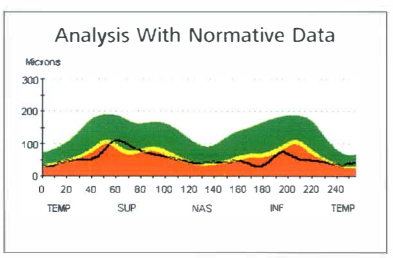


Stay ahead.



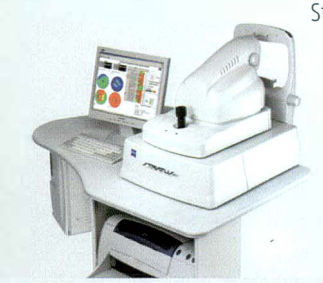






Stratus OCT™ Direct Cross-Sectional Imaging, Measurement and Analysis. Proven performance from the technology leader.

With the widest range of capabilities and proven, time-tested performance, Stratus OCT provides a unique and comprehensive level of diagnostic insight across a broad spectrum of ophthalmic diseases. Stratus OCT gives you the power to quantify retinal nerve fiber layer (RNFL) and macular thickness, and compare them to a normative database. Only Stratus OCT offers unsurpassed imaging and measurement capabilities to help you better evaluate your patients with retinal disease and glaucoma. And dramatic Stratus OCT cross-sectional images vividly document retinal abnormalities to help your patients better understand their diagnosis and treatment options and the impact of your interventions.



So whether your focus is glaucoma, AMD, diabetic retinopathy, pre- and post-cataract surgery assessment, or all of the above, contact us today for more information on Stratus OCT. You'll stay at the forefront of disease management and clinical practice.

ZEISS

Carl Zeiss Meditec Inc. 800-342-9821 www.meditec.zeiss.com

Amniotic Membrane — A Gift of Nature

Lee Sao Bing
Department of Ophthalmology, National University Hospital, Singapore

Human amniotic membrane is a wonderful natural material that not only protects the foetus but also has been used in a variety of ways to treat diseases. The first publication of its use was for skin transplantation in 1910.[1] Amniotic membrane has been used for clinical applications in dermatology, dental surgery, ear, nose and throat surgery, orthopaedics, and ophthalmology. Examples of its use include management of burns, reconstruction of the oral cavity, and tympanoplasty.

The first documented use in ophthalmology was for treating conjunctival defects in 1940.[2] However, there was no further documentation of its use in ophthalmology until the 1990s when Batle and Perdomo reported its use as a conjunctival substitute.[3] Since then, there has been an increase in its use in ophthalmology to treat various ocular surface diseases and as an aid in glaucoma surgery.

How Does Amniotic Membrane Work?

Amniotic membrane is the innermost layer of the foetal membranes. The membrane consists of a single layer of epithelial cells attached to a basement membrane and an avascular stromal matrix. Cryopreservation of human amniotic membrane retains its properties and renders the epithelial cells non-viable and thus non-immunogenic.[4]

Amniotic membrane has several properties that enables its use for various ocular surface diseases:

- Anti-inflammatory and anti-angiogenic factors.
  The stromal matrix excludes inflammatory cells and sequesters T lymphocytes when used as a patch in vivo.[5] Anti-inflammatory factors such as tissue inhibitors of matrix metalloproteinases, interleukin-10 and interleukin-1 receptor antagonists are present in human amniotic membrane.[6]
- Inhibits fibrosis.
  Amniotic membrane downregulates transforming growth factor-β and receptor expression by fibroblasts and thus reduces fibrosis.[7] This unique property enables the membrane to promote scarless healing of the foetus in utero.
- Promotes epithelialisation.
  Amniotic membrane acts like a basement membrane and facilitates migration of epithelial cells.[8]
- Antimicrobial and antiviral properties.
  Antibacterial properties of this tissue have been reported since the early 1990s.[9] This may explain why it has been shown to help in infectious keratitis.[10]

How Can Amniotic Membrane be Used in Ophthalmology?

The use of amniotic membrane in ophthalmology has proliferated in the past 10 years, although its use for some indications has been more helpful than for others. It is very effective for promoting epithelialisation in various ocular surface problems. This includes non-healing persistent epithelial defects and high-risk keratoplasty, as in neurotrophic corneas. It has also been useful in a setting of chronic inflammation and diffuse ocular surface failure such as in Stevens-Johnson syndrome and ocular cicatricial pemphigoid.

Although amniotic membrane is effective in treating recurrent pterygia, some authors may argue that the traditional conjunctival graft provides a better outcome. Most authors will agree that conjunctival grafting is better for primary pterygia. The use of amniotic membrane has shown some degree of success in the treatment of bullous keratopathy, filter leaks, and infectious keratitis. However, it has been less effective for conditions in which the eye is extremely dry, causing the amniotic membrane to dessicate and fail.

In this issue of *Asian Journal of Ophthalmology*, Shenoy et al present a retrospective analysis of the use of human amniotic membrane in patients with trachoma.[11] In this disease, the affected eye may be extremely dry and may have persistent inflammation and progressive scarring. This limits the success of amniotic membrane transplantation for eyes with severe disease.

While the amniotic membrane is not able to treat all ocular surface diseases to the same extent, its ability to protect and to heal is indisputable and is incomparable for certain diseases with no better treatment solution. *Asian J Ophthalmol. 2006;8:189*

Correspondence: Dr Lee Sao Bing, Department of Ophthalmology, National University Hospital, Singapore.
Tel: (65) 6772 5317; Fax: (65) 6777 7161;
E-mail: saobing.lee@nuheye.com

References

1. Davis JW. Skin transplantation with a review of 550 cases at the Johns Hopkins Hospital. Johns Hopkins Med J. 1910;15:307.
2. De Roth A. Plastic repair of conjunctival defects with fetal membrane.

Arch Ophthalmol. 1940;23:522-5.
3. Batle JF, Perdomo F J. Placental membranes as a conjunctival substitute. Ophthalmology. 1993;100:A107.
4. Dua HS, Gomes JA, King AJ, Maharajan VS. The amniotic membrane in ophthalmology. Surv Ophthalmol. 2004;49:51-77.
5. Shimmura S, Shimazaki J, Ohashi Y, Tsubota K. Antiinflammatory effect of amniotic membrane transplantation in ocular surface disorders. Cornea. 2001;20:408-13.
6. Hao Y, Ma DH, Hwang DG, Kim WS, Zhang F. Identification of antiangiogenic and antiinflammatory proteins in human amniotic membrane. Cornea. 2000;19:348-52.
7. Lee SB, Li DQ, Tan DT, Meller DC, Tseng SC. Suppression of TGF-beta signaling in both normal conjunctival fibroblasts and pterygial body fibroblasts by amniotic membrane. Curr Eye Res. 2000;20:325-34.
8. Lee SH, Tseng SC. Amniotic membrane transplantation for persistent epithelial defects with ulceration. Am J Ophthalmol. 1997;123:303-12.
9. Talmi YP, Sigler L, Inge E. Antibacterial properties of human amniotic membranes. Placenta. 1991;12:285-8.
10. Kim JS, Kim JC, Hahn TW, Park WC. Amniotic membrane transplantation in infected corneal ulcer. Cornea. 2001;20:720-6.
11. Shenoy R, Bialsiewicz A, Al-Muniri A, Thakral A. Preserved human amniotic membrane transplantation in patients with trachoma: a retrospective analysis. Asian J Ophthalmol. 2006;5:191-4.

Preserved Human Amniotic Membrane Transplantation in Patients with Trachoma: a Retrospective Analysis

Radha Shenoy,[1] Alexander Bialsiewicz,[1] Abdullah Al-Muniri,[2] Archana Thakral[1]
[1]Department of Ophthalmology and School of Ophthalmic Technicians, and [2]Department of Epidemiology and Medical Statistics, Sultan Qaboos University College of Medicine and Health Sciences, Muscat, Oman

Aim: To evaluate the outcomes of human amniotic membrane transplantation for rehabilitation of ocular surface disorders, with special reference to cicatrising trachoma.

Patients and Methods: Cryopreserved human amnion from healthy donors was grafted in 19 eyes of 14 patients with cicatrising trachoma, 4 eyes of 2 patients with Stevens-Johnson syndrome, and 2 eyes of 1 patient with chemical burns. Follow-up was performed 1 week, 6 weeks, and 6 months after surgery. Outcomes evaluated were re-epithelialisation of the corneal surface, visual acuity, recurrence of symblepharon, and corneal vascularisation. The results for patients with trachoma were compared with those for patients with Stevens-Johnson syndrome and chemical burns.

Results: Twenty five eyes of 17 patients underwent human amniotic membrane transplantation. The success rate for eyes with trachoma did not differ from those without trachoma. After 6 months, 15 of 19 eyes with trachoma (79%) had developed recurrence of symblepharon compared with 2 of 6 eyes without trachoma (33.3%) [$p = 0.06$] and 13 of 15 eyes (86.6%) with cicatricial trachoma experienced a recurrence of corneal vascularisation compared with 2 of 6 eyes without trachoma ($p = 0.18$). Persistent long-term re-epithelialisation was observed in 1 of 19 eyes with trachoma (5.3%) compared with 4 of 6 eyes without trachoma (66.7%) [$p = 0.005$]. Three of 19 eyes with trachoma perforated under the graft and had to be enucleated.

Conclusion: The outcome for human amniotic membrane grafting in ocular surface reconstruction depends on the extent and severity of the primary disease, and its efficacy was limited for rehabilitation of cicatrising trachoma.

Key words: Amnion, Eye burns, Reconstructive surgical procedures, Stevens-Johnson syndrome, Trachoma, Transplantation

Asian J Ophthalmol. 2006;8:191-4

Introduction

Blinding trachoma is still a persistent health problem in developing countries. Approximately 145 million people are affected by the disease, while another 540 million people (10% of the world's population) living in trachoma-endemic areas are at risk of infection. The economic burden of trachoma is comparable to the expected costs of eradicating blindness caused by this disease.[1-4] Antibiotics, improved personal and community hygiene and sanitation, and surgery for trichiasis are strategies recommended for the global campaign for elimination of trachoma.[2,3]

Correspondence: Dr Radha Shenoy, Department of Ophthalmology and School of Ophthalmic Technicians, Sultan Qaboos University College of Medicine and Health Sciences, 123 Al Khod, Muscat, Oman.
Tel: (96 8) 2414 3516; Fax: (96 8) 2441 4100;
E-mail: shenoyvs@omantel.net.om

Little is known about the availability of rehabilitatory measures for the estimated 6 million people affected by trachoma–induced blindness or severe loss of vision, who constitute 15% of the world's total blind population.[1-4] Human amniotic membrane has been found to be an effective functional substrate facilitating the proliferation and differentiation of epithelial cells and it has been widely used for the reconstruction of compromised ocular surfaces.[1-3] The efficacy of this biological tissue for the management of indolent corneal ulcers, epithelial defects, symblephara resulting from scarring and contraction of the conjunctiva in trachomatous eyes has not previously been studied. This retrospective evaluation was performed to ascertain the outcome of human amniotic membrane grafting in eyes with cicatrising trachoma and other mucus deficiency syndromes in Oman, a country in which trachoma has been hyperendemic until recently.

Patients and Methods

Patients

Demographic data of age, sex, surgical indications, and morphological details of postoperative re-epithelialisation of the cornea, recurrence of symblepharon, corneal vascularisation, and visual outcomes after 1 week, 6 weeks, and 6 months were collected from the medical records of patients who underwent amniotic membrane grafting between 2000 and 2005 in a tertiary care teaching hospital in the Sultanate of Oman. Twenty five eyes of 17 patients underwent amnion grafting for indolent corneal defects due to trachoma (n = 19), Stevens-Johnson syndrome (n = 4), or lye burns (n = 2).

Methods

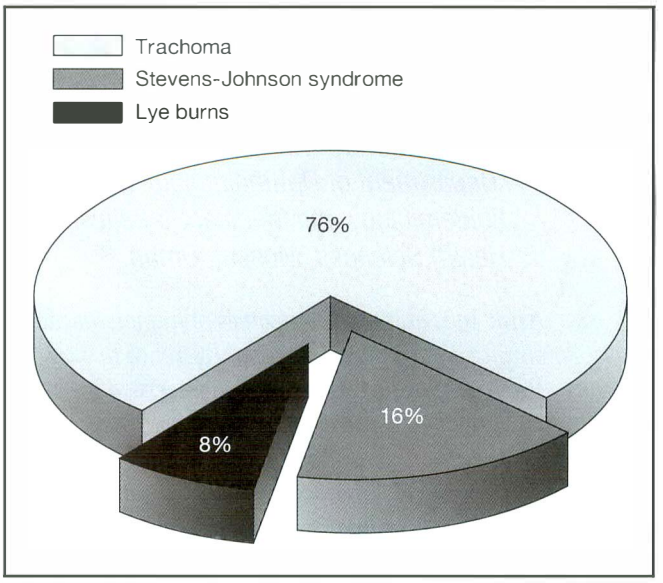
Placentas were obtained at the time of caesarean section from consenting patients who were serologically screened antenatally for exclusion of infections of retrovirus, syphilis, toxoplasma, mycobacteria, rubella virus, and hepatitis B and C virus. Placentas were harvested under sterile conditions and washed with Earle's balanced salt solution (BSS) containing penicillin 50 μg/mL, streptomycin 50 μg/mL, neomycin 100 μg/mL, and amphotericin B 2.5 μg/mL. Amniotic membrane was dissected from the chorion under sterile conditions, washed with BSS, and flattened on a nitrocellulose paper with the epithelial surface uppermost. The paper with the membrane was cut into 2- x 2-cm pieces. The amniotic membrane was fixed to the paper with non-absorbable sutures and stored at -80°C in 1:1 Dulbecco Modified Eagle's medium and glycerol to which gentamicin 80 mg/mL was added.

After a 360° peritomy and cauterisation of the limbal vessels, any symblephara present were released. The amniotic membrane was thawed to room temperature and washed with BSS, placed epithelial side up to cover the debrided corneal and scleral surfaces, and sutured to the sclera with 7-0 vicryl sutures. The margins were covered with Tenon's capsule and conjunctiva wherever possible, and a scleral shell was applied for 3 days. Topical antibiotics and tear substitutes were given in the postoperative period. The patients were followed up at 1 week, 6 weeks, and 6 months. Trichiatic cilia were electro-epilated either before or at the time of surgery.

Results

Twenty five eyes of 17 patients underwent reconstruction of the ocular surface using cryopreserved human amniotic membrane transplantation for indolent corneal ulcers of varied etiology. Nineteen eyes had cicatrising trachoma with mucus deficiency syndrome, 4 eyes had Stevens-Johnson syndrome, and 2 eyes had grade 2 lye burns (Figure 1). The male to female ratio was 1:2, and the average age of the patients was 46.5 years (SD, 2.3 years).

Figure 1. Distribution of ocular diseases in eyes undergoing amniotic membrane transplantation.



Preoperative visual acuity ranged from hand movements (n = 7) to 1/50 (n = 9), 20/250 (n = 4), 20/200 (n = 3), and 20/80 (n = 2). Extensive conjunctival and corneal scarring was observed in 18 eyes — trachoma (n = 12; 67%), Stevens-Johnson syndrome (n = 4; 23%), lye burns (n = 2; 11.1%). Thirteen eyes had anterior symblephara and 12 eyes had posterior symblephara with complete obliteration of the fornices (Table 1). Refractory corneal ulcers were present in all eyes. Sixteen eyes (64%) had paracentral and/or central ulcers — trachoma (n = 12), Stevens-Johnson syndrome (n = 2), lye burns (n = 2) — and the ulcers extended to more than one-third of the corneal surface in 18 eyes (72%) — trachoma (n = 12), Stevens-Johnson syndrome (n = 4), lye burns (n = 2). Eleven eyes with trachoma (57.9%) showed circumferential superficial and deep corneal vascularisation. All eyes had an abnormal Schirmer's test (wetting, 6 mm at 5 minutes) and tear film break-up time (mean, 2 seconds).

Outcomes of Surgery

Fourteen eyes (56%) experienced complete re-epithelialisation within 28 to 35 days (mean, 31 days; SD, 2.3 days) — trachoma

Table 1. Preoperative extent of symblepharon and its distribution in relation to ocular diseases in eyes undergoing amniotic membrane transplantation (n = 25).

Disease	Anterior symblepharon		Posterior symblepharon	Total
	<1/3 surface	>1/3 surface		
Trachoma	2	6	11	19
Stevens-Johnson syndrome	3	0	1	4
Lye burns	2	0	0	2
Total (%)	7 (28)	6 (24)	12 (48)	25 (100)

Table 2. Outcomes according to ocular disease (n = 25).

Outcome	Trachoma (%) [n = 19]	Non-trachoma			Total eyes (%)	p Value*
		Stevens-Johnson syndrome (%) [n = 4]	Lye burns (%) [n = 2]	Total (%) [n = 6]		
Permanent epithelialisation	1 (5.3)	2 (50)	2 (100)	4 (66.7)	5 (20)	0.005
Symblepharon	15 (79)	2 (50)	0	2 (33.3)	17 (68)	0.06
Corneal vascularisation	13 (68.4)	2 (50)	0	2 (33.3)	15 (60)	0.18
Perforation	3 (15.8)	0	0	0	3 (12)	0.55

* Significance between eyes with and without trachoma.

(n = 9), Stevens-Johnson syndrome (n = 3), lye burns (n = 2). However, epithelialisation persisted in 7 eyes at 6 weeks — trachoma (n = 3), Stevens-Johnson syndrome (n = 2), lye burns (n = 2) — and in 5 eyes at 6 months — trachoma (n = 1), Stevens-Johnson syndrome (n = 2), lye burns (n = 2) [p = 0.005].

Fifteen eyes had healing of corneal ulcers with vascularisation (Table 2). Fifteen eyes with trachoma and 1 eye with Stevens-Johnson syndrome developed moderate symblephara. One eye with Stevens-Johnson syndrome developed mild symblepharon (less than one-third of the ocular surface and fornix) with no restriction of motility after initial epithelialisation.

Three eyes with trachoma and corneal thinning (16%) perforated under the graft within the first postoperative week and had to be enucleated due to the lack of availability of a tectonic graft. Recurrence of symblephara and or corneal vascularisation following amniotic membrane grafting occurred in 20 of 25 eyes (Table 3). Three of 7 eyes (42.8%) with persistent epithelialisation at 6 weeks had 1 line improvement in visual acuity postoperatively, while the remaining 4 eyes (57.1%) showed no change.

Discussion

Blindness is a serious public health problem requiring attention in the Sultanate of Oman.[1-3] Trachoma and corneal infection contribute significantly, accounting for 31.6% of blindness in the country.[1-3] The chlamydial infection that causes trachoma is contracted in early childhood and progresses into adulthood. Although active infection is uncommon in adulthood, persistent inflammation and progressive scarring continues, with angiogenesis and addition of extracellular matrix, resulting in conjunctival and corneal scarring, symblephara formation, entropion, trichiasis, and secondary dry eye conditions.[1-4] Severe dry eye, in conjunction with microtrauma caused by trichiasis and uncorrected entropion, which often go unrecognised due to impaired corneal sensitivity, contribute significantly to non-healing corneal ulcers in affected individuals.[1-4]

Table 3. Summary of amniotic membrane graft procedures (n = 25).

	Trachoma*	Non-trachoma		Total
		Stevens-Johnson syndrome	Lye burns	
Grafted	19	4	2	25
Unsuccessful (%)	18 (90)	2 (10)	0	20
Failure rate (%)	94.7	50.0	0	80

* p = 0.005.

Human amniotic membrane consists of a single layer of epithelium on a basement membrane with an avascular stromal matrix. Amniotic membrane facilitates epithelialisation, and reduces formation of scars and adhesions, particularly in stem cell-deficiency disorders,[4-13] and has been used successfully for reconstructive surgery. Amniotic membrane provides a suitable substrate for epithelialisation, facilitates migration of newly formed epithelial cells, reinforces adhesion of the basal cells, and promotes cell differentiation. In addition, growth factors produced by the basement membrane also stimulate epithelialisation.[4-14] The membrane suppresses transforming growth factor-β, DNA synthesis, and subsequent myofibroblast differentiation. These properties, together with the inhibition of new vessel invasion by the avascular stroma, prevent scar formation. However, cryopreservation decreases the production and potency of growth factors, limiting the ability of the graft to exhibit these properties.[13]

Success rates for human amnion transplantation have been reported as 66.7% for chemical burns,[9] 30.0% for severe dry eye or previous conjunctival surgery,[10] and 70.3% for excision of pterygium.[11] However, Zhou et al concluded that microenvironments such as symblephara, dry eyes, and preoperative lack of healthy conjunctiva influenced the long-term results of amniotic membrane graft;[12] this result was also noted in the present study with respect to the trachoma patients. Short-term re-epithelialisation of the ocular surface was achieved in 14 eyes in patients with trachoma and other disorders. However, the long-term success rate relating to corneal vascularisation and recurrence of symblephara was low. Surface epithelialisation occurred only in 3 of 19 eyes with trachoma, with 2 of the 3 eyes developing mild symblephara after 6 months. Four eyes without trachoma developed surface epithelialisation, with recurrence of mild anterior symblephara in only 1 eye with Stevens-Johnson syndrome.

The integrity of the ocular surface is maintained by the corneal, limbal, and conjunctival epithelial cells, together with the tear film. Alterations in the morphology and/or function of these structures, as occurs in trachoma, Stevens-Johnson syndrome, and alkali burns, results in a compromised ocular surface requiring surgical reconstruction. Human amniotic membrane has been successfully grafted in patients with stem cell deficiencies.[4-15] However, since amniotic membrane graft cannot repopulate or prevent loss of stem cells or goblet cells, its efficacy for ocular surface reconstruction depends on the nature and extent of the primary disease, the severity of the dry eye condition, and the availability of goblet and stem cells.[4-15] The facilitating properties of the amniotic membrane for wound healing, re-epithelialisation, and reduced scarring is lessened by cryopreservation, which inhibits growth factor release.[7-13] Persistent inflammation and progressive conjunctival scarring associated with extracellular matrix deposition and angiogenesis, together with the above mentioned factors, probably explain the limited efficacy of the membrane for rehabilitation of trachomatous eyes in this study.[1-4]

References

1. Khandekar R, Mohammed AJ, Negrel AD, et al. The prevalence and causes of blindness in the Sultanate of Oman: the Oman Eye Study (OES). Br J Ophthalmol. 2002;86:957-62.
2. West SK. Trachoma: new assault on an ancient disease. Prog Retin Eye Res. 2004;23:381-401.
3. Abu El-Asrar AM, Al-Kharasi SA, Missotten L, et al. Expression of growth factors in the conjunctiva from patients with active trachoma. Eye. 2006; 20:362-9.
4. Frick KD, Basillion EV, Hanson CL, et al. Estimating the burden and economic impact of trachomatous visual loss. Ophthalmic Epidemiol. 2003;10:121-32.
5. Lee SH, Tseng SC. Amniotic membrane transplantation for persistent epithelial defects with ulceration. Am J Ophthalmol. 1997;123:303-12.
6. Tseng SC, Prabhasawat P, Lee SH. Amniotic membrane transplantation for conjunctival surface reconstruction. Am J Ophthalmol. 1997; 124:765-74.
7. Prabhasawat P, Barton K, Burkett G, et al. Comparison of conjunctival autografts, amniotic membrane grafts, and primary closure for pterygium excision. Ophthalmology. 1997;104:974-85.
8. Kim JC, Tseng SC. Transplantation of preserved human amniotic membrane for surface reconstruction in severely damaged rabbit corneas. Cornea. 1995;14:473-84.
9. Arora R, Mehta D, Jain V. Amniotic membrane transplantation in acute chemical burns. Eye. 2005;19:273-8.
10. Jain S, Rastogi A. Evaluation of the outcome of amniotic membrane transplantation for ocular surface reconstruction in symblepharon. Eye. 2004;18:1251-7.
11. Prabhasawat P, Tesavibul N. Preserved amniotic membrane transplantation for conjunctival surface reconstruction. Cell Tissue Bank. 2001;2: 31-9.
12. Zhou SY, Chen JQ, Chen LS, et al. Long-term results of amniotic membrane transplantation for conjunctival surface reconstruction. Zhonghua Yan Ke Za Zhi. 2004;40:745-9.
13. Sridhar MS, Sagwan VS, Bansal AK, et al. Amniotic membrane transplantation in the management of shield ulcers of vernal keratoconjunctivitis. Ophthalmology. 2001;108:1218-22.
14. Ucakhan OO, Koklu G, Firat E. Nonpreserved human amniotic membrane transplantation in acute and chronic chemical eye injuries. Cornea. 2002;21:169-72.
15. Kozak I, Trbolova A, Sevcikova Z, et al. Superficial keratectomy, limbal autotransplantation and amniotic membrane transplantation in the treatment of severe chemical burns of the eye. Acta Veterinaria Brno. 2002; 71:85-91.

Refractive Errors and Other Eye Diseases in Primary School Children in Petaling Jaya, Malaysia

Sagili Chandrasekhara Reddy, Mohamed Hassan
Department of Ophthalmology, Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia

Aim: *To determine the prevalence of refractive errors and other eye diseases in primary school children in Petaling Jaya, Malaysia.*
Patients and Methods: *A total of 1214 primary school children, aged from 7 to 12 years, were examined for refractive errors, colour vision defects, and other eye diseases. Visual acuity, anterior segment examination, colour vision test, and fundus examination were performed.*
Results: *One or more ocular abnormalities were found in 499 children (41.1%); refractive errors in 33.3%, colour vision defects in 2.6%, squint in 2.5%, epicanthus tarsalis in 1.6%, ocular melanosis in 3.8%, ptosis in 1.1%, trichiasis in 0.2%, chalazion in 0.2%, allergic conjunctivitis in 0.2%, conjunctival naevus, capillary haemangioma of the face in 0.1%, squamous papilloma of lower lid in 0.1%, and unilateral optic atrophy in 0.1%.*
Conclusions: *Myopia is the most common refractive error in primary school children. Examination of primary school children at the time of their admission by an ophthalmologist will help in early detection of refractive errors and other eye diseases so that they can be treated and visual impairment can be minimised.*

Key words: *Blepharoptosis, Child, Color vision defects, Refractive errors, Strabismus*

Asian J Ophthalmol. 2006;8:195-8

Introduction

Eyes are the windows of learning and visual appreciation of objects contributes to learning in any individual's life. Visual disability in childhood can be minimised, or even prevented, if the causes are detected early and treated before they become irreversible. Refractive errors are one of the major causes of low vision and preventable blindness, and the most common reason for patients to consult ophthalmologists and optometrists throughout the world. Detection of visual impairment in school children is important. Poor vision in childhood affects performance in school, and has a significant impact on the future life of the child in terms of education and development. Correction of refractive errors will minimise visual disabilities and improve the child's life in general.

Several studies on the screening of school children for eye diseases such as refractive errors, strabismus, colour blindness, cataract, vitamin A deficiency, trachoma, stye, blepharitis, and chalazion are available.[1-13] Teoh and Yow from Malaysia have reported that 7.1% of children had refractive errors and 2.2% had squint.[13] However, these authors did not mention the type and severity of refractive errors, or the presence of any other eye diseases. Since there is a paucity of data on the prevalence of eye diseases in primary school children from Malaysia, this study was undertaken to determine the prevalence of refractive errors, colour blindness, and other eye diseases among children studying in a national primary school.

Correspondence: Professor Sagili Chandrasekhara Reddy, Department of Ophthalmology, International Medical University, Clinical school, Jalan Rasah 70300 Seremban, Negeri Sembilan, Malaysia.
Tel: (60 6)767 7798; Fax: (60 6) 767 7709;
E-mail: screddy98@hotmail.com

Patients and Methods

Patients

All children attending Sri Petaling National Primary School, Petaling Jaya, Selangor, Malaysia, were included in this study. The children were examined at the school between September 2001 and November 2002. The study was approved by the ethics committee of the Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia.

Methods

After taking an ocular history, the visual acuity was tested using the Snellen chart. The pinhole test was done whenever necessary.

Students wearing spectacles were tested for visual acuity with and without their glasses. The power of the lenses was measured using a Zeiss SBM 70 Focimeter (Carl Zeiss, Jena, Germany). Colour vision was tested using Ishihara Pseudoisochromatic colour plates (Kanehara Co Ltd, Tokyo, Japan). Each student was asked to read the numbers on the charts at normal reading distance, and the results were interpreted as per the instructions supplied by the manufacturer. Detailed examination of the eyelids, conjunctiva, sclera, cornea, anterior chamber, iris, pupil, and lens was carried out using torchlight and a magnifying loupe (Eagle +1.75X; Eagle Vision, Memphis, USA). Later, the Hirschberg (corneal light reflex) test was done to detect any squint. The cover test was done to find out whether the squint was in one eye only or alternating in both eyes. Ocular movements were tested in all the cardinal positions to check for paralysis of the extraocular muscles. Fundus examination was done in the same room (with the lights off) with direct ophthalmoscope. If there was any abnormal fundus finding, eyes were dilated with tropicamide 1% eye drops and the fundus was re-examined.

Children with vision <6/6 and other ocular abnormalities were referred to the eye clinic at the University of Malaya Medical Center for cycloplegic refraction, further evaluation, and treatment. The child and parents were informed about the action of the eye drops instilled in the eyes for refraction, and consent was given. Homatropine 2% eye drops, one drop in each eye at 15-minute intervals, were instilled 3 times. The refraction was done with the streak retinoscope, 1 hour after the first drop of homatropine was placed in the eyes. To constrict the pupil, pilocarpine 2% eye drops, 1 drop in each eye, was administered at the end of the refraction test. Three days later, subjective correction was done and glasses were prescribed when needed.

A child with defective vision (≤6/9) who improved to 6/6 clear vision with -0.25 or +0.25 D or more power lens correction was considered to have refractive error. Even though the power is minimal, glasses are prescribed for these children to have clear vision. Visual acuity of ≤6/12 in one or both eyes with best correction glasses was taken as amblyopia, in accordance with other reported studies.[8-10]

Students who did not come for an eye examination were reminded by sending a letter, signed by headmaster and research project investigator, twice to their home address. Those who did not attend for an eye examination despite the second letter were designated as defaulters.

Statistical Analysis

The findings were recorded on a data sheet, and analysed by using the Statistical Product and Service Solution (SPSS) programme. Pearson correlation coefficient was used to analyse the significance of the relationship between refractive error and the age of the children. A p value of ≤0.05 was considered to be significant.

Results

1214 of 1310 students from years 1 to 6 at Sri Petaling National Primary School were examined for a response rate of 92.67%. There were 661 boys (54.4%) and 553 girls (45.6%). The children were aged between 7 and 12 years (children start school at the age of 7 years in Malaysia). There were 714 Malays (58.8%), 308 Chinese (25.4%), and 192 Indians (15.8%).

One or more ocular abnormalities were found in 499 students (41.1%). Uncorrected visual acuity (UCVA) 6/6 was noted in 810 students, and UCVA ≤6/9 was noted in 1 eye in 52 students and in both eyes in 352 students. These 404 students were diagnosed to have refractive error since there was no other eye disease to account for their diminished vision. Thus, 33.3% of students had refractive errors. At the time of the study, 149 of the 404 students with refractive errors (36.9%) were wearing glasses. Of 404 students with refractive errors, 201 were Malays (49.7%), 125 were Chinese (30.9%), and 78 were Indians (19.3%); 199 were boys (49.2%) and 205 were girls (50.7%). Forty two children did not attend for refraction, so refraction was done for 362 students. The likely reasons for default were that the parents felt that nothing was wrong with their child's vision or that the parents were too busy to bring their child for an eye check.

The types of refractive errors are shown in Table 1. Myopia (myopia + myopic astigmatism) was noted in 321 children, hypermetropia (hypermetropia + hypermetropic astigmatism) in 16 children, and mixed astigmatism in 25 children. 138 children had astigmatism (myopic astigmatism, hypermetropic astigmatism, or mixed astigmatism). Among the 25 children with mixed astigmatism, 4 had myopic sphere with hypermetropic cylinder and 21 had hypermetropic sphere with myopic cylinder.

The prevalence of myopia was highest in children aged 10 years (49 of 72 children; 68.1%), which was statistically significant

Table 1. Refractive errors in primary school children according to age (n = 362).

Refractive error	Age (years)						Total
	7	8	9	10	11	12	
Myopia	14	31	29	49	37	53	213
Simple myopic astigmatism	3	—	—	—	—	—	3
Compound myopic astigmatism	6	15	13	18	26	27	105
Hypermetropia	2	1	1	2	3	2	11
Simple hypermetropic astigmatism	—	—	—	—	—	—	—
Compound hypermetropic astigmatism	1	1	1	—	2	—	5
Mixed astigmatism	8	6	3	3	5	—	25
Total	34	54	47	72	73	82	362

Table 2. Severity of refractive errors in primary school children (n = 362).

Spherical equivalent (D)	Right eye	Left eye
Mild myopia (-0.25 to -1.75)	210	224
Moderate myopia (-2.00 to -5.00)	86	84
Severe myopia (>-5.00)	18	16
Mild hypermetropia (+0.25 to +1.75)	18	18
Moderate hypermetropia (+2.00 to +5.00)	3	1
Severe hypermetropia (>+5.00)	—	—
Zero spherical equivalent	27	19
Total	362	362

(p = 0.01). Myopia (excluding the children with astigmatism) was seen in 213 children (17.5%), hypermetropia (excluding the children with astigmatism) was seen in 11 children (0.9%), and astigmatism (both myopic and hypermetropic) was seen in 138 children (11.4%).

The majority of children with myopia had mild myopia in either eye (Table 2). Even though the myopia was mild, glasses were prescribed for improvement of vision to 6/6 in these eyes.

Amblyopia was noted in 56 children (4.6%). Amblyopia was noted in one eye in 26 students (2.1%), and in both eyes in 30 students (2.5%); 51 children with amblyopia had refractive error (49 myopia and 2 mixed astigmatism) and 5 had squint (3 convergent squint and 2 divergent squint).

In addition to refractive errors, other ocular abnormalities were found (Table 3). In some children, more than one ocular abnormality was noted in one or both eyes. Ocular melanosis (melanosis of sclera present since birth) was the most common eye disease observed in this study, affecting 3.78% of children. Colour vision defects were seen in 32 children (2.63%), predominantly occurring in boys (31); 1 girl had red-green deficiency. Defective distant vision in one or both eyes was noted in 13 children (10 of 27 children with red-green deficiency and 3 of 5 children with total colour blindness). The visual acuity was 6/9 to 6/18 in 9 children, 6/24 to 6/60 in 3 children, and ≤6/60 in 1 child. Six of the 13 children wore glasses with good improvement of vision (6/6 to 6/9 in both eyes). The remaining 19 children had 6/6 vision in both eyes, suggesting that the majority of children with colour vision defects have good distance vision. None of these children had any positive family history of colour blindness. All the children and their parents were counselled in choosing a future career, which excluded jobs dealing with colour discrimination such as armed forces service, railways, telecommunication, textile industry, and computer graphic applications.

Table 3. Other eye diseases in primary school children (n = 1214).

Disease	Number of children (%)
Epicanthus tarsalis	19 (1.56)
Congenital ptosis	13 (1.07)
Trichiasis	3 (0.24)
Chalazion	2 (0.16)
Papilloma	2 (0.16)
Capillary haemangioma of eyelids	1 (0.08)
Allergic conjunctivitis	2 (0.16)
Conjunctival naevus	1 (0.08)
Ocular melanosis	46 (3.78)
Primary optic atrophy	1 (0.08)
Divergent squint	27 (2.22)
Convergent squint	3 (0.24)
Total colour blindness	5 (0.41)
Red-green deficiency	27 (2.22)

Discussion

The prevalence of ocular morbidity (one or more ocular abnormalities) was found in 41.1% of children in this study; this is higher than the rates of 9.4%,[2] 11.0%,[12] 13.0%,[5] and 24.4%[4] found in other studies. The prevalence of refractive errors (33.3%) in this study is much higher than that in other studies reported from different countries (Table 4). The higher prevalence of ocular morbidity seen in the present study could be due to the higher prevalence of refractive errors when compared with other studies. The prevalence of myopia was observed to be highest in 10-year-old children (68.1%) in this study, which was statistically significant. A similar finding was noted in a Hong Kong study, with the highest incidence of myopia occurring in 11-year-old children amongst a study group aged 7 to 11 years.[14] The prevalence of amblyopia due to refractive errors in this study was 4.2% (51 of 1214 children). This was less than the previously reported figures of 5.0% from China,[8] 9.0% from Nepal,[9] and 6.5% from Chile.[10] The prevalence of colour vision defects of 2.63% in this study is similar to that of 1.7% reported by Reddy[4] and 1.6% reported by Cummings.[6]

The prevalence of 2.5% for squint in this study is higher than the figures reported by Reddy[4] and Wedner et al,[11] but is similar to that reported by Turacli et al,[5] Pokharel et al,[9] and Teoh and Yow,[13] and lower than that reported by Laatikainen and Erkkila,[1] Simpson et al,[2] Chaturvedi and Aggarwal,[7] and Maul et al[10] (Table 5).

Table 4. Prevalence of refractive errors in school children from different countries.

Study	Country	Number of children	Age group (years)	Percent with refractive errors
Laatikainen and Erkkila[1]	Finland	411	7-15	17.5
Simpson et al[2]	New Zealand	984	7	10.5
Jensen and Goldschmidt[3]	Denmark	1216	5-13	11.8
Reddy[4]	India	3675	11-15	17.5
Turacli et al[5]	Turkey	23,810	5-12	11.0
Cummings[6]	UK	1809	8-10	31.0
Chaturvedi and Aggarwal[7]	India	679	5-15	7.4
Zhao et al[8]	China	5884	5-15	12.8
Pokharel et al[9]	Nepal	5067	5-15	2.9
Maul et al[10]	Chile	5303	5-15	15.8
Wedner et al[11]	Tanzania	1386	7-19	1.0
Nepal et al[12]	Nepal	1100	5-16	8.1
Teoh and Yow[13]	Malaysia	650	7	7.1
Present study	Malaysia	1214	7-12	33.3

Table 5. Prevalence of squint in school children from different countries.

Study	Country	Number of children	Percent with squint
Laatikainen and Erkkila[1]	Finland	411	4.6
Simpson et al[2]	New Zealand	988	3.9
Reddy[4]	India	3675	0.3
Turacli et al[5]	Turkey	23,810	2.5
Chaturvedi and Aggarwal[7]	India	679	7.4
Pokharel et al[9]	Nepal	5067	2.1
Maul et al[10]	Chile	5303	9.9
Wedner et al[11]	Tanzania	1386	0.5
Teoh and Yow[13]	Malaysia	650	2.2
Present study	Malaysia	1214	2.5

Vitamin A deficiency has been reported in 3.7% of children from India,[4] and in 0.4% of children from Nepal.[12] However, there were no children with signs of vitamin A deficiency affecting the eyes in this study. This could be due to the good nutritional status of children in Malaysia.

It is ideal to screen preschool children to correct refractive errors at an early age, but this may not be practical at a national level. Therefore, to reduce the cost and encourage participation in the programme, inclusion of vision screening and an eye health check as part of the school health care services is recommended. This can be organised by medical officers working in the Ministry of Health to take place immediately after admission of children into primary school. All children with defective vision and other eye problems should be referred to the nearest ophthalmologist for further assessment and management. It is important to provide health education about common eye diseases to the general public, school teachers, and primary health care workers so that they can detect the eye problems of children and seek advice from ophthalmologists. Refractive errors can be detected early and corrected so that vision can be improved and amblyopia can be reduced. At the same time, eye diseases such as ptosis, squint, and cataract can be detected and treated to prevent ocular morbidity in children.

Acknowledgements

The authors are grateful to The University of Malaya for funding this project by the short-term research grant (Vote F 0126/2001D); the Director of Education, Selangor State, Malaysia for giving permission to examine the school children; and the Headmaster and teachers of Sri Petaling National Primary School who helped in conducting this study.

References

1. Laatikainen L, Erkkila H. Refractive errors and other ocular findings in school children. Acta Ophthalmol. 1980;58:129-36.
2. Simpson A, Kirkland C, Silva PA. Vision and eye problems in seven year olds: a report from the Dunedin Multidisciplinary Health & Development Research Unit, New Zealand. N Z Med J. 1984;97: 445-9.
3. Jensen H, Goldschmidt E. Visual acuity in Danish school children, Odense, Denmark. Acta Ophthalmol. 1986;64:187-91.
4. Reddy SC. Ocular morbidity and color blindness among school children in Kakinada. The Antiseptic. 1987;84:611-6.
5. Turacli ME, Aktan SG, Duruk K. Ophthalmic screening of school children in Ankara, Turkey. Eur J Ophthalmol. 1995;5:181-6.
6. Cummings GE. Vision screening in junior schools. Public Health. 1996; 110:369-72.
7. Chaturvedi S, Aggarwal OP. Pattern and distribution of ocular morbidity in primary school children of rural Delhi. Asia Pac J Pub Health. 1999;11:30-3.
8. Zhao J, Pan X, Sui R, et al. Refractive Error Study in Children: results from Shunyi District, China. Am J Ophthalmol. 2000;129: 427-35.
9. Pokharel GP, Negrel AD, Munoz SR, et al. Refractive Error Study in Children: results from Mechi Zone, Nepal. Am J Ophthalmol. 2000;129: 436-44.
10. Maul E, Barroso S, Munoz SR, et al. Refractive Error Study in Children: results from La Florida, Chile. Am J Ophthalmol. 2000;129:445-54.
11. Wedner SH, Ross DA, Balira R, et al. Prevalence of eye diseases in primary school children in a rural area of Tanzania. Br J Ophthalmol. 2000;84:1291-7.
12. Nepal BP, Koirala S, Adhikary S, et al. Ocular morbidity in school children in Kathmandu. Br J Ophthalmol. 2003;87:531-4.
13. Teoh GH, Yow CS. Prevalence of squint and visual defects in Malaysian primary one school children. Med J Malaysia. 1982;37:336-7.
14. Fan DS, Lam DS, Lam RF, et al. Prevalence, incidence, and progression of myopia of school children in Hong Kong. Invest Ophthalmol Vis Sci. 2004;45:1071-5.

Development of Myopia in Medical School

Hamid Fesharaki, Bahareh Kamali, Mojtaba Karbasi, Mohamad Fasihi
Eye Department, Farabi Hospital, Isfahan, Iran

Aim: *To determine the effect of extensive educational effort involving near work on the progression and emergence of new cases of myopia in medical students.*
Patients and Methods: *This longitudinal study was performed on 262 eyes of 131 randomly selected first-year medical students attending Isfahan University, Isfahan, Iran. Eye examinations, including objective and subjective refraction, were performed at the time of study enrolment and again after 5.5 years. Eyes with ≥0.25 D spherical equivalent myopic error were considered to be myopic. Data from the initial and follow-up examinations were compared and analysed using Student's t test and chi-squared test.*
Results: *The prevalence of myopia increased from 46.5% to 64.0% during the study period. The mean myopic progression was 0.20 D per year. Myopic development occurred in 52.5% of the participants.*
Conclusion: *Medical students are at risk for myopic development in medical school. This should be taken into account for prognostic purposes and in relation to refractive surgery.*

Key words: *Disease progression, Iran, Longitudinal studies, Medical students, Myopia*

Asian J Ophthalmol. 2006;8:199-202

Introduction

The prevalence of myopia has been found to vary from 16% to 70% in different ethnic populations, with a higher prevalence in Asian countries such as Taiwan, Singapore, Japan, Hong Kong, and China. The reasons for these differences are of interest.[1] It has been suggested that parental history and genetic inheritance play a role in the development of myopia.[2-5] In addition, the visual environment has been shown to affect the growth of the eye in animal models of myopia.[6] Myopic progression in juvenile-onset myopia usually stops in the mid-teen years and refractive errors stabilise in 75% of teenagers.[2] Some studies indicate an association between extensive education-related near-work activity and high prevalence of myopia,[7-10] and recent studies provide clear evidence that environmental factors and ethnic differences can affect myopic progression.[11-13]

The effect of working habits on myopic progression in different populations and ethnic groups needs further clarification. There have been few previous longitudinal studies of myopic progression in young adults undertaking higher education.[14-16] This study was performed at the Eye Department, Farabi Hospital, Isfahan University, Isfahan, Iran, from 1998 to 2003 to determine the incidence and progression of myopia in young adult medical students attending Isfahan University.

Correspondence: Dr Hamid Fesharaki, Associate Professor of Ophthalmology, Isfahan University of Medical Sciences, Isfahan, Iran.
Tel: (98 311) 624 3664; Fax: (98 311) 627 2713;
E-mail: h_fesharaki@med.mui.ac.ir

Patients and Methods

From 300 first-year medical students who enrolled at Isfahan University in 1998, 150 were randomly selected to participate in this longitudinal study. The purpose of the study was discussed with each participant and personal and ocular history was collected, including the age of onset of myopia and any history of eye surgery or trauma. The study process was consented to by all of the selected participants and ethical approval was obtained. Participants were requested to stop wearing soft contact lenses for 7 days prior to each eye examination.

Measurement of refractive error included cycloplegic autorefractometry (Topcon RMA8000; Topcon Corporation, Tokyo, Japan) and retinoscopy (Heine Optotechnik, Herrsching, Germany) and subjective refraction using a Snellen chart and trial lenses to check the uncorrected and best corrected visual acuity of each eye. Tropicamide 1% was used for cycloplegic refraction. Two of the authors performed the eye examinations. The entire eye examination procedure, including measurement of refractive error, was repeated 5.5 years later, when the students were in the sixth year of their medical course. Eyes with a spherical equivalent myopic error of ≥0.25 D were considered to be myopic and progression of myopia was defined as a myopic shift of ≥0.37 D between the first and second examination. Of the 150 participants enrolled, 131 (262 eyes) remained in the study. Seventeen were excluded due to loss to follow-up and 2 because of refractive surgery and irregular astigmatism. Data collected at the initial

Table 1. Myopic status of medical students at time of enrolment.

Myopic status (D)	No. of eyes (%)
None	140 (53.5)
Low (0.25 to 3.00)	88 (33.5)
Moderate (>3.00 to 6.00)	30 (11.5)
High (>6.00 to 8.00)	4 (1.5)
Pathologic myopia	0
Total	262 (100)

and follow-up examination were compared and analysed using Student's *t* test and chi-squared test.

Results

The ages of the 84 male and 47 female students who completed the study ranged from 17 to 25 years (mean, 19.8 years; SD, 1.2 years) at the start of the study. The prevalence of myopia increased significantly from 46.5% to 64.0% ($p < 0.001$); the mean initial myopic error was 1.97 D (SD, 1.86 D; range, 0.25 to 7.25 D) and the mean final myopic error was 2.98 D (SD, 2.13 D; range, 0.25 to 9.00 D). Of the 122 eyes that were myopic at the beginning of the study, 65% had juvenile-onset myopia (onset at 7 to 16 years) and 35% became myopic after the age of 16 years. The myopic status of the students at the beginning of the study is given in Table 1.

A high proportion of eyes that were myopic at the beginning of the study (81%) showed myopic progression during the study period (Table 2), with an average myopic shift of 1.13 D (SD, 0.81 D; range, 0.37 to 4.38 D). In addition, 33% of eyes that were not myopic at the beginning of the study became myopic during the study period (adult-onset myopia) [Table 2]. For the latter eyes, the mean myopic shift was 1.05 D (SD, 0.78 D; range, 0.25 to 4.25 D) and most showed a myopic shift of 0.37 to 4.25 D (Table 2). Overall, 52.5% of participants showed myopic progression or onset of myopia while attending medical school. The diopter extent of myopic shift in relation to initial myopic error is shown in Table 3. Statistical significance was observed only between groups 1 and 3 in the left eye ($p = 0.005$). No statistically significant difference was found between men and women with respect to myopic progression or the incidence of adult-onset myopia. No statistically significant difference was found overall between the right and the left eyes in terms of the proportion of eyes that showed myopic development and the extent of myopic shift.

Discussion

Most students who were myopic when enrolled in medical school had late juvenile and early adult-onset myopia. The age of onset was >14 years in 81% of participants, most of whom experienced myopic progression while attending medical school. Approximately one-third of the students who were not myopic at the time of enrolment became myopic while at medical school. The results of the present study differ from those reported for juvenile-onset myopia in the general population, which indicate that juvenile-onset myopia usually stops in the mid-teen years and that stabilisation of refractive errors occurs in 75% of teenagers.[2] However, the results of the present study are comparable to the results of 3 previous longitudinal studies of similar groups experiencing high educational demands (Table 4),[14-16] confirming the influence of near-work activity on adult myopic development. In comparison with the present study, Lin et al, in a study of medical students in Taiwan, found a higher prevalence of myopia (92.8%) and a higher

Table 2. Frequency distribution of myopic progression in young adults during attendance at medical school.

Initial status	Right eye		Left eye	
	Myopic shift (D)	No. of eyes (%)*	Myopic shift (D)	No. of eyes (%)*
Myopia, ≥0.25 D	>2.00 to 4.38	7 (2.67)	>2.00 to 3.50	6 (2.29)
	>1.50 to 2.00	6 (2.29)	>1.50 to 2.00	10 (3.81)
	>1.00 to 1.50	18 (6.87)	>1.00 to 1.50	11 (4.19)
	>0.75 to 1.00	7 (2.67)	>0.75 to 1.00	8 (3.05)
	>0.50 to 0.75	7 (2.67)	>0.50 to 0.75	6 (2.29)
	0.37 to 0.50	7 (2.67)	0.37 to 0.50	6 (2.29)
	0.25	5 (1.90)	0.25	6 (2.29)
	0.00	5 (1.90)	0.00	7 (2.67)
Total	—	62 (23.64)	—	60 (22.88)
No myopia, <0.25 D	>2.00 to 4.25	4 (1.52)	>2.00 to 2.25	2 (0.76)
	>1.50 to 2.00	1 (0.38)	>1.50 to 2.00	4 (1.52)
	>1.00 to 1.50	2 (0.76)	>1.00 to 1.50	3 (1.14)
	>0.75 to 1.00	4 (1.52)	>0.75 to 1.00	2 (0.76)
	>0.50 to 0.75	3 (1.14)	>0.50 to 0.75	3 (1.14)
	0.37 to 0.50	5 (1.90)	0.37 to 0.50	6 (2.29)
	0.25	4 (1.52)	0.25	3 (1.14)
	0.00	46 (17.55)	0.00	48 (18.32)
Total	—	69 (26.33)	—	71 (27.09)

* Expressed as a percentage of total number of left and right eyes.

Table 3. Comparison of myopic progression in students grouped according to myopic error at time of enrolment.

Group	Initial myopic error (D)	No. of eyes (right)	Mean (D)	SD	No. of eyes (left)	Mean (D)	SD
1	≤0.50	23	0.9783	0.7976	13	0.5769*	0.5138
2	0.75-2.75	21	1.2329	1.0339	29	1.2078	0.9511
3	≥3.00	18	1.2783	0.7611	18	1.2708*	0.7367
Total	≥0.25	62	1.1317	0.8716	60	1.0900	0.8451

* $p < 0.005$.

Table 4. Comparison of results of longitudinal studies of adult myopic development.

Authors	Myopic progression (D/year)	Incidence of adult-onset myopia (%)	Study population	Age (years) Mean (SD)	Follow-up (years)
Kinge et al[14]	0.17	59	196 engineering students	20.6 (1.1)	3.0
Lin et al[15]	0.12	42	345 medical students	19.8 (1.2)	5.0
McBrien and Adams[16]	0.38	39	166 clinical microscopists	Range: 21-63	2.0
Present study	0.20	31	131 medical students	19.8 (1.2)	5.5

initial mean myopic error (4.26 D; SD, 2.66 D) but a lower mean rate of myopic progression (0.12 D per year).[15]

The effect of confounding factors on the results of the present study is likely to be low because no clear effect of heredity has been found for juvenile- and adult-onset myopia and the study population was relatively homogenous with respect to age, race, education, intelligence, and diet. Most of the myopic students had low to moderate myopia, there were only 2 students with high myopia, and none showed pathologic fundus changes. Astigmatism was present in 40% of eyes (mean, 0.60 D; SD, 0.60 D; range, 0.50 to 3.25 D) and 75% of occurrences were of the with-the-rule type. No other genetic predisposition such as glaucoma, ocular hypertension, cataract, lens subluxation, retinal detachment, esodeviation, or history of premature birth was found in the study population. The results of the study are probably not significantly affected by the loss of 12.5% of participants to follow-up because this group did not differ significantly from the rest of the study population in terms of initial refractive error.

The rate of anisomyopia of ≥0.50 D was 8.4% among students with myopia at the start of the study; this rate was found to be 16.8% at the end of the study ($p = 0.001$). A tendency for further progression in right eyes was observed in these asymmetrically progressed myopic cases. Comparable studies of groups of young adults of similar ethnicity who were engaged in minimal near work activity would help to define further the effect of near work on myopic development. Stepwise longitudinal studies would be useful for determining the extent of myopic development due to near work at different ages.

The findings of this study confirm the association between intense near work activity and the development of myopia. They also indicate that about 50% of students in medical school are at risk for myopic onset or progression. This should be taken into account for prognostic purposes and in relation to refractive surgery.

Acknowledgements

The authors thank participating medical students of the Eye Department, Alzahra Hospital, Isfahan University, Isfahan, Iran, and Mr Ahmad Azzizadeh for statistical guidance. This study was supported by Isfahan University of Medical Sciences (Research Planning Approval Number 77133).

References

1. McCarty CA, Taylor HR. Myopia and vision 2020. Am J Ophthalmol. 2000;129:525-7.
2. American Academy of Ophthalmology. Optics and refraction. Basic and clinical science course, Section 3. San Francisco: American Academy of Ophthalmology; 2004-2005. p. 122-3.
3. Zadnik K, Satariano WA, Mutti DO, et al. The effect of parental history of myopia on children's eye size. JAMA. 1994;271:1323-7.
4. Saw SM, Carkeet A, Chia KS, et al. Component dependent risk factors for ocular parameters in Singapore Chinese children. Ophthalmology. 2002;109:2065-71.
5. Liang CL, Yen E, Su TY, et al. Impact of family history of high myopia on level and onset of myopia. Invest Ophthalmol Vis Sci. 2004;45:3446-52.
6. Zhu X, Winawer JA, Wallman J. Potency of myopic defocus in spectacle lens compensation. Invest Ophthalmol Vis Sci. 2003;44: 2818-27.
7. Ting PW, Lam CS, Edwards MH, Schmid KL. Prevalence of myopia in a group of Hong Kong microscopists. Optom Vis Sci. 2004;81: 88-93.
8. Saw SM, Hong RZ, Zhang MZ, et al. Near-work activity and myopia in rural and urban schoolchildren in China. J Pediatric Ophthalmol Strabismus. 2001;38:149-55.
9. Saw SM, Chua WH, Hong CY, et al. Nearwork in early-onset myopia. Invest Ophthalmol Vis Sci. 2002;43:332-9.
10. Midelfart A, Aamo B, Sjohaug KA, et al. Myopia among medical students in Norway. Acta Ophthalmol (Copenh). 1992;70:317-22.
11. Hyman L, Gwiazda J, Hussein M, et al. Relationship of age, sex, and ethnicity with myopia progression and axial elongation in the correction of myopia evaluation trial. Arch Ophthalmol. 2005;123: 977-87.
12. Wu HM, Seet B, Yap EP, et al. Does education explain ethnic differences in myopic prevalence? A population-based study of young adult males in Singapore. Optom Vis Sci. 2001;78: 234-9.
13. Fan DS, Lam DS, Lam RF, et al. Prevalence, incidence and

progression of myopia of school children in Hong Kong. Invest Ophthalmol Vis Sci. 2004;45:1071-5.
14. Kinge B, Midelfart A, Jacobsen G, et al. The influence of near-work on development of myopia among university students. A three-year longitudinal study among engineering students in Norway. Acta Ophthalmol Scand. 2000;78:26-9.
15. Lin LL, Shih YF, Lee YC, et al. Change in ocular refraction and its components among medical students — a 5-year longitudinal study. Optom Vis Sci. 1996;73:495-8.
16. McBrien MA, Adams DW. A longitudinal investigation of adult onset and adult progression of myopia in an occupational group. Invest Ophthalmol Vis Sci. 1997;38:321-33.

Bilateral Visual Loss Due to Antihypertensive Agent-induced Retinal Hypoperfusion Injury

Arijit Mitra, George M Saleh, Eleni Tsetsemeglou, Paul Hart, Peter Fison
Epsom and St Helier University Hospitals NHS Trust, London, UK

This report is of a patient with congestive cardiac failure and uncontrolled hypertension who presented with sequential sudden loss of vision after administration of a single dose of angiotensin II receptor blocker in combination with a diuretic.

Key words: *Heart failure, congestive, Hypertension, Ischaemia, Retina*

Asian J Ophthalmol. 2006;8:203-4

Introduction

Sudden reduction of blood pressure after administration of multiple hypertensive medications can result in hypoperfusion and ischaemic injury to susceptible tissues. This report is of a patient with sequential sudden loss of vision due to bilateral global arterial hypoperfusion after a single dose of angiotensin II receptor antagonist was added to diuretic therapy.

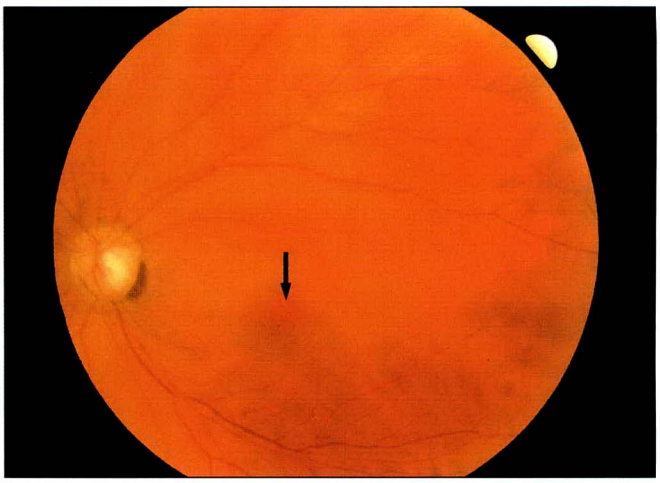
Case Report

A 74-year-old man was treated by his physician for congestive cardiac failure and uncontrolled hypertension in 2004. He had a history of diabetes mellitus, transient ischaemic attacks, bilateral carotid artery stenosis, and advanced primary open angle glaucoma with peripheral visual field loss in both eyes. The physician prescribed furosemide 80 mg and candesartan 8 mg per day to replace amlodipine 5 mg that he had previously been taking.

After one dose of the angiotensin II receptor antagonist, the patient noticed 'waviness' of vision in his left eye; by the next morning, the visual acuity in his left eye was reduced to hand movements. Fundus examination showed oedema of the macular area with a cherry red spot, suggesting ischaemic injury of the retina (Figure 1). Temporal arteritis and retinal emboli were excluded.

Candesartan was discontinued and amlodipine was restarted. Twenty four-hour blood pressure monitoring 5 days later revealed a significantly elevated blood pressure, with an average reading of 170/95. To treat the hypertension, amlodipine was substituted with valsartan 80 mg per day. Following a single dose of valsartan, the patient noticed rapid loss of vision in his right eye to hand movements. Fundus examination once more revealed central retinal artery occlusion. Fundus fluorescein angiogram performed within 24 hours of the second episode showed normal perfusion in the left eye (Figure 2) but global hypoperfusion in the right eye, particularly involving the cilioretinal artery region and superotemporal branch of the central retinal artery (Figures 3 and 4). There was no sign of thromboembolic occlusion of the cilioretinal or central retinal artery. Magnetic resonance imaging of the brain showed no evidence of cerebral watershed ischaemia. A few weeks after the acute episodes his visual acuity remained hand movements in both eyes and he was experiencing visual hallucinations of the Charles Bonnet syndrome type.

Figure 1. Fundus examination showed oedema of the macular area with a cherry red spot, suggesting ischaemic injury of the retina of left (first) eye. The arrow indicates the cherry red spot, with surrounding oedema.



Discussion

Vascular insufficiency is the most common cause of sudden painless unilateral loss of vision. Retinal ischaemia can result either from

Correspondence: Dr Arijit Mitra, Wolverhampton Eye Infirmary, Compton Road, Wolverhampton, West Midlands, WV3 9QR, UK.
Tel: (44 79) 0595 6498; Fax: (44 19) 0271 1985;
E-mail: drarijitmitra@yahoo.com

Figure 2. Fundus fluorescein angiogram of the left eye showing normal perfusion 8 days after the first episode.

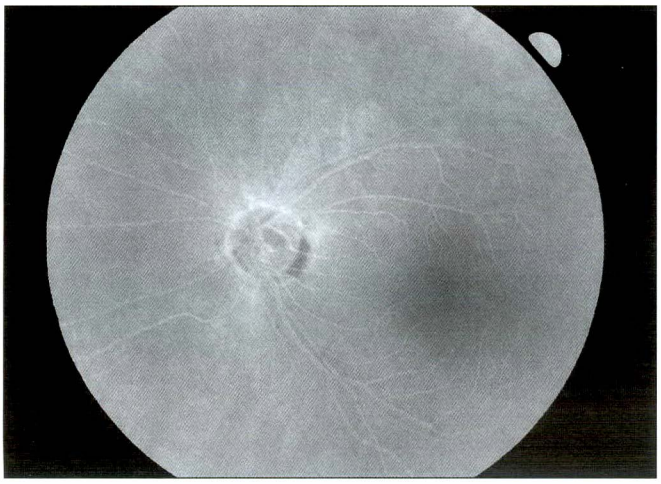
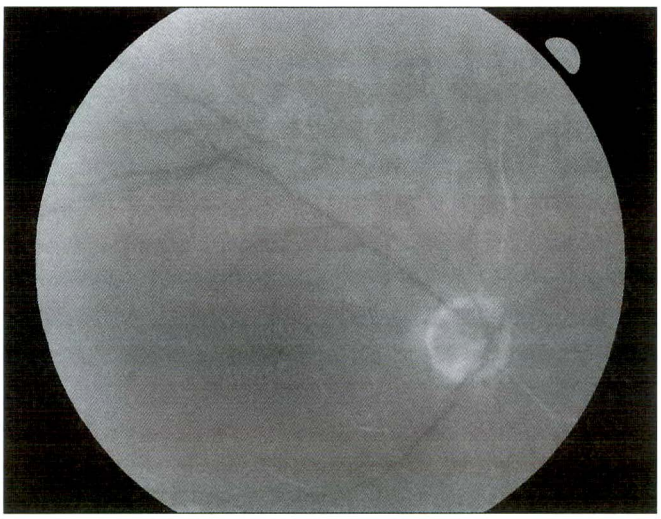


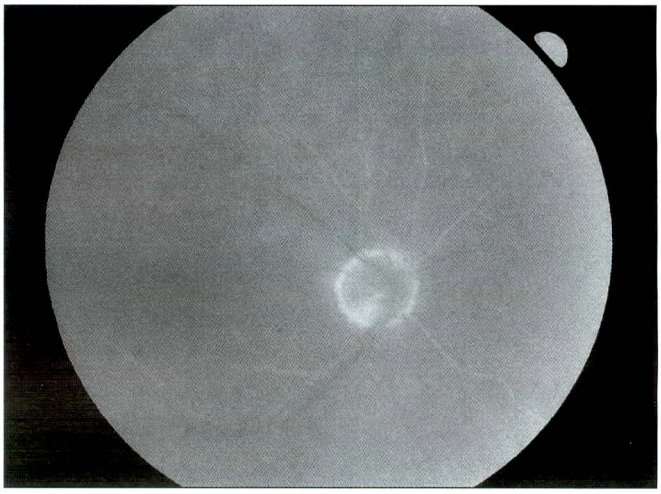
Figure 3. Fundus fluorescein angiogram of the right eye (3 minutes 53 seconds after injection of dye) showing global hypoperfusion, particularly of the cilioretinal artery region and superotemporal branch of the central retinal artery.



direct occlusion of the retinal vasculature by an embolism or in situ thrombosis, or from decreased blood flow secondary to systemic or local hypoperfusion.[1] Chronic hypertension can induce hypertrophy of the tunica muscularis of the arterial wall causing narrowing of the lumen and increase in peripheral vascular resistance.[1,2] In such patients, sudden lowering of blood pressure can compromise cerebral and retinal perfusion, leading to ischaemic damage to susceptible tissues.[1]

The reported patient was already taking diuretics for congestive cardiac failure and angiotensin II inhibitors were added for

Figure 4. Fundus fluorescein angiogram of the right eye (6 minutes 11 seconds after injection of dye) still showing global hypoperfusion, particularly of the cilioretinal artery region and superotemporal branch of the central retinal artery.



persistently high blood pressure. First-dose hypotension (maximum 24-hour fall in mean arterial pressure greater than 10% from baseline) is common with angiotensin II receptor antagonists, especially when combined with diuretics. The synergistic interaction of the 2 drugs may have induced hypoperfusion of the retinal arteries,[3] which may have led to infarction in the presence of an already compromised retinal circulation (due to longstanding glaucoma and carotid artery stenosis). The left eye, which was involved first, regained good perfusion (as would be expected from a hypoperfusion injury) but the visual acuity failed to improve due to permanent ischaemic damage of the retinal tissues.

This report highlights that care should be taken when normalising high blood pressure, especially in a patient with chronic hypertension, in the presence of other vasculopathies. In the absence of manifestations of hypertensive encephalopathy (severe headache, vomiting, visual field disturbances, transient paralysis, stupor, or coma) or clinical findings arising from changes resulting from elevated blood pressure (papilloedema, retinal haemorrhages, and exudates), a more conservative approach should be taken.[1]

References

1. Mainville N, Connolly WE. Acute visual loss after initiation of antihypertensive therapy: case report. CMAJ. 2003;169:313-5.
2. Steigerwalt RD Jr, Laurora G, Incandela L, et al. Ocular and orbital blood flow in cigarette smokers. Retina. 1998;18:539-45.
3. Bindschedler M, Degen P, Flesch G, et al. Pharmacokinetic and pharmacodynamic interaction of single oral doses of valsartan and furosemide. Eur J Clin Pharmacol. 1997;52:371-8.

Oral Radioactive Iodine for the Treatment of Orbital Metastasis of Carcinoma of the Thyroid

Juliette O Howden,[1] Anne Maree Kean,[2] Raf Ghabrial[1,3]
[1]Save Sight Institute, University of Sydney, [2]Department of Endocrinology, Prince Alfred Hospital, and [3]Sydney Oculoplastic Surgery, Sydney, Australia

This report describes a case of orbital metastasis as the primary manifestation of thyroid carcinoma. A 70-year-old woman presented with a 1-month history of left ptosis and painful swelling in the periorbital region associated with reduced vision. Orbital biopsy revealed metastatic follicular carcinoma of the thyroid. She underwent a total thyroidectomy and has been treated with 4 doses of oral radioactive iodine. Following 2 doses of radioactive iodine, the left orbital mass began to regress and the ptosis was clinically reduced.

Key words: *Iodine radioisotopes, Orbital neoplasms, Thyroid neoplasms*

Asian J Ophthalmol. 2006;8:205-7

Introduction

Metastatic carcinomas comprise 6% of all orbital tumours.[1] Breast and lung cancers are the most common sources but thyroid cancer orbital metastases have been reported.[2-5] Ninety four percent of thyroid carcinomas are of the differentiated type, which is iodine-avid.[6] Radioactive iodine is used to treat the orbital metastasis in these patients, unless the mass is very large, causing optic nerve compression, or there is intractable orbital pain. Iodine^{131} (^{131}I) undergoes decay, releasing high-energy electrons that induce highly localised radiation cytotoxicity.[6] This report is of a patient who presented with an occult orbital metastatic thyroid carcinoma. Treatment with radioactive iodine has gradually led to tumour regression and improvement of symptoms.

Case Report

A 70-year-old woman presented with a 1-month history of left ptosis and painful swelling in the periorbital region associated with reduced vision in 2004. Her past ocular history included recurrent anterior uveitis between the ages of 20 and 50 years. She had no history of systemic malignancy. At examination, the visual acuity in her left eye was 6/9 with a pinhole. There was a fullness in the left superotemporal region with hypoglobus and resistance to retropulsion. There was mild proptosis and some limitation of up gaze. The temporal fossa was tender to palpation. Fundoscopy was within normal limits. The right eye was asymptomatic and normal on clinical examination. Computed tomography (CT) of the orbits revealed a swelling in the left lacrimal fossa and associated bony destruction of the lateral orbital wall and roof (Figure 1). Orbital biopsy was undertaken and the histopathological appearance was consistent with metastatic follicular carcinoma of the thyroid. The thyroglobulin was 3072 μg/L (normal range, 3-42 μg/L).

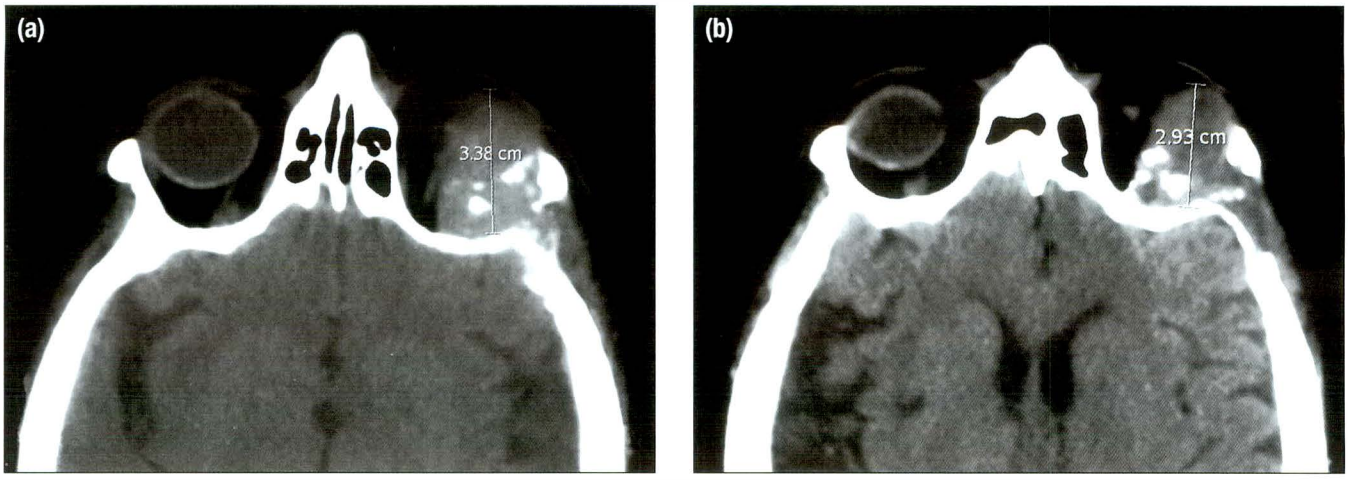
Clinically there was a bilateral multinodular goitre that was asymptomatic. The regional lymph nodes were not involved. She underwent a total thyroidectomy. The histopathology showed a follicular variant of papillary carcinoma with capsular invasion but no vascular invasion. A bone scan at this time showed uptake in the left supraorbital region but no other evidence of metastasis.

She has since been treated with 4 doses of oral ^{131}I at 3-monthly intervals and the first post-treatment ^{131}I scan is shown in Figure 2. As the thyroglobulin became significantly elevated when thyroxine was withdrawn on the first 2 occasions, and the mass increased in size and became more clinically evident despite corticosteroid cover, the third and fourth doses of ^{131}I were given while the patient was receiving recombinant thyroid-stimulating hormone and thyroxine. Lithium was administered concurrently to increase and prolong ^{131}I uptake.

Just prior to the third ^{131}I dose, she had a flare of anterior uveitis in the left eye, which settled with appropriate treatment. Clinically, the left orbital mass has been regressing slowly since the second dose of ^{131}I (Figure 1). Concurrent radiological evidence showed the mass decreased in size and has remained stable. After 3 doses of ^{131}I the left visual acuity was 6/24 with a pinhole, intra-ocular pressure of 23 mm Hg, the eye and temporal region were comfortable, extraocular movements were improved, and proptosis

Correspondence: Dr Raf Ghabrial, Sydney Oculoplastic Surgery, Level 7, 229 Macquarie St, Sydney, NSW 2000, Australia.
Tel: (61 2) 9222 9901; Fax: (61 2) 9222 9902;
E-mail: rafg@bigpond.com

Figure 1. Axial computed tomography scan of the orbits (a) pretreatment, showing a destructive lesion centred on the left sphenoid triangle with intraocular and intracranial extension and a maximum anteroposterior dimension of 3.38 cm — there is mass effect on the orbital structures; and (b) 5 months after 2 doses of iodine^{131}, when there was a reduction in the maximum anteroposterior dimension of the soft tissue component of the left orbital lesion to 2.93 cm.

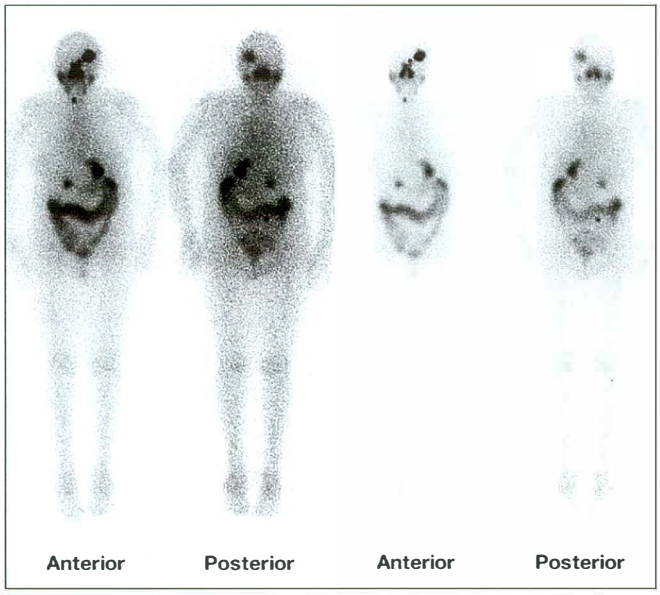


was reduced. The fourth post-treatment ^{131}I scan shows persistent iodine-avid disease in the left retro-orbital region.

Discussion

Thyroid cancer accounts for only 1% of all new malignancies.[6] Ninety four percent of tumours are differentiated thyroid carcinomas, either of the more common papillary type or the follicular type. Medullary carcinoma, accounting for 5% of cases, is derived from the neuroendocrine parafollicular cells that secrete calcitonin. Anaplastic carcinoma is the most aggressive of the thyroid carcinomas and accounts for only 1%.[6] Differentiated thyroid carcinoma affects women more often than men, with a ratio of 2:1.[6]

Figure 2. Whole body iodine^{131} scan following the first iodine^{131} treatment dose. There is significant iodine^{131}-avid tissue in the left orbital region, left nasolacrimal region, and right thyroid bed.



Thyroid carcinoma of all histological types can metastasise to the orbit. A previously excised goitre reported as benign may have contained a malignancy as these multifocal lesions require careful sectioning. Orbital metastasis is rare and is usually a presenting rather than a later manifestation of thyroid carcinoma.[1] In approximately 30% of all orbital metastases, the orbital tumour is the presenting sign of systemic cancer.[7]

Orbital metastatic carcinomas are common, comprising 6% of all orbital tumours;[1] the most common sources are breast and lung cancers.[1,7] Thyroid cancer comprised only 4 of 83 cases in the series by Henderson.[1] Thyroid carcinoma orbital metastases have been reported recently.[2-5] Less commonly, thyroid carcinoma metastasises to the globe;[8-10] this is the reverse of the usual predilection of most metastatic carcinomas for the globe versus the orbit, in a ratio of 7:1 in one series.[10]

Treatment of orbital metastatic thyroid cancer depends on the histological type. Most differentiated thyroid carcinomas, papillary or follicular, are iodine-avid;[6] in this situation, radioactive iodine is the appropriate first-line treatment, with or without prior excision. It can take some time for the mass to regress after radioactive iodine treatment and may take 1 to 2 years for the thyroglobulin to normalize,[6] even in the context of a negative follow-up radioiodine scan. In one patient, exophthalmos resolved but there was still iodine-avid disease in the orbit 5 years after treatment.[5] External beam radiotherapy is indicated for unresectable, residual, or metastatic differentiated thyroid carcinomas that do not concentrate ^{131}I.[11] Surgery is the treatment for the rarer medullary thyroid cancer, and the treatment of anaplastic thyroid cancer is generally palliative in the form of radiotherapy with or without chemotherapy.[11]

It is important to consider metastatic disease, especially in older patients presenting with unilateral proptosis. In the rare case of

orbital metastatic thyroid carcinoma, radioactive iodine is usually the treatment of choice.

References

1. Henderson JW. Orbital tumors. 3rd ed. New York: Raven Press; 1994. p. 361-76.
2. Basu S, Nair N, Aravind N. Unilateral proptosis with thyrotoxicosis resulting from solitary retroorbital soft tissue metastasis from follicular carcinoma thyroid. Clin Nucl Med. 2001;26:136-8.
3. Daumerie C, De Potter P, Godfraind C, et al. Orbital metastasis as primary manifestation of thyroid carcinoma. Thyroid. 2000;10:189-92.
4. Bernstein-Lipschitz L, Lahav M, Chen V, et al. Metastatic thyroid carcinoma masquerading as lacrimal gland tumor. Graefes Arch Clin Exp Ophthalmol. 1990;228:112-5.
5. Umbreit JN, Lyss AP. Metastatic thyroid adenocarcinoma presenting as exophthalmos. Mo Med. 1988;85:792-4.
6. Sherman SI. Thyroid carcinoma. Lancet. 2003;361:501-11.
7. Goldberg RA, Rootman J, Cline RA. Tumors metastatic to the orbit: a changing picture. Surv Ophthalmol. 1990;35:1-24.
8. Slamovits TL, Mondzelewski JP, Kennerdell JS. Thyroid carcinoma metastatic to the globe. Br J Ophthalmol. 1979;63:169-72.
9. Ahmadi MA, Nicholes D, Esmaeli B. Late choroidal metastasis secondary to papillary thyroid carcinoma. Am J Ophthalmol. 2001; 132:796-8.
10. Ferry AP, Font RL. Carcinoma metastatic to the eye and orbit. I. A clinicopathologic study of 227 cases. Arch Ophthalmol. 1974;92: 276-86.
11. Guidelines for the management of thyroid cancer in adults. British Thyroid Association. London: Royal College of Physicians; 2002. p. 13, 21.

Bilateral Panuveitis in Neurosarcoidosis with Spinal Cord Involvement

Nurettin Akyol,[1] Adem Türk,[1] Zekeriya Alioglu,[2] Engin Sözen,[3] Halil Ibrahim Imamoglu[1]
[1]Department of Ophthalmology, [2]Department of Neurology, Karadeniz Technical University, Faculty of Medicine, Trabzon, and [3]Department of Ophthalmology, Rize State Hospital, Rize, Turkey

Sarcoidosis is a granulomatous disease of multiple organ involvement without a known cause. Central nervous system involvement is seen in nearly 10% of all patients. In neurosarcoidosis, basal leptomeninges and cranial nerves are the most common sites of involvement, while spinal cord involvement is rare. Ocular involvement is mostly bilateral and insidious, and is an important clinical indicator for diagnosis. This report is of a 61-year-old man with bilateral uveitis who had an established diagnosis of sarcoidosis and who later developed loss of strength in his feet. The patient was diagnosed with neurosarcoidosis with spinal cord involvement and findings of bilateral panuveitis were noted. Treatment consisted of topical and systemic administration of corticosteroids.

Key words: *Glucocorticoids, Granulomatous disease, chronic, Sarcoidosis, Uveitis*

Asian J Ophthalmol. 2006;8:208-10

Introduction

Sarcoidosis is a systemic disease characterised by granulomas of unknown cause in several organs. Neurological involvement is seen in 10% of patients. Any part of the nervous system may be involved, although the spinal cord is a rare site of involvement. Ocular involvement often plays an important role in establishing the diagnosis.[1,2] This report describes a patient with neuroscoidosis with spinal cord involvement and bilateral panuveitis.

Case Report

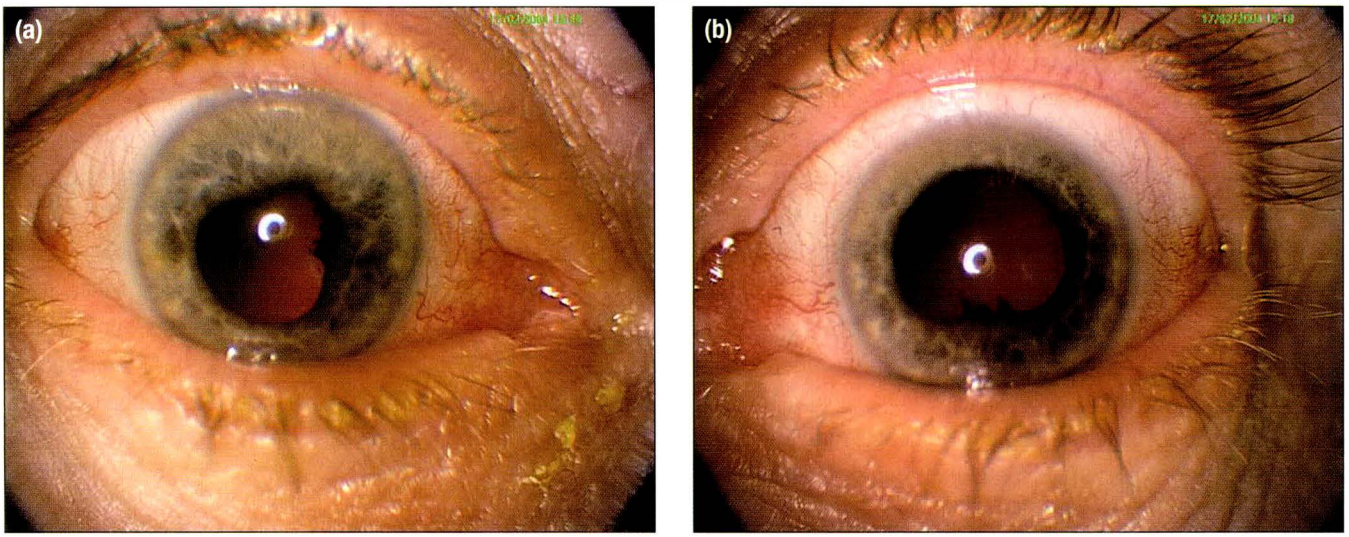
A 61-year-old man was admitted to the Department of Chest Diseases, Karadeniz Technical University Faculty of Medicine, Trabzon, Turkey, in 2003 after presenting with fatigue. Chest examination revealed hilar adenopathy and parenchymal involvement. Transbronchial fine needle biopsy revealed granulomatous inflammation, as well as high levels of serum angiotensin converting enzyme (ACE), hyperglobulinaemia, and high levels of calcium in the urine suggesting a diagnosis of grade II sarcoidosis.

Six months after diagnosis, the patient was referred to the Neurology Outpatient Clinic with loss of strength in his feet. Apart from sarcoidosis, he did not have any comorbidity. Findings at physical examination were normal. At neurological examination, there was level 4/5 loss of strength, together with impairment of joint position and vibration sensations in both lower extremities. Lower extremity reflexes were vivid and Babinsky test was bilaterally positive. Neurological examination correlated with spinal cord involvement of sarcoidosis. Cerebrospinal fluid (CSF) examination showed high levels of protein and immunoglobulin G, although the ACE level was not significantly elevated. Other CSF findings were normal and tests for other diseases that might involve neurological systems were also negative.

Ophthalmological examination was performed and visual acuity was recorded as 20/20 for the right eye and 20/23 for the left eye. Ocular movements and intraocular pressure were normal. Biomicroscopy demonstrated mutton fat keratic precipitates in both eyes, cellular reaction (+2) in the anterior chamber, and posterior synechia and cells in the vitreous (Figure 1). In the lower peripheral parts, the vitreous of both eyes, there was a snow ball-like appearance. Fundoscopic examination revealed foci correlating with retinitis in the lower peripheral parts of both retina. Cranial magnetic resonance imaging (MRI) revealed hyperdense lesions in periventricular white matter in T2-weighed series. Thoracolumbar MRI identified regions with pathological signal intensities at levels T7 and T12-L1 of the spinal cord that correlated with neurosarcoidosis (Figure 2). After examination

Correspondence: Dr Adem Türk, Department of Ophthalmology, Karadeniz Technical University, Faculty of Medicine, 61080 Trabzon, Turkey.
Tel: (90 462) 377 5471; Fax: (90 462) 325 2270;
E-mail: doktorademturk@yahoo.com

Figure 1. Panuveitis in (a) the left eye; and (b) the right eye.



for the differential diagnoses, a diagnosis of neuro-ophthalmic sarcoidosis was established.

Topical high-dose dexamethasone and intravenous methylprednisolone 1000 mg/day was administered for 3 days followed by 500 mg/day for 2 days, after which oral prednisolone 1 mg/kg/day was given. After 2 weeks, there was improvement of ocular findings. However, the neurological findings persisted. The patient was treated with topical and oral corticosteroids and continued to be followed up.

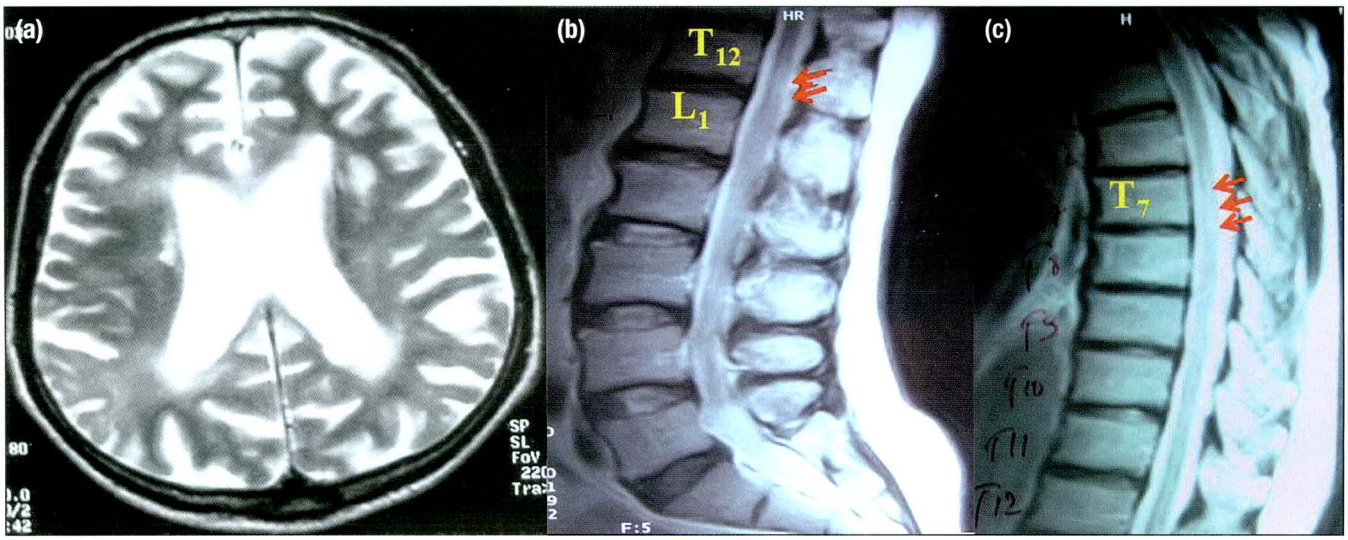
Discussion

Sarcoidosis is a systemic disease that may involve any of the body's organs. The clinical presentation of the disease varies between patients and depends on the disease activity and the involved organ. The primary symptoms include ocular complaints, skin lesions, cough, general fatigue, and fever. Nearly 25% of the patients are asymptomatic.[3,4]

Ocular involvement is common in sarcoidosis and is helpful for establishing the diagnosis in most patients. The most commonly observed ocular findings include uveitis, dry eye, intravitreal opacities, retinal perivasculitis, and patch type chorioretinal exudates.[3-5] The risk for neurosarcoidosis increases in patients with involvement of the posterior chamber of the eye.[6] The involvement of the posterior segment in this patient supports this relationship.

Uveitis due to sarcoidosis is usually bilateral with similar findings and similar clinical courses in both eyes. The mild inflammatory

Figure 2. T2-weighted magnetic resonance imaging of (a) the cranium showing periventricular hyperintense lesions; (b) the thoracolumbar region showing pathological signal intensities correlating with sarcoidosis at levels T12-L1; and (c) the thoracic region showing pathological signal intensities correlating with sarcoidosis at level T7.



response that is observed in uveitis begins insidiously without causing symptoms, yet it may have a progressive course. Visual acuity decreases in the presence of significant inflammation.

Tests such as erythrocyte sedimentation rate, Mantoux skin test, serum ACE level, chest X-ray for bilateral hilar lymphadenopathy, and lymph node biopsies may help to make the diagnosis, despite their lack of specificity.[7] The diagnosis of sarcoidosis in this patient was based on the identification of several markers, including biopsy. The differential diagnosis was based on CSF findings and blood tests.

Neurological involvement is not common in patients with sarcoidosis. Despite the fact that the disease can involve any part of the nervous system, spinal cord involvement is rare. Due to the nonspecific clinical presentation and neuroradiological features of the disease, it may be difficult to make a diagnosis, especially for asymptomatic patients. The diagnosis for most patients with neurosarcoidosis is supported by neurological impairment and biopsy findings. Occasionally, neurological signs may be observed without other systemic finding.[1,2,8]

MRI is a sensitive technique for diagnosing the intracranial abnormalities that stem from sarcoidosis. Despite the fact that lumbar puncture is helpful in differentially diagnosing other neurological disturbances, primarily those of infectious origin, CSF findings are not specific to sarcoidosis.[8] This patient had previously been diagnosed with sarcoidosis and the diagnosis of neurosarcoidosis was based on the findings from MRI and CSF.

Most patients with neurosarcoidosis who have cranial neuropathy show spontaneous improvement; it is usually sufficient to follow these patients without treatment. However, for patients with severe symptoms or for those with spinal neurosarcoidosis, it may be necessary to administer steroids. For patients with severe involvement or frequent recurrences, immunosuppressive agents or radiotherapy should be considered.[8,9]

The treatment for ocular involvement of sarcoidosis is generally topical corticosteroids. Systemic steroids are rarely required.[10] This patient responded to steroid treatment, although there was no improvement of neurological findings.

In conclusion, sarcoidosis is a disease of unknown cause; depending on the organ involved, it may run a malignant course if the diagnosis and treatment are delayed. For patients with systemic involvement, ocular findings provide guidance for the diagnosis. Patients who are suspected to have sarcoidosis should also be evaluated for ocular findings.

References

1. Sharma OP. Neurosarcoidosis: a personal perspective based on the study of 37 patients. Chest. 1997;112:220-8.
2. Zajicek JP, Scolding NJ, Foster O, et al. Central nervous system sarcoidosis-diagnosis and management. Q J Med. 1999;92: 103-17.
3. Ohara K. Ocular sarcoidosis. Nippon Rinsho. 2002;60:1807-12.
4. Cieslicka E, Cieslicki J, Ziora D, Romaniuk W, Oklek K. Symptomatic eye changes during the course of sarcoidosis. Klin Oczna. 1998;100:179-83.
5. Heuser K, Kerty E. Neuro-ophthalmological findings in sarcoidosis. Acta Ophthalmol Scand. 2004;82:723-9.
6. Turner RG, James DG, Friedmann AI, et al. Neuro-ophthalmic sarcoidosis. Br J Ophthalmol. 1975;59:657-63.
7. Spalton DJ, Sanders MD. Fundus changes in histologically confirmed sarcoidosis. Br J Ophthalmol. 1981;65:348-58.
8. Nowak DA, Widenka DC. Neurosarcoidosis: a review of its intracranial manifestation. J Neurol. 2001;248:363-72.
9. Oritsu M. Steroid treatment of neurosarcoidosis. Nippon Rinsho. 1994;52:1659-63.
10. Kogure M. Treatment of ocular manifestation of sarcoidosis. Nippon Rinsho. 1994;52:1654-8.

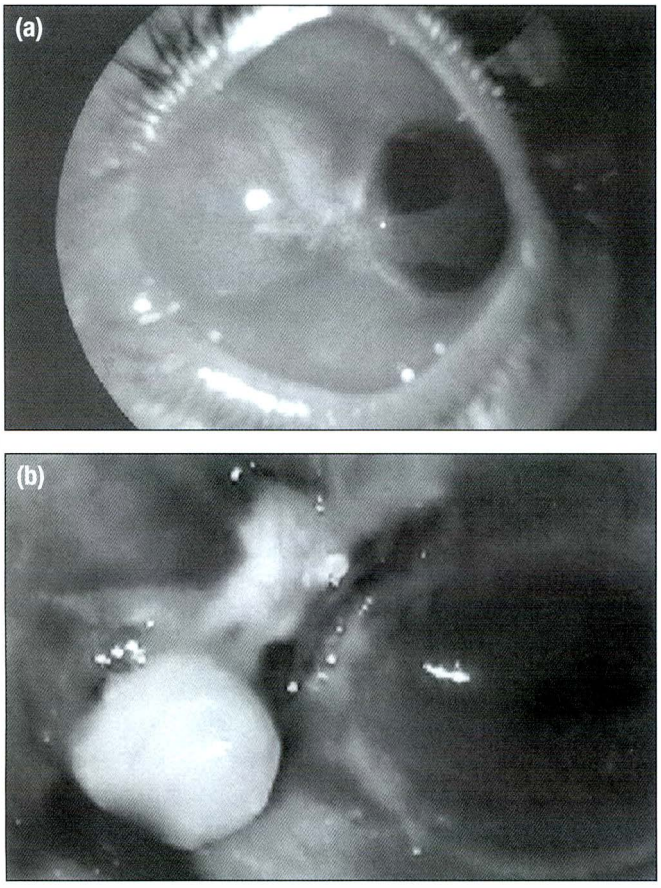
Sympathetic Ophthalmia Following Traumatic Phacocele

Dear Editor,

Phacocele is the herniation of the crystalline lens, outside the outer coating of the eyeball. This term is used for the subconjunctival location of the lens. Sympathetic ophthalmia is a rare, bilateral inflammatory process involving the uveal tract occurring secondary to penetrating trauma or ocular surgery. The overall incidence is estimated to be approximately 0.1% to 0.3% following ocular trauma.[1] Traumatic phacocele is rarely encountered following blunt injury[2] and the resulting sympathetic ophthalmia has not been documented.

A 38-year-old man presented with decreased vision in the left eye after a trauma 2 days earlier. Examination revealed hand movement vision, diffuse subconjunctival haemorrhage, and a large firm subconjunctival cyst in the superonasal quadrant 0.5 mm from the limbus (Figure 1a). The cornea was clear. Iridodialysis was present inferiorly for 5 clock hours. The pupil was peaking nasally and the lens was absent in the retropupillary area. Vitreous haemorrhage precluded fundus view. Ultrasound B-scan did not show evidence of retinal detachment. The right eye was unremarkable. Intraoperatively, the conjunctiva was incised along the limbus from 7 o'clock to 12 o'clock and the lens was removed. Examination of the bed of the cyst revealed a scleral laceration 2 mm, circumferential to the limbus extending from 7 o'clock to 11 o'clock, with prolapse of uveal tissue and vitreous (Figure 1b). Uveal tissue was excised and the scleral laceration was repaired. The patient was left aphakic. Postoperatively, the patient was administered topical steroid. After 6 weeks, his best-corrected visual acuity was 20/60.

Figure 1. The left eye showing (a) a subconjunctival cyst containing the lens; and (b) uveal prolapse and scleral tear. Iridodialysis is seen inferiorly.



He presented 8 weeks later with decrease in vision for 1 week. Ocular examination revealed mutton fat keratic precipitates, 4+ cells, and hand movement vision in both eyes. Uveal pigment was found at the sutured scar in the left eye (Figure 2). The lens was cataractous in the right eye (Figure 3). Grade 3 vitritis was seen in both eyes. Systemic evaluation was normal. The diagnosis of sympathetic ophthalmia was made clinically. He was administered systemic, topical, and subconjunctival steroids, but was lost to follow-up.

Phacocele is a rare complication of blunt trauma to the eye.[2] Scleral rupture can occur due to direct or indirect effect of the trauma. The temporo-inferior quadrant is the most frequent site of impact, which compresses the globe against the trochlea rupturing the globe in the superonasal quadrant. The lens, when dislocated by the force, is directed through the dehiscence into the subconjunctival space. Surgical management consists of removing the lens and repairing the scleral rupture.[2,3]

Sympathetic ophthalmia is a rare bilateral granulomatous panuveitis, following scleral rupture with uveal prolapse resulting in exposure of sequestered uveal antigen.[2,4] The mainstay of treatment is high doses of both local and systemic steroids.[5] Although phacocele and sympathetic ophthalmia are well known entities, to

Figure 2. The left eye showing uveal tissue incarcerated in the surgical scar.

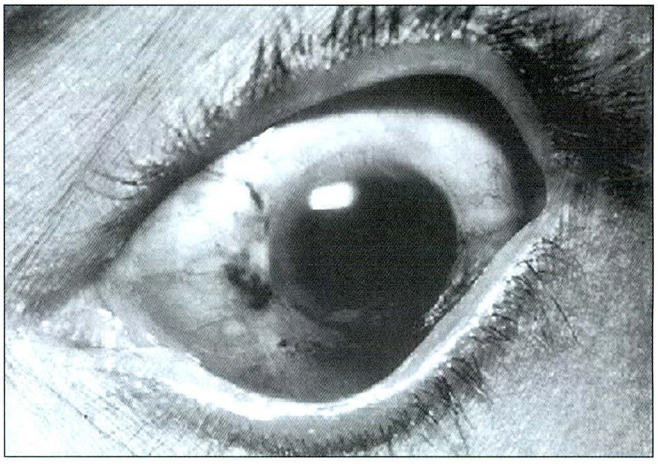
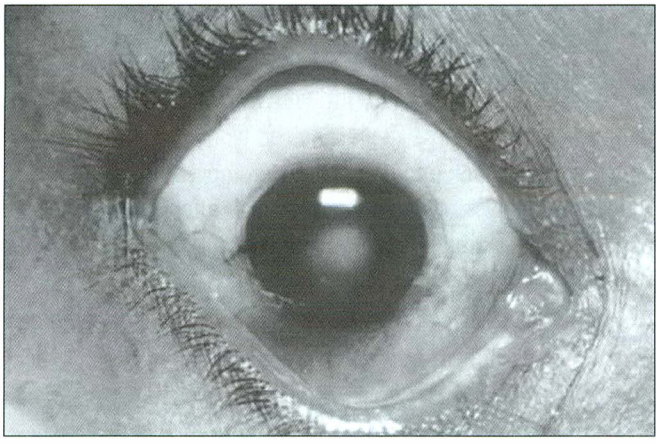


Figure 3. The right eye with cataractous lens and uveitis.



our knowledge this is the first case of sympathetic ophthalmia occurring after phacocele. The cause of sympathetic ophthalmia may be attributed to the scleral rupture with uveal prolapse and phacocele, or incarceration of uveal tissue in the surgical scar.

References

1. Chan CC, Roberge FG, Witecup MS, et al. 32 cases of sympathetic ophthalmia. A retrospective study at the National Eye Institute, Bethesda, Md., from 1982 to 1992. Arch Ophthalmol. 1995;113:597-600.
2. Sathish S, Chakrabarti A, Prajna V. Traumatic subconjunctival dislocation of the crystalline lens and its surgical management. Ophthalmic Surg Lasers. 1999;30:684-6.
3. Cherry PMH. Rupture of the globe. Arch Ophthalmol. 1972;88: 498-507.
4. Panuveitis. In: The Foundation of the American Academy of Ophthalmology, Basic and Clinical Science Course. Intraocular Inflammation and Uveitis. Section 9. 2001-2002:190-3.
5. Hakin KN, Pearson RV, Lightman SL. Sympathetic ophthalmia: visual results with modern immunosuppressive therapy. Eye. 1992: 6;453-5.

Subashini Kaliaperumal, Arvind Gupta, Renuka Srinivasan, Vasudev Anand Rao

Department of Ophthalmology, Jawaharlal Institute of Postgraduate Medical Education and Research, Pondicherry 605 006, India

E-mail: arvind_ophthal@yahoo.co.in

Comment

Sympathetic ophthalmia (SO) is a rare potentially blinding autoimmune bilateral granulomatous panuveitis occurring after any ocular penetration. During the past 2 decades, there has been a changing trend from accidental causes to surgical-related causes. SO affects 0.1% to 0.5% of non-surgical penetrating eye injuries, and less than 0.1% of surgical eye wounds. Kilmartin et al reported an overall incidence of 0.03/100,000 population in the UK.[1] Eighty percent of cases occur within 3 months of injury to the eye and 90% occur within 1 year. The mechanism is believed to be a dynamic T-cell reaction to exposed self-antigens (e.g., the retinal S antigen) following surgical or accidental trauma, which interact with HLA-DR4 complex triggering a choroid-directed inflammatory response. The HLA-DRB1*04 and DQA1*03 genotypes in Caucasian patients (similar to Japanese patients)[2] and certain cytokine gene polymorphisms are markers of increased SO susceptibility and severity.[3]

The authors described a patient with SO occurring 2 months after traumatic accidental globe rupture with prolapse of uveal tissue. In traumatic penetrating injuries, sequestered uveal antigens are believed to sensitise the host, thereby decreasing tolerance and resulting in an inflammatory response.[4] This is the most likely cause of SO in the patient described. Interestingly, the scleral rupture provided an exit passage for subconjunctival migration of the traumatic dislocated lens resulting in a phacocele.

There have been very few reports of phacocele in the English literature, which usually occurs in patients with compromised scleral integrity following previous ocular surgery precipitated by traumatic blunt injuries. However, as the authors have pointed out, the likely cause for SO is the loss of uveal tissue rather than sequestration of lens material in the subconjunctival space.

The modern management of sympathetic ophthalmia with immediate and aggressive use of systemic immunosuppressive therapy (including systemic and local or intravitreal steroids) has resulted in a reduced incidence of enucleation. It would be interesting to compare the management and dosages of medications used for this patient and the final outcome.

References

1. Kilmartin DJ, Dick AD, Forrester JV. Prospective surveillance of sympathetic ophthalmia in the UK and Republic of Ireland. Br J Ophthalmol. 2000;84:259-63.
2. Kilmartin DJ, Wilson D, Liversidege J, et al. Immunogenetics and clinical phenotype of sympathetic ophthalmia in British and Irish patients. Br J Ophthalmol. 2001;85:281-6.
3. Atan D, Turner SJ, Kilmartin DJ, et al. Cytokine gene polymorphism in sympathetic ophthalmia. Invest Ophthalmol Vis Sci. 2005;46: 4245-50.
4. Rao NA. Mechanisms of inflammatory response in sympathetic ophthalmia and VKH syndrome. Eye. 1997;11:213-6.

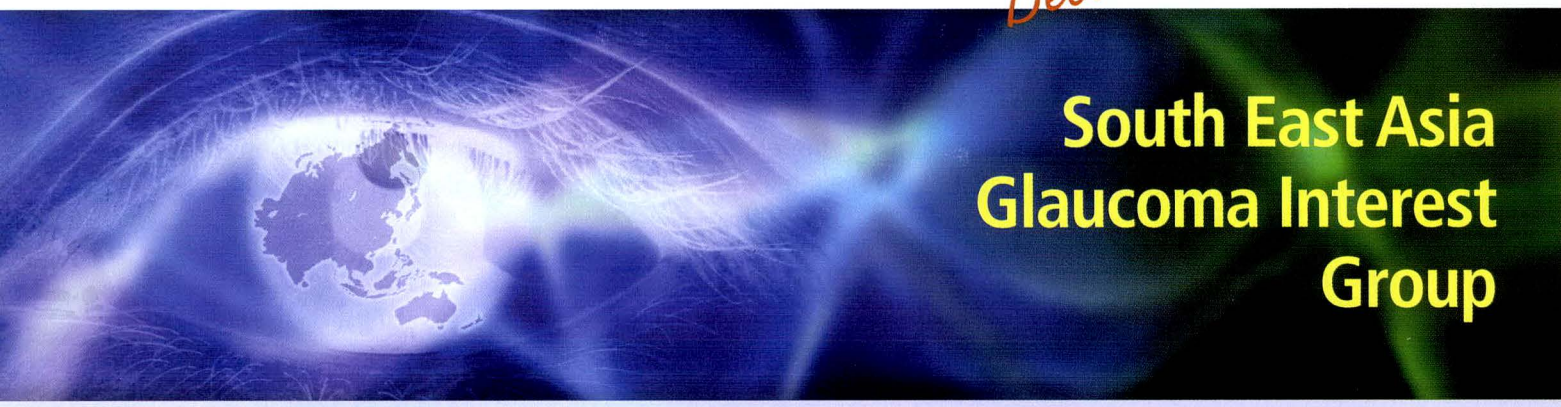
Dr Stephen Teoh

Uveitis, Ocular Immunology and Retina Service, The Eye Institute Tan Tock Seng Hospital, Singapore

E-mail: Stephen_Teoh@ttsh.com.sg

SEAGIG
South East Asia Glaucoma Interest Group

Become a Member!



South East Asia Glaucoma Interest Group

The South East Asia Glaucoma Interest Group (SEAGIG) was established in 1997 by a group of glaucoma specialists to tackle some of the problems associated with glaucoma specific to the Asian region. The intention is to facilitate contact between glaucoma specialists in Asia and around the world, to encourage collaborative research and service projects, to increase the opportunities for exchange of skills and knowledge in the rapidly advancing field of glaucoma, and to assist ophthalmologists and other eye care workers to remain up to date with advances in all aspects of glaucoma diagnosis and management.

The objectives of SEAGIG are to promote excellence in the diagnosis and care of patients with glaucoma at both individual and community levels:

- improving the care of patients with all types of glaucoma and related diseases
- increasing the understanding of such diseases through educational activities
- facilitating, conducting, and funding research programmes to expand knowledge about glaucoma
- working with universities, medical schools, hospitals, and other institutions to advance these aims
- maintaining and promoting relationships with any organisation with similar goals
- raising, disbursing, and administering funds in furtherance of these objectives.

SEAGIG has recently produced Asia Pacific Glaucoma Guidelines to assist ophthalmologists to manage optimally patients with glaucoma in Asia. The guidelines may be viewed online at: www.seagig.org/apgg.html. All members will recieve a free copy of the guidelines.

SEAGIG membership benefits include:

- Journal subscription
- Glaucoma Guidelines
- 10% discount on registration fee to SEAGIG/Asian-Oceanic Glaucoma Society conferences.

Asian Journal of OPHTHALMOLOGY

Asian Journal of OPHTHALMOLOGY is the official publication of SEAGIG and is indexed in EMBASE/ Excerpta Medica. Now in its seventh volume, this quarterly peer-reviewed Journal publishes articles of interest to ophthalmologists in the Asia-Pacific region, including Original Articles, Review Articles, Case Reports, Technical Notes, Pictorial Ophthalmology, Ophthalmology Society Updates, Letters to the Editor, and News and Views. Papers should be submitted to: ***editor@seagig.org****. The Journal can be viewed online at:* ***www.seagig.org****.*

Scientific Communications

SEAGIG Membership

- **Glaucoma Member** — any ophthalmologist in active practice who has completed a Glaucoma Fellowship and/or whose work consists of at least 50% glaucoma *US$60*
- **Ophthalmic Member** — any doctor working as an ophthalmologist in the country of residence of that doctor *US$50*
- **Trainee Member** — any medical practitioner participating in an ophthalmic vocational training programme *US$40*
- **Medical Member** — any medical practitioner with an interest in glaucoma *US$20*
- **Research Member** — any person who is a vision scientist *US$20*
- **Associate Member** — any person who is an eye care worker who is not a medical practitioner *US$20*
- **Corporate Member** — any associations and organisations wishing to support the objectives of the Society *US$250*

Glaucoma Members, Ophthalmic Members, Trainee Members, and Corporate Members will receive Asian Journal of OPHTHALMOLOGY free as part of their membership. All SEAGIG members receive a **1-year online subscription** to Asian Journal of OPHTHALMOLOGY, access to the members only part of the SEAGIG website, the **Asia Pacific Glaucoma Guidelines,** and a **10% discount on the attendance rates of SEAGIG/Asian-Oceanic Glaucoma Society conferences**.

To join: Visit the SEAGIG website at **www.seagig.org**.

Update in Glaucoma and Cataract

From the Third Global Chinese Ophthalmic Conference in conjunction with the XI National Congress of Chinese Ophthalmological Society held in Beijing, China, 31 August to 4 September 2006

Vascular Aspects of Glaucoma and the Clinical Implications

Catherine Jui-ling Liu
Taipei Veterans General Hospital
National Yang-Ming University School of Medicine
Taipei, Taiwan

It was proposed more than a century ago that vascular factors play a role in the pathogenesis of glaucomatous optic neuropathy (GON). Proponents of the vascular theory consider GON as a consequence of insufficient blood supply due to increased intraocular pressure (IOP) or other risk factors reducing ocular blood flow (OBF). This is supported by accumulating evidence obtained from clinical observation, population studies, and animal experiments. Recent studies using modern technology to determine OBF in various ocular vasculatures have demonstrated that, compared with normal controls, patients with either primary open angle or angle closure glaucoma have decreased blood flow, decreased flow velocity, and increased vascular resistance. The OBF reduction increases in extent as GON progresses, but it may occur before the nerve damage can be identified. With the IOP controlled, glaucomatous eyes with reduced OBF are more likely to progress to GON than those without impaired OBF.

It is important to be aware that glaucoma medication that lowers IOP but simultaneously reduces OBF may unfavourably influence the disease outcome. Treatment modalities that reduce IOP without decreasing blood pressure or increasing ocular vascular resistance may increase OBF as a result of increased ocular perfusion pressure. Carbonic anhydrase inhibitors may increase retinal circulation by directly acting on the capillaries in addition to their IOP-lowering effect. The usefulness of other treatment such as ginkgo biloba extract in glaucoma management awaits further study.

Regulation of Extracellular Matrix Hydrolysis in the Control of Intraocular Pressure

Iok-Hou Pang
Glaucoma Research
Alcon Research, Ltd.
Forth Worth
Texas, USA

Elevated intraocular pressure (IOP) is a major risk factor for glaucoma. Although the exact aetiology of ocular hypertension remains unclear, an excessive accumulation of extracellular matrix (ECM) in the trabecular meshwork (TM), which hinders the outflow of aqueous humor, likely contributes to the mechanism. Matrix metalloproteinases (MMPs), when activated, can hydrolyse many types of ECM and should improve aqueous outflow. Research into compounds that increase MMPs in the TM has been performed, and selected compounds have been tested for their effect on IOP. Enzyme-linked immunosorbent assay was used to evaluate expression of MMPs by cultured human TM cells. Drug effects on IOP were assessed by human ocular perfusion organ culture.

Various compounds were shown to increased MMP-3 production in TM cells. Among these compounds, 2 were tested in perfused human donor eyes.

In non-glaucomatous eyes, tert-butylhydroquinone (tBHQ; 10 M) increased outflow facility 1 to 4 days after the start of perfusion ($p < 0.05$; $n = 6$). At day 4, the increase in outflow was approximately 60% above the vehicle-treated contralateral eyes. Perfusates of the tBHQ-treated tissues had elevated proMMP-3 levels. Maximal increase (approximately 200% of control) occurred at day 2 after perfusion. In glaucomatous eyes, tBHQ also increased the aqueous outflow facility by approximately 60% at day 3 after perfusion.

Propentofylline (PPF; 100 M), also significantly enhanced outflow facility of non-glaucomatous eyes 1 to 3 days after treatment started ($p < 0.05$). Similar to tBHQ, PPF significantly increased proMMP-3 concentration in the perfusate by 20% to 40% after the initiation of treatment ($p < 0.05$).

To improve aqueous outflow by the hydrolysis of excessive ECM in the TM has been proposed for some time. Until recently, no clinically practical methods were available to accomplish this. The small molecules described in this study show the conceptual feasibility of this approach. Their mechanism of action is different from most of the current glaucoma drugs and may allow them to have an additive effect with current medications, Hence, these results suggest that the induced hydrolysis of ECM in the TM with small molecules may provide a future useful therapy for glaucoma.

Selective Laser Trabeculoplasty as a Primary Treatment for Glaucoma

S Melamed
The Sam Rothberg Glaucoma Center
Tel Aviv University Medical School
Tel Aviv, Israel

Selective laser trabeculoplasty (SLT) has been developed as an alternative to argon laser trabeculoplasty (ALT). In recent years, this treatment modality has become the

most accepted method of treatment for open angle glaucoma.

The advantages of SLT are the result of the specific targeting of melanin granules in the trabecular meshwork by the laser. It is a double-frequency YAG laser with an exposure time of 3 nanoseconds, and there is no necrotic response of tissue as there is much less heat dissipation when compared with ALT.

In recent years, many investigators have reported positive results with SLT, which include effective IOP reduction and reduced complications of intraocular pressure spike and peripheral anterior synechiae formation.

Selective Laser Trabeculoplasty as Adjunctive Therapy

En-Ping Chen
Glaucoma Service
St Erik's Eye Hospital
Karolinska Institutet
Stockholm, Sweden

A retrospective study was performed to obtain clinical data of selective laser trabeculoplasty (SLT) for 90° during a 3-year period. SLT with 25 laser effects on 90° of the trabecular meshwork was performed in 76 eyes of 76 patients. The time interval between the laser treatment and the loss of intraocular pressure (IOP) control was analysed by Kaplan-Meier survival analysis. The influence of exfoliation, pigmentation of the trabecular meshwork, use of topical medication, and previous argon laser trabeculoplasty (ALT) and SLT treatment on the results of SLT were analysed.

The 95% confidence interval of the duration in which the IOP had been regulated after a first-time SLT was 196 to 297 days. Repeated SLT had a similar duration of IOP regulation as first-time SLT. First-time SLT was more effective in patients administered ≤2 topical medications. Age, previous ALT, existence of exfoliation, or pigmentation of the trabecular meshwork did not influence the period of IOP control after first-time SLT. It was concluded that SLT was effective as adjunctive therapy. Repeat 90° SLT provided a similar duration of IOP control to first-time SLT.

Improving Surgical Efficiency with Advanced Technology

David Lubeck
Arleo Eye Institute
USA

The technologies available for phacoemulsification today greatly contribute to more efficient, more reliable and safer surgery with better surgical outcomes. The superior fluidic performance of the latest generation technology phacoemulsification system enables us to better utilise vacuum and flow rates for lens removal. In some cases, vacuums in excess of 600 mm Hg are achieved, using extremely fast rise times, yet the actual flow rate through the anterior chamber can be minimised. When this technology is properly used it dramatically improves the efficiency of the lens emulsification and evacuation. In some cases, it is possible to virtually eliminate the use of ultrasound power for lens removal.

Alternative energy removal modalities such as sonic oscillations (NeoSoniX) have proven to be a useful complement to ultrasound. The oscillations contribute to the overall reduction of ultrasound power by reorienting the lens material on the phaco tip. This is particularly effective for evacuating harder lens fragments during the quadrant removal or chop phase of the procedure.

More recently, great success has been achieved with AquaLase technology for lens removal. A pre-chop technique has been successfully combined with the newly designed AquaLase Tip. This has provided a significant improvement in surgical efficiency, with a good safety profile.

Advancements in dual bevel and high performance steel blade keratomes and sideport knives enable better wound construction. This helps to minimise wound leakage as well as reducing fluid volume and turbulence. All these factors contribute to a more predictable surgery and better outcomes.

The combined use of a dispersive and cohesive viscoelastic provides optimal endothelial protection while achieving necessary space maintenance. Soon there will be one viscoelastic product that will effectively fulfil both roles.

Finally, one of the most exciting developments has been the 2.2-mm coaxial incision phaco system. This micro-coaxial approach provides the ability to deliver a 6.0-mm state-of-the-art aspheric single-piece natural lens through a 2.2-mm incision. Consistently good results are achieved with the new apodised lens technology and the natural version is awaited.

Proper selection and use of improved surgical tools along with sound surgical technique enable surgeries to be performed with few or no complications. This means higher efficiency and effectiveness, which translates to better immediate and long-term patient postoperative outcomes for patients.

6th International Glaucoma Symposium

28-31 March 2007, Athens, Greece

The 6th International Glaucoma Symposium will be held in Athens, Greece, from 28 to 31 March 2007. Participants in this meeting will have a unique opportunity to meet with colleagues and exchange ideas in a friendly and congenial environment. There will be ample opportunities to discuss state-of-the-art approaches to glaucoma and to consult with the experts in both informal discussions and formal panel sessions.

Topics will include:

- advances in glaucoma surgery (lasers, implants, minimally invasive surgery)
- advances in medical therapy for glaucoma (sustained release, new concepts, new drug studies)
- epidemiology and demographics of glaucoma
- genetics and molecular biology of glaucoma
- glaucoma grand rounds
- glaucoma in the presence of comorbidities (uveitis, myopia, penetrating keratoplasty)
- imaging in glaucoma
- new ideas in glaucoma diagnosis and management
- non-intraocular pressure factors in glaucoma: corneal thickness, neuroprotection, blood flow
- pathophysiological mechanisms in glaucoma (aqueous outflow and inflow, retinal ganglion cell apoptosis, glaucoma models)
- pseudoexfoliation glaucoma
- visual function (perimetry and electrophysiology).

The deadline for the submission of abstracts is 2 November 2006.

Scholarship for Young Physicians

The 6th International Glaucoma Symposium offers up to 20 scholarships for young physicians. The grant consists of free registration to the symposium plus a partial travel grant to physicians from East Europe, Greece, and Turkey aged 35 or younger on 1 March 2006, who are presenting (first) authors, not yet in a permanent position and whose abstract has been accepted. Application for Scholarship will be possible online and only applications accompanied by an abstract will be considered.

Applications must be accompanied by a fax (send to: 41 22 732 2850) or e-mail (send to: glaucoma@kenes.com) with a copy of your passport showing your date of birth and a letter from the head of your department, proving that you are truly a resident and that without the scholarship you will not be able to attend the conference.

Applications for scholarships will only be considered if submitted by the time of abstract submission deadline, which is 2 November 2006.

Scholarship recipients will be selected on the basis of the quality of their abstracts by the Scholarship & Travel Grant Committee.

Applicants who have been granted a scholarship will be informed well before the early registration fee deadline and will receive a travel grant check at the Congress.

For further details, contact the Symposium Secretariat:

Symposium Organizers

17 Rue du Cendrier, PO Box 1726, CH-1211 Geneva 1, Switzerland

Tel: (41 22) 908 0488

Fax: (41 22) 732 2850

E-mail: **glaucoma@kenes.com**

SEAGIG

South East Asia Glaucoma Interest Group

SEAGIG - CHENNAI 2006

Under the aegis of

GLAUCOMA SOCIETY OF INDIA

INTERNATIONAL GLAUCOMA CONVENTION DECEMBER 1-3,2006, CHENNAI, INDIA.

Hosted by

SANKARA NETHRALAYA

Venue :

Chennai Convention Centre
CTC Complex, Nandambakkam,
Chennai - 600 089

FOR MORE INFORMATION PLEASE CONTACT

Dr L .Vijaya
Organizing Chairperson, SEAGIG - CHENNAI 2006
Director, Glaucoma Services
SANKARA NETHRALAYA
18 College Road, Chennai 600 006, India
Tel: (91 44) 2827 1616 / 2823 3556 Fax: (91 44) 2825 4180
E-mail: drlv@snmail.org ; seagig_2006@yahoo.com

Programme Highlights

Symposia and Sessions on:

- Angle Closure Glaucoma
- Evidence Based Ophthalmology
- Controversies in Glaucoma
- Diagnostics
- Video Surgery Session
- Pediatric Glaucoma and Secondary Glaucoma
- Patient Management beyond the RCT's
- Instruction Courses

  How to write a paper
  Angle Evaluation
  Disc & Field Assessment
  Imaging Techniques

Faculty

We have a stellar cast of glaucoma experts who will share their experiences with us.

Confirmed International Faculty:

Alan Robin	USA	Marlene Moster	USA
Andre Mermoud	SWITZERLAND	Ming Guang He	CHINA
Aung Tin	SINGAPORE	Paul Foster	UNITED KINGDOM
Clement C Y Tham	HONG KONG	Paul TK Chew	SINGAPORE
Deepak Edward	USA	Paul Palmberg	USA
Ho Ching Lin	SINGAPORE	Prin Rojana Pongpun	THAILAND
Ivan Goldberg	AUSTRALIA	Robert Ritch	USA
Jimmy Lai	HONG KONG	Seng Kheong Fang	MALAYSIA
Jost Jonas	GERMANY	Ted Krupin	USA
Kuldev Singh	USA	Vincent Michael Patella	USA
Lu Da Wen	TAIWAN	Yoshiaki Kitazawa	JAPAN

Glaucoma Experts from India

SEAGIG Chennai 2006

Registration can be done online at www.seagigchennai.org

Abstract Submission

We invite abstracts for poster and free paper presentations. Important dates in this regard are:

15th June 2006 : Abstract submission opens

15th August 2006 : Abstract submission closes

Abstract submission can be done online at
www.seagigchennai.org

Platinum Sponsors

ALLERGAN

Alcon® INDIA

Gold Sponsor

Pfizer

November 2006

3-4
21st Annual Clinical and Scientific Meeting of the Neuro-Ophthalmology Society of Australia
Sydney, Australia
Contact: Mark Paine, 55 Victoria Parade, Fitzroy 3065, Sydney, Australia
E-mail: markpaine@ozemail.com.au
or
michael@icn.usyd.edu.au

3-5
5th International Retina Angiograph Symposium
Kyoto, Japan
Contact: Congress Secretariat
Tel: (49 6221) 646 3306
E-mail: Academy@HeidelbergEngineering.com

11-14
2006 American Academy of Ophthalmology's Joint Meeting with the Asia Pacific Academy of Ophthalmology
Las Vegas, USA
Contact: American Academy of Ophthalmology, PO Box 7424,
San Francisco, CA 94120-7424, USA
Tel: (1 415) 561 8500
Fax: (1 415) 561 8533
E-mail: meetings@aao.org

December 2006

1-3
SEAGIG 2006 Meeting of the South East Asia Glaucoma Interest Group/Asian Oceanian Glaucoma Society and the Glaucoma Society of India
Chennai, India
Contact: Dr L Vijaya, Director, Sankara Nethralaya, No.18, College Road, Chennai 600 006, Tamil Nadu, India
Tel: (91 44) 2827 1616
Fax: (91 44) 2825 4180
E-mail: seagig_2006@yahoo.com
or
drlv@snmail.org

February 2007

24-28
22nd Congress of Asia-Pacific Academy of Ophthalmology
Lahore, Pakistan
Contact: Congress Secretariat
E-mail: secretariat@apao2007.com
or
info@apao2007.com
Website: http://www.apao2007.com

March 2007

2-5 March
Asia ARVO
Singapore
Contact: Karen Chee, Singapore Eye Research Institute, 11 Third Hospital Avenue, #05-00, SNEC Building, Singapore 168751
Tel: (65) 6322 8311
Fax: (65) 6323 1903
E-mail: karen.chee.s.l@seri.com.sg
Website: www.seri.com.sg

28-31
6th International Glaucoma Symposium
Athens, Greece
Contact: Avital Rosen
Tel: (41 229) 080 488
Fax: (41 227) 322 850
E-mail: glaucoma@kenes.com

Phaco Training and ICO Fellowships

Mahatme Eye Bank & Eye Hospital, Nagpur, India, offers training programmes for ophthalmologists, as follows:
1. Three-month International Council of Ophthalmology fellowship in phaco.
2. Two-week hands-on phaco training.
3. Thirteen-month fellowship postgraduation.
4. Postgraduate courses in ophthalmology.
5. International Council of Ophthalmology examination assessment centre.

This training is available throughout the year. For further information, please contact:
Dr Vikas Mahatme, Mahatme Eye Bank & Eye Hospital, 16 Central Excise Colony, Ring Road, Nagpur-440015, India.
Tel: (91 712) 223 4345/222 2556
Fax: (91 712) 224 2202
E-mail: contact@mahatmehospital.com
Website: www.mahatmehospital.com

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

April 2007

28-2 May
2007 Annual Symposium and Congress of the American Society of Cataract and Refractive Surgery
San Diego, CA, USA
Contact: ASCRS-ASOA, 4000 Legato Road, Suite 850, Fairfax, Virginia 22033
Tel: (1 703) 591 2220
Fax: (1 703) 591 0614
E-mail: ascrs@ascrs.org/asoa@asoa.org

May 2007

20-23
Annual Meeting Of The European Strabismological Association
Mykonos, Greece
Contact: Organizing Secretariat, Aktina-City Congress
Tel: (30 210) 323 2433
Fax: (30 210) 323 2338
E-mail: dch@citycongress.com
Website: www.esa-strabismology.com

June 2007

9-12
2007 Congress of the European Society of Ophthalmology
Vienna, Austria
Contact: Britta Sjöblom
Tel: (46 84) 596 650
Fax: (46 86) 619 125
E-mail: britta.sjoblom@congrex.se

November 2007

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

Asian Journal of OPHTHALMOLOGY is the official publication of the South East Asia Glaucoma Interest Group. The Journal is a peer-reviewed publication and is published bimonthly in February, April, June, August, October, and December. All articles that contribute to the development and understanding of ophthalmology in Asia will be considered. The Journal is indexed in EMBASE/Excerpta Medica.The Journal welcomes contributions within the categories of original research, invited papers, review articles, case reports, conference reports, and letters to the editor. Submissions may be made online at www.seagig.org or sent by e-mail to manuscripts@seagig.org or may be sent on disk to the following address:

The Editor
Asian Journal of OPHTHALMOLOGY
Scientific Communications International Ltd
Suite C, 10/F, Wo On Building
10 Wo On Lane, Central, Hong Kong
E-mail: editor@seagig.org

Manuscript Criteria

Submitted manuscripts should adhere to the stated format. Manuscripts that do not conform to the approved format will be returned without review. Authors should fulfil the authorship criteria stated in the *Uniform requirements for manuscripts submitted to biomedical journals: writing and editing for biomedical publication,* which should also be followed for general guidance.[1] A covering letter stating that the submitted material has not been previously published and is not under consideration for publication elsewhere should be included. The receipt of submissions will be acknowledged. All accepted papers become the permanent property of *Asian Journal of OPHTHALMOLOGY* and may not be published elsewhere without written permission from the Journal.

Categories of Articles

Editorials — Length should not exceed 1000 words; the total number of Tables and Figures should not be more than 2, and references not more than 10.

Original Articles — Length should not exceed 2500 words; the total number of Tables and Figures should not be more than 6, and references not more than 40. Headings of Introduction, Patients and Methods, Results, and Discussion should be included.

Review Articles — Length should not exceed 2500 words; the total number of Tables and Figures should not be more than 6, and references not more than 40. Section headings should be provided.

Case Reports — Length should not exceed 1000 words; the total number of Tables and Figures should not be more than 2, and references not more than 10.

Conference Reports — Length should not exceed 2500 words; the total number of Tables or Figures should not be more than 6, and references not more than 40. Headings for different sections should be provided.

Letters to the Editor — Communications on all aspects of ophthalmology are encouraged. Length should not exceed more than 300 words, and references should number no more than 5.

Manuscript Preparation

The manuscript should be arranged as follows:

Title Page

The title page should contain the following:

- the title of the article, which should be concise but informative
- a short running title of fewer than 40 characters (including spaces)
- the first name, middle initials, and last name of all authors, with their 2 highest academic degrees; a maximum of 6 authors is permitted
- the name of the department(s) and institution(s) to which each author is affiliated
- the full name, address, telephone and fax numbers, and e-mail address of the designated author for correspondence.

Abstract and Key Words

The abstract for original articles must summarise the purpose, procedures, main findings, and principal conclusions of the investigation, and must be structured with the following subheadings: Aim(s), Patients and Methods, Results, and Conclusion(s). Abstracts for all other articles must be unstructured, but should include the key points discussed in the paper. Abstracts should be no longer than 250 words. The key words must be Medical Subject Headings taken from Medline/*Index Medicus*.

Body Text

For original articles, the following sections should be included:

Introduction

The rationale for the study should be summarised and pertinent background material outlined. This should not include findings or conclusions.

Patients and Methods

This section should describe the methodology in sufficient detail to leave the reader in no doubt as to how the results are derived.

Manuscripts that contain the results of human or animal studies should make clear that a high standard of ethics was applied. Invasive studies of humans should state that the research protocol was approved by the local ethics committee.

Results

The results should be presented in logical sequence in the text, Tables, and Figures; repetitive presentation of the same data in different forms should be avoided. This section should not include material appropriate to the Discussion. Results must be statistically analysed where appropriate, and the statistical guidelines of the International Committee of Medical Journal Editors should be followed.[1]

Discussion

Data given in the Results section should not be repeated here. This section should present the implications and limitations of the study. The Discussion may also include

an evaluation of methodology and of the relationship of new information to the existing body of knowledge in the field. Conclusions should be incorporated into the final paragraph and should be consistent with — and completely supported by — data in the text.

Acknowledgement(s)

Acknowledgements can be made to people who have offered assistance in the research or preparation of the manuscript and who do not fulfil authorship criteria. Research or project support should also be stated, as well as any conflicts of interest.

References

The references should be numbered in numerical order in the text and the reference list, and should not appear in alphabetical order. References should follow the *Uniform requirements for manuscripts submitted to biomedical journals: writing and editing for biomedical publication*[1] and should appear in the text, Tables, and Figures as Arabic numerals in superscript. Journal titles should be abbreviated in accordance with Medline/*Index Medicus*. Authors are responsible for the accuracy of references and must verify them against the original documents. All authors and editors must be listed. The following are examples of reference style:

Standard journal article

Cheung JC, Wright MM, Murali S, Pederson JE. Intermediate-term outcome of variable dose mitomycin C filtering surgery. Ophthalmology 1997;104:143-149.

Supplement

Taylor A, Jacques PF, Epstein EM. Relations among aging, antioxidant status, and cataract. Am J Clin Nutr 1995;62 (6 Suppl): 1439-1447.

Books and other monographs

Kupfer C, Underwood B, Gillen T. Leading causes of visual impairment worldwide. In: Albert DM, Jakobiec FA, editors. Principles and practice of ophthalmology. Philadelphia: WB Saunders Company; 1994: 1250-1251.

Tables

Tables must be typed in Table format after the references or provided in a separate Microsoft Word file. All Tables must be cited in numerical order in the text. A brief title should be supplied for each Table and a short heading provided for each column. Explanatory matter should be placed in footnotes, not in the heading. Abbreviations should be avoided in Tables. If abbreviations are necessary, they must be explained in a footnote. Statistical measures of variation such as standard deviation, standard error of the mean, and confidence interval should be identified in the column headings.

Figures and Illustrations

All Figures must be cited in numerical order in the text, and a brief legend provided after the references. Figures should be provided in electronic format in a separate file, not as part of the body text. The resolution of Figures must be at least 350 dpi. When symbols, arrows, numbers, or letters are used to identify part of an illustration, each one should be identified and clearly explained in the legend. If only hard copies of Figures are submitted, each one should have a label pasted on the back indicating the number of the Figure, the author's name and the top of the Figure (Figures must not be written on and paper clips must not be used).

Illustrations can include photographs, photomicrographs, charts, and diagrams. Photographs of persons must be retouched to make the subject unidentifiable, and be accompanied by written permission from the subject to use the photograph. Footnotes for Figures and Tables must use the following symbols, in this order: *, †, ‡, §, ‖, ¶, **, ††, ‡‡, §§, ‖‖, ¶¶.

The corresponding author is responsible for obtaining written permission to use any copyrighted text or illustrations.

General Style

The following style should be used:

- all papers should be written in English; spelling should comply with the Concise Oxford English Dictionary
- Arabic numerals should be used for all numbers, except for numbers below 100 at the beginning of sentences, which should be spelled out
- abbreviations should not appear in the title or abstract and their use in the text should be limited; abbreviations should be defined at the first mention in the text unless they are standard units of measurement
- Système International (SI) measurements must be used for all laboratory values
- generic drug names must be used unless the specific trade name of a study drug is directly relevant to the discussion.

Editorial Processing

All manuscripts submitted to *Asian Journal of OPHTHALMOLOGY* will be assessed by peer review to determine their suitability for publication. Some manuscripts may require revision before acceptance for publication; in this case, reviewers' comments will be forwarded to the author. The Editor makes the final decision regarding acceptance of a manuscript for publication. Papers should be submitted exclusively to *Asian Journal of OPHTHALMOLOGY*. If accepted for publication, copyright for the manuscript and all parts will be assigned to *Asian Journal of OPHTHALMOLOGY*.

Accepted manuscripts will be copy edited according to the Journal style and returned to the author for approval prior to publication. Authors are responsible for all statements made in their work, including changes made by the copy editor.

Reference

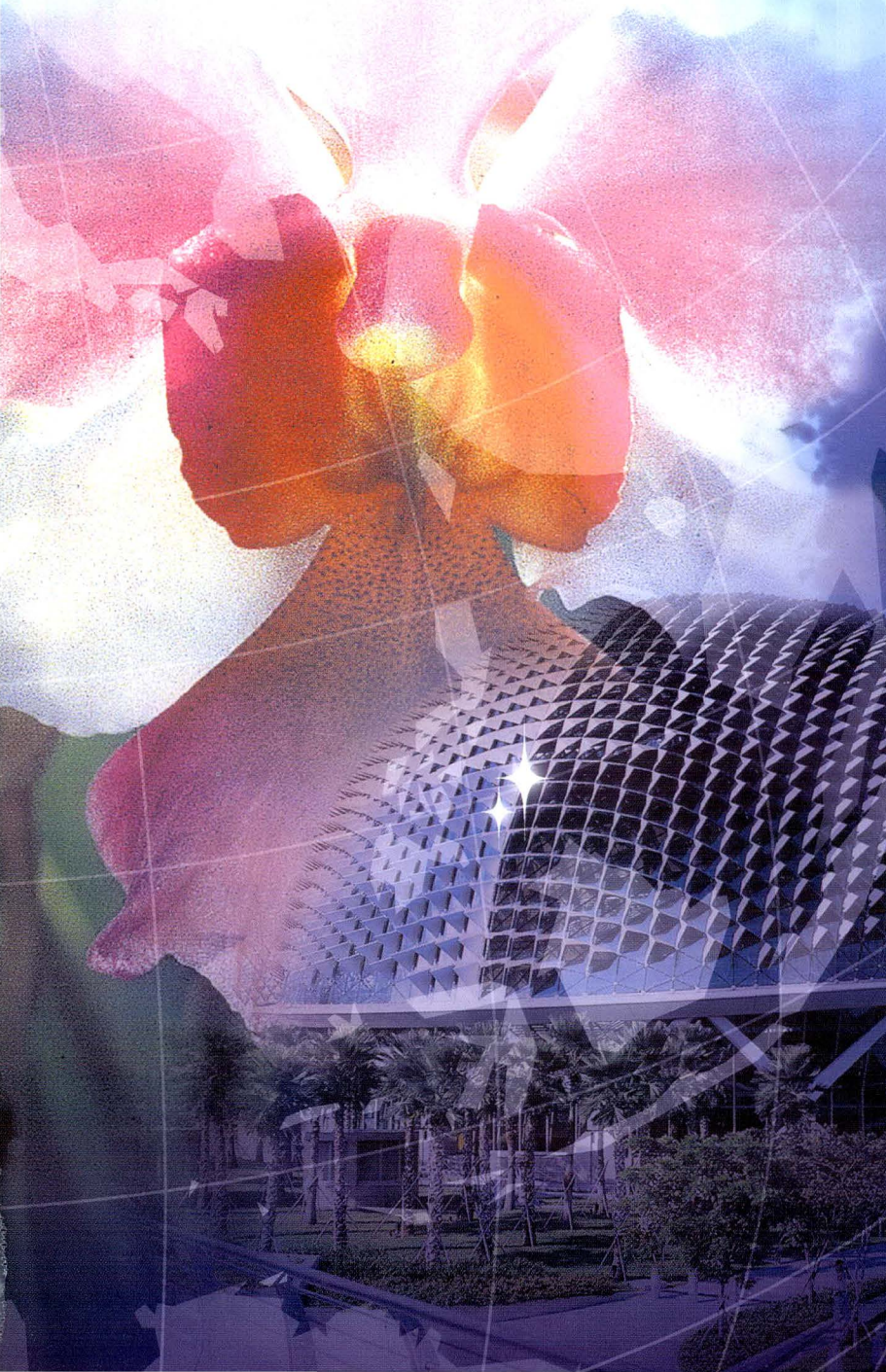
1. International Committee of Medical Journal Editors (ICMJE). Uniform requirements for manuscripts submitted to biomedical journals: writing and editing for biomedical publication. ICMJE; 2004. Available from: http://www.icmje.org

See You in Singapore in 2007!

Asia ARVO®

MEETING ON RESEARCH IN VISION AND OPHTHALMOLOGY

2 - 5 March 2007
Singapore



Asia-ARVO is a biennial meeting held in locations throughout Asia to highlight the important clinical and translational aspects of vision and ophthalmic research. Previously known as SERI-ARVO for meetings held in 2003 and 2005, the name was changed in 2005 to reflect ARVO's commitment to partnering with the many countries in Asia that have expanding eye research programs.

CALL FOR ABSTRACTS

The Organising Committee invites abstracts from both **ARVO** and **NON-ARVO** members for **clinical, basic or epidemiological** research to be considered for oral or poster presentation.

Poster Presentations
Similar to the annual ARVO meetings, Asia ARVO encourage presenters to submit their work as Poster Presentations, which allows interaction between the presenter and the delegates.

Free Paper Presentation
A small majority of abstracts may be accepted for oral presentations in the Free Paper sessions. Presenters will be given 8 minutes to present their research findings and 2 minutes for discussion.

The deadline for ON-LINE abstract submission is 30 September 2006.

For more information, please contact:
Ms Karen Chee
Singapore Eye Research Institute
11 Third Hospital Avenue #05-00
Singapore 168751
Tel: +65 6322 4500 Fax: +65 6323 1903
Email: event@seri.com.sg
Website: www.seri.com.sg

Platinum Sponsors

ALLERGAN Alcon®

Silver Sponsor

THE VISION CARE INSTITUTE™ of Johnson & Johnson Vision Care, Inc.

Supported by

UNIQUELY Singapore
visitsingapore.com

FOR POWERFUL INCREMENTAL IOP REDUCTION AFTER INITIAL MONOTHERAPY

1-drop-daily XALACOM provides your patients with

- ***Powerful incremental intraocular pressure (IOP) reduction[1,2]***
- ***Tolerability that supports therapeutic goals***
- ***Added power without the added drops***

XALACOM is indicated for the reduction of IOP in patients with open-angle glaucoma and ocular hypertension who are insufficiently responsive to topical beta-blockers.

XALACOM is contraindicated in patients with reactive airway disease, including bronchial asthma, a history of bronchial asthma, or severe chronic obstructive pulmonary disease; sinus bradycardia; second- or third-degree atrioventricular block; overt cardiac failure; cardiogenic shock; or hypersensitivity to any component of this product.

XALACOM has been reported to cause changes to pigmented tissues. The most frequently reported changes have been increased pigmentation and growth of eyelashes. The iris pigmentation changes may be permanent.

In the 6-month registration trials, the most frequent adverse events were eye irritation, including stinging, burning, and itching (12.0%); eye hyperaemia (7.4%); corneal disorders (3.0%); conjunctivitis (3.0%); blepharitis (2.5%); eye pain (2.3%); headache (2.3%); and skin rash (1.3%).

Please refer to product insert for full prescribing information.

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

Once Daily Xalacom™
latanoprost/timolol maleate
1 Drop for Incremental Power

Pfizer

Suite 701, Pacific Place,
88 Queensway,
Hong Kong