Advances in the Diagnosis and Treatment of Uveitis

Although uveitis patients comprise less than 1-2% of ophthalmological general consults, it is a condition that results in significant morbidity and visual loss. Hence it is important to establish a working diagnosis to enable timely and appropriate treatment, so as to reduce the risk of blindness.

**Evolution in Diagnostics**

Over the last 80 years, there have been significant shifts in the etiologies of uveitis. Pre-1950, infections such as syphilis, tuberculosis, herpes and toxoplasmosis were reported to account for up to 70% of uveitis, but their diagnosis was mainly clinical and not based on laboratory tests. As diagnostic methods improved through the 60s and 70s, most of these were in fact found to be autoimmune-related. Recently, improvements in molecular diagnostics with polymerase chain reaction (PCR) testing ironically revealed many of the idiopathic uveitis cases to have an infective etiology. There is therefore a return to infection as the initial cause for uveitis, although it may evolve into an autoimmune condition over time.

The clinical picture of uveitis is not always constant as it is dependent on the interaction between the inciting antigen and the immune response of the individual. As such, very atypical presentations can be seen in immunosuppressed patients, such as those with HIV or on immunosuppressant therapy. The clinical criteria for diagnosis would not be applicable in this group and laboratory tests also have to be interpreted differently in view of the immune status. These patients may also develop abnormal immune responses when their immune status improves, for example HIV patients on HAART therapy.

At the National Healthcare Group Eye Institute, the latest molecular diagnostic methods like PCR are utilised in diagnosing tuberculosis, toxoplasmosis, herpes simplex, varicella zoster or cytomegalovirus infections. In addition, serology tests for other infective agents such as syphilis, Lyme, and cat-scratch diseases are performed if the history and clinical presentation are suggestive. Fortunately, most of the infective uveitides can be diagnosed with simple laboratory tests, with the exception of tuberculosis. The diagnosis of tuberculous uveitis is dependent upon clinical presentation, chest radiography, Mantoux test and frequently the new T-spot TB test. T-spot TB is currently the only available test for diagnosis of latent TB in immunosuppressed and HIV patients.

Toxoplasmosis retinitis diagnosed on serology and PCR.

Continued next page
Dear Readers,

A Happy New Year from all of us at the NHG Eye Institute! We open 2009 with Uveitis and Oculo-Plastics, two undeservedly lesser known sub-specialties outside of Ophthalmology, despite massive diagnostic and therapeutic advances over the past few years.

As such, we have aimed for a stronger emphasis on updates, rather than focus on esoteric entities within each realm, in that vein, our Optoms' Corner article revisits our "how not to miss uveitis" tutorial, and we have for the first time produced a poster for the general public on the condition.

Looking ahead, we have already cranked up the machinery for the NHG Eye Institute 2nd International Ophthalmology Congress. HMDP funding for 3 world-renowned experts from the sub-specialties of glaucoma and neuro-ophthalmology has been secured, so I daresay that we can promise yet another fulfilling conference, for all those who can make it to Suntec City in Singapore from 15 - 17 October.

Till the next issue, then.

FOCUS Editorial Team
Dr Wong Hon Tym (Chief Editor)
Dr Jeanne Joyce Ogle (Editor)
Ms Tan Mui Leng (Secretary)
A/Prof Goh Lee Gan (Advisor)

We would appreciate your frank feedback on any part of this newsletter, be it on the format or content. Please email your comments to tei@nhg.com.sg or mail to Ms Tan Mui Leng, NHG Eye Institute @ Tan Tock Seng Hospital, 11 Jalan Tan Tock Seng, Singapore 308433. Please indicate if you would grant us the permission to publish your letter. If you would like to receive our 4 monthly newsletter, please send an email with your name to tei@nhg.com.sg with the subject heading "FOCUS Subscribe".

Evolution in Therapeutics
The treatment for infective uveitides includes antibiotics, anti-virals and antitoxoplasmosis therapy. Response to therapy is usually rapid if the diagnosis is correct. In addition, we usually need to cover the patients with topical or systemic steroids to control the intraocular inflammation that may even worsen immediately after commencing anti-microbial therapy.

The treatment of non-infectious uveitis i.e. autoimmune uveitis is usually more difficult and protracted. The long-term use of systemic steroids, with its associated side effects, is no longer acceptable and there are good evidence-based immunosuppression regimes for different uveitis entities. The use of steroid-sparing agents such as methotrexate, azathioprine, cyclosporine A and mycophenolate mofetil (cellecept) enables us to reduce systemic prednisolone to a lower and safer level. With proper monitoring, these immunomodulating agents are safe and they are our key to controlling sight-threatening uveitis conditions. The typical duration of treatment is however longer, at about 12 to 24 months. All patients on these medications are therefore counselled for the side effects and the expected duration of treatment, and monitored closely.

In addition to immunomodulating agents, biologics are a new class of drugs directed at specific immune cytokines. Biologics such as infliximab, daclizumab and adalimumab are effective but very costly, and therefore mostly used in recalcitrant uveitis when second line agents fail. Such cases are rare but if the drugs are used in a timely fashion, biologics could be the only sight saving therapy we can offer.

Despite the recent advances in molecular science, the diagnosis and management of uveitis remains challenging. As uveitis predominantly affects young and middle-aged working adults, rapid and accurate diagnosis with timely management to reduce the risk of permanent visual loss is paramount.


Syphilis - Posterior placoid chorioretinitis.
Drug-Induced Uveitis

While infective agents and autoimmune disorders are well known triggers of ocular inflammation, drugs are often-overlooked cause as the incidence is low (<0.5%). The mechanisms are unknown and may be due to direct toxicity or indirectly via immune complexes. Several drugs have been postulated to cause uveitis.

Rifabutin
A rifampin derivative, Rifabutin is used for prophylaxis and treatment of Mycobacterium avium in patients with AIDS. It has the strongest association of all drugs associated with uveitis, as suggested by a high prevalence, bilaterality, recurrence of uveitis with rechallenge, increasing severity with dose escalation, and improvement upon withdrawal.

The reaction appears to be dose- and duration-related, and may be potentiated in patients on macrolides and/or anti-fungal (azole) agents. Rifabutin-induced uveitis ranges from mild to fulminating, presenting with hypopyon, vitritis and even panophthalmitis. However, they respond well to topical corticosteroids and as such, cessation of rifabutin therapy may not be necessary.

Biphosphonates
These inhibitors of bone resorption, prescribed for osteoporosis, hypercalcemia, painful bone metastases and Paget’s disease, have long been associated with ocular inflammation.

Anterior uveitis is the most common ocular complication especially with pamidronate, and less so with clodronate and zoledronate. It typically occurs within 24-48 hours but sometimes only a week later. It ranges from mild, requiring topical corticosteroids or no therapy, to severe, requiring systemic corticosteroids. Patients should thus be monitored for 24-48 hours following pamidronate infusion.

Other complications include episcleritis, and scleritis. These may occur 1-6 days after administration, necessitating corticosteroid therapy or cessation of treatment in severe cases. It is postulated that biphosphonate-induced inflammation may be due to stimulated release of interleukins-1 and -6 resulting in lymphocyte proliferation and enhancement of immune-complex-mediated disease, and unlike rifabutin, it appears unrelated to the dose, route or activity of disease.

Cidofovir
A potent anticytomegaloviral acyclic nucleoside (cytosine) analog used in the treatment of cytomegalovirus retinitis in AIDS patients. Cidofovir has been found to cause granulomatous ocular inflammation in 20-25% of patients. Treatment with HAART may further increase this risk, possibly due to a heightened immune response. Cidofovir-related uveitis responds to topical corticosteroids, and discontinuation of therapy may not be warranted. However hypotony is a more severe side effect that limits its use.

Others
Other drugs that have been purported to induce uveitis include:

- Sulfonamides
- Quinidine
- Streptokinase
- Diethylcarbamazine
- Etanercept - an anti-tumour necrosis factor (TNF) biologic agent used with good results in rheumatoid arthritis and ankylosing spondylitis. It has been reported to cause bilateral severe non-granulomatous anterior uveitis.

In summary, biphosphonates and rifabutin are the most likely agents to cause ocular inflammation. However we should be mindful of the other reported drugs as differential diagnoses of uveitis.

Drugs documented to cause ocular inflammation

<table>
<thead>
<tr>
<th>Systemic Drugs</th>
<th>Topical Drugs</th>
<th>Intracameral Drugs</th>
<th>Vaccines</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Rifabutin</td>
<td>1. Metipranolol</td>
<td>1. Cidofovir (intravenous)</td>
<td>By Dr Stephen Teoh, NHG Eye Institute @ TTSH</td>
</tr>
</tbody>
</table>

SPOTLIGHT ON

NHG Eye Institute’s Uveitis Team

Dr Lim Wee Kiak
Fellowship-trained in the world renowned National Eye Institute, USA, Dr Lim Wee Kiak, Member of the Parliament, heads NHG Eye Institute’s Ocular Inflammation, Immunology and Uveitis Service. He is currently a world leading expert in the field and has authored 26 scientific peer-reviewed publications and 9 book chapters. He is also a member of the International Uveitis Study Group, International Ocular Inflammation Society and American Uveitis Society.

Dr Stephen Teoh
Dr Stephen Teoh completed his HMDP Fellowship at the Bristol Eye Hospital, UK, under the mentorship of Professor Andrew Dick in 2006. He undertook training in surgical vitreoretinal, uveitis, ocular immunology and inflammation. This was followed by a clinical observership in HIV-related uveal inflammation at the Wilmer Eye Institute, Johns Hopkins Hospital, USA, under the supervision of Dr James P. Dunn and Professor Douglas Johns. Dr Teoh runs his subspecialty clinics at NHG Eye Institute @TTSH, as well as the Communicable Disease Centre (CDC).

Dr Ho Su Ling
Dr Ho completed her ophthalmology surgical training programme in Ireland and was awarded the Registrar’s Prize by the Royal Academy of Medicine and the Barbara Knox Medal by the Irish College of Ophthalmologists. She was fellowship trained in Uveitis by Professor John V. Foremore, University of Aberdeen, Scotland and was instrumental in setting up the uveitis database in the clinic in Aberdeen. Her current projects include looking into pseudoexfoliation syndrome in the Asian population and planning of electronic medical record (EMR) system for NHG Eye Institute.

Dr Lennard Thean
Dr Thean, Senior Consultant @ NUHS is the team’s link to the subspeciality of uveitis. Having received fellowship training in both glaucoma and uveitis, Dr Thean, also a visiting consultant @ A4H brings a wealth of experience to both these areas. Acknowledging his expertise in the area of uveitic glaucoma, Dr Thean has been invited to give numerous talks on this topic both locally and regionally.
What is “Uveitis”? 
The wall of the human eye is comprised of 3 layers: the outermost white layer is the sclera, the innermost layer is the retina, and sandwiched between is the uvea. Within the uvea, there are blood vessels which bring nutrition to the eye. “Uveitis” (pronounced as “yoo-vee-i-tis”) is an inflammation of the uvea.

What Causes Uveitis?
- Infective agents such as bacteria, fungi, viruses or parasites.
- It can also be triggered by an overactive immune response of the eye towards injury or illness, leading to uncontrolled self-destruction of the eye tissue.
- In approximately 30% of cases, the cause is unknown.

Who is Prone to Getting Uveitis?
- The peak age falls between the third to fourth decades of life, the most productive years for many of us. This has significant socioeconomic impact on patients as well as the community.
- People who suffer from immune or “rheumatic” diseases are also at higher risk.

What are the Symptoms of Uveitis?
- Redness, pain and blurring of vision, usually in one eye.
- “Floaters” may also be experienced.
- Some patients may have associated joint pains, rashes or fever.

The uvea (coloured blue in the illustration) is composed of iris, ciliary body and choroid.
How does uveitis damage the eye?

Swelling of the Macula
The macula is the area of the retina that provides sharp central vision. Prolonged inflammation and swelling of this area in uveitis can lead to loss of central vision.

Cataract
Chronic untreated uveitis causes clouding of the lens, ie cataract (arrowed in image), leading to visual loss.

Exudative Retinal Detachment
Some types of uveitis can cause fluid to collect underneath the retina (pale, uneven patches in image) causing significant visual loss.

Bleeding in the eye
Uveitis can lead to growth of abnormal blood vessels which have a high tendency to bleed in the eye (arrowed in image), obscuring vision and causing permanent scarring.

Floaters
Inflammation of the vitreous (the “jelly” of the eye) leads multiple fluffy clumps, obscuring vision.

Glaucoma
Uveitis can cause damage to the drainage mechanism of the eye, leading to raised eye pressure. If left untreated, blindness will occur.

How do eye specialists treat uveitis?
- For most episodes of uveitis, visual loss can be prevented if prompt treatment is given.
- Blood tests and X-rays may need to be taken to determine the exact cause in severe cases.
- Eye specialists may prescribe steroids, antibiotics and painkillers either via eyedrops, tablets or injections. Very severe cases may need admission and surgery. Such patients are almost always treated in conjunction with other specialists, such as rheumatologists.

Consult your family doctor or an eye care professional if in doubt.

General Hotline: 6357 7735  Website: www.tei.nhg.com.sg
Blepharitis:
More than an irritation...

Blepharitis, a frequent cause of irritable red eyes, is a common condition. It may involve the anterior eyelid margin (anterior blepharitis) and/or the posterior eyelid margin (posterior blepharitis). The former manifests with eyelid crusting, erythema and scales around the root of lash follicles. The latter presents with inspissations of the meibomian gland orifices, duplication of the orifices, keratin plaques and telangiectastic vessels. Due to the presence of meibomian gland dysfunction, posterior blepharitis results in dry eyes due to tear film instability. The tear film breakup time is often abnormal and corneal punctate epithelial erosions may be observed in these cases.

Blepharitis is often a chronic recurring condition that is difficult to manage. It may occur de novo, but acne rosacea, a chronic skin disease involving hypertrophy of the sebaceous glands, needs to be excluded (Fig 1). The nose is commonly affected by telangiectasia, erythema and enlarged sebaceous glands units giving an appearance of a large nose (rhinophyma). Co-management with a dermatologist is advisable.

Chronic blepharitis may result in recurrent styes and chalazia (see the other article on page 7) that can be treated with warm compress and topical antibiotic cream initially. If these fail, intralesional steroid injections or an incision and drainage procedure may be considered. Rarely, the stye or chalazion causes pre-septal cellulitis, necessitating admission. Sebaceous cell carcinoma may masquerade as chronic blepharitis and needs to be excluded especially if the inflammation and chalazia are non-responsive to treatment or unilateral.

In addition, chronic blepharitis may cause trichiasis (misdirected eyelashes) due to metaplasia of the pilo-sebaceous unit of the eyelid. In severe cases, chronic inflammation and scarring result in cicatricial entropion and lash ptosis (inturned or vertical eyelashes). These complications are difficult to manage in view of the chronic nature of the underlying disease.

Epilation of the aberrant lashes usually provides only temporary relief, and electrolysis is not always successful as the lashes may re-grow. If the extent of trichiasis is large, resection of the anterior lamella of the trichiatric lash-bearing eyelid margin (after a lid split at the gray line) can be a viable option (Fig 2). Mild cases of cicatrical entropion may be managed with a recession of the anterior lamella (after a lid split) but can still recur if the underlying disease progresses. Moderate cases of upper eyelid cicatrical entropion can be treated fairly effectively with a wedge resection of the upper tarsus in conjunction with an upper blepharoplasty to evert the eyelashes (Fig 3).

Full thickness pentagonal wedge resection of the affected eyelid area has also been described. In severe cases whereby there is deficiency of the tarsus and/or deficient posterior lamella due to scarring, tarsal fracture, eversion and augmentation with a posterior lamellar graft such as nasal cartilage or hard palate may be necessary.

Chronic blepharitis should be managed aggressively to prevent these complications. Associated dry eyes should also be treated concurrently with lubricants and anti-inflammatory agents.

By Dr Yip Chee Chew, Alexandra Hospital.

**SPOTLIGHT ON**

**NHG Eye Institute’s Oculoplastics Team**

**Dr. Yip Chee Chew**
Now heading our Oculoplastics Service, Dr Yip, Head and Senior Consultant @ AH is one of the few doctors awarded a two-year Ministry of Health HMDP scholarship. He was trained at the University of Cincinnati and the University of California, Los Angeles – two prominent centres in Eye Plastic and Reconstructive Surgery. He publishes widely and has experience in the functional and cosmetic treatment of eyelid, orbital and lacrimal diseases. He is also a reviewer for the American Journal of Ophthalmology and a certified trainer and supervisor for Oculoplastics advanced surgical training, as well as a course instructor at the American Academy of Ophthalmology Annual Meeting.

**Dr. Shantha Amrith**
Dr Shantha, a senior consultant at NHG Eye Institute @ NUHS, obtained her FRCS Ophth from Royal College of Surgeons, Edinburgh in 1979, followed by two Ophthalmic Plastic and Reconstructive Surgery fellowships in Sydney Eye Hospital, Australia and University of Cincinnati, USA. She possesses a vast breadth of experience in managing a wide variety of patients with various lid, lacrimal and orbital pathologies for reconstructive as well as cosmetic surgery.

**Dr Gangadhara Sundar**
Dr Ganga, a consultant at NHG Eye Institute @NUHS, is active in furthering the cause of the specialty in the South-East Asian region. His special interests include pediatric oculoplastics, aesthetic and functional reconstructive surgery, anophthalmic sockets and orbital reconstruction.

**Visiting Consultants**
Dr Raymond Phua, NHG Eye Institute @NUHS
Dr Fong Kee Siew @ NHG Eye Institute @TTSH
Styes and Chalazia

Styes and chalazia are common problems affecting both the young and elderly, who may present either to the GP or the ophthalmologist.

A stye, also known as an external hordeolum, is a lash follicle abscess caused by Staphylococcus aureus infection of the follicle and its associated gland of Zeis or Moll. It is common in patients with a history of blepharitis. A chalazion is a chronic lipogranulomatous inflammatory lesion caused by blockage of the meibomian gland orifices with stagnation of sebaceous secretions. Acne rosacea or seborrhoeic dermatitis are skin disorders commonly associated with recurrent chalazion formation.

The typical presentation of a stye is an acute and tender swelling in the eyelid margin (Fig. 1) which appears as a small pus-filled lump pointing through the skin, and it can cause pain and redness. The history given for a chalazion is usually that of a small, gradually enlarging painless visible lump in the eyelid. This may occur months prior to the patient’s consultation at the clinic. The chalazion usually appears on the internal surface of the eyelid (i.e. on the conjunctival side) as a non-tender roundish lesion. Eversion of the lid may show a polypoidal granuloma if it has ruptured through the conjunctiva.

The initial management for a small stye would be lid hygiene, application of warm compress, and an antibiotic ointment such as chlortetracycline, fusidic acid or tobramycin in young children. Most styes would self-drain, although if it points at a lash follicle, pulling out the eyelash would accelerate the process. If untreated, the inflammation may deteriorate to become preseptal and even orbital cellulitis, whereby systemic antibiotics are required.

Some chalazia may resolve spontaneously. If they continue to enlarge or fail to settle within a few months, then incision and curettage (I & C) can be done. Alternatively, an intra-lesional injection of triamcinolone may be considered.

I & D or I & C of the stye/chalazion can be done in the treatment room. Prepare a set containing local anaesthesia (lignocaine 2%, with or without adrenaline), a syringe with a small gauge needle (preferably 27G or smaller), a chalazion clamp, a blade, a curette and sterile gauze.

Mark the location of the lump if it is relatively small. Apply a drop of topical anaesthesia into the eye. Local anaesthesia is infiltrated into the area to be clamped. Once securely clamped, an incision is made across the swelling (parallel to the lid margin if the lump “points” cutaneously, and perpendicular to the lid margin if the lump “points” internally). The contents are then curedt. Upon release of the clamp, immediate firm pressure is applied to the eyelid with gauze for about 5 to 10 minutes to aid haemostasis. The patient is discharged with an antibiotic ointment.

In cases of recurrent chalazia/styes, a referral could be made to the ophthalmologist for further assessment. The ophthalmologist will also assess if systemic tetracycline may be required to control the problem. It is also crucial that sebaceous gland carcinoma should be excluded in such cases.

By Dr Eugenie Poh, NHG Eye Institute@ TTSH

Distinguishing between Conjunctivitis and Uveitis

Do you know the difference?

<table>
<thead>
<tr>
<th></th>
<th>Conjunctivitis</th>
<th>Uveitis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>History</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Laterality</td>
<td>Often bilateral</td>
<td>Usually unilateral</td>
</tr>
<tr>
<td>Visual acuity</td>
<td>May be slightly reduced</td>
<td>Reduced</td>
</tr>
<tr>
<td>Pain</td>
<td>Mild discomfort, grittiness</td>
<td>Yes</td>
</tr>
<tr>
<td>Other symptoms</td>
<td>Usually none</td>
<td>Joint pains, rashes, fever</td>
</tr>
<tr>
<td>Presence of systemic diseases</td>
<td>Yes, examples include herpes zoster, multiple sclerosis</td>
<td></td>
</tr>
<tr>
<td><strong>Examination</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Redness</td>
<td>Most intense in the fornices</td>
<td>Most intense circumcorneally</td>
</tr>
<tr>
<td>Pupil reaction</td>
<td>Normal</td>
<td>Sluggish or irregular shape</td>
</tr>
<tr>
<td>Red reflex</td>
<td>Normal</td>
<td>May be reduced due to anterior chamber activity, cataract or vitritis</td>
</tr>
<tr>
<td>Discharge</td>
<td>Significant amount</td>
<td>Minimal if any</td>
</tr>
<tr>
<td>Intraocular pressure</td>
<td>Normal</td>
<td>Normal or high</td>
</tr>
</tbody>
</table>

The assumption that every red eye is conjunctivitis may lead to missing the diagnosis of uveitis. Here are a few simple tips to help differentiate between the two conditions.

By Jonah Huang, NHG Eye Institute @ TTSH
The NHG Eye Institute held its inaugural International Ophthalmology Congress at TTSH on 23-25 October 2008, in conjunction with the National Healthcare Group’s Annual Scientific Congress.

Themed “Advances in Vitreoretina and Uveitis”, the Congress was honoured to have Mr Hawazi Daipi, Senior Parliamentary Secretary, Ministry of Manpower and Health to grace its opening ceremony. Our esteemed faculty included Professors John Forresten(UK), Andrew Dick(UK) and Neil Bressler(USA). Over 300 participants from the global ophthalmology community (Indonesia, Malaysia, China, India, Hong Kong, Philippines, Thailand, Taiwan, Brunei, Israel, Italy, Australia, UK and USA) attended symposia, workshops and a free paper session.

NHG Eye Institute
International Ophthalmology Congress Free Paper Research Prize Winners

<table>
<thead>
<tr>
<th>Category</th>
<th>Category Description</th>
<th>Winners</th>
<th>Title of Paper</th>
</tr>
</thead>
<tbody>
<tr>
<td>NHG Eye Institute</td>
<td>– Allergan Research Prize</td>
<td>Dr Nicola GAN</td>
<td>Immune recovery uveitis in Singapore HIV patients on HAART with cytomegalovirus retinitis</td>
</tr>
<tr>
<td>NHG Eye Institute</td>
<td>– Allergan Merit Prize</td>
<td>Dr Francine YANG</td>
<td>Outcomes of retained lens fragments after cataract surgery</td>
</tr>
<tr>
<td>NHG Eye Institute</td>
<td>– Bausch &amp; Lomb Research Prize</td>
<td>Dr Jocelyn CHUA</td>
<td>Increased SPARC expression in primary angle closure glaucoma iris</td>
</tr>
<tr>
<td>NHG Eye Institute</td>
<td>– Bausch &amp; Lomb Merit Prize</td>
<td>Dr Johnson TAN</td>
<td>Tele-Ophthalmology: a prospective study on the quality and accuracy in the diagnosis of important causes of chronic blurred vision</td>
</tr>
<tr>
<td>NHG Eye Institute</td>
<td>– Novartis Research Prize</td>
<td>Dr Veluchamy BARATHI</td>
<td>Analysis of regulation of mouse scleral fibroblast (SF) after induction of experimental myopia</td>
</tr>
<tr>
<td>NHG Eye Institute</td>
<td>– Novartis Merit Prize</td>
<td>Dr Jay SIAK</td>
<td>Nuclear factor kappa B &amp; a potential molecular target for pterygium treatment</td>
</tr>
<tr>
<td>NHG Eye Institute</td>
<td>– Alcon Research Prize</td>
<td>Dr Ajeet WAGLE</td>
<td>Ophthalmic manifestations in dengue fever patients during epidemics caused predominantly by different dengue serotypes</td>
</tr>
<tr>
<td>NHG Eye Institute</td>
<td>– Alcon Merit Prize</td>
<td>Dr Colin TAN</td>
<td>Pneumatic retinopexy revisited</td>
</tr>
<tr>
<td>NHG Eye Institute</td>
<td>– Hoya Research Prize</td>
<td>Mr Wei Kiong NGO</td>
<td>Long-term changes in intracocular pressures after cataract extraction in non-glaucomatous eyes</td>
</tr>
<tr>
<td>NHG Eye Institute</td>
<td>– Hoya Merit Prize</td>
<td>Ms Lynn FOO</td>
<td>Pain experienced during ophthalmic laser procedures</td>
</tr>
</tbody>
</table>

Block these dates! 15 - 17 October 2009!
Look out for the 2nd International Ophthalmology Congress featuring Glaucoma & Neuro-Ophtalmology!

WHAT’S ON

NHG Eye Institute
International Ophthalmology Congress

TEST YOUR EYEQ

A 15 year old boy presented to the clinic with the complaint of a rash on his right lower eyelid.

a) What is the diagnosis?

b) What is the management?

c) What possible eye complications could it be associated with?

Answers:

1) Herpes simplex vesicular eruption on right lower eyelid
2) Oral and topical acyclovir
3) Herpetic conjunctivitis, keratitis, iritis with or without glaucoma, vitritis, retinitis

Quiz Master: Dr Eugenie Poh, NHG Eye Institute@ TTSH

Block these dates! 15 - 17 October 2009!