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CERTIFICATE COURSE FOR GENERAL PRACTITIONERS & PARAMEDIC

Commonly Encountered Infections In Daily Practice

常見的感染及傳染病

Course No. C124

Jointly organized by:

The Federation of Medical Societies of Hong Kong
香港醫學組織聯會

The Hong Kong Society for Infectious Diseases
香港傳染病醫學會

Objective: To provide updated information on commonly encountered infections in Hong Kong to local healthcare providers.

Date & Time	Topic	Speaker
3 Oct 2007	Respiratory Tract Infection 呼吸道感染	Dr. Ada Lin 連慰慈醫生
10 Oct 2007	Urinary Tract and Other Intra-abdominal Infection 泌尿系統及其他腹內感染	Dr. WS Leung 梁偉成醫生
17 Oct 2007	CNS Infection 中樞神經系統感染	Dr. Wilson Lam 林緯遜醫生
24 Oct 2007	Skin, Soft Tissue, Bone and Joint Infection 皮膚，皮下組織，骨骼及關節感染	Dr. Iris Li 李慧芯醫生
31 Oct 2007	Common Communicable Diseases in Hong Kong 本港常見的傳染病	Dr. Ada Lin 連慰慈醫生
7 Nov 2007	Infection Related to Travelers 與旅遊關聯的感染	Dr. Wilson Lam 林緯遜醫生

Date : 3 October 2007 - 7 November 2007
Time : 7:00 p.m. - 8:30 p.m.
Venue : Lecture Hall, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong
Course Fee : HK$750 (6 Sessions)
Language : Cantonese with supplemented by English
Certificate : Awarded to participants with a minimum attendance of 70%
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Editorial

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Editor

Dr. Timothy YY Lai

Patients with various eye conditions are frequently encountered in the primary care setting and it is important for general practitioners to be equipped with ophthalmic knowledge in order to offer proper management and arrange referral. In this issue of Medical Diary, members of the Hong Kong Ophthalmological Society will share their experience in the management of some common and important ophthalmic diseases.

Tearing and children with allergic conjunctivitis are two common conditions in clinical practice. In two separate articles, Dr. Dylan D.N. Chan, Dr. Alan C.K. Ng, and Dr. Woon-Man Lam will be discussing the diagnosis of these two conditions and how treatment can help to alleviate the symptoms suffered by these patients.

Since the visual system is an extension of the central nervous system, ophthalmic disorders can be one of the earliest presentations of a variety of neurological disorders. In a review article, Dr. Carmen K.M. Chan will provide an overview on some important neuroophthalmic conditions which might be encountered by general practitioners. Besides the central nervous system, other systemic disorders may also have a close interaction with eye diseases and Dr. Dexter Y.L. Leung will highlight the association between cardiovascular diseases and a specific entity of glaucoma known as normal tension glaucoma.

Myopia is the commonest ophthalmic condition in Hong Kong and patients with myopia of greater than 6 diopters are classified as high myopia. Retinal complications can be associated with high myopia, resulting in significant visual impairment and some of these sight-threatening retinal complications will be reviewed in my article. An important aspect of the visual system is the critical developmental period during childhood and any sensory deprivation during this period can result in amblyopia. The practical tips and importance of making prompt diagnosis and treatment of amblyopia will be presented by Dr. Wilson W.K. Yip and Prof. Dorothy S.P. Fan.

With the rapid advancements in ophthalmic surgical techniques and increasing patients' expectations, clinicians will need to be aware of the new treatment options for presbyopia, as well as alterative therapies for eye diseases including Traditional Chinese Medicine (TCM). Previously, the only treatment option for individuals with presbyopia is to wear glasses for near correction. Advancements in surgical techniques and instrumentations in the past decade have allowed for the development of safe and effective treatment of presbyopia and Dr. Arthur C.K. Cheng will present some of these treatment modalities. With increasing popularity of TCM among the general public, it is important for us who are practising Western Medicine to be aware of the use of TCM. Dr. Jane C.C. Yeung will be sharing with us her experience of TCM in Ophthalmology as an ophthalmologist and as an undergraduate student in TCM.

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Those with diabetes, hypertension or CHD.

Reference: 1. Data on file. Pfizer Inc., New York, NY. **Detailed information available upon request.**

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Management of Tearing in Adults

Dr. Alan CK Ng
MBBS (HK), MRCS (Edin), MMed (Ophth)(Singapore)

Dr. Dylan DN Chan
FHKAM (Ophthalmology)

Dr. Alan Ng Dr. Dylan Chan

INTRODUCTION

Tearing, also known as epiphora means overflow of tears. The clinical spectrum ranges widely from occasional overflow to constant bothersome tearing.[1] Tearing is due to a disruption in the balance between tear production and drainage. This complex system is dependent on the interaction of anatomy and physiology.

NORMAL TEAR FLOW

In order for the eye to remain healthy, it must remain moist. The lacrimal gland is a specialised gland located under the outer one-third of the upper eyelid that produces tears. Blinking spreads the tears over the surface of the eye and the orbicularis muscle "pumps" excess tears into the tear duct drainage system that drains tears into the inferior meatus of the nose (Fig 1). That is why the nose runs when one cries.

CAUSES OF NORMAL TEARING

When the tear volume exceeds that of the normal drainage capacity of the drainage system, the excess tears will "spill over" onto one's cheek. This can be due to the followings:

- OVERPRODUCTION (lacrimation) of tears due to irritation as in cases of blepharitis (infection of the eyelids), trichiasis (in-turning of the eyelashes), entropion (in-turning of the eyelid), epiblepharon (congenital anomaly of the lid which pushes the lashes against the eyeball) (Fig 2), corneal and conjunctival diseases or even reflex tearing secondary to dry eyes etc.
- PUMP FAILURE in which the pump action of the orbicularis muscle is disturbed like in cases of facial nerve palsy.
- DRAINAGE OBSTRUCTION (obstructive tearing) may occur at the level from the punctum down to the outlet of the nasolacrimal duct in the nose due to various pathologies.

ASSESSMENT OF TEARING

A careful history is essential to provide clues to the diagnosis. Irritation and redness usually signify eyelid or anterior segment (conjunctiva and cornea) pathologies whereas history of facial nerve palsy may point to a pump failure. A full ocular examination is warranted to decide whether the patient is having an overproduction of tears or suffering from an obstructive cause of tearing. In cases of suspected drainage obstruction, further examinations may be needed to define its level and nature. These tests include:

- Syringing and probing -- Syringing involves the use of a syringe filled with saline, which is injected through the punctum. Only free-flowing tear drainage will enable the patient to taste the salty water. Probing maybe required once a blockage in the tear drainage is identified from "syringing". A small probe is guided through the punctum and into the tear drainage system to locate the site of blockage. In addition, probing has a therapeutic role in the management of tear drainage obstruction. This will be discussed below.
- Jone's tests -- Flourescein is instilled in the conjunctival sac. Stain can be recovered from the nose of a patent system.
- Dacryocystography - A special X-ray/CT procedure that is done with contrast media to view the tear drainage of the eye.
- Nasal examination - To rule out any pathology in the nose.
- Further imaging -- If mass obstruction is suspected along the tear drainage system, further imaging is mandatory to rule out any malignancies.

MANAGEMENT

Treatment is directed to correct the underlying cause. This article concentrates on the management of obstructive tearing.

Acute Dacryocystitis

If the lacrimal gland is producing tears properly but the "tear duct" that drains the tears from the eye into the nose becomes nonfunctioning, obstructive tearing occurs. In addition to the bothersome symptoms of tearing as discussed above, the stagnant tears within the system can create a conducive environment to the development of an infection. An acute infection with the "tear duct" causes a painful swelling in the inner corner of the eyelids, which is known as dacryocystitis (Fig 3). The infection can spread to involve surrounding structures.

This is a medical emergency in which systemic antibiotics are necessary. An incision and drainage of the lacrimal sac is needed in some cases. Traditionally, the infection and inflammation of acute dacryocystitis secondary to nasolacrimal obstruction should be well controlled before a definitive dacryocystorhinostomy

(discuss below) is to be done. With the advent of endoscopic technique, primary treatment of dacryocystitis with endoscopic (endonasal) dacryocystorhinostomy is a suggested way of management.[2]

Chronic Tearing Secondary to Obstructive Causes

(A) Surgical management

If tearing per se causes severe symptoms, surgery can be performed to re-establish the drainage system. This depends on the level of the obstruction.

(I) At the level of the punctum (punctal stenosis):

Stenosis of the lacrimal punctum can be due to numerous causes including infections like herpes simplex, herpes zoster, chlamydia, actinomyces, human papilloma virus etc. Direct or thermal trauma, usage of topical or systemic chemotherapy and irradiation may also lead to stenosis of the punctum. Some underlying causes should be managed and stabilised prior to definitive surgical correction.

The surgery of choice is punctoplasty (to open up the punctum) (Fig 4). Assuming the rest of the system is free of pathology, this procedure re-establishes the normal tear drainage. The punctum is found with a punctum seeker and dilated sufficiently. Scissors are then used to excise the edge of the punctum with a three-snips manouvre.[3] This maintains a patent punctum for tears to drain through.

(II) At the level of the nasolacrimal duct (Nasolacrimal Duct Obstruction):

Obstruction of the nasolacrimal duct is most commonly due to idiopathic or involutional causes. Infectious, cicatricial, traumatic and neoplastic causes are rare.
The definitive management of such an obstruction is done by creating a new tear duct by making a hole in the bone (lacrimal bone and part of the frontal process of the maxilla) between the tear sac and the middle meatus of the nose. This operation is called "dacryocystorhinostomy" which can be performed externally or endoscopically, depending on the availability of equipment and expertise.

External Dacryocystorhinostomy

This procedure is done from outside, thus termed "external" dacryocystorhinostomy. A small incision of about 15-20mm is usually placed medial to the medial canthus (the corner of the eye). An ostium is then made on bones underlying the lacrimal sac by a bone rongeur. The lacrimal sac is incised and then connected to the nasal mucosa by stitches, creating a new tear drainage pathway. Silicone tubes (stents) are sometimes placed in the newly created tear drainage pathway for a few months to prevent scarring of the tear duct, which might otherwise result in failure of the surgery. Success rate of this procedure is high. Most studies report success rates of approximately 90% or higher despite variations in technique and exclusion criteria.[4]

Endoscopic Dacryocystorhinostomy

Nowadays, with the use of nasal endoscopes, surgeons can perform the procedure from inside. A 3.5mm rigid endoscope is inserted into the nasal cavity to the middle meatus (Fig 5). The nasal mucous membrane is incised and removed or retracted, to allow for the creation of an ostium on the lacrimal bone and the adjacent bones mechanically by bone rongeur. A vertical incision is made in the lacrimal sac, which creates a new drainage pathway for tears to drain into the nasal cavity (Fig 6). Silicone tubes can be inserted to assist long-term patency just as in external dacryocystorhinostomy. Proponents of endoscopic dacryocystorhinostomy cite increasing success rates approaching that of external dacryocystorhinostomy. The major advantage of the procedure over the external approach is the avoidance of an external scar[5] and also preventing the complication of cribiform plate fracture. Camera et al recently reported a success rate of 99% with the adjunctive use of mitomycin C.[6] Some surgeons suggest the use of laser to create the bony ostium in endoscopic dacryocystorhinostomy, but its reported success rate is much lower than mechanical endoscopic dacryocystorhinostomy.

(III) At the level of the canaliculus:

Canaliculus is the part in the tear drainage system that links up the punctum and the lacrimal sac. Canalicular malfunction may occur in cases with physical or irradiational injuries, infections such as herpes simplex or trachoma and systemic diseases like Steven Johnson Syndrome involving the canaliculus. Many of these cases can be successfully managed with an external or endoscopic dacryocystorhinostomy as mentioned above with meticulous dissection of the obstructed canaliculus together with placement of silicone tubes. However, some cases may require a by-pass surgery. This is done by inserting a by-pass tube, known as Lester Jones' tube, which connects the globe surface directly to the middle meatus of the nose via an ostium created by a dacryocystorhinostomy. The Lester Jones' tube is a tube of about 15 millimetres in length and a few millimetres in width, and is hollow. It allows tears to run into the nose through it. When in its final position it is nearly invisible. (Fig 7)

(B) Non-invasive management

Probing of the tear drainage system may be offered to patients who do not wish to undergo a formal surgical procedure. A probing procedure is an office procedure, which takes about 10 minutes. A dilator first dilates the punctum. Then a thin, blunt metal wire is gently passed through the tear duct to open any obstruction. Sterile saline is then irrigated through the duct into the nose to make sure that there is an open path (Fig 8). It has the merits of low morbidity rate, ease of use and low cost.[7] However, its major draw back is the high failure rate and rate of recurrence. Tsai et al[8] suggests the use of anti-metabolite in this procedure, which increased the success rate at least in the first 9 months post operatively.

Balloon catheterisation of the lacrimal duct is another option. In this procedure, a tube is advanced through the blocked tear duct, utilising an inflatable balloon to help dilating the drainage pathway into the nose. The inflatable balloon is similar to the type of balloon used in coronary artery angioplasty procedures. Silicone tubing is then inserted in the dilated tear duct system, which is generally removed 4 to 6 months later. Again, this procedure has a relatively high failure rate.[9]

CONCLUSION

The management of tearing in adults depends on the cause of the symptom. It is important to discuss with patients about possible diagnostic tests that may be necessary to evaluate the condition and their possible results. Patients are to be well informed of the treatment protocols and options.

Fig 1 Normal tear drainage

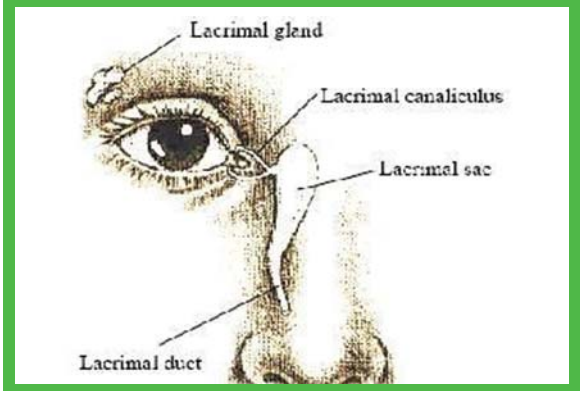

Fig 2 Irritating eyelashes causing tearing

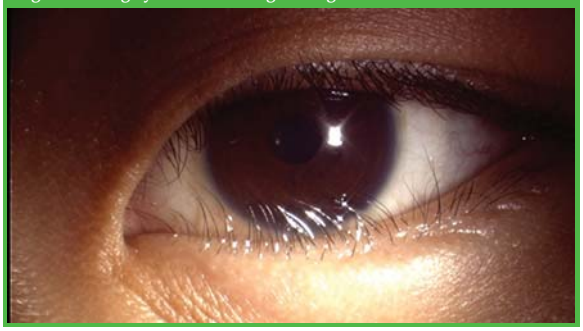

Fig 3 Acute dacryocystitis

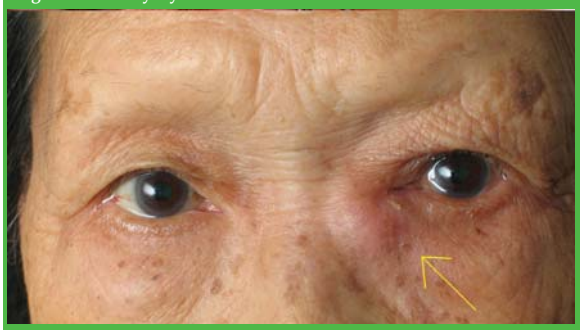

Fig 4 Punctoplasty

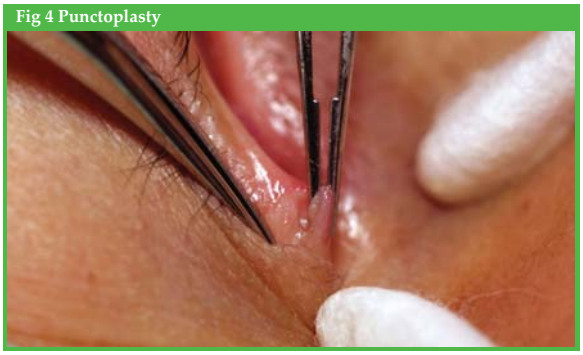

Fig 5 Endoscopic dacryocystorhinostomy

Fig 6 Creation of the bony ostium (endoscopic view)

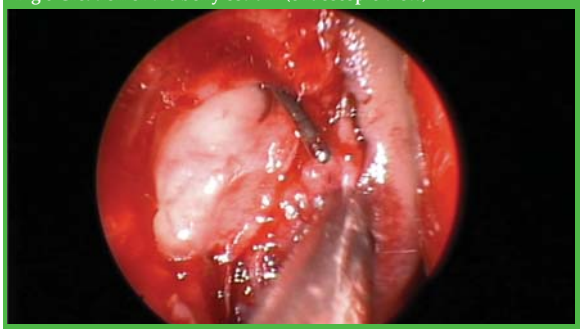

Fig 7 Lester Jones tube in situ

Fig 8 Upper - Cannular for saline injection. Middle - Punctum dilator. Lower - Lacrimal probe

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Botulinum toxin type A (BTA) is a purified protein, extensively tested for safety and administered in miniscule quantities into very specific muscles to weaken them and relax wrinkles. A simple non-surgical procedure that, within days, smoothes moderate to severe lines relaxing the muscles of the forehead, brows and crows feet. This intensive workshop will introduce BTA's applications. Emphasis is placed on Facial Anatomy, mechanism of action, storage, preparation and varied dilution methods. We will explore several techniques for BTA cosmetic and other injection methods. Safety and efficacy issues will be discussed. You will identify and perform facial mapping areas to be injected on each other.

a. Introduction
b. Indications and consent
c. Selection of patients
d. Complications
e. Advice pearls
f. Live demonstration and hands-on

3. Skin fillers

This intensive training workshop will focus on the art and science of Dermal Filler injection techniques. Emphasis will be on establishing patient selection criteria for injectable fillers, understanding indications for appropriate use of injectable fillers, contraindications, understanding potential risks and proper injection techniques.

a. Introduction and types
b. Indications and consent
c. Selection of patients
d. Complications
e. Advice pearls
f. Live demonstration and hands-on

Course fee **US$800**

CERTIFICATE COURSE IN CHEMICAL PEELING

Day two – 5 Oct 07

This session will highlight the use of superficial chemical treatments. Understanding the skin and common skin conditions is critical to the safe application of peel solutions. The assessment, evaluation, and classification of skin types as well as pathophysiology, anatomy and physiology of the skin are also featured. Emphasis will be on how facial skin and other body skin differ, the ageing process, and what works and what doesn't. You will have an understanding of product ingredients, how they work and how to choose the right treatment protocol for your clients. You will learn about chemical peels, eg, glycolic, salicylic, TCA, and how to use them safely.

1. Skin conditioning
   - Indications and contraindications
   - Side effects
   - Results
   - Skin regimen
2. Chemical peelings
   - Indications and patients selection
   - Contraindications
3. Peeling chemistry
   - Glycolic acid
   - Salicylic acid
   - Resorcin
   - Pyruvic
   - TCA
     - Easy Peel
     - Krystal Peel
     - Blue Peel
4. Phenol peelings
5. Other peelings
6. Complications
7. Live demonstration and hands-on
   - Glycolic Acid Peel
   - Easy Peel
   - Salicylic Acid Peel

Course fee **US$800**

CERTIFICATE COURSE IN LASERS

Day three – 6 Oct 07

This workshop will focus on the introduction and basic concepts of different IPL/Laser applications, physics, mechanism of action and parameters for permanent laser hair reduction, photofacial, lasers for tattoos, pigmented lesions, non-ablative rejuvenation, acne, resurfacing and laser vein removal. Emphasis will be on safety and efficacy issues, interpreting patient skin type, complications and patient selection procedures.

1. Introduction to and the physics of lasers
2. Laser safety
3. Types of lasers
4. Photo rejuvenation techniques
   - Intense Pulsed Light (IPL)
   - Light Emitting Diode (LED)
   - Photodynamic Therapy (PDT)
5. Physical peelings
   - Cryopeeling
   - Microdermabrasion
   - Sandbrasion
   - Dermabrasion
6. Live demonstration and hands-on

Contact

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Ocular Allergy in Children

Dr. Koon-man Lam

MBChB, MRCS, FCOphHK, FHKAM(Ophthalmology)
Ophthalmologist in private practice

Dr. Koon-man Lam

Case Scenario

In a beautiful morning in May, a 6 years old boy came in my clinic with his mother. The child was blinking frequently when he walked in. In many cases, blinking was just a bad habit in children, without any actual eye problems. Sometimes, young kids blinked because their eyelashes touched their eyes and made them itchy. In children with tendency to squint, they blinked to bring their eyes back to the forward looking direction. But it was another story for this child. His mother complained that he rubbed his eyes incessantly for some time already. He was also afraid of sunlight. Itchiness was the major symptom bothering the boy. Sometimes the eyes did get red. Moreover, he sneezed every morning and he was using some nasal spray for that already. When he was younger still, his airway was hypersensitive but was much better now. On closer look under the slitlamp, the conjunctiva was injected mildly. There were some punctate epithelial erosions on the cornea. When his upper eyelids were everted, I saw a lot of nodules arranged in a cobblestone style on the conjunctiva, which I called giant papillae. The major concern of the mother was the frequent blinking. With all the above findings, my major focus was his vernal keratoconjunctivitis.

This case history described one possible and not uncommon presentation of ocular allergy in children.

Introduction

Ocular allergy is a very common condition in children. In developed nations, it was reported to prevail at around 15 to 20% of children.[1] They present to both primary physicians and ophthalmologists, as the allergy sometimes is not easy to be treated and is associated with significant ocular problems like corneal scarring.

The most frequent presenting symptom is itchiness. In the mildest case, it can be the only hint that the child has ocular allergy as they may not have other signs. Other symptoms include red eyes, frequent rubbing of eyes, tearing, photophobia, foreign body sensation, burning and lid swelling. Many children have allergic rhinitis, eczema or asthma or all of them at the same time.

Broadly speaking, ocular allergy presents in three different clinical entities, though sometimes it may not be easy to distinguish among them. They are allergic rhinoconjunctivitis, vernal keratoconjunctivitis and atopic keratoconjunctivitis.[2]

Allergic rhinoconjunctivitis

Allergic rhinoconjunctivitis is the most common form of ocular allergy. As you can tell from the name, there are both nasal and ocular symptoms. It is a type 1 hypersensitivity reaction to airborne antigens.[3] It can recur in a seasonal pattern, with pollens being the most common allergens. It can also occur perennially, when the child is allergic to house dust mites, fungal allergens or other unknown airborne particles. Itchy eyes, lid oedema associated with sneezing and nasal discharge is a very common collection of presenting symptoms. Conjunctiva is commonly mildly injected with mild chemosis. Tiny papillae may be found on the upper tarsal conjunctiva. But giant papillae is not a feature of this disease.

It is the mildest form of ocular allergy, as it does not cause corneal ulcer or scarring or other significant ocular complications.

Vernal keratoconjunctivitis

This entity is a more severe form of ocular allergy. It is usually bilateral. Boys living under warm and dry climates are more commonly, though not exclusively, affected. It is also commonly associated with asthma, eczema or allergic rhinitis. Similar to allergic rhinoconjunctivitis, the disease can be seasonal or perennial. If it is seasonal, spring and summer are the peak seasons of occurrence. Actually, the word "vernal" means "in the season of spring". The severity of the disease usually decreases when the child grows up.

Similar to other ocular allergy, itchiness, tearing, photophobia, burning sensation are the usual presenting symptoms. Sometimes the patient may have thick mucus discharge. Papillae commonly form on the upper tarsal conjunctiva. These papillae enlarge in more severe cases, giving rise to the "cobblestone" appearance, which ophthalmologists called giant papillae. Nodules with overlying white plaques may form around the circumference of the cornea, i.e. along the limbus. They are called Trantas dots. Corneal ulcers, which ophthalmologists call shield ulcer, sometimes occur, resulting in corneal scarring. "Pseudogerontoxon", similar to arcus senilis in appearance, sometimes appears along the limbus. The corneal rigidity may also decrease in these patients, resulting in keratoconus.

The pathophysiology is more complicated than allergic conjunctivitis.[4] It involves tissue remodelling, with

eosinophils and lots of other immunomediators participating, forming the giant papillae.

Atopic keratoconjunctivitis

This is potentially the most serious entity among the three forms of ocular allergy. The patient usually has atopic dermatitis. The disease may not resolve with age, as vernal keratoconjunctivitis may do. Some serious complications, like keratoconus, cataract and retinal detachment are associated.

The skin of the eyelids is typically thickened, macerated and fissured. Blepharitis is a common association. Inferior conjunctiva is more commonly affected with conjunctival injection, chemosis and papillae formation. Conjunctival scarring causing symblepharon may occur in advanced cases. Corneal ulcer and scarring may also occur similar to vernal keratoconjunctivitis.

Treatment of Allergic Conjunctivitis

Treating ocular allergy involves a stepwise approach. Avoidance of allergens is the first step, though in many cases no specific allergen can be identified.

In the mildest case of allergic conjunctivitis, with just mild itchiness and conjunctival injection, topical antihistamine with or without mast cell stabiliser may be the only required medications. Antihistamine eyedrops like levocabastine, emedastine, antazoline, cause faster relief of symptoms than pure mast cell stabilisers like sodium cromoglycate, nedocromil, lodoxamide. Mast cell stabilisers are not effective in controlling acute exacerbations but may have some prophylactic effect and can be used as steroid sparing eyedrops. Artificial tears may wash away the allergens and relieve the symptoms a bit. Cold compress may also have some soothing effect. Systemic antihistamine, like chlorpheniramine, may be helpful when there is lid oedema or nasal symptom.

Since the pathophysiology of ocular allergy may involve other immunomediators and cellular mediators, medications combining the actions of antihistamine eyedrops and mast cell stabilisers and having other anti-inflammatory actions are developed.[5] Examples include ketotifen and olopatadine. They have antihistamine action and the effect of mast cell stabilisers at the same time. On the other hand, they can prevent activation of neutrophils, eosinophils, macrophages and reduce the release of leukotrienes, platelet activating factors and many other factors. Because they combine the actions of antihistamine and mast cell stabilising eyedrops, patients may be able to use less medications. This can improve the compliance of the patients.

In cases refractory to the above treatment, steroid eyedrops should be considered in the next step. Steroid eyedrops might also be considered at the early stage of vernal or atopic keratoconjunctivitis particularly when there is keratopathy. Cases requiring topical steroids are best to be handled by ophthalmologists due to the potential side effects of topical steroids and patients will require careful monitoring. Topical steroid is well known to cause ocular side effects like cataract and raised intraocular pressure leading to steroid-induced glaucoma. Monitoring intraocular pressure is particularly difficult in children. Some steroid eyedrops which cause less increase in intraocular pressure have been developed.[6] One example is 0.2% loteprednol. With treatment, the giant papillae in vernal keratoconjunctivitis may shrink in size as inflammation subsides, and they often do not disappear.

In the steroid-resistant cases some more aggressive treatment using topical immunosuppressant eyedrops may be required such as topical tacrolimus and topical cyclosporin[7,8]. These eyedrops are not widely available and are reserved for recalcitrant cases.

Blepharitis associated with atopic keratoconjunctivitis should also be treated properly, like using topical antibiotics and emphasising lid hygiene. When the disease is complicated by developing corneal ulcer which is resistant to medical treatment, or if there are other ocular complications, surgical management may be required.

Conclusions

Ocular allergy is a very common condition which is encountered very frequently, if not daily, even in a tertiary eye centre. The mildest cases can be treated in a primary setting. If the patient is not responsive to simple treatment, or if there are complications, prompt referral to ophthalmologists will be the next step. Treating these children usually is a prolonged process which may take even years. But it is worth all the efforts if vision impairing complications can be prevented.

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APPLIED SCIENCE

Department of Applied Science

PROFESSIONAL CERTIFICATE COURSE FOR CLINIC NURSES

Professional Certificate in Clinic Operation
診所運作專業證書

香港專業教育學院沙田分校應用科學系所舉辦的第一屆【診所營運專業證書課程】已於2007年7月正式畢業。本課程旨為在職之診所醫護人員提供一個全面的持續進修及提升醫療管理的途徑，從而配合香港市民對醫護界的高質素要求。課程設計共一百個小時，內容包含護理學、配藥學、顧客服務、診所管理及資訊學，因課程中的理論與實習互相配合，使學員容易掌握重點之餘並學以致用。此外,為使學生有更靈活的學習空間，本課程更提供了網上學習模式，同學即使留在家中亦可温習及做練習。加上沙田專教學院提供完善設備的課室及實驗室上課，使學員有良好的學習環境，從而提高了學習的效率

相片一: 方主席在畢業禮上發言，向畢業生勉勵

在結業禮上學生代表陳碧瑜小姐道出「即使我從事醫護行業已二十多年，這課程不但令我可以温固知新，還可以使我學習到一些忽略了的知識，使我更充實，縱使課程比想像中吃力…..」此外，香港醫學組織聯會主席方道生醫生亦提出「社會不斷的進步，病人對醫護人員質素的要求亦不斷提高，這課程的而且確提供一個好好的機會給在職的醫護人員，我們期待這課程可幫助更多的同工……」在此我們祝願畢業生可以在自己的工作崗位作出更大的貢獻，亦期望有更多的醫生鼓勵及支持員工的進修，相信最終會有更多病人及有需要的人士得益。第二屆【診所營運專業證書課程】將於本年十月開課，有興趣人士請登入http://stas.vtc.edu.hk 或 http://www.fmshk.org/courses/index.htm，下載報名表格。

相片二: 全體畢業生與老師及嘉賓合照

相片三: 沙田專教院署理院長祁小姐、學生代表陳小姐、醫學組織聯會主席方醫生、應用科學系系主任陳小明合照

Neuro-ophthalmology for General Practitioners: A Revision

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This article has been selected by the Editorial Board of the Hong Kong Medical Diary for participants in the CME programme of the Medical Council of Hong Kong (MCHK) to complete the following self-assessment questions in order to be awarded one CME credit under the programme upon returning the completed answer sheet to the Federation Secretariat on or before 30 September 2007.

What is neuro-ophthalmology?

The term "Neuro-ophthalmology" (眼神經科) is probably unfamiliar to most people, including a lot of doctors. In the higher surgical training curricula of the Royal College of Ophthalmologists (UK) and the Royal College of Surgeons of Edinburgh, it is one of the seven established subspecialties of ophthalmology, the others being oculoplastic; cornea; cataract and refractive surgery; glaucoma; retina and paediatric ophthalmology.

Neuro-ophthalmologists mainly deal with optic nerve disorders, abnormalities of the pupil, extraocular movement, eye lid position/movement and intracranial conditions which affect the eyes. In addition, patients with visual loss of unknown cause often end up in neuro-ophthalmology clinics. Essentially neuro-ophthalmology is the fusion between neurology and ophthalmology. In the US, neuro-ophthalmologists can either be neurology-or ophthalmology-trained. Currently neuro-ophthalmology services are offered at some Hospital Authority eye clinics and patients are usually referred from the general eye clinic of the respective hospitals, like other subspecialty services.

Relevance of Neuro-ophthalmology to the General Practitioner

Although GPs are not expected to manage neuro-ophthalmological conditions by themselves, they have an important role to play as the gatekeeper: by making a basic assessment and referring appropriately, they can potentially not only save the sight but also the life of a patient.

The diagnosis of a lot of neuro-ophthalmological conditions can be made by taking a good clinical history. Often all the equipment required to make a basic assessment in a busy GP clinic are a pocket Snellen chart, pen torch and an ophthalmoscope (see Table 1). We will see how this is applied to specific clinical situations below.

Table 1: The Basic Neuro-ophthalmological Assessment

- History
- Inspection
- Examination (directed)
  - Visual acuity
  - Pupils
  - Extra-ocular movement
  - Visual field
  - Fundus
- Other associated signs (other cranial nerves)

Specific clinical situations:

A. Visual loss

In the history, ascertain whether the visual loss is in one or both eyes, the onset and duration; and whether there are any associated symptoms. Patients are often unable to distinguish between visual loss in e.g. in the right eye or the right side of visual field of both eyes. One should therefore ask them to cover either eye to see if the visual problem persists. Past medical history, particularly diabetes and hypertension, is relevant, since they can cause diabetic/hypertensive retinopathies and are risk factors for e.g. retinal artery/vein occlusion and non-arteritic anterior ischaemic optic neuropathies (the risk of developing these conditions is further increased by smoking). A drug history may be contributory: it is well recognised that ethambutol and isoniazid can cause optic neuropathy and chloroquine/ hydroxychloroquine can cause retinopathy.[1] More recently it has been identified that erectile dysfunction drugs[2] and amiodarone[3] may be also be associated with optic neuropathies and vigabatrin can lead to visual field defects.[4]

In the examination, apart from testing the visual acuity of each eye (with appropriate glasses and pinhole if necessary), the pupillary light reflex is very important as it is one of the only objective signs of optic nerve dysfunction. There is no relative afferent defect if the visual loss is bilateral and equal; and cortical visual loss (posterior to the midbrain in the visual pathway) does not affect the pupillary reaction. Testing of visual fields is particular relevant if the patient complains of bilateral visual loss.

If the patient has unilateral visual loss, it is most appropriate to refer to the ophthalmologist, and urgent

referral is warranted if e.g. retinal detachment or temporal arteritis is suspected. However if the patient has homonymous visual field defects in both eyes, particularly if there are other neurological deficits, referral to a neurologist or an accident and emergency department may be appropriate, depending on the clinical situation.

B. Extraocular movement abnormalities

These often manifest as diplopia. It is a priority to ascertain whether the diplopia is monocular or binocular: ask the patient to cover up either eye and see if the double vision persists. If it does, this is monocular diplopia, most likely caused by a problem with the optic media in the affected eye and referral to an ophthalmologist is warranted. If the diplopia is binocular then use the approach in Table 2 to further narrow down the diagnosis.

Table 2: Clinical Approach to Diplopia

- History
  - Monocular or binocular?
  - Vertical or horizontal?
  - Onset and duration
  - Variable?
  - Associated problems e.g. ptosis?
- Examination
  - Inspection (squint?)
  - Cranial nerve pattern or not?
  - Lids and pupils
  - Other cranial nerve functions:5, 7, 8

The effects on extraocular movement resulting from third (oculomotor), fourth (trochlear) and sixth (abducens) nerve palsies would be well known to the reader. In terms of symptoms, sixth nerve palsy results in a convergent squint and horizontal diplopia, which is worse when looking at distant objects than at near. It is the most common isolated cranial nerve palsy and it can also be a false localising sign i.e. increased intracranial pressure can cause sixth nerve palsy without a lesion along the nerve itself. Third and fourth nerve palsies will result in vertical or oblique diplopia. Traditionally, third nerve palsies are divided into 'medical' or 'surgical', depending on whether the pupil is involved. However 'surgical' third nerve palsy can spare the pupil.[5] Therefore, in clinical practice, unless the patient is in the typical age group (middle aged or elderly), with vasculopathic risk factors and presents with a complete, pupil-sparing third nerve palsy, a 'surgical' cause has to be considered and prompt, appropriate imaging is warranted.

If the extraocular movement abnormalities fall into a cranial nerve pattern, it is important to check for other associated neurological defects, which can pinpoint the location of the lesion (see Table 3).

Table 3: Localization of lesions causing 3rd/ 4th and 6th nerve palsies

Neurological signs	Probable location of lesion
Cranial nerves 2, 3, 4 and 6 palsies + proptosis	Orbit
Cranial nerves 3, 4, 5 (ophthalmic division) and 6 palsy	Cavernous sinus
Cranial nerves 5, 6, 7 & 8 palsies + cerebellar signs	Cerebellopontine angle

If the extraocular movement defects do not fall into a cranial nerve pattern, it may be caused by thyroid eye disease (most common), myasthenia gravis or orbital lesions. Thyroid eye disease (ophthalmic Graves) can occur not only in the thyrotoxic state, it can also occur in patients who are euthyroid or hypothyroid. Thyroid eye disease can often be diagnosed clinically and if necessary, confirmed by the typical appearance of enlarged extraocular muscles with sparing of muscle tendons on computer tomography of the orbit. In recent years, smoking has been identified to be the most important risk factor for developing eye disease in patients with Graves disease.[6]

Ocular myasthenia gravis (OMG) will result in variable diplopia and ptosis and the pupil is never involved. Traditionally OMG can be diagnosed with the Tensilon test. However, more recently the 'Ice pack' test has largely replaced Tensilon test for the clinical diagnosis of OMG in patients with ptosis. It involves placing an ice pack on the ptotic eye for 2 minutes which should improve the ptosis. It is easier and safer to perform than the Tensilon test, has a high specificity and sensitivity[7], but it is of limited use in patients with only subtle extraocular movement limitation without ptosis.

C. Unequal sized pupils (Anisocoria)

The pupil size is controlled by the sympathetic and parasympathetic nervous systems. Disruption of the sympathetic pathway will result in Horner's syndrome, which manifests as a small, poorly reactive pupil (miosis), mild ptosis +/- lack of sweating on the ipsilateral side of the fact (anhydrosis). Disruption of the parasympathetic pathway will result in an enlarged pupil, e.g. in pupil-involving oculomotor nerve palsy or in Adie's syndrome. Adie's syndrome (also called Homes-Adie tonic pupil syndrome) is a condition which typically affects young adult females, unilateral in 80% of the cases,[8] and manifests as a large pupil, which is poorly reactive to light, but reacts to accommodation ("light- near dissociation"). This condition may be associated with hyporeflexia or areflexia.

The most common causes of unequal sized pupils seen by ophthalmologists are local causes at the iris itself- e.g. traumatic/ iatrogenic damage to the iris, dilating eye drops, glaucoma eye drops (pilocarpine), acute iritis etc. In particular, a red painful eye, associated with headache, nausea, hazy cornea and a poorly reactive, semi-dilated pupil should suggest acute angle closure glaucoma and warrants urgent referral to an ophthalmologist or an accident and emergency department.

D. Ptosis

The classification of the causes of ptosis or droopy eyelids can be simplified into 1) muscular/ mechanical, 2) neurological (third nerve palsy and Horner's syndrome) and 3) neuro-muscular (myasthenia gravis). When seeing a patient with ptosis, the following aspects of the history need to be ascertained:
a) Whether the ptosis is unilateral or bilateral
b) The duration- old photos including the photo on the HKID card, give a good clue
c) Whether it is associated with abnormalities of the pupil and extraocular movement; and
d) Whether it is variable, which might suggest myasthenia gravis

The most common cause of ptosis in the elderly is aponeurotic ptosis, which results from dehiscence, disinsertion or stretching of the levator aponeurosis. It is usually insidious in onset, bilateral, and affected patients will have widened upper lid creases, deep superior sulci with good preservation of levator

function. We also have to be aware that local causes, e.g. severe conjunctivitis can cause lid swelling and mild ptosis in the acute stage, as can a retained contact lens.

E. Optic disc abnormality

In practice, it is difficult to get a clear view of the optic discs through undilated pupils and with a direct ophthalmoscope, at it is the case in the GP's office or at the accident and emergency department.

'Papilloedema' is a term reserved for bilateral optic disc swelling secondary to intracranial pressure. Although it may be associated with transient visual obscurations, the vision usually remains normal until the late stage and there may be other signs or symptoms of increased intracranial pressure.

Optic disc swelling due to all other causes are simply described as "disc swelling", even when it is bilateral. Disc swelling can be 'real' and pathological, for example in anterior ischaemic optic neuropathy, optic nerve compression, malignant hypertension or optic neuritis. However it is to be noted in typical idiopathic optic neuritis and that associated with multiple sclerosis, about two thirds are 'retrobulbar' in nature[9] i.e. although the patient complains of visual loss with objective visual dysfunction (i.e. relative afferent pupillary defect), the disc appearance can be entirely normal- to put it succinctly, it is a condition in which 'the patient sees nothing and the doctor sees nothing;'

There are some patients with physiologically elevated discs, which is termed 'pseudo- disc swelling'. This can occur in patients with hypermetropia, tilted discs or patients with optic nerve head drusen. Usually these patients are asymptomatic and the 'disc swellings' are incidental findings. Distinguishing between real and pseudo-disc swelling sometimes pose difficulties even for an experienced ophthalmologist, therefore general practitioners are certainly are not expected to make the distinction and referrals to ophthalmologists are warranted if optic disc swelling is suspected.

Urgent neuro-ophthalmological conditions

Of particular importance to the general practitioner are urgent neuro-ophthalmological conditions which warrant prompt referral and treatment. (Table 4)

Table 4: Urgent neuro-ophthalmological conditions

Conditions	Clues to diagnosis
Temporal arteritis	Elderly, general malaise, weight loss Headache, scalp tenderness, jaw claudication Tender and pulseless temporal arteries Visual disturbance (transient or persistent) Raised ESR and CRP
Intracranial aneurysm (Especially of posterior communicating artery)	'Surgical' 3rd nerve palsy: incomplete/ pupil involving/ headache Atypical age group and no vasculopathic risk factors
Carotid dissection	Acute onset Horner's syndrome Painful neck (Vertebral dissection can also cause Horner's syndrome, but maybe associated with other signs of lateral medullary syndrome)

Conclusion

A lot of neuro-ophthalmological diagnoses can be made by taking a careful history and by performing a directed examination using basic tools available at the GP's office. The general practitioner can really make a difference by making appropriate referrals after a preliminary assessment. Save sight, save life and impress your colleagues with your diagnostic skills!

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MCHK CME Programme Self-assessment Questions

Please read the article entitled "Neuro-ophthalmology for General Practitioners: A Revision" by Dr. Carmen KM Chan, and complete the following self-assessment questions. Participants in the MCHK CME Programme will be awarded 1 CME credit under the Programme for returning completed answer sheets via fax (2865 0345) or by mail to the Federation Secretariat on or before 30 September 2007. Answers to questions will be provided in the next issue of The Hong Kong Medical Diary.

Questions 1-10: Please choose the best answer:

1. The following drugs are recognised to be associated wtih optic neuropathies, except
A. Sildenafil
B. Amiodarone
C. Hydroxychloroquine
D. Isoniazid
E. Ethambutol

2. Diplopia
A. Monocular diplopia is caused by a problem with extraocular movements
B. Sixth nerve palsy causes vertical diplopia
C. Sixth nerve palsy causes a divergent squint
D. Isolated trochlear nerve palsy causes vertical diplopia
E. A young healthy person with a pupil-sparing, incomplete third nerve palsy does is not need further investigation because it is a 'medical' third nerve palsy

3. **Extraocular movement abnormalities:**
   A. Thyroid eye disease typically causes enlargement of extraocular muscles with sparing of muscle tendons on CT scans of the orbit.
   B. The most important risk factor for developing eye disease in patients with Grave's disease is the control of thyroid functions
   C. Thyroid eye disease only affects patients who are thyrotoxic
   D. In ocular myasthenia, the ptosis is made worse by placing an ice pack on the lid.
   E. The Ice Pack test is best suited for cases of ocular myasthenia with subtle extraocular movement abnormalities

4. **Pupils**
   A. Visual loss due to occipital lobe lesions causes an afferent pupillary defect
   B. Horner's Syndrome results in mydriasis (dilated pupil)
   C. The pupil in Adie's Syndrome is usually small.
   D. 'Surgical' third nerve palsy always involves the pupil
   E. A fixed dilated pupil can be a presentation of acute angle closure glaucoma

5. **Which of the following conditions is NOT a cause of anisocoria (unequal pupil size)?**
   A. Acute iritis in one eye
   B. Acute angle closure glaucoma in one eye
   C. Isolated optic atrophy in one eye
   D. Third nerve palsy in one eye
   E. Horner's Syndrome in one eye

6. **Adie's Syndrome affects both eyes in what percentage of patients?**
   A. 10%
   B. 20%
   C. 50%
   D. 80%
   E. 90%

7. **The following may be a cause of ptosis except:**
   A. Isolated fourth nerve palsy
   B. Horner's Syndrome
   C. Conjunctivitis
   D. Myasthenia gravis
   E. Retained contact lens

8. **Aponeurotis ptosis: the following statements are true except**
   A. The upper lid crease is widened
   B. It usually affects the elderly
   C. It is usually bilateral
   D. The levator function is poor
   E. It is insidious in onset

9. **Optic disc swelling**
   A. Idiopathic optic neuritis causes disc swelling in most cases
   B. Papilloedema can be caused by anterior ischaemic optic neuropathy
   C. Patients with elevated discs due to optic nerve head drusen usually have visual symptoms
   D. Papilloedema is usually associated with profound visual loss in the early stages
   E. Papilloedema is usually bilateral

10. **The following may suggest a diagnosis of temporal arteritis:**
    A. Elderly patient
    B. Recent weight loss
    C. Jaw claudication
    D. Tender temporal arteries
    E. All of the above

ANSWER SHEET FOR SEPTEMBER 2007

Please return the completed answer sheet to the Federation Secretariat on or before 30 September 2007 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Neuro-ophthalmology for General Practitioners: A Revision

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1 [ ] 2 [ ] 3 [ ] 4 [ ] 5 [ ] 6 [ ] 7 [ ] 8 [ ] 9 [ ] 10 [ ]

Name (block letters):_____ HKMA No.: _____

HKID No.:___ ___ - ___ ___ ___ ___ X X (x)

Other Membership No. (please indicate): _____

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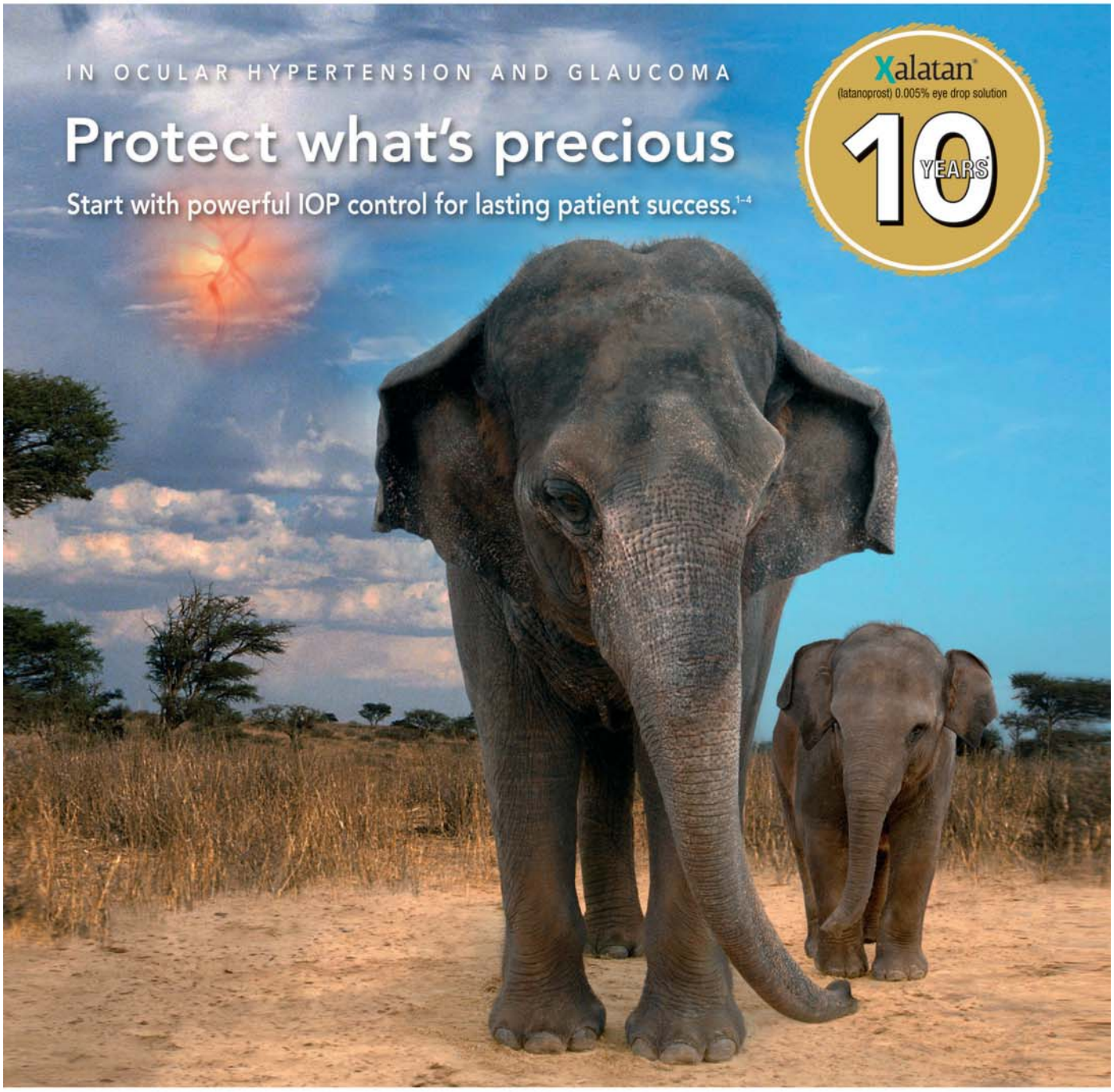
Answers to August 2007 issue

Systemic Adjuvant Therapy for Invasive Breast Cancer

1. F 2. F 3. F 4. T 5. F 6. T 7. T 8. T 9. T 10. F



XALATAN is indicated for the reduction of elevated IOP patients with open-angle glaucoma (OAG), ocular hypertension (OH) and chronic-angle closure glaucoma (CACG)[5].

XALATAN should be used with caution in aphakic patients, in pseudophakic patients with torn posterior lens capsule, or in patients with known risk factors for macular oedema[5].

In clinical trials with XALATAN, the most common ocular adverse effects included conjunctival hyperaemia, transient punctate epithelial erosions and increased iris pigmentation[5].

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*** Regulatory approval in Hong Kong: June 1997**

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Normal Tension Glaucoma - a Sick Eye in a Sick Body

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For centuries, glaucoma is being recognised as an irreversible optic neuropathy as a result of damages caused by an elevated intraocular pressure (IOP). Nowadays, however, we know that glaucoma occurs in patients with IOP entirely in the "normal" range (typically taken as less than 21 mmHg). For this group of glaucomas, termed as Normal Tension Glaucoma (NTG), numerous studies have demonstrated that factors other than IOP may play a role in its pathogenesis. Many of these factors are general medical diseases and hence, NTG can be of interest to our general practitioner audience. In fact, Felix Lagrange of Bordeaux was the first to note that glaucomatous optic neuropathy (GON) may in fact be a "sick eye in a sick body" as early as in 1922.[1]

Role of Blood Pressure

Kummell in 1911 was one of the first to describe the relationship between high blood pressure (BP) and glaucoma.[2] Thereafter, numerous studies demonstrated the correlation between arterial hypertension and glaucoma;[3-11] as well as between arterial hypotension and glaucoma.[10, 12-15]

Patients with NTG or primary open angle glaucomas were shown to be more likely to suffer from hypertension than normal controls. We know that the relationship between hypertension and mean ocular perfusion pressure is not straightforward and simple hypertension does not lower the mean ocular perfusion pressure. In fact, assuming the same vascular tone and intact autoregulatory mechanism, a modest increase in BP may increase the ocular perfusion pressure a bit. The ***treatment*** of hypertension, however, has been shown to be the culprit in resulting NTG and optic nerve ischaemic damages. Hayreh and co-workers performed an important work in 24-hour ambulatory BP monitoring and diurnal IOP curves. They noted a significantly lower night time mean diastolic BP and a significantly greater mean percentage decrease in diastolic BP in NTG patients. Moreover, patients with arterial hypertension taking oral hypotensive therapy who developed nocturnal hypotension as a result, were significantly associated with glaucoma visual field deterioration.[12,16] NTG patients with deteriorating visual fields have lower nocturnal BP than those who were stable in visual fields, and it was interesting to note that the other BP parameters did not differ significantly between the two groups.[17] Patients with pre-existing greater nocturnal systolic, diastolic, and mean arterial BP dips were more likely to develop visual field deterioration despite good IOP control.[18] Currently it is not entirely sure what would be the critical level of BP dip that would result in NTG and its deterioration. Further large scale prospective studies will answer this important question. For the time being, we do not recommend routine 24-hour BP check for all NTGs; however, in those NTG subjects with continual optic nerve glaucoma damage despite a good IOP control, in the presence of hypertension, we may consider a 24-hour ambulatory BP monitoring to look for possibility of a nocturnal dip (>20% from baseline is considered as large dippers). In case such a dip exists, a close liaison with our fellow physicians may be justified to fine tune to BP control to avoid the optic nerve "dying in the night".

Role of Vasospasm

Vascular dysregulation, such as inappropriate constriction or insufficient dilatation in the micro-circulation to stimuli such as coldness, has been proposed as a risk factor for glaucoma.[19] Studies have shown that patients with NTG had reduction of nail-fold capillary blood-flow velocity upon cold provocation, significantly more so than other open angle glaucomas and normal subjects.[20] A number of endothelium-derived vasoactive substances maintain and modulate the vascular tone throughout the body and the eye. In the ophthalmic vascular bed, a constant basal release of nitric oxide (NO) maintains the circulation in constant vasodilatation, whereas endothelin-1 (ET-1) was shown to cause marked vasoconstriction. Imbalance of the level of these agents, possibly in concert with various other vasoactive substances, was postulated as one of the models for pathogenesis leading to optic nerve damage.[21-22] The role of treatment with Calcium-channel blockers in this respect is uncertain. On the other hand, the use of the Chinese herbs Gingko Biloba Extract showed initial beneficial evidence[23-25] though of course much more studies will be needed.

Role of Migraine

Functional vasospasm of the brain vessels is linked to the pathogenesis of migraine. In fact, both NTG and migraine are associated with systemic vascular dysregulation.[26] Phelps and Corbett found a higher prevalence of migraine-like headaches in NTG, compared to other open angle glaucomas and normal controls.[27] These findings were supported by data from

the Blue Mountain Eye Study, which suggested association between migraine and glaucoma in patients of 70-79 years of age.[28] The Collaborative Normal Tension Glaucoma Study, one of the largest studies on NTG, analysed the risk factors for progression in the disease and found migraine to be an independent risk factor for more rapid progression.[29] In the same study, IOP lowering benefitted females with migraine but without eliminating all migraine-associated risks.[29] It may seem prudent to ask patients with NTG for symptoms of migraine. Further studies will be needed to see whether treatment of accompanying migraine is of benefit to the NTG as well.

Role of Sleep Apnoea Syndrome (SAS)

Walsh and Montplaisier were among the first who reported a combination of familial glaucoma and sleep apnoea syndrome across two generations of a family.[30] Later studies suggested a significant association between SAS and incidence of NTG and open angle glaucoma.[31-33] Respiratory Disturbance Index (RDI) during night sleep correlated with visual field loss variance and glaucomatous optic disc damages.[34] Current hypotheses include that the glaucoma damages result directly from impaired perfusion to the optic nerve head due to SAS, or alternatively from SAS-induced arterial hypertension and arteriosclerosis.[35] Patients with SAS were shown to have increased plasma ET-1 levels which may be implicated in vasoconstriction and hypoperfusion.[36]

Although currently we do not have data to justify a sleep laboratory study for SAS in all NTG patients, it may seem prudent that we should actively look for signs and symptoms of SAS in NTG subjects and liaise with specialists to treat any SAS.

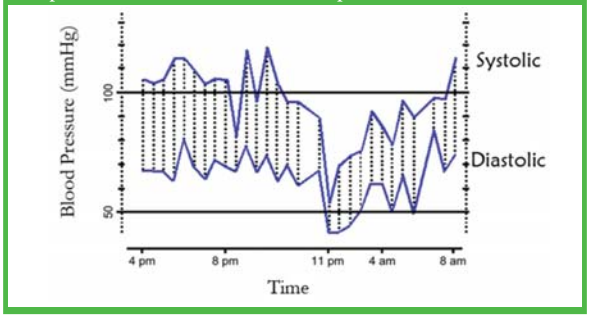
Role of Silent Cerebral Infarcts (SCI)

Stroman and coworkers performed Magnetic Resonance Imaging (MRI) in NTG subjects and found significantly more diffuse cerebral small-vessel ischaemic changes when compared to controls.[37] Later studies indicated that the prevalence of ischaemic MRI changes can be found in as much as 34% of NTG patients.[38-39] It has been proposed that these findings may reflect a vascular cause in some NTG patients, possibly related to cerebral small-vessel ischaemia. Further studies are warranted. Meanwhile, we do not recommend a routine neuroimaging for patients with NTG. However, in case neuroimaging is done for NTG to rule out any "space occupying lesion" along the visual pathway as a potential cause of visual field defect at hand, we recommend to actively look for the presence of cerebral ischaemic changes, or silent cerebral infarcts (SCI). As SCI is a potent independent risk factor for future stroke, liaison with neurologists may be indicated for stroke prophylaxis.[40]

Messages: NTG is a Sick Eye in a Sick Body

Vascular factors may play a role in pathogenesis. Therefore,
1. look for arterial hypertension and hypotension. Consider 24-hour ambulatory BP measurement and avoid nocturnal arterial hypotension.
2. look for vasospasm (e.g. Raynaud's phenomenon)
3. look for migraine, consider liaise with neurologists for treatment
4. look for Sleep Apnoea Syndrome, consider liaise with specialists for treatment
5. look for Silent Cerebral Infarcts, consider liaise with neurologists for stroke prophylaxis

Figure 1. Twenty-four hour ambulatory blood pressure monitoring in a glaucoma patient showing a typical "nocturnal dip" in blood pressure. (adopted from Pache & Flammer, Surv Ophthalmol 51: 179-212, 2006)



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Retinal Complications of High Myopia

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Introduction

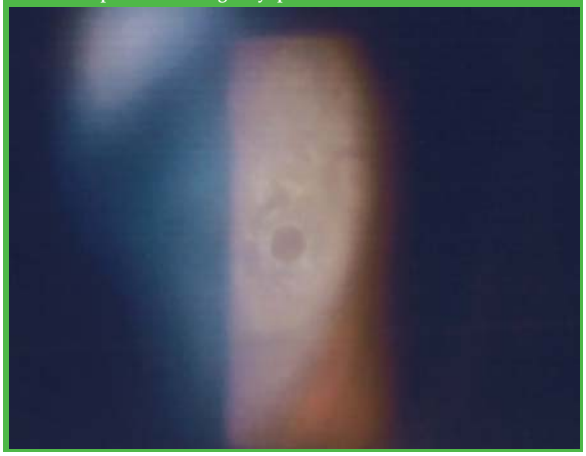
High myopia or pathological myopia is associated with globe elongation and a refractive error of at least 6 diopters (D) and/or axial length of greater than 25.5 mm.[1-3] The prevalence of high myopia varies considerably in different ethnic groups and has been estimated to be around 10% in Asian populations.[1,2] Excessive axial elongation of the globe in high myopia can cause mechanical stretching and thinning of the choroid and retinal pigment epithelium layers, resulting in various retinal degenerative changes.[4] It is well known that individuals with high myopia have increased risks of retinal complications such as peripheral retinal degenerations, retinal tears, retinal detachment, posterior staphyloma, chorioretinal atrophy, retinal pigment epithelial atrophy, lacquer cracks, choroidal neovascularisation (CNV) and macular haemorrhage.[4-6] In a cross-sectional community-based epidemiological study in Hong Kong, 56.1% and 11.3% of subjects with high myopia were found to have one or more peripheral retinal degenerative lesion or posterior pole lesion respectively.[7] Some of these retinal lesions may be associated with severe irreversible visual loss and therefore it is important for clinicians to be aware of the retinal pathologies in high myopia. This review aims to provide an overview on some of the important retinal complications associated with high myopia.

Peripheral retinal degenerations and rhegmatogenous retinal detachment

Epidemiological studies have demonstrated increased prevalence of peripheral retinal degenerations in association with high myopia and increased axial length.[4-13] Among the different types of peripheral retinal degenerations in high myopia, lattice degeneration is the most important peripheral retinal degeneration which can predispose to rhegmatogenous retinal detachment (RRD).[14] This is because retinal tears can develop at the posterior and lateral margins of the lattice degeneration caused by strong vitreoretinal adhesions following posterior vitreous detachment. Symptoms of posterior vitreous detachment and retinal break formation include sudden or gradual increase in the number of floaters and/or flashes. In patients with RRD, they may also develop symptoms of curtain-like progressive visual field loss and blurring of vision. Dilated fundus examination should be carried out in patients with these symptoms as soon as possible to detect for the development of retinal break or retinal detachment.

Laser photocoagulation is used for the treatment of eyes which have developed retinal hole or break. This can be performed in the majority of patients under topical anaesthesia as an out-patient procedure. Several rows of laser are applied onto the retina to surround the retinal defect in order to seal off the retinal break (Fig. 1). Since around 30% of eyes with acute RRD have been found to have lattice degeneration, prophylactic laser treatment can also be performed in patients with peripheral retinal degenerations,[15] especially those with a history of retinal detachment in the fellow eye.

Figure 1. Retinal hole surrounded by fresh laser photocoagulation marks in a patient with high myopia



In eyes with retinal detachment, laser photocoagulation alone is insufficient to treat the condition and vitreoretinal surgery is required. Surgical modalities for RRD include pneumatic retinopexy, scleral buckling surgery with cryopexy, and pars plana vitrectomy with intravitreal tamponade such as gas or silicon oil. The goal of the surgery is to identify and seal off all retinal breaks. For patients in whom the macula is still attached, they will generally have favourable visual outcome postoperatively. However, for patients in which the central of the macula i.e. the fovea is detached, the visual prognosis of the patient is more variable and some patients might develop irreversible visual loss despite successful retinal detachment surgery. Therefore, prompt ophthalmic consultation is advised for early detection of retinal detachment in order to prevent irreversible visual loss.

Myopic foveoschisis and macular hole

Due to excessive axial elongation of the globe in high myopia, patients can develop posterior bulging or ectasia

of the globe known as posterior staphyloma. Recent advancement in retinal imaging technology using optical coherence tomography (OCT) has demonstrated that highly myopic patients with posterior staphyloma are predisposed to develop macular pathologies such as myopic foveoschisis and macular hole. Myopic foveoschisis is the splitting of the retinal layers in the macula and can result in metamorphopsia and blurring of vision.[16] Macular surgery may be performed in myopic foveoschisis to prevent further deterioration of vision.[17,18] In more advanced stage, myopic macular hole can develop which may be associated with retinal detachment and patients will suffer from severe visual loss with reduced visual acuity. Various surgical procedures have been performed for macular hole with or without retinal detachment and they include pars plana vitrectomy with gas or silicone oil tamponade, macular buckling, and scleral shortening surgeries.[19-21] However, despite these interventions, reopening of the macular hole and retinal redetachment may still develop and some patients will require multiple surgeries to achieve attachment due to the loss of chorioretinal tissue and retinal pigment epithelial atrophy.

Lacquer cracks

Lacquer cracks are formed by spontaneous ruptures in the Bruch's membrane and small haemorrhages may develop within the lacquer cracks. Lacquer cracks predispose patients with high myopia to have sudden visual loss as macular CNV may develop in close proximity to the lacquer cracks. Small ingrowth of fibrovascular tissue may also give rise to small elevated pigmented circular lesions and are known as Fuchs' spots.[22]

Choroidal neovascularisation in high myopia

Among various lesions associated with high myopia, macular CNV is one of the most vision threatening complications(Fig. 2).[23] It develops in around 5 to 10% of eyes with high myopia and is the commonest cause of CNV in young individuals and accounts for around 60% of CNV in young patients aged 50 years or younger.[24-26] The incidence of myopic CNV in patients with pre-existing myopic CNV in the fellow eye is even higher, as more than 30% of patients will develop CNV in the second eye within eight years after the first eye.[26]

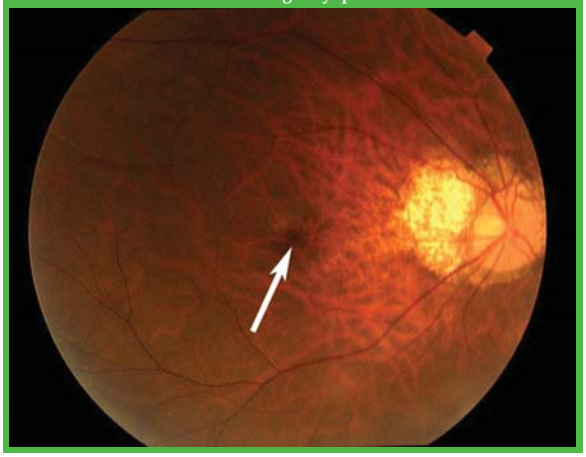
Patients with new onset myopic CNV may develop metamorphopsia, central or paracentral scotoma and reduction in visual acuity. On clinical examination, myopic CNV appears as a flat, small, greyish subretinal membrane beneath or in close proximity to the fovea. Fluorescein angiography (FA) is used to document fluorescein leakage in the CNV and to assess the location of the CNV for treatment planning.

The natural history of myopic CNV is generally poor and a large proportion of patients may have visual acuity of 20/200 or less after five years.[27,28] Poor prognostic factors for patients with myopic CNV include age of greater than 40 years, larger CNV, and worse initial visual acuity.[29,30] Based on studies on the natural history of myopic CNV, active interventions should be considered to avoid gradual visual deterioration. This is particularly important for patients with poor prognostic factors like older age of onset, larger CNV and worse visual acuity at initial presentation.

Direct thermal laser photocoagulation of myopic CNV has been attempted for treatment but this will lead to considerable visual loss due to expansion of the laser scar in the long term and therefore thermal laser treatment is no longer performed for myopic CNV. Other treatment modalities such as submacular surgery and macular translocation surgery for myopic CNV have also been performed with some success but the procedures are technically demanding and are potentially associated with high CNV recurrence rate.[31,32] The most commonly used method in the treatment of myopic CNV currently is photodynamic therapy (PDT) with verteporfin. It is a two-steps procedure involving infusion and activation of a photosensitising drug. The selectivity and efficacy of PDT on the abnormal CNV are caused by differential clearance of the photosensitising drug within the blood stream and preferential binding to low-density lipoprotein receptors on CNV endothelial cells.[33] Studies have shown that PDT with verteporfin can result in stabilisation of vision following treatment.[34-36] However, only around 20-30% of patients will have improvement in vision after PDT with verteporfin. Combined PDT with intravitreal triamcinolone acetonide has also been attempted to further improve the outcome of PDT for myopic CNV but no significant difference was observed compared with eyes which had PDT monotherapy.[37]

More recently, the use of angiogenesis therapy with anti-vascular endothelial growth factor (VEGF) agents like intravitreal bevacizumab has demonstrated encouraging results in the treatment of myopic CNV as patients had visual gain after treatment.[38,39] In a recent study by Chan et al,[38] three monthly injections of bevacizumab resulted in a mean improvement of 2.6 lines at 6 months with 68% of patients having visual improvement of two or more lines. With the increasing availability of other anti-VEGF agents like ranibizumab, targeted angiogenesis therapy will play an increasing role in the management of myopic CNV and may become the treatment of choice for myopic CNV in the near future.

Figure 2. Macular haemorrhage (white arrow) associated with choroidal neovascularisation in high myopia



Conclusions

Individuals with high myopia are subject to various retinal pathologies including peripheral retinal degenerations, retinal detachment, and posterior pole chorioretinal lesions. Since these retinal pathologies might be associated with serious sight-threatening complications, patients with high myopia should be educated about the symptoms of retinal complications such as retinal detachment, macular hole, and myopic CNV. Patients should be advised to seek medical care promptly should such symptoms arise. Prompt referral to ophthalmologists will be useful in preventing severe visual loss as effective surgical and medical treatments are available for these retinal complications especially in the early stages.

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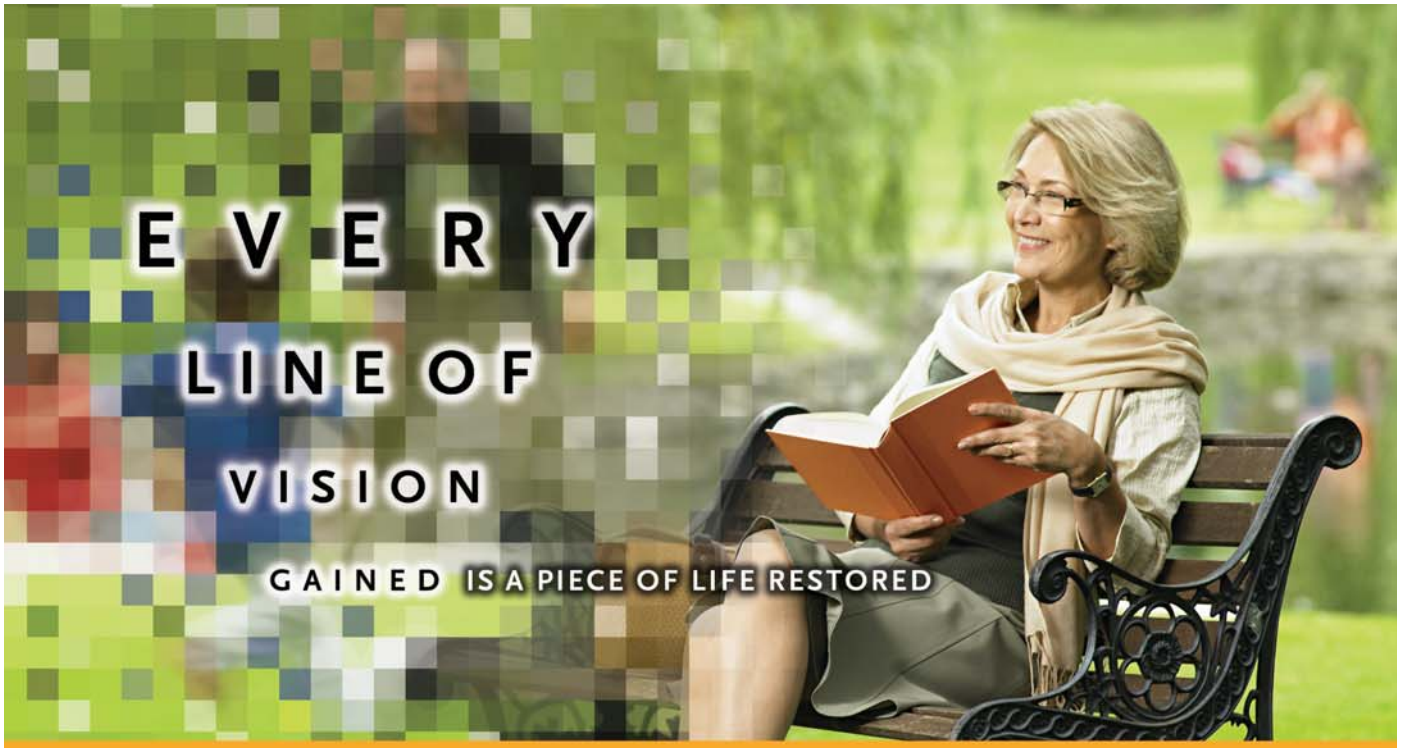

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Amblyopia: An overview

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Introduction

Amblyopia, or commonly referred as lazy eye, can be defined as a reduction in vision with no demonstrable abnormality of the visual pathway that is not immediately resolved by refractive correction.[1] With a prevalence of 3-5%, amblyopia represents a major public health problem. In fact, amblyopia is the leading cause of monocular vision loss in the United States in people younger than 40 years.[2] It develops during maturation of the visual pathway and is largely reversible during the first seven to eight years of life. This is known as the 'critical period'. In some situations the critical period may be extended.[3]

Causes

Amblyopia may develop when the images coming into one or both eyes are either blurred or obscured. There is an abnormal binocular cortical interaction and results in a loss of acuity, contrast sensitivity and/or positional disorder.[4] Amblyopia is usually classified by cause.[1] However, it is not uncommon for the types to co-exist. It can be grouped into three major categories.

(1) Strabismic amblyopia: when it is due to the presence of a squint
(2) Refractive amblyopia: it can be subdivided into
a) Anisometropic amblyopia: where there is a large difference in refractive error between the two eyes
b) Meridional amblyopia: where there is a significant degree of astigmatism
c) Ametropic amblyopia: where the refractive error is so significant that neither eye receives a good quality images.
(3) Stimulus deprivation amblyopia: where, for example, a cataract or ptosis obscures the visual axis

Strabismus means the eyes are not looking in the same visual direction and is not necessarily giving rise to amblyopia, especially in intermittent type with freely alternating fixation. In Hong Kong, intermittent exotropia (divergent squint) is the most common type of squint which is less commonly associated with amblyopia. However, if the condition becomes constant and/or has a strong fixation preference, amblyopia may arise while esotopia (convergent squint) is more commonly associated with amblyopia.

Refractive amblyopia is the result of a blurred visual image being formed on the retina. Among the three subtypes, anisometropic amblyopia is the most common one and hypermetropic (long-sightedness) anisometropia is more amblyogneic. A difference of more than +1.0 D is generally considered to be of significance and should be corrected with spectacles. Myopic anisometropia is less commonly resulting in amblyopic since they can see clear at some point in the near fixation. Meridional amblyopia is due to the presence of high astigmatism which means the refractive power along the two perpendicular axes has a significant difference. There is no general consensus as to how much astigmatism is amblyogneic. The presence of 1.5 to 2.0 D of astigmatism will generally require spectacle correction. Ametropic amblyopic usually refers to the condition that there are high degrees of refractive error in either one or both eyes.

Stimulation deprivation amblyopia is due to the presence of structurally abnormality preventing the formation of clear image in the visual pathway. The most commonly cause is ptosis that obscuring the visual axis. Other causes include cataract, lid or orbital masses.

Screening

Amblyopia is a deficit of vision that has to be treated within the critical period. Effective treatment depends on early detection, and a broad consensus of professional opinion supports vision screening of infants and young children. Screening programmes have been set up to detect this largely asymptomatic condition and refer children for treatment. There are several issues concerning screening that have not yet been fully resolved and these include when to screen, how to screen and who should do the screening. Currently there are a variety of recommendations for vision screening programmes and a number of different approaches to providing the services. The battery of tests carried out usually includes monocular visual acuity testing with an age-appropriate test with or without assessment of extraocular muscle function, binocular status, and colour vision assessment. No single method of screening has been demonstrated to be superior in detecting amblyopia and all methods have significant limitations.

Most screening programmes to detect strabismus and amblyopia, or their precursors, have been with children between 3 and 6 years.[5-9] There has been a great deal of debate regarding the optimum time for visual screening. Amblyopia screening as such is not generally practical in infancy. However, an alternative approach is to screen for early strabismus and strabismogenic factors that are precursors and predictors.[10]

Vision screening for preschool children has employed traditional visual acuity - based methods to evaluate visual functions.[11] These tests typically involve reading optotypes - such as Allen Symbols, LEA figures, HOTV Letters, and Snellen Acuity - using numbers or letters. The advantage of these optotype visual acuity-based techniques for preschool vision screening is that they provide a direct measure of visual function. However, successful screening by means of optotype visual acuity testing requires an older and more cooperative child and is less effective in younger children who have limited attention spans. Results of these testings are also highly dependent on the skills and experience of the examiners. Newer technologies such as photoscreening and automated refraction have prompted a re-evaluation of visual screening. However, they detect problems associated with the development of amblyopia, i.e. amblyogenic factors, instead of detecting amblyopia directly. While the natural history of many amblyogenic factors is not well understood, not all patients with amblyogenic factors will develop amblyopia. However, the risk of developing amblyopia appears to increase along with the magnitude of amblyopic factors.

Recommendations regarding who should carry out screening for amblyopia vary. In America practice varies from state to state and preschool vision screening is carried out by a variety of professional, volunteer and lay professional, volunteer and lay personnel.[12,13] In Sweden nurses carry out a vision screen as part of other healthcare surveillance checks.[14] In some places trial of home vision screening kits for parents have been conducted.[13,15] In the UK orthoptists have been shown to compare favourably with other screening personnel. In a comparative trial in Newcastle estimates of 100% sensitivity and 97.1% specificity were calculated for orthoptists undertaking vision screening in three year olds. Health visitors achieved better specificity at 100% but managed a sensitivity of only 50%.[16] Nurse and lay screeners can achieve similar sensitivity, when specificity is set at 0.90, for detecting preschool children in need of a comprehensive eye examination.[17]

In an attempt to provide standards for which conditions should be detected, the American Association for Pediatric Ophthalmology and Strabismus (AAPOS) Vision Screening Committee has reviewed the literature and introduces a set of standard risk factors that should be detected with preschool vision screening. These risk factors are derived from consensus, reviews of surveys of paediatric ophthalmologists, evaluation of "gold-standard" criteria from large vision screening programmes.[11, 18-20]

Vision in Preschoolers Study Group[21] also defined their targeted groups with slightly different criteria. Children were referred to specialists in Singapore according to their local guidelines.[22]

Chan et al suggested referral criteria for Hong Kong children should be set as hyperopia of ≥+2.00 D, myopia of ≥-1.00 D, astigmatism of ≥1.00 D and anisometropia of ≥ 1.25 D.[23] Sensitivity using only these criteria for abnormal refraction in identifying children with amblyopia, esotropia, exotropia and subnormal vision (<6/12) was respectively 100%, 84.6%, 45.2% and 95.7%. The overall sensitivity for the identification of visual problems was 86.1% and the overall specificity was 76.0%.

Treatment

Currently, the most commonly employed treatments of amblyopia include correction of the refractive error with spectacles, patching of the better eye and penalisation of the better eye with atropine, along with treatment of other associated underlying disorders.

For anisometropic amblyopia, constant wear of a pair of corrective spectacles alone can improve visual acuity pair of (VA) in many cases with moderate amblyopia.[24] This can be combined with patching at the beginning or when the VA shows no further improvement after wearing glasses alone. This will depends on the preference of the ophthalmologist, the density of the amblyopia and also the compliance of spectacles. In case of meridional and ametropic amblyopia, there are still no well designed studies but the same treatment principles are usually adopted.

However, in children without documented amblyopia but having just mild to moderate refractive error that may be amblyogenic, there is still some controversy as to when spectacles should be recommended. In our experience, a simple rule of 1-2-4 can be considered. When there is hypermetropic anisometropia of +1.0 D, astigmatism of 2.0 D or myopia of -2.0 D or a hypermetropia of +4.0 D, spectacles can be considered even though there may not be amblyopia. Spectacles are not a must in these conditions if the uncorrected visual acuity is satisfactory. However, in the presence of 2.0 D astigmatism or myopia, the uncorrected vision may not be good enough for learning, especially Chinese characters as they are more complex in structure.

Patching conceptually forces the patient to use the amblyopic eye by occluding the better eye. However, the regimen of patching is not well established. Some will prefer aggressive treatment with full time patching that run a risk of occlusion amblyopia, i.e. development of amblyopia in the sound eye because of occlusion depriving it of visual stimulation. More commonly, it is done in a dosing method. A general rule is the hours of patching daily equal the years of age of the patient and this is then stepped up or down according to the response of the patient.

In the recent reports by the Pediatric Eye Disease Investigator Group, the regimen may be simplified further. In their study group of patients aged between 3 and 7 with moderate amblyopia, defined as visual acuity ranging from 20/40 (6/12) to 20/100 (6/30), patching is slightly more effective than atropinisation and a 10 or more hours of patching showed a more rapid response than lesser hours of patching for patients in the range of 20/80 (6/24) to 20/100 (6/30).[25] Although by 6 months, the difference in improvement between the two treatment groups is not significant.[25] For patients with moderate amblyopia, 2 hours of daily patching is equally effective as 6 hours of daily patching.[26] For severe amblyopia with a range of 20/100 (6/30) to 20/400

(6/120), 6 hours of daily patching has similar effect as full-time patching.[27] Moreover, in other reports by the same group, patching is also effective in treating some amblyopic patients aged between 7 and 18 years old.[28, 29]

Although patching and atropinization is effective in treating amblyopia, the vision still may not be fully restored. A two-year follow up of the moderate amblyopia group treated either by patching or atropine showed that the amblyopic eye was still 2 lines worse than the sound eye.[30]

Another issue regarding treatment of amblyopia is recurrence after cessation of treatment. Nearly one fourth of successfully treated amblyopic children had a recurrence within the first year off treatment.[31] The risk factors include patients treated with 6 or more hours of daily patching and then stopped abruptly without weaning to 2 hours of patching, better visual acuity at the time of cessation of treatment, a greater number of lines improved during the previous treatment and a history of recurrence.[31, 32] These patients may need to be followed up more carefully.

The other issues that are still not answered in these studies include the treatment protocol for patients below the age of 3 years and the effect of different treatment methods for severe amblyopia. In young toddlers, long patching hours may have a higher risk of occlusion amblyopia.

Summary

Amblyopia can usually be treated effectively with either glasses or occlusion therapy or both. This depends on early detection by personnels with relevant experience since they are usually asymptomatic in the case of refractive amblyopia. Patients with strabismic amblyopia may be brought to attention earlier because of the squint condition may lead parents to seek for medical advice. Only through effective screening and a high index of suspicion, we can salvage the vision of our children.

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Surgical Correction for Near Sightedness

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Presbyopia, which literally means "old eyes", is a normal and expected consequence of the aging process. The crystalline lens within your eyes is composed of proteins. These proteins are soft and flexible when you are younger. Beginning as you approach your 40's, presbyopia occurs as the protein composition of the crystalline lens changes, making it harder and less flexible. When the crystalline lens loses its ability to flex, it is no longer able to change its shape and effectively bend light rays as sharply, and the ability to focus on near objects is diminished. As mentioned above, the clinical symptoms of presbyopia typically commence between age forty and progressively worsen through age sixties. When presbyopia begins, people who already wear glasses may need bifocals or trifocals, and those who have never worn glasses may require reading glasses.

Presbypia can now be surgically corrected. This can be achieved with the cornea approach or lens approach.

The Cornea Approach - PresbyLASIK

LASIK has been shown to be effective in the correction of myopia, hyperopia and astigmatism. It achieves the effect by changing the curvature of the anterior corneal surface. However, lasik has no effect on the eye's focusing muscles or on the crystalline lens, so it corrects presbyopia with a different principle. Traditionally, LASIK helps patients with presbyopia by creating monovision.

Mononvision: Currently, patients with presbyopia can consider monovision when undergoing LASIK. The concept of monovision is very simple. One eye is corrected for near vision and the other eye is corrected for distance vision. The brain figures out which eye to use and when. This is usually achieved by undercorrecting the non-dominant eye leaving behind 1 - 2D of myopia. Since presbyopia is a problem with near vision and myopia is a problem of far vision. Leaving a small degree of myopia will cancel the effect of presbyopia during near vision. The disadvantage of this technique is that there will only be 2 points of focus. While both eyes will be used during daily activities when intermediate vision is required, only one eye is used reading at the very near and the very far distance. This can result in a reduction of stereopsis and the quality of the intermediate distance will also be affected.

Multifocality: Other than monovision, LASIK can also create a multifocal cornea in very much the same way bifocal and trifocal works. Multifocal LASIK eye correction surgery involves making multiple curvatures on the cornea to provide multiple focal points for incoming light. The ablation profile makes use of the accommodation reflex which includes pupil constriction, convergence and accommodation. In prebyopia, accommodation is lost but pupil constriction and convergence are still functioning. By changing the refractive power over the cornea with respect to different pupil sizes, different degrees of myopia can be corrected.

- Aspheric design: This is analogous to a progressive lens as there are no clear zones for distance and near vision. There is a gradual change in curvature from near to distant vision. Depending on the pupil size at different stages of constriction, the point of optimal focus gradually shifts from distant to near.

- Centre Near design: This is analogous to a bifocal or trifocal design. In a centre near design, the central portion of the cornea will have a higher power than the periphery. When the pupil constricts during accommodation, the higher power central portion of the cornea is selected and gives an extra power for reading small print at near.

The Lens Approach

Other than the cornea, the lens also accounts for 25% of the refractive power of the eye. It is also the component inside the eye that gets harden in patients with cataract. During lens surgery, this natural lens is replaced by an intraocular lens (IOL) by a process called phacoemulsification. Current technology allows very accurate calculations of lens power so that the implanted IOL can also correct the patients' refractive error. For this reason, there are patients who opt for lens surgery with minimal or no cataracts. These procedures are now called Refractive Lens Exchange

During phacoemulsification, the surgeon first makes a small incision at the edge of the cornea and then creates an opening in the membrane that surrounds the natural lens. This thin membrane is called the capsule. Next, a small ultrasonic probe is inserted through the opening in the cornea and capsule. The probe's vibrating tip breaks up or "emulsifies" the cloudy lens into tiny fragments that are suctioned out of the capsule by an attachment on the probe tip. After the lens is completely removed, the probe is withdrawn leaving only the clear (now empty) bag-like capsule, which will act as support for the intraocular lens (IOL).

Phacoemulsification allows lens surgery to be performed through a very small incision in the cornea. Stitches are seldom needed to close this tiny entry, which means that there is less discomfort and quicker recovery of vision than with other surgical techniques. Small incisions do not change the curvature of the cornea like larger ones that were required with older surgical techniques. This allows for more rapid rehabilitation of vision and possibly less dependence on glasses for good distance vision. After the lens is removed, the IOL is then implanted.

In the past, IOL design is single focus (monofocal) with only one clear point of focus. Usually, ophthalmologists will target this point of focus at about 1 - 2 metres as this is the distance that most work are done. To see clearly at other distances, one needs reading spectacles and distant spectacles.

Presbyopic intraocular lenses (IOLs) technology has made presbyopia corrective surgery a big step forward. Presbyopic intraocular lenses or multifocal IOLs, also known as accommodating IOLs or multifocal IOLs, can eliminate the need for glasses in most patients. Most patients drive, read, enjoy sports, and work on the computer with little or no need for glasses. There are basically 2 designs.

Multifocal IOL: These lenses have highly specialised optical properties that can divide light to bring it into focus at more than one point at the same time. This allows the eye to see both near and far, usually without glasses.

Accommodating IOLs: These IOLs have a single focal point, however, the focal point can shift position in space so that objects at distance are clear when the eye focuses on them, but when the eye looks at a near object the IOL will shift its focal point to bring the near object into focus. Accommodating Monofocal IOLs achieve this by physically moving inside the eye in response to the focusing action of the muscles of the eye.

There are currently many surgical options available in the correction of presbyopia. However, no single method is the best option that fits all patients at the moment. The surgical decision of the best prebyopic correction will have to be worked out on a case-by-case basis.

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Traditional Chinese Medicine and Ophthalmology

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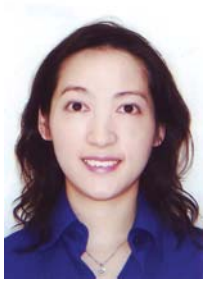

Dr. Jane C C Yeung

Imagine, distinctively different eye conditions such as dry eyes, floaters, age-related macular degeneration and optic neuropathy can be classified under the same "syndrome" in Traditional Chinese Medicine (TCM)?

Without some knowledge of TCM, one can never make sense out of the above statement.

History of TCM and basic TCM theories

TCM has been in practice since ancient times. One of the earliest and one of the most important literatures in TCM is the "Huang-di-nei-jing"(黃帝內經) written in between 475 B.C. and 265 B.C.[1] It describes correctly the gross anatomy of the body, gives advice on a healthy diet and lifestyle, and emphasises the importance of exercise. All of which are still applicable today. This book is the first documented source of Chinese medical theory.

Whilst Western medicine has a strong scientific basis of anatomy, physiology, biochemistry, and molecular biology, TCM takes a philosophical approach towards the body and maintaining health.

Basic TCM theories include the followings: [2,3,4]

1) Holistic ideology (整體觀念)
Humans are part of the universe. Everything within or outside of the body is ultimately interconnected

2) The Vital Substances: Essence (精), Qi (氣), Blood (血) & Body Fluids (津液) theory
These substances within the body are fundamental to life and provide the material and functional basis of the body

3) Yin-yang (陰陽) theory
The development of all phenomena in the universe is the result of the interplay of two opposite stages, symbolised by Yin and Yang. Although Yin and Yang are opposite, they are also interdependent and are in a constant state of dynamic balance.

4) Five-element theory (五行)
The five elements are seen as existing in a dynamic and balanced relationship with each other. By way of analogy, phenomena and matter are classified in terms of the five elements.

5) 5) Zang Fu Theory (臟腑)
The concept of "organ" in Chinese medicine is not equivalent to that in Western medicine. Although the internal organs, collectively known as "Zang-fu organs", do share some of the functions as the same-named organs as understood in Western medicine, there are major departures in terms of functions and inter-relationships. Zang-fu organs are better thought of as complex functional systems. These systems are inter-connected by "meridians"

6) Meridian Theory (經絡)
The meridian system is the system of inter-connecting pathways throughout the body in which qi and blood circulate, connecting all parts of the body into an organic whole

Good health is seen as a reflection of internal harmony of the body. TCM believes that good health is due to a kinetic balance between both Yin aspects (nourishing substances) and Yang aspects (functional activities) of the human body, with good level of vital substances generated from normal physiological functioning of various Zang-fu organs.

How do the eyes fit into this body "Universe"?

All parts of the body are inter-connected. The eyes are nourished by the Essence and Qi produced by all Zang-fu systems.[4, 5] Of all the Zang-fu systems, the eyes are most closely related to the Kidney and Liver Systems.

Eye and Kidney System

The most important function of the "Kidney" is to store Essence - which is the most basic substance of life. It is the basis of body Yin and Yang. The level of Essence normally lowers with advancing age but its consumption will be speeded up with exhaustion, stress, and chronic illness. We can only see if the eye is well nourished by Essence.

Eye and Liver System

According to Huang-di-nei-jing, the eye is the sense organ of the Liver System ("肝開竅於目"). The Liver Meridian is the only meridian that runs directly to the eyes.[4, 5] The function of the Liver is to store blood (as in Western medicine) and regulate the circulation of Qi.

Back to the first question - how can distinctively different eye conditions be classified under the same "syndrome"?

On a macro level, when the level of Essence drops, all

functions of the body are also reduced; when the flow of Qi is disrupted, all functions of the body are disrupted. Similar analogy in Western medicine can be thought of as anaemia or hypoxia and metabolic disorder.

On a micro level, a body part will manifest a certain symptom when internal harmony is lost.

For example, if the stored level of Essence or stored blood drops, malnourished conjunctiva would manifest as dry eye; malnourished vitreous-floaters; malnourished retina - macular degeneration; malnourished optic nerve - optic neuropathy. Apart from the eyes, this affected person may have other symptoms, such as easy fatigue, dizziness, tinnitus etc. All together these symptoms are collectively known as a syndrome/ pattern(證) that describes the pathological process of illness. For the above example, the syndrome / pattern in TCM terms will be that of "deficiency in Kidney essence and Liver blood" (腎精衰虛, 肝血虧損) or simply "impaired renal and liver functions" (肝腎不足).[5] Of course, this does not imply that the liver and renal function tests results are abnormal.

This concept of "syndrome/ pattern" is unique in TCM. Treatment then aims to treat this abnormal "pattern / state of health" but not to treat specific "disease" or "symptom".

TCM treatment

The aim of TCM treatment is to restore internal harmony in terms of Yin-yang, Vital Substances, between Zang-fu systems. What is deficient, you replenish; what is in excess, you reduce or down-regulate. .

The most common forms of TCM treatment are herbs and acupuncture.

Herbal TCM treatment

Herbs are categorised primarily in terms of their actions on the body and are designated as warm (溫), hot (熱) (that correlate with Yang), neutral, cold (寒), cool (涼)(that correlate with Yin). Chinese medicine theory also holds that herbs enter specific meridians. It is understood that each individual herb has an affinity with one or more specific organs and would reach the organs via their meridians to produce therapeutic effect. This is probably the earliest form of understanding of receptors and target organs[6].

Herbs are rarely used individually. TCM herbal treatment usually is in the form of "medicinal formula"(方劑). The choice of herbs follows TCM theory and the prescription is individualised according to the patient's condition, age, gender …There are hundreds of known medicinal formulae that treat many diseases and their various syndromes. Practitioners may simply choose the key representative formula for a given condition or syndrome and then modify it according to the patient's presenting condition.

Acupuncture

The free flow of Qi is the foundation of a balanced mind and body i.e. a healthy person. Diseases are the products or indications of interrupted flow of Qi or the weakness of Qi. Acupuncture is to restore its free flow[7]. Combination of local points around the malfunctioning organ and distant points along its corresponding meridian are often chosen and these points are stimulated using thin needles in a treatment session.

In the treatment of eye diseases by acupuncture, local points are dangerous with risks of globe perforation / orbital haemorrhage / intra-orbital or even intra-ocular retained foreign body. Therefore distant points along the Liver and Kidney meridians are preferred

As with TCM treatment for other diseases, one would be always given diet and healthy lifestyle advice on top of specific treatment in order to achieve long term success in maintaining health.

Is TCM treatment safe?

All therapeutic drugs can be poisonous. Although often thought of as a form of "natural healing", herbal remedies are of no exception in causing possible side effects. Unlike conventional drugs, the quality of herbal products can be influenced by a range of natural factors, such as climate change, soil quality, harvesting, processing etc. It is difficult to always guarantee good quality herbs.

Even with good quality herbs, in their dried / processed forms, herbs can look very alike. Serious adverse effects have been reported due to misidentification of herbs.

One other major problem for TCM products is contamination with conventional Western drugs, which quite often are not shown on the product labels. We hear it on news from time to time that certain over-the-counter herbal product has to be withdrawn from the market due to this reason.

A good herb if taken in excessive amount or misused can result in adverse effects.

Prolonged use of Ginseng (人參) has been associated with a "ginseng abuse syndrome" including symptoms like hypertension, oedema, morning diarrhoea, skin eruptions, insomnia depression and amenorrhoea.

Prolonged use of Licorice (甘草)can result in hypertension, encephalopathy and pseudohyperaldosteronism.[8]

A good herb being used correctly can sometimes cause serious side effects in susceptible individuals. Specific example in causing ocular side effects is Ma-huang (麻黃)(Epherdra species). These contain the tertiary alkaloid epherdrine. It causes mydriasis (dilatation of the pupil) and may trigger acute angle closure glaucoma which can cause blindness if not treated within a short time.

Special precautions are needed peri-operatively if a patient is known to use TCM. Some examples are shown in the table below (quoted from American Academy of Ophthalmology):

Nutritional Supplements: Actions & Possible Side Effects

Supplement	Action	Side effect(s)	Suggested Preop Discontinuation*
Echinacea	Activates cell-mediated immunity	Allergic reactions initially, then decreased immunosuppression	No data
Ephedra	Increases heart rate and blood pressure; also arrhythmias (direct sympathomimetic)	Myocardial infarction/cerebrovascular accident. Interacts with monoamine oxidase inhibitors	24 hours at least
Fenugreek 葫蘆巴	Stimulates pancreas to release insulin; lowers low density lipoprotein; interferes with iron absorption	Alters thyroid hormone balance	2-3 weeks
Feverfew 小白菊	Slows platelet aggregation, prostaglandin synthesis (inhibits arachidonic acid)	Anticoagulant with aspirin, mucosal ulcers	2 weeks
Garlic	Inhibits platelet aggregation. Increased fibrinolysis; +/- antihypertensive effect	Increased bleeding risk (especially if other antiplatelet, indocin or warfarin)	7 days at least
Ginkgo 銀杏	Inhibits platelet-activating factor, peripheral vasodilator, antioxidant & gamma-aminobutyric acid activity, induces CYP450 3A	Increased bleeding risk (especially if other antiplatelet), subdural haematoma, subarachnoid haemorrhage, hyphema, seizures, decreased anticonvulsants	36 hours at least
Ginseng 人參	Lowers blood glucose. Inhibits platelet aggregation. Increased prothrombin time/partial thromboplastin time in animals. Inhibits cyclic adenosine monophosphate.	Hypoglycaemia; increases bleeding risk; decreases anticoagulant effect of warfarin; psychoactive central effect (monoamine oxidase inhibitor)	7 days at least
Kava	Sedation; decreases anxiety	May increase sedative effect of anesthetics; possibly addictive/withdrawal effects; hepatotoxicity.	24 hours at least
Licorice 甘草	Increases cortisol	Hypokalaemia; increases effect of cardiac glycosides, steroids, antihypertensives, can cause hypertension encephalopathy (pseudoaldosteronism)	Several days at least
St. John's wort	Inhibits neurotransmitter uptake; induces cytochrome P450 enzymes & P-glycoprotein intestinal transport	Decreases cyclosporine, warfarin, steroids, protease inhibitors, and possibly benzodiazepines, theophylline, calcium channel blockers,	5 days at least

SOURCE: Adapted from Ang-Lee et al (JAMA, 2001)[8]

Conclusions

Whether you accept it or not, a significant proportion of population in Hong Kong uses TCM on a daily basis.[8] TCM development is unstoppable and the government policy for the future development of Chinese medicine was enshrined in the Basic Law of the Hong Kong Special Administrative Region Article 138 "the Government of the Hong Kong Special Administrative Region shall, on its own, formulate policies to develop western and traditional Chinese medicine and to improve medical and health services. Community organizations and individuals may provide various medical and health services in accordance with law." Some knowledge of TCM is advisable for all of us for the better care of our patients.

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CALL FOR SUBMISSION OF ARTICLES FOR PUBLICATION IN THE HONG KONG MEDICAL DIARY

The Editorial Board of the Hong Kong Medical Diary invites you to send in your interesting articles for publication in the Hong Kong Medical Diary (500 -1,500 words per article). Abstracts from recent local or international meetings/symposia are also welcome. You can send in your manuscript by facsimile at 2865 0345, through mail or via email at sue.cheng@fmshk.org. The Editorial Board of the Hong Kong Medical Diary will give you a prompt reply.

Rosiglitazone and Risk of Myocardial Infarction: Clear Danger or Media Hype?

Dr. Norman Chan

FRCP, MD

Clinical Director, Qualigenics Diabetes Centre, Hong Kong Resort International

Dr. Norman Chan

The recent publication of the meta-analysis of rosiglitazone by Nissen and Wolski in the *New England Journal of Medicine* has created much controversy in the medical world which has been much publicised in the media causing widespread panic in the diabetes community. The Endocrinologic and Metabolic Drugs Advisory Committee and the Drug Safety and Risk Management Advisory Committee of the Food and Drug Administration (FDA) convened to discuss this very issue on 30th July, 2007. The FDA suggested that a subgroup of patients with type 2 diabetes at risk for these events include those on nitrate and those receiving concomitant insulin therapy. They concluded that rosiglitazone should stay in the market with label warnings and extensive educational efforts to be instituted immediately.

With this controversial rosiglitazone issue dominating the medical headlines in recent weeks, it is not surprising that in a recent Expert Forum on Cardiovascular Disease in Type 2 diabetes held in Singapore (12th August, 2007, Raffles Convention Centre), much of the discussion was centred on this issue. The two speakers were Dr. Low Lip Ping, a highly regarded Cardiologist who is the current Chairman of the Board of Directors at the Singapore Heart Foundation, and Dr. Richard Nesto from Boston, USA. Dr. Nesto is a Co-principal Investigator of BARI-2D, a NIH trial evaluating various treatment strategies to improve survival in patients with diabetes and coronary heart disease. More than 60 Cardiologists and Endocrinologists from major Asian countries attended this one-day meeting.

Dr. Low started the meeting with a concise overview of the growing prevalence of diabetes globally particularly in Southeast Asia and its associated cardiovascular risk. He highlighted the importance of insulin resistance in the aetiology of coronary heart disease. In a subsequent lecture, Dr. Low discussed effective strategies in prevention of diabetes development citing several landmark studies of patients with impaired glucose tolerance, including the Diabetes Prevention Program (DPP), the STOP-NIDDM and the recently published DREAM study. He also emphasised the importance of preventing diabetes progression and discussed results of the ADOPT study in which rosiglitazone was clearly more effective in preserving beta-cell function when compared to metformin or glyburide over a period of 5 years. Hyperglycaemia, however, should not be the only target in the management of type 2 diabetes. Dr. Low further discussed the importance of multiple risk factor intervention in reducing cardiovascular mortality using the evidence from the STENO-2 study.

The key lecture was the one discussing the controversial rosiglitazone issue delivered by Dr. Richard Nesto, who ended the day by giving a lecture on heart failure in diabetes and its mechanisms in insulin-treated and glitazone-treated patients. Of interest, Dr. Nesto does not consider screening with Echocardiogram cost-effective and he suggested that clinical assessment is often sufficient to decide when glitazone therapy should be avoided. Dr. Nesto pointed out that none of the cases of glitazone-induced fluid retention or heart failure in the large prospective studies involving rosiglitazone have resulted in death and often resolved after drug withdrawal.

The main discussion of the day was focused on rosiglitazone and risk of myocardial infarction. Dr. Nesto started the lecture by quoting comments from a leading journalist, Scott Gottlieb, published in *The Wall Street Journal(May 29, 2007; Page A15)*:

"I can't help but wonder if the NEJM is functioning more like the mainstream press than a scientific journal at this point."
"When they use shortcuts and shoddy analysis to fabricate criticism and doubt of drug regulation, they're no better than some politicians they increasingly comport with."

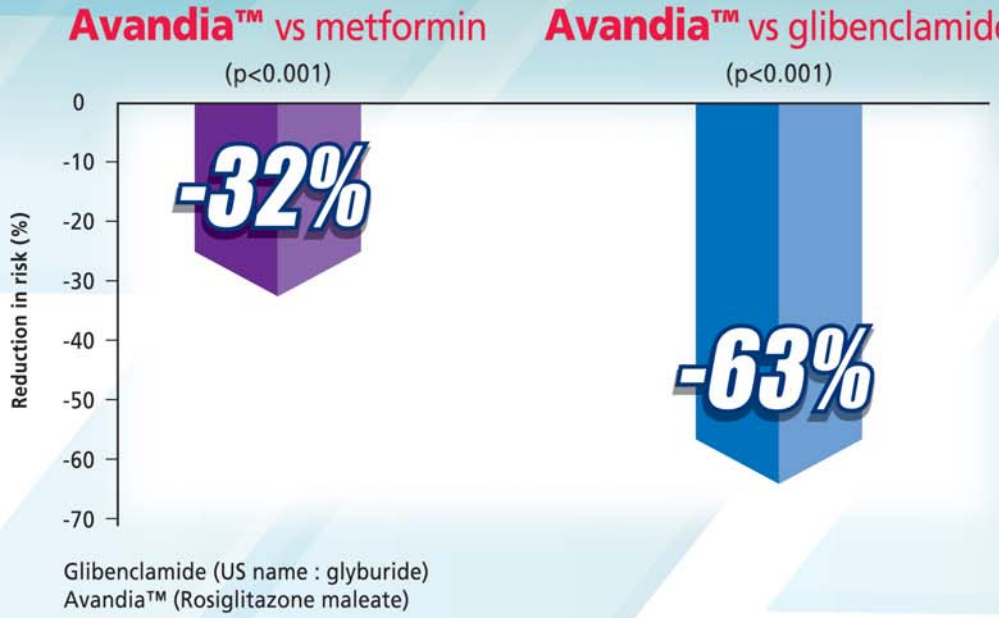
The numerous weaknesses and limitations of meta-analysis were discussed by Dr. Nesto who suggested that meta-analysis was only useful for generating hypothesis and not making cause-effect implications. Dr. Nesto specifically pointed out that many of the small studies included in the Nissen meta-analysis were of 6-month duration. He made analogy to the PROACTIVE study in which another glitazone, pioglitazone, was compared to placebo over a 3-year period in type 2 diabetic patients who already had cardiovascular events. The result of the PROACTIVE study showed that the pioglitazone-treated arm had a lower incidence of primary composite endpoint compared to the placebo-arm at the end of 3 years. However, if the data were analysed at the end of 6 months, the result was totally the opposite favouring the placebo arm. Another important weakness in Nissen's meta-analysis was the very small number of endpoints. Dr. Nesto also eluded to studies examining effects of metformin as an example of how conflicting results may result from meta-analysis. In the UKPDS, metformin subgroup showed significant reduction in most diabetes-related endpoints when compared to other class of drug treatment. In contrast, in the Cochrane review of all non-UKPDS metformin studies, the results showed that patients treated with metformin had more vascular complications compared to other classes of drugs. Finally, Dr. Nesto reminded us that it was not so long ago that calcium channel blockers were considered to be a dangerous class of anti-hypertensive drugs causing gastrointestinal haemorrhage as suggested by certain meta-analysis.....

Overall, this symposium has provided great insight into the limitations of meta-analysis. Definitive answers to this rosiglitazone debate will come from the ongoing RECORD study which is due to be completed in 2009.

ADOPT

A Diabetes Outcome Progression Trial

Landmark ADOPT study shows Avandia™ (rosiglitazone) is superior to both sulphonylurea and metformin in sustaining long-term glycaemic control in type 2 diabetes



Glibenclamide (US name : glyburide)
Avandia™ (Rosiglitazone maleate)

Kahn SE, Haffner SM, Heise MA, et al. N Eng J Med 2006; 355: 2427-2443

Avandia™
rosiglitazone maleate

ACTIVE INGREDIENT: Rosiglitazone maleate. **INDICATIONS:** AVANDIA is indicated as an adjunct to diet and exercise to improve glycemic control in patients with type 2 diabetes mellitus. AVANDIA is indicated as monotherapy and in combination with a sulfonylurea, metformin, or insulin. AVANDIA is also indicated for use in combination with a sulfonylurea plus metformin.

DOSAGE AND ADMINISTRATION: AVANDIA may be administered either at a starting dose of 4 mg as a single daily dose or divided and administered in the morning and evening. For patients who respond inadequately following 8 to 12 weeks of treatment, as determined by reduction in FPG, the dose may be increased to 8 mg daily as monotherapy or in combination with metformin, sulfonylurea, or sulfonylurea plus metformin. AVANDIA may be taken with or without food. **Monotherapy:** The usual starting dose of AVANDIA is 4 mg administered either as a single dose once daily or in divided doses twice daily. In clinical trials, the 4 mg twice daily regimen resulted in the greatest reduction in FPG and HbA1c. **Combination Therapy:** When AVANDIA is added to existing therapy, the current dose(s) of the agent(s) can be continued upon initiation of AVANDIA therapy. **Sulfonylurea:** When used in combination with sulfonylurea, the usual starting dose of AVANDIA is 4 mg administered as either a single dose once daily or in divided doses twice daily. **Metformin:** The usual starting dose of AVANDIA in combination with metformin is 4 mg administered as either a single dose once daily or in divided doses twice daily. It is unlikely that the dose of metformin will require adjustment due to hypoglycemia during combination therapy with AVANDIA. **Insulin:** For patients stabilized on insulin, the insulin dose should be continued upon initiation of therapy with AVANDIA. AVANDIA should be dosed at 4 mg daily. Doses of AVANDIA greater than 4 mg daily in combination with insulin are not currently indicated. **Sulfonylurea Plus Metformin:** The usual starting dose of AVANDIA in combination with a sulfonylurea plus metformin is 4 mg administered as either a single dose once daily or divided doses twice daily. **Maximum Recommended Dose:** The dose of AVANDIA should not exceed 8 mg daily. Doses of AVANDIA greater than 4 mg daily in combination with insulin are not currently indicated. No dosage adjustments are required for the elderly or when AVANDIA is used as monotherapy in patients with renal impairment. Since metformin is contraindicated in such patients, concomitant administration of metformin and AVANDIA is also contraindicated in patients with renal impairment. Rosiglitazone is not recommended in patients with moderate to severe hepatic impairment, and in patients under 18 years of age.

CONTRAINDICATIONS: History of hypersensitivity to rosiglitazone or any other ingredient of the preparation.

WARNINGS AND PRECAUTIONS: AVANDIA is active only in the presence of insulin and should not be used in the treatment of type 1 diabetes. AVANDIA is not recommended in patients with severe cardiac failure unless the expected potential benefit is believed to outweigh the potential risk. AVANDIA, like other thiazolidinediones, can cause fluid retention, which can exacerbate or lead to signs or symptoms of congestive heart failure. The fluid retention may very rarely present as rapid and excessive weight gain. All patients, particularly those receiving concurrent sulphonylurea or insulin therapy, those with mild to moderate heart failure (NYHA class I and II), and those at risk for heart failure, should be monitored for signs and symptoms of adverse reactions relating to fluid retention, including heart failure. Postmarketing reports of new-onset or worsening diabetic macular oedema with decreased visual acuity have been reported with AVANDIA. Many of these patients reported concurrent peripheral oedema. In some cases the visual events resolved or improved following discontinuation of the drug. Prescribers should be alert to the possibility of macular oedema if patients report disturbances in visual acuity. Patients taking AVANDIA may be at risk of dose-related hypoglycaemia if receiving combination regimens that contain a sulphonylurea or insulin. A reduction in the dose of the concomitant agent may be necessary. Close monitoring of glycaemic control and rosiglitazone dose adjustment may be needed when AVANDIA is co-administered with CYP2C8 inhibitors or inducers.

INTERACTIONS: In vitro data demonstrate that rosiglitazone is predominantly metabolised by CYP2C8, and to a lesser extent, CYP2C9. Co-administration of AVANDIA with CYP2C8 inhibitors (e.g. gemfibrozil) resulted in increased rosiglitazone plasma concentrations. Since there is a potential for an increase in the risk of dose-related adverse reactions, a decrease in AVANDIA dose may be needed when CYP2C8 inhibitors are co-administered. Co-administration of AVANDIA with a CYP2C8 inducer (e.g. rifampicin) resulted in decreased rosiglitazone plasma concentrations. Therefore, close monitoring of glycaemic control and changes in diabetic treatment should be considered when CYP2C8 inducers are co-administered.

PREGNANCY AND LACTATION: AVANDIA may result in resumption of ovulation in premenopausal, anovulatory women with insulin resistance. These patients may be at risk for pregnancy. AVANDIA has been reported to cross the human placenta and to be detectable in foetal tissues. There are no adequate data to support the use of AVANDIA during pregnancy and lactation in humans. AVANDIA should be used during pregnancy only if the potential benefit justifies the potential risk to the foetus. AVANDIA should be used during lactation only if the potential benefit justifies the potential risk to the infant.

ADVERSE REACTIONS: *Clinical Trials:* Oedema was generally dose-related, mild to moderate in nature and was more frequently observed when AVANDIA was used in combination with a sulphonylurea or insulin. Anaemia was generally dose-related and mild to moderate in nature. Hypercholesterolaemia. Weight gain was generally dose-related. Hypoglycaemia was generally mild to moderate in nature and was dose-related when AVANDIA was used in combination with a sulphonylurea or insulin. Increased appetite. Congestive heart failure/pulmonary oedema - An increased incidence of heart failure has been observed when AVANDIA (at both 4 mg and 8 mg) was added to treatment regimens that include a sulphonylurea or insulin. There were too few events to confirm a dose relationship; however, the incidence of heart failure appeared higher with 8 mg AVANDIA compared to 4 mg AVANDIA (total daily dose). Events typically associated with cardiac ischaemia - A small number of events typically associated with cardiac ischaemia was observed with AVANDIA in combination with insulin and these events occurred at a higher frequency with the combination (2.77%) compared with insulin alone (1.36%). In a retrospective analysis of data from pooled clinical studies, the overall incidence of events typically associated with cardiac ischaemia was higher for AVANDIA containing regimens, 1.99% versus comparators, 1.51% [Hazard ratio 1.31 (95% confidence interval 1.01 - 1.70)]. In a large observational study where patients were well-matched at baseline, the incidence of the composite endpoint of "myocardial infarction and coronary revascularization" was 1.75 events per 100 person years for AVANDIA containing regimens and 1.76 events per 100 person years for other anti-diabetic agents [Hazard ratio 0.93 (95% confidence interval 0.80 - 1.10)]. A causal relationship between cardiac ischaemic events and AVANDIA has not been established. Constipation is generally mild to moderate. ***Postmarketing Data:*** Anaphylactic reaction. Congestive heart failure/pulmonary oedema- AVANDIA as monotherapy and in combination with other antidiabetic agents. Hepatic dysfunction, primarily evidenced by elevated hepatic enzymes- A causal relationship to AVANDIA has not been established. Angioedema, urticaria, rash, pruritis, macular oedema, bone fractures.

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Clinical Quiz

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MBBS, FRCR, FHKCR, FHKAM (Radiology)

Associate Consultant, Department of Radiology, Queen Mary Hospital

Dr. Helen KS Tung

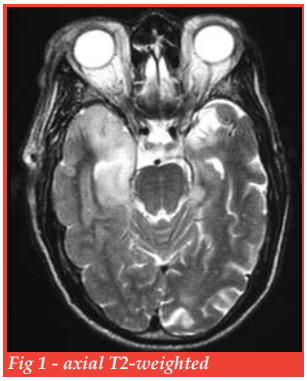

Fig 1 - axial T2-weighted

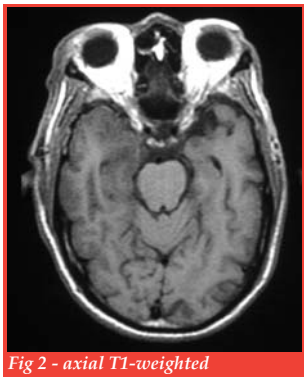

Fig 2 - axial T1-weighted

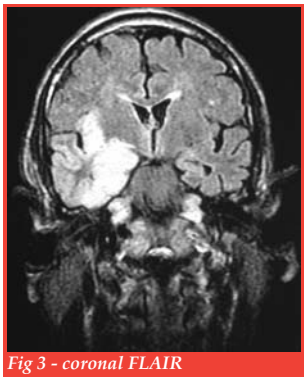

Fig 3 - coronal FLAIR

Clinical History :

These are the MRI images of a 65-year-old man who presented with fever and acute confusion with fluctuating GCS.

Questions:

- What are your findings?
- What is the most likely diagnosis?

(See P. 37 for answers)

News from Member Societies:

The Hong Kong Ophthalmology Society
Updated office-bearers for the year 2007-2009 are as follow: President: Dr. KWOK Kwan Ho, Alvin, Vice-President: Dr. YUEN Suk Yin, Nancy, Hon Secretary: Dr. LAI Yuk Yau, Timothy, Hon Treasurer: Dr. CHAN Ding Nai, Dylan.

Hong Kong Burns Society
Updated office-bearers for the year 2007-2008 are as follows: President: Dr. CHUNG Hon Ping, Hon Secretary: Dr. NG Wai Man, Hon Treasurer: Dr. LAM Lai Kun.

The Hong Kong College of Anaesthesiologists
New office-bearers for the year 2007-2009 are as follow: President: Prof. Michael IRWIN, 1st Vice-President: Dr. CHOW Yu Fat, 2nd Vice-President: Dr. LIU Tak Chiu, John, Hon. Secretary: Dr. CHAN Kin Cheong, Simon, Hon Treasurer: KOO Chi Hung, Assistant Secretary: Dr. LEE Ha Yun, Libby, Assistant Treasurer: Dr. LEE Yeuk Ying, Samantha.

Hong Kong Society of Nephrology
Updated office-bearers for the year 2007-2008 are as follow: Chairman: Dr. WONG Kui Man, Andrew, Hon Secretary: Dr. LUI Sing Leung, Hon Treasurer: Dr. LEUNG Chi Bon, Council Representative: Dr. WONG Kim Ming, Francis.

The Hong Kong Psychogeriatric Association (HKPGA)

The Hong Kong Psychogeriatric Association (HKPGA) was founded in 1998 by a group of professionals working in the field of psychogeriatrics. Our objectives are: to promote, through a multidisciplinary approach, the study and advancement of the science and practice of psychiatry of the elderly as well as the ancillary sciences and branches of medicine and health care; to further public education; to contribute to the improvement of mental health care for Hong Kong senior citizens; and to collaborate with the relevant local and overseas organizations to achieve the above. The current President of HKPGA is Dr Chan Wah-fat and Patron is Dr Hon Leong Che-hung.

The HKPGA has been active in academic and public activities; in the past year these included a scientific symposium on Alzheimer's Disease and a workshop on the Clinical Dementia Rating (CDR) Scale by Prof John Morris of the Washington University and another scientific symposium on mild cognitive impairment (MCI) by Prof Ronald Petersen of the Alzheimer's Disease Center at the Mayo Clinic. These symposia were well received by the attendees. Upcoming activities include a study tour for HKPGA members to Osaka, Japan in Oct 12-19, 2007 for mutual exchange of experience on dementia care and to attend the Silver Congress of the International Psychogeriatric Association (IPA). Two scientific meetings by Prof Bengt Winblad of the Karolinska Institute in Sweden would be held in November, 2007. Details of our activities and other useful information could be found at our official website: www.hkpga.org

Hong Kong College of Cardiology

History, aims, objectives

Since its foundation in 1992, the Hong Kong College of Cardiology has been continuing its mission to promote the advancement of cardiovascular medicine and practice for public benefit through scientific, professional, educational activities and international communication and cooperation. Its ultimate goal is working towards the improvement of heart health for people of Hong Kong. It also acts as a body for consultation in related matters of educational or public interests.

President Message by Prof. Lau Chu Pak

As my forth year being the President of the College the main objective was to strive for a closer relationship with our mainland counterparts as well as continuing the beneficial interactions with professional communities around the world. Looking forward to next few months, many conferences and community activities would be held, such as World Heart Day on 30 September 2007 which will be co-organized with Tung Wah Group of Hospitals and the Cardiovascular Interventional Summit / Hong Kong at the end of November 2007 which will be co-organized with the Guangdong Provincial Peoples Hospital. An increasing participation was drawn from the professional community in Hong Kong, Mainland China and other countries to take part in these international cardiac meetings.

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
						★ 內地-香港康復醫學學術會議 ★ 4th Exercise Prescription Certificate Course (Module 1-3) 1
2	★ Management of Small Renal Tumour 3	★ FMSHK Officers' Meeting 4	5	★ HKMA Council Meeting 6	7	★ 4th Exercise Prescription Certificate Course (Module 4-5) ★ Refresher Course for Health Care Providers 2007/2008 (I) - Common ENT Problems in General Practice ★ Trailwalker 5th Practice Session (Stage 4, 5 & 6) ★ 9th Joint Annual Scientific Meeting 8
★ HKMA Structured CME Programme at Queen Elizabeth Hospital Year 07/08 (VI) - Orthopaedic ★ HKMA Badminton Tournament ★ Joint Professional Table Tennis Tournament 9	10	★ HKMA Newsletter Editorial Meeting 11	★ Hong Kong Neurosurgical Society Monthly Academic Meeting - Current Management for Brain Metastasis ★ HKMA Golf Tournament 12	★ HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2007 (IX) ★ (1) Obstructive Sleep Apnea Hypopnea Syndrome: Influences of Upper Airway Patency in an Animal Model (2) Beyond the Bronchoscopic View: Part I & Part II 13	14	★ 4th Exercise Prescription Certificate Course (Module 6-8) 15
★ Paediatric Grand Rounds - "Two Challenging Cases with Simple Presentation" 16	17	18	19	★ FMSHK Executive Committee Meeting ★ Annual General Meeting ★ Workshop on "NLP Approach on Communication and Counseling" (Code No. SMIG-02-07) 20	21	★ 4th Exercise Prescription Certificate Course (Module 9-10) 22
★ Trailwalker 5th Practice Session (Stage 1, 2 & 3) ★ HKMA Badminton Tournament 23 ★ PALS Course 2007 ★ HKMA Structured CME Programme at Kwong Wah Hospital Year 07/08 (VI) - Respiratory Medicine ★ HKMA Tennis Tournament Kick Off 30	24	25	26	27	★ PALS Course 2007 28	★ PALS Course 2007 ★ 4th Exercise Prescription Certificate Course (Module 11-14) ★ Health Research Symposium 2007 29

Medical Diary of September

Date / Time	Function	Enquiry / Remarks
1 SAT 2:00 pm	**內地-香港康復醫學學術會議** Organised by: The Federation of Medical Societies of Hong Kong & Chinese Medical Association Speaker: Various # Guangzhou Yuehua Hotel, Guangzhou	Ms. Karen CHU Tel: 2821 3515 Fax: 2865 0345
	4th Exercise Prescription Certificate Course (Module 1-3) Organised by: The Hong Kong Medical Association; Department of Health & Physical Fitness Association of Hong Kong, China Chairman: Dr. Y.S. CHAN & Dr. C.F. YEUNG Speaker: Dr. CHOI Kin, Dr. Patrick CHONG & Prof. Stanley HUI # Lecture Theatre, Ruttonjee Hospital, Wanchai	Miss Gloria CHEUNG Tel: 2527 8285 (Registration fee is required) 2 CME Points
3 MON 7:30 pm - 8:30pm	**Management of Small Renal Tumour** Organised by: Hong Kong Urological Association Chairman: Dr. CHAN Shu Yin Eddie Speaker: Dr. LO Ka Lun # Seminar Room, G/F., Block A, Queen Elizabeth Hospital, Kowloon	Dr. CHAN Kwok Keung Sammy / Ms. Siddy MA Tel: 2958 6006 Fax: 2958 6076
4 TUE 8:00 pm - 10:00pm	**FMSHK Officers' Meeting** Organised by: The Federation of Medical Societies of Hong Kong # Gallop, 2/F., Hong Kong Jockey Club Club House, Shan Kwong Road, Happy Valley, Hong Kong	Ms. Carmen CHEUNG Tel: 2821 3512 Fax: 2865 0345
6 THU 8:00 pm	**HKMA Council Meeting** Organised by: The Hong Kong Medical Association Chairman: Dr. K CHOI # HKMA Head Office, 5/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Christine WONG Tel: 2527 8285
8 SAT 2:00 pm	**4th Exercise Prescription Certificate Course (Module 4-5)** Organised by: The Hong Kong Medical Association; Department of Health & Physical Fitness Association of Hong Kong, China Chairman: Dr. Y.S. CHAN & Dr. C.F. YEUNG Speaker: Dr. LEUNG Tat Chi & Dr. CHOW Chun Chung # Lecture Theatre, Ruttonjee Hospital, Wanchai	Miss Gloria CHEUNG Tel: 2527 8285 (Registration fee is required) 2 CME Points
2:30 pm	**Refresher Course for Health Care Providers 2007/2008 (I) - Common ENT Problems in General Practice** Organised: The Hong Kong Medical Association & Our Lady of Maryknoll Hospital Speaker: Dr. SK LAU # Training Room II, 1/F., OPD Block, Our Lady of Maryknoll Hospital, 118 Shatin Pass Road, Wong Tai Sin, Kowloon	Ms. Clara TSANG Tel: 2354 2440 2 CME Points
7:30 pm	**Trailwalker 5th Practice Session (Stage 4, 5 & 6)** Organised by: The Hong Kong Medical Association Speaker: Dr. Y.H. CHOW	Miss Dorothy KWOK Tel: 2527 8285
1:00 pm - 6:20 pm	**9th Joint Annual Scientific Meeting** Organised by: The Hong Kong Society of Gastroenterology, The Hong Kong Society of Digestive Endoscopy, Hong Kong Society for Coloproctology, Hong Kong Association for the Study of Liver Diseases & The Hong Kong Society of Gastrointestinal Motility Chairman: Dr. YUEN Man Fung Speaker: Various # Level 7, Langham Place Hotel, Mongkok, Kowloon	Ms. Celia TAM Tel: 2869 5133 Fax: 2869 9533 CME Accreditation: Various
9 SUN 2:00 pm	**HKMA Structured CME Programme at Queen Elizabeth Hospital Year 07/08 (VI) - Orthopaedic** Organised by: The Hong Kong Medical Association & Queen Elizabeth Hospital Speaker: Dr. C.Y. LO & Dr. P.H. CHIN # Lecture Theatre, G/F., Block M, Queen Elizabeth Hospital, Kowloon	Miss Viviane LAM Tel: 2527 8452 (Registration fee is required) 3 CME Points
2:00 pm	**HKMA Badminton Tournament** Organised by: The Hong Kong Medical Association Chairman: Dr. S.N. CHEONG # MacLehose Medical Rehabilitation Centre, Hong Kong	Ms. Dora HO Tel: 2527 8285
	Joint Professional Table Tennis Tournament Organised by: The Hong Kong Medical Association Chairman: Dr. H YEUNG & Dr. H KOO # Cornwall Street Park, Shek Kip Mei, Kowloon	Ms. Dora HO Tel: 2527 8285
11 TUE 8:00 pm	**HKMA Newsletter Editorial Meeting** Organised by: The Hong Kong Medical Association Chairman: Dr. H.H. TSE # HKMA Head Office, 5/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Tammy TAM Tel: 2527 8941
12 WED 7:30 am	**Hong Kong Neurosurgical Society Monthly Academic Meeting - Current Management for Brain Metastasis** Organised by: Hong Kong Neurosurgical Society Chairman: Dr. HO Wai Shing Wilson Speaker: Dr. WONG Kai Sing Alain # Seminar Room, G/F., Block A, Queen Elizabeth Hospital, Kowloon	Dr. Y.C. PO Tel: 2990 3788 Fax: 2990 3789 2 CME Points (College of Surgeons of HK)
2:00 pm	**HKMA Golf Tournament** Organised by: The Hong Kong Medical Association Chairman: Dr. L HOU # Hong Kong Golf Club	Ms. Dora HO Tel: 2527 8285
13 THU 2:00 pm	**HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2007 (IX)** Organised by: The Hong Kong Medical Association & Hong Kong Sanatorium & Hospital Speaker: Dr. W.S. WONG # HKMA Dr. Li Shu Pui Professional Education Centre, 2/F., Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Miss Viviane LAM Tel: 2527 8452 (Registration fee is required) 1 CME Point
6:30 pm - 8:00 pm	**(1) Obstructive Sleep Apnea Hypopnea Syndrome: Influences of Upper Airway Patency in an Animal Model** **(2) Beyond the Bronchoscopic View: Part I & Part II** Organised by: Hong Kong Thoracic Society/ACCP (HK & Macau Chapter) Chairman: Dr. TSANG Wah Tak Kenneth & MOK Yun Wing Thomas Speaker: Various # LG1, Lecture Room, Ruttonjee Hospital, Wanchai	Dr. C.Y. TAM / Dr. Maurine M.L.WONG Tel: 2468 5407 Fax: 2468 6188 1 CME Point
15 SAT 2:00 pm	**4th Exercise Prescription Certificate Course (Module 6-8)** Organised by: The Hong Kong Medical Association; Department of Health & Physical Fitness Association of Hong Kong, China Chairman: Dr. Y.S. CHAN & Dr. C.F. YEUNG # Lecture Theatre, Ruttonjee Hospital, Wanchai	Miss Gloria CHEUNG Tel: 2527 8285 (Registration fee is required) 2 CME Points
16 SUN 2:00 pm - 5:00 pm	**Paediatric Grand Rounds - "Two Challenging Cases with Simple Presentation"** Organised by: Hong Kong College of Paediatricians Speaker: Various # Hospital Authority Lecture Theatre, 147B Argyle Street, Kowloon	Miss Karen YU Tel: 2871 8773 Fax: 2785 1850 3 CME Points (Cat A, HKCPaed)
20 THU 8:00 pm - 10:00pm	**FMSHK Executive Committee Meeting** Organised by: The Federation of Medical Societies of Hong Kong # Council Chambers, 4/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Carmen CHEUNG Tel: 2821 3512 Fax: 2865 0345

Medical Diary of September

Date / Time	Function	Enquiry / Remarks
20 THU 7:00 pm 6:30pm to 9:30pm	**Annual General Meeting** Organised by: Hong Kong Society for Coloproctology # World Trade Centre Club Hong Kong, 38/F., World Trade Centre, 280 Gloucester Road, Causeway Bay, Hong Kong **Workshop on "NLP Approach on Communication and Counseling" (Code No. SMIG-02-07)** Organised by: College of Nursing, Hong Kong	Miss Christina LO Tel: 2595 6416 Fax: 2515 3195 Email: cloyy@ha.org.hk Secretariat Tel: 2572 9255 Fax: 2838 6280 3 CNE Points
22 SAT 2:00 pm	**4th Exercise Prescription Certificate Course (Module 9-10)** Organised by: The Hong Kong Medical Association; Department of Health & Physical Fitness Association of Hong Kong, China Chairman: Dr. Y.S. CHAN & Dr. C.F. YEUNG # Room 3-006, Tang Shiu Kin Hospital, Wanchai	Miss Gloria CHEUNG Tel: 2527 8285 (Registration fee is required) 2 CME Points
23 SUN 7:00 am 2:00 pm	**Trailwalker 5th Practice Session (Stage 1, 2 & 3)** Organised by: The Hong Kong Medical Association Chairman: Dr. Y.H. CHOW **HKMA Badminton Tournament** Organised by: The Hong Kong Medical Association Chairman: Dr. S.N. CHEONG # MacLehose Medical Rehabilitation Centre, Hong Kong	Miss Dorothy KWOK Tel: 2527 8285 Ms. Dora HO Tel: 2527 8285
28 FRI (Sep 29,30,31, Oct 1,2)	**PALS Course 2007** Organised by: Hong Kong College of Paediatricians Speaker: Various # Hong Kong Academy of Medicine, 99 Wong Chuk Hang Road, Aberdeen, Hong Kong	Miss Karen YU Tel: 2871 8773 Fax: 2785 1850
29 SAT 2:00 pm 9:00 am - 6:00 pm	**4th Exercise Prescription Certificate Course (Module 11-14)** Organised by: The Hong Kong Medical Association; Department of Health; Physical Fitness Association of Hong Kong, China Chairman: Dr. Y.S. CHAN & Dr. C.F. YEUNG # Lecture Theatre, Ruttonjee Hospital, Wanchai **Health Research Symposium 2007** Organised by: Health, Welfare and Food Bureau Speaker: Various # Hong Kong Academy of Medicine, 99 Wong Chuk Hang Road, Aberdeen, Hong Kong	Miss Gloria CHEUNG Tel: 2527 8285 (Registration fee is required) 2 CME Points Ms. Lenora YUNG Tel: 2871 8841 Fax: 2871 8898
30 SUN 2:00 pm 7:30 pm	**HKMA Structured CME Programme at Kwong Wah Hospital Year 07/08 (VI) - Respiratory Medicine** Organised by: The Hong Kong Medical Association & Kwong Wah Hospital Speaker: Dr. S.P. LAM & Dr. K.S. YEE # Lecture Theatre, 10/F., Yu Chun Keung Memorial Medical Centre, Kwong Wah Hospital, Kowloon **HKMA Tennis Tournament Kick Off** Organised by: The Hong Kong Medical Association Chairman: Dr. C.W. CHIN # Kowloon Tong Club	Miss Viviane LAM Tel: 2527 8452 (Registration fee is required) 3 CME Points Ms. Dora HO Tel: 2527 8285

Calendar of Events

Meetings

19-22/10/2007	**16th Asian Congress of Surgery & 3rd Chinese Surgical Week** Organised by: Asian Surgical Association & The Chinese Surgical Society of the Chinese Medical Association # Grand Epoch City, Beijing, China Enquiry: ASA Congress Secretariat Tel: 2855 4235 / 2855 4993 Fax: 2818 1186 Email: info@AsianSurgAssoc.org Website: www.AsianSurgAssoc.org
20/10/2007 1:00 pm - 5:30 pm	**The Federation's Annual Scientific Meeting 2007 - Targeted Therapy in Cancer** Organised by: The Federation of Medical Societies of Hong Kong # M/F, Lecture Theatre, Hospital Authority Building, Kowloon Enquiry: Ms. Karen CHU Tel: 2821 3515 Fax: 2865 0345 Website: www.fmshk.org
27-28/10/2007	**2nd Joint Scientific Meeting of The Royal College of Radiologists & Hong Kong College of Radiologists and 15th Annual Scientific Meeting of Hong Kong College of Radiologists** Organised by: The Royal College of Radiologists & Hong Kong College of Radiologists Speaker: Various # Hong Kong Academy of Medicine, Jockey Club Building, 99 Wong Chuk Hang Road, Aberdeen, Hong Kong Enquiry: Secretariat, Tel: 2871 8788 Fax: 2554 0739 Email: enquiries@hkcr.org Website: http://www.hkcr.org
17-18/11/2007	**Annual Scientific Meeting in Anaesthesiology 2007 - Expanding the Boundaries** Organised by: The Hong Kong College of Anaesthesiology & The Society of Anaesthetists of Hong Kong # Hong Kong Convention and Exhibition Centre Enquiry: CMPMedica Pacific Limited Tel: 2559 5888 Fax: 2559 6910 Email: meeting.hk@asia.cmpmedica.com Website: www.hkca.edu.hk/asm2007.htm
24-25/11/2007	**4th Asian Pacific Diabetic Limb Problems** Organised by: Various # Wiliam MW Mong Block, Li Ka Shing Faculty of Medicine, The University of Hong Kong, 21 Sassoon Road, Hong Kong Website: http://www.diabeticlimb.hk/

Courses

11/10/2007	"脊柱創傷護理"**(Code No. SCNSG-07-04)** Organised by: College of Nursing, Hong Kong Enquiry: Secretariat Tel: 2572 9255 Fax: 2838 6280

Answer to Clinical Quiz

Answer :

Radiographic Findings:
T1-weighted hypointense, T2-weighted hyperintense lesions are noted in the medial right temporal lobe, right insula and external capsule. Smaller similar lesion is also observed in the medial left temporal lobe. The gray matter is predominantly involved and gyral thickening is seen.

Diagnosis:
Herpes encephalitis

Herpes encephalitis typically involves the medial temporal lobe and orbital surface of the frontal lobes. The involvement may extend to the insular cortex, posterior occipital cortex, and the cerebral convexity. However, the basal ganglia are spared. Bilateral involvement is frequent. Foci of haemorrhage occasionally can be observed. Gyral thickening is seen. Sometimes patchy parenchymal or gyral enhancement is present.

MRI is preferred for imaging and follow-up studies of herpes encephalitis.

Dr. Helen KS Tung
MBBS, FRCR, FHKCR, FHKAM (Radiology)
Associate Consultant, Department of Radiology, Queen Mary Hospital

THE FEDERATION OF MEDICAL SOCIETIES OF HONG KONG

The Federation's Annual Scientific Meeting 2007

Targeted Therapy in Cancer

20 October, 2007 (SAT)
1:00 p.m. - 5:30 p.m.
Lecture Theatre, M/F,
Hospital Authority Building,
147B Argyle Street, Kowloon

- ***Overview and Breast Cancer***
  Prof. Richard J. Epstein
  Department of Medicine, Queen Mary Hospital, The University of Hong Kong
- ***Head and Neck Cancer***
  Dr. Daniel TT Chua
  Department of Clinical Oncology, Queen Mary Hospital, The University of Hong Kong
- ***Lung Cancer***
  Dr. James CM Ho
  Department of Medicine, Queen Mary Hospital, The University of Hong Kong
- ***Gastrointestinal Tract Cancers***
  Prof. Benjamin CY Wong
  Department of Medicine, Queen Mary Hospital, The University of Hong Kong
- ***Haematological Malignancies***
  Dr. James CS Chim
  Department of Medicine, Queen Mary Hospital, The University of Hong Kong
- ***Childhood Malignancies***
  Dr. Godfrey CF Chan
  Department of Paediatrics, Queen Mary Hospital, The University of Hong Kong

Registration Fee
HK$100 Members of member societies
HK$200 Non-member

Registration
Registration forms can be obtained by calling our Secretariat at 2527 8898 or the homepage http://www.fmshk.org Please send registration form and cheque to: The Federation of Medical Societies of Hong Kong, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong. Registration will be on first-come-first-serve basis.

CME/CPE: Please refer to our homepage http://www.fmshk.org for details