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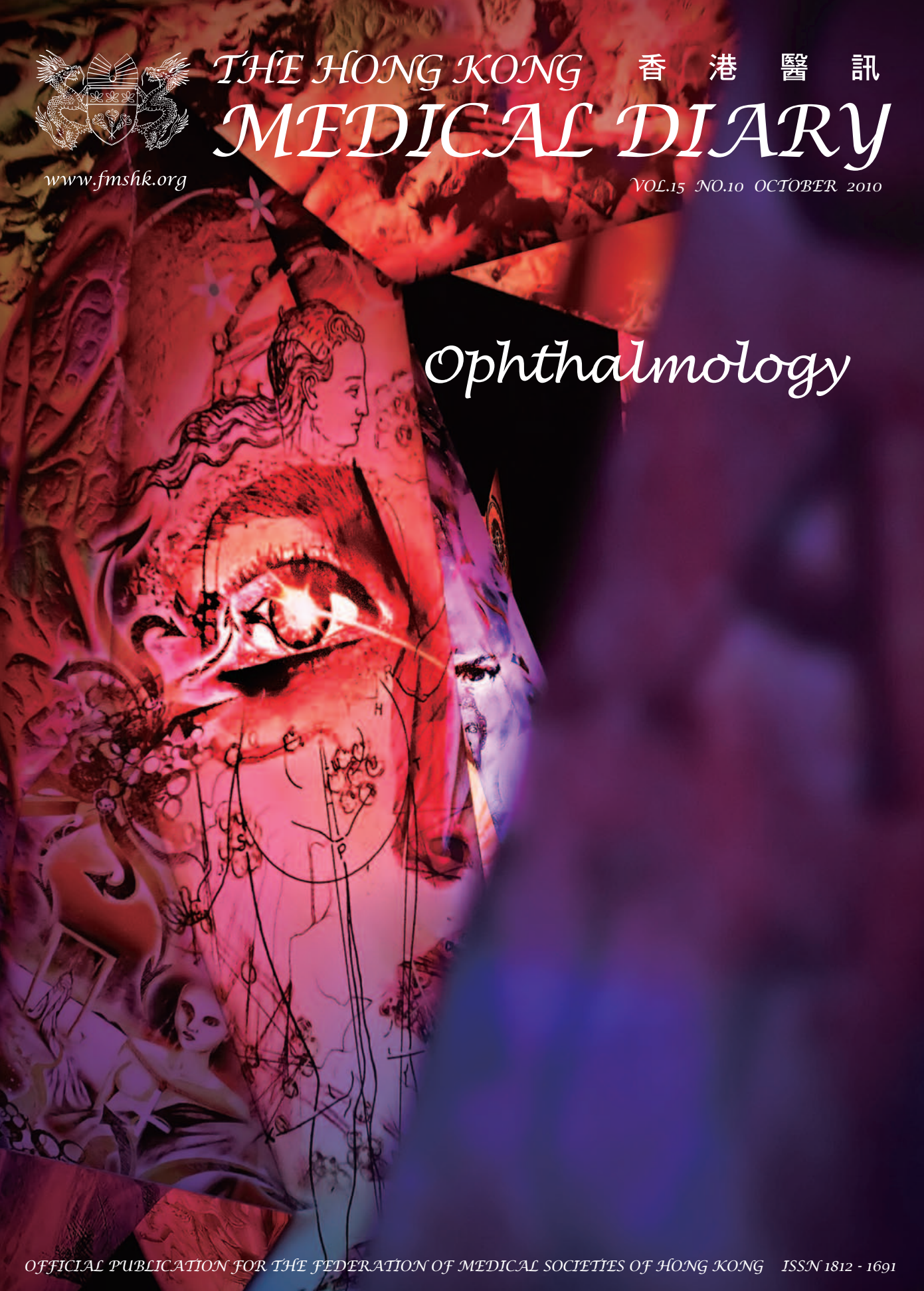
Ophthalmology



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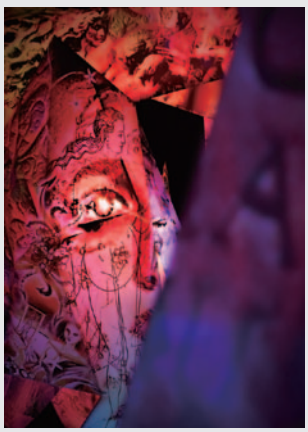

ISO 1600, f2.8 0.5sec 35mm
Dr Amy Lai-man PANG.
Innsbruck 2008.

BEING WATCHED WHILST WATCHING

Imagery of the human eye has been a recurring and persistent theme in art throughout human history. The famous Eye of Horus, the ancient symbol of the Egyptian falcon deity, was popular in numerous art pieces and sculptures in ancient times, and is still often drawn upon, for example in tattoos.

The human eye, the window to the soul, inspires both joy, fear and stirring emotion in the artists' audience. The art creations thus seem to communicate with the viewer, taking on a life of its own.

This photograph of a creation in Innsbruck Austria, which shows numerous eyes in different perspectives. The viewer of this art piece has the subconscious feeling, and a strong one at that, of being watched whilst watching. This photograph captures the latter emotion most vividly.

This photo was taken in a very dim light condition and changing color tone. No tripod was allowed. The key to this photo is to know your camera performance, your ability of stabilization and to choose the perspective and composition while being aware of the best color contrast to execute. What is the highest ISO with acceptable noise level? How long you can hold still? Turn on the noise reduction function in your camera before shooting. To stabilize your body, you may find something stable to lean upon. Hold deep breath while shooting. For this photo, to make the foreground blurry in order to divert attention to the object of interests "the eyes", and to complete the composition, choose a large aperture. Focus on the "eyes". Multiple shooting is advised, so that one could rest on either the trough or peak phases of the body vibrating rhythm, a sine wave. Post-processing, burn down the periphery, lighten with increased contrast of the eyes.

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Editorial

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Editor

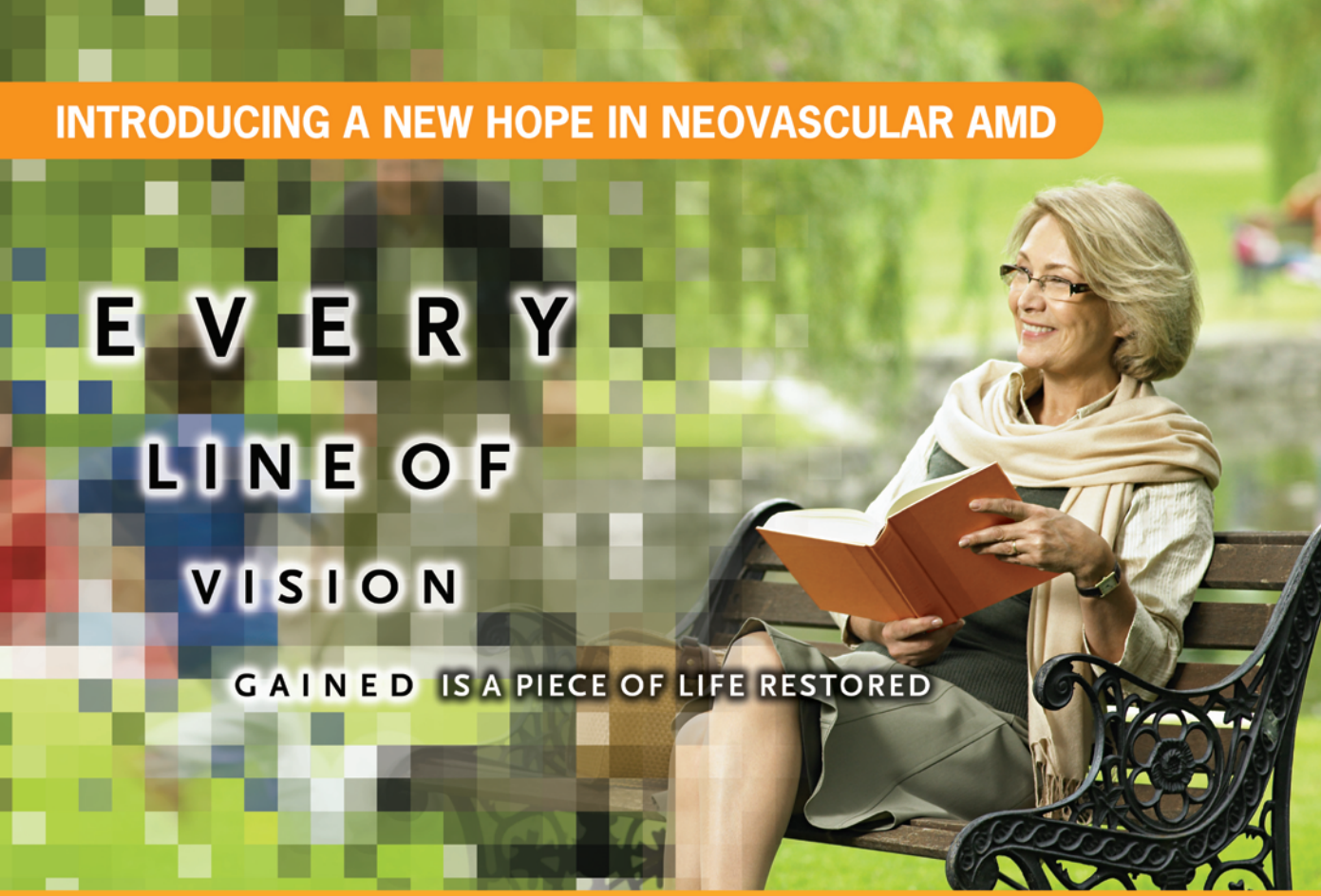
Dr. Timothy YY LAI

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 referrals. Among various eye conditions seen by clinicians, dry eye syndrome is probably the most frequently encountered ocular condition in clinical practice. It has been estimated that around 10-20% of the adult population in Hong Kong are developing ocular symptoms caused by dry eyes. In the first article of this ophthalmology issue of the Hong Kong Medical Diary, Dr. Jeffery C. F. Pong will review the clinical types, causes, assessment and treatments of dry eyes. Strabismus is also another frequently encountered ophthalmic condition in clinical practice and the management of patients with strabismus lies in the importance of making a proper diagnosis. In the second article, Dr. Jane C. C. Yeung will discuss the classifications, assessment, investigations and surgical and non-surgical treatments of strabismus.

Many systemic diseases can also affect the eyes and thyroid eye disease or Grave's ophthalmopathy is one of the most common and important extra-thyroidal manifestations of autoimmune thyroid disease. It is also one of the most common causes of proptosis or exophthalmos in adults. The clinical spectrum of thyroid eye disease can range from mild discomfort symptoms to significant visual morbidity resulting in irreversible blindness. In the third article of this issue, Dr. Kelvin K. L. Chong will comprehensively review the epidemiology, pathophysiology, diagnosis and treatment of thyroid eye disease.

Patients with vitreo-retinal diseases such as retinal detachment, epiretinal membrane and proliferative diabetic retinopathy commonly require vitrectomy for treatment of their retinal diseases. Recent developments in small-gauge microsurgical instruments and systems have enabled the development of vitrectomy without the use of sutures. The application of sutureless vitrectomy has reduced surgical time, enabled faster postoperative recovery, as well as less postoperative discomfort for the patients. In the fourth article of this issue, Dr. Gary K. Y. Lee and I will discuss the recent developments and application of 23-gauge sutureless vitrectomy surgery.

Glaucoma is one of the most common causes of blindness worldwide and is characterised by optic neuropathy associated with increase in intraocular pressure. Although most forms of glaucoma are primary in nature, the development of secondary glaucoma has also been associated with the use of various medications. In the penultimate article of this issue, Dr. Nafees Baig will review the mechanisms, pathophysiology, and treatment of drug-induced glaucoma. In addition to the use of anti-glaucomatous medications, non-pharmaceutical approaches might also be beneficial in the management of glaucoma. In the last article of this issue, Prof. Clement C. Y. Tham will give an overview on how exercise can be an effective non-pharmaceutical approach in the management of glaucoma.



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Thyroid Eye Disease: a Comprehensive Review

Dr. Kelvin KL CHONG

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Dr. Kelvin KL CHONG

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 31 October 2010.

Nomenclature of Thyroid Eye Disease[1]

Thyroid eye disease (TED) is also known as Graves' ophthalmopathy / orbitopathy, thyroid associated ophthalmopathy / orbitopathy (TAO), or thyrotoxic/ endocrine exophthalmos. It is the most important extrathyroidal manifestation of autoimmune thyroid diseases such as Graves' Disease and Hashimoto thyroiditis. It is also the most common orbital disorder in adults worldwide and the commonest causes of unilateral or bilateral axial proptosis (exophthalmos), acquired strabismus or lid retractions. TED may lead to visual dysfunction, ocular discomfort, facial disfigurement and significantly decreased quality of life.[2]

Disease Spectrum of Thyroid Eye Disease

Patients with TED may not have any clinical or biochemical evidence of thyroid dysfunction or autoimmunity. They may have only orbital involvement known as ophthalmic Graves' Disease or euthyroid Graves' Ophthalmopathy; or they may also have isolated thyroid dysfunction with minimal (subclinical) TED.

Epidemiology of Thyroid Eye Disease[3,4]

TED tends to have a bimodal presentation during the fourth or sixth decade. While the female to male ratio of patients with all forms of clinical TED is about 9:1; for those with the severe form of disease it drops to 3:1. A previous research from a Caucasian TED cohort of 120 patients, 90% had Graves' Disease, 7% was euthyroid, 3% had Hashimoto thyroiditis and 1% had primary hypothyroidism.

Chronology of Thyroid Eye Disease[5]

Around 4% of TED patients present more than 6 months before diagnosis of thyroid problems, while 19% within 6 months before. In about 20% of patients, there are concurrent ocular and endocrine features on presentation, while 22% and 35% had ocular manifestations within 6 months or more after being treated for thyroid dysfunction respectively.

Risk Factors of Thyroid Eye Disease[1,6]

The main risk factors for the development of TED in patients with thyroid disease include the male gender, advancing age of onset, smoking, use of radioactive iodine (RAI) and post-ablative hypothyroidism. Previous studies have demonstrated that smokers have a higher risk of developing GD, as well as additional chances of TED developing or worsening and a reduced response to immunosuppressants or orbital radiotherapy.[6]

Thyroid Eye Disease in Paediatric Patients[7, 8]

TED is not uncommon in the paediatric population but is usually far less severe than their adult counterpart. Among 83 Chinese children aged 16 or below with GD, 63% had ocular findings including 38.6% had lower lid retraction, 13% had punctate epithelial corneal erosion, 12% had mild proptosis (<3mm), and 1.2% had limited extraocular movement. None of the children developed visual threatening complications.

Pathophysiology of Thyroid Eye Disease[9, 10]

With genetic susceptibilities[11] and permissive environmental triggers, individuals with the risks factors are prone to develop autoantibodies which can stimulate the thyroid gland to cause goitre and hyperthyroidism (**Fig. 1A**), some attack the orbits leading to TED/TAO while a minority are responsible for pretibial myxoedema (**Fig. 1B**).[12] A T-cell mediated response appears to be important in orbital autoimmunity. In addition, autoantibodies to Thyroid Stimulating Hormone receptor and Insulin-like Growth Factor-1 receptor were found to be implicated in TED.[13] The physical co-localisation and functional linkage of the two receptors were recently reported.[14] Orbital fibroblasts are the primary cells responding to autoimmune stimuli leading to adipogenesis, accumulation of glycosaminoglycan (GAG) particularly hydrophilic hyaluronan causing tissue oedema, infiltration by lymphocytes, mast cells and secondary involvement of the extraocular muscles

with subsequent tissue fibrosis and remodelling. The anatomically confined orbital space further exacerbates compressive effects and congestive changes.[15] While extraocular muscle enlargement has been used to be considered as the cardinal feature in TED, there is now growing molecular, radiological and clinical evidences that adipogenesis is universal in all patients with TED particularly the younger patients. The dual differentiating pathways of orbital fibroblasts, or more recently circulating fibrocytes[16] may determine such fat or muscle predominant phenotypes of TED.

There are still many missing links between the thyroid and orbit in TED. How these two anatomically and embryologically distinct organs are related during the autoimmune attack is still unknown. The diagnosis of GD and Hashimoto thyroiditis with TED can be chronologically discordant. The asymmetric involvement (**Fig 2A**), predilection of certain EOM (inferior > medial > superior > lateral recti >obliques) are atypical of systemic conditions. As opposed to other autoimmune diseases, inflammation in TED will eventually quiet down but fibrotic and congestive features often persist and progress. Reactivation of TED does occur in rare circumstances.

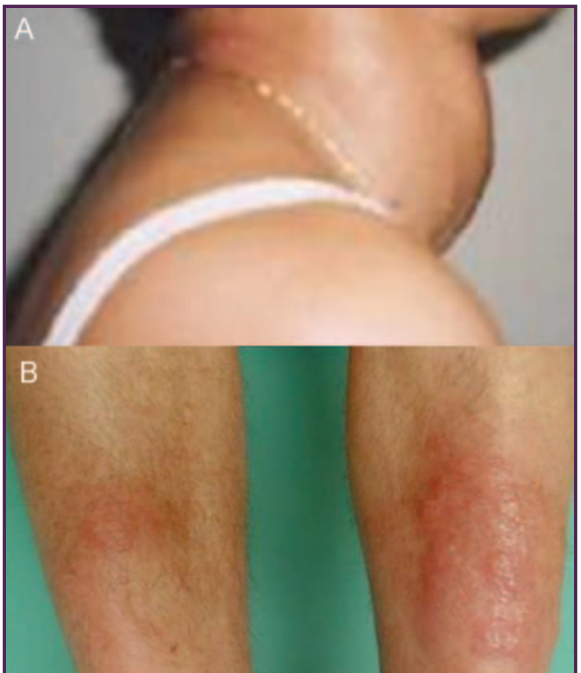

Figure 1 (A) Severe goiter in a patient with Graves' disease. (B) Bilateral pretibial myxedema (more severe on the left).

Clinical Features of Thyroid Eye Disesae[3-5]

Typical signs of TED from a Caucasian cohort include eyelid retraction (90%) (**Fig. 2B**), lid lag (50%) (**Fig. 2C&D**), exophthalmos (60%) (**Fig. 2E**), restrictive myopathy (40%) (**Fig. 2F**), optic nerve dysfunction (5%) (**Fig. 2G**) while classical presentation with the above (except optic nerve dysfunction) comprised only 5%. Physical findings may be grouped as extraocular: lid puffiness, lid retraction, lid lag, lagophthalmos (incomplete eyelid closure) (**Fig. 2H**), axial non-pulsatile proptosis (exophthalmos), restrictive strabismus, acquired lower lid epiblepharon in Asians, and conjunctival injection (**Fig. 3A**) , exposure keratopathy (**Fig. 3B**), chemosis (**Fig. 3C**), raised intraocular pressure (IOP), optic disc swelling, retinal venous congestion or choroidal folds. Vision loss in TED can be caused by optic nerve dysfunction (dysthyroid optic neuropathy), exposure keratopathy, raised IOP or globe subluxation.

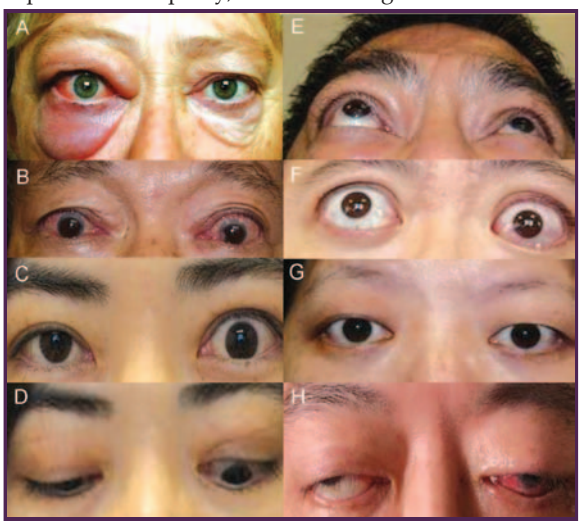

Figure 2 (A) Asymmetric active TED with lid edema and erythema over the right side only. (B) Symmetric active TED with bilateral upper lid retraction (more severe on the left), injection, lid swelling and chemosis. (C) Left upper lid retraction at primary gaze. (D) Left upper lid lag on downgaze. (E) Asymmetric exophthalmos (more severe on the right) on worm's eye view. (F) Severe restrictive myopathy affecting upgaze (left more severe); patient on attempt to look up. (G) Bilateral DON in a young patient with minimal inflammatory feature or proptosis. (H) Bilateral incomplete eyelid closure; left eye had poor Bell's reflex (the left eye did not roll up on lid closure)

Dysthyroid Optic Neuropathy[17]

Dysthyroid optic neuropathy (DON) is usually due to apical compression by enlarged extraocular muscles (**Fig. 4A**). Other mechanisms involved include inflammation, ischaemia or mechanical stretching. Patients often present with diplopia (particularly abduction deficits secondary to medial rectus enlargement) while patients with fat predominant subtype may have relative normal motility but severe proptosis and straightening of optic nerve on axial orbital scans (**Fig. 4B**). Signs of optic neuropathy include drop in vision, colour vision, visual field and afferent papillary defect or optic disc swelling. Patients with existing diabetes or of Asian origin (due to shallow orbits) have higher chances of developing DON. Medical decompression (e.g. intravenous methylprednisolone 1gm daily for 3 days) and often surgical (medial wall or fat) decompression are required subsequently.

Lid Retraction

Lid retraction is the commonest sign in TED (**Fig. 2B to 2D**). Practitioners should note that the normal upper lid rests at 1-2mm below the superior limbus (corneal-scleral junction) and the lower lid should be just below the inferior limbus. On the other hand,

while the commonest cause of lid retraction is also TED, its differential diagnoses include myasthenia gravis, myotonia dystrophy, Marcus Gunn jaw winking, metabolic diseases (uraemia, cirrhosis), Parinuad's (midbrain) syndrome, Parkinson's disease, contralateral ptosis or aberrant third nerve regeneration.

Proptosis (exophthalmos)

Patients with TED have axial non-pulsatile proptosis (exophthalmos) secondary to orbital venous congestion, accumulation of GAG and adipogenesis. Exophthalmos can be quantified by the exophthalmometer or radiologically with axial orbital scans.

Diplopia

Diplopia or double vision is often the most debilitating visual symptom in TED. Strabismus is restrictive (fibrotic) rather than paralytic in nature. As mentioned earlier, the inferior rectus is the most commonly involved, followed by the medial, superior then lateral rectus. Movement is therefore usually worst in elevation or abduction (**Fig. 2F**). Radiologically involved muscles are often enlarged but not infrequently sparing the tendons.

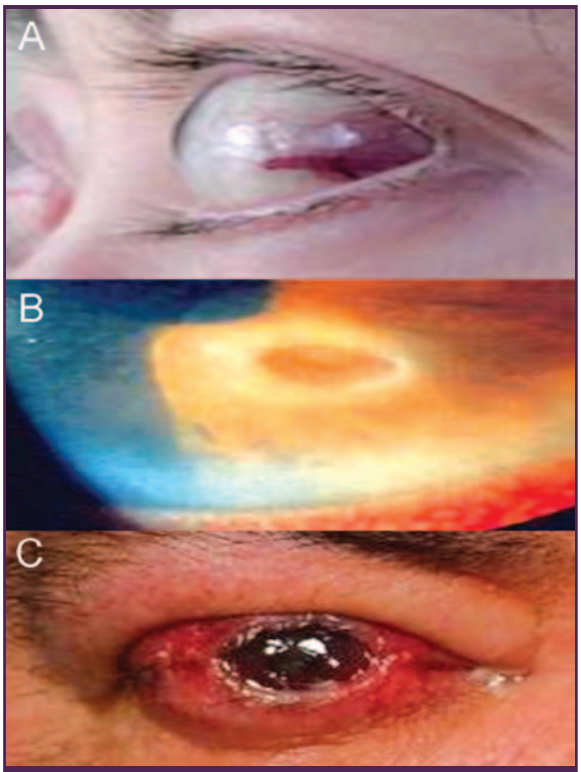

Figure 3 (A) Conjunctival injection seen around insertion of horizontal (in this case, lateral) recti muscles. (B) Exposure keratopathy and early microbial keratitis. (C) Severe conjunctival chemosis and injection during active TED

Diagnosis, Grading and Investigation of Thyroid Eye Disease[18, 19]

TED can be graded according to the severity (tissue remodelling or deformities) against activity (inflammation). The NOSPECS grading (Normal, Only sign, Soft tissue involvement, Proptosis, Extraocular Motility, Corneal exposure, Sight-threatening) can be used to define the degree of deformities, while the clinical activity score (CAS) utilises the parameters of inflammation (erythema, swelling, tenderness, loss of function).

Diagnosis of TED is largely clinical based on history and typical ocular examination findings. However, all patients should have appropriate endocrinological referrals for evaluation with thyroid function test for serum sensitive thyrotropin (sTSH) and free thyroxine (free T3, free T4) levels. Thyroid-related antibodies including anti-thyroglobulin, anti-microsomal and TSH receptor antibodies may be used adjunctively. Other ancillary ocular investigations involve the use of visual field (automated perimetry), colour vision assessment (Ishihara pseudoisochromatic plates), Hess chart (for extraocular movement) and binocular field (of single vision).

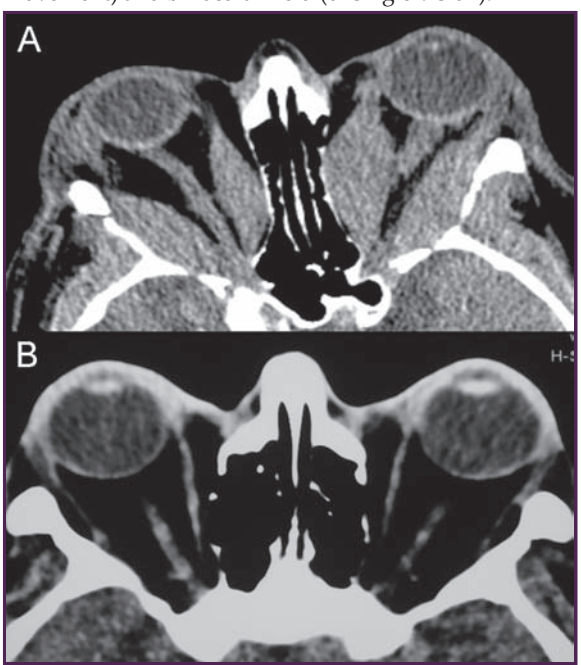

Figure 4 (A) Diffuse, symmetric, tendon-sparing extraocular muscles enlargement in axial CT scan of the orbits. (B) Symmetric severe proptosis, straightening of optic nerves with mainly enlargement of fat compartment and minimal muscle involvement often seen in young female patients with TED who are typically non-smokers.

For patients with features suggestive of infiltrative TED (motility restriction and/or proptosis), DON or before surgical decompression, orbital imaging should be considered. Non-contrast computerised tomography (CT) of the orbits performed in axial and coronal (direct or reformatted) planes readily reveals proptosis, EOM enlargement (**Fig. 4A**), apical compressions and bony anatomical variants for preoperative planning. Alternatively, orbital magnetic resonance imaging is superior in delineating the soft tissue component and T2 relaxation times on extraocular muscles have been used to assess the degree of oedema for disease activity or response to immunosuppressants. Orbital scintigraphy, positron emission tomography (PET) CT scan or more recently digital infrared thermal imaging have been introduced to measure disease activity for research purposes. The threshold of imaging should

be lower if there are atypical features suggestive of alternative diagnosis.

Differential Diagnoses of Thyroid Eye Disease

Differential diagnoses of TED include other orbital disorders such as carotid-cavernous fistula, idiopathic orbital inflammation (pseudotumour), orbital or preseptal cellulitis, and orbital tumour. Appropriate orbital imaging might be warranted to differentiate these conditions.

Management of Thyroid Eye Disease[20]

The multidisciplinary approach in managing TED requires close collaboration between physicians and ophthalmologists for early diagnosis, triaging, referral, symptomatic topical and systemic anti-inflammatory therapies, as well as staged surgical rehabilitation.

General Recommendations

Although the thyroid status does not always correlate with the presence, severity or activity of TED, we do recommend early stabilisation of thyroid functions (see section on prognosis). Ophthalmologists are often consulted on the risk of using RAI in patients with GD. It was reported that about 15-20% of patients receiving RAI have progression or development of TED.[21] Patients who are current smokers, with unstable thyroid function, high levels of thyroid-stimulating immunoglobulin (TSI) and in particular active (CAS≥3/10) TED are at risk. This may be controlled with a short tapering course of moderate dose (0.5mg/kg/day) oral prednisolone. RAI causes progression of TED by releasing intrathyroidal autoreactive lymphocytes and antigens while subsequent hypothyroidism (even subclinical) may promote accumulation of GAG by slowing down its turnover. Frequent biochemical monitoring and timely thyroxine supplement are therefore crucial.

All patients except those with the mildest form of TED would benefit from topical lubricants including eye drops and gel during day time and thicker ointment at bed time with or without taping the eyelids closed. Sunglasses may be valuable for those with photophobia or pending surgical rehabilitation. Stick-on (Fresnel) or spectacles-incorporated prism lens may alleviate small to moderate amount of diplopia.

Immunosuppressants

Immunosuppressants are occasionally required for patients with active (CAS>=3) TED. Systemic corticosteroid with oral prednisolone of 1mg/kg/day for 1-2 weeks is usually the first-line treatment of active TED. This is followed by slow tapering depending on the response. The treatment regime of using intravenous administration is more variable and common regimes include 500-1000mg pulse methylprednisolone for 3 days followed by oral prednisolone, or 500mg pulse methylprednisolone weekly for 6 weeks then 250mg for another 6 weeks. Recent reports suggest that intravenous corticosteroid were better tolerated and more effective than the oral route, though idiosyncratic hepatic failure and arrhythmia might occur at cumulative doses over 8g. Periocular steroid injections, e.g. triamcinolone acetonide (40mg/ml) can be used in asymmetric orbitopathy, mild relapses during tapering, or when the patient is contraindicated or reluctant for systemic steroids. In patients with persistent active disease despite a full course of steroid treatment, steroid-sparing agents (e.g. methotrexate, azathioprine, rapamycin, cyclosporin, cyclophospamide) or newer biologics (e.g. rituximab,[22] adalimumab) may be administered by rheumatologists.

Radiotherapy[23]

Orbital radiotherapy (fractionated external beam irradiation of 20Gy over 10 sessions) has been shown to have comparable effects with oral prednisolone for moderate to severe orbitopathy. It may be used with systemic corticosteroids concurrently or after surgical decompression for resistant or residual disease activity although younger patients or those with diabetic retinopathy are relatively contraindicated.

Anti-glaucomatous Medication

Anti-glaucomatous eye drops may be required to control secondary increased intraocular pressure. Topical alpha-agonists (e.g. brimonidine) have an added benefit of decreasing conjunctival and episcleral congestions compared with other eye drops.

Surgical Intervention

In general, stability in an endocrine and orbital status for at least 6-9 months is often recommended before surgical rehabilitation. A staged approach is undertaken as follows: orbital decompression, followed by strabismus surgery, correction of lid retraction, then finally blepharoplasty and other aesthetic operations.[24] Orbital decompression and/or expansion are classified into bone removal orbital decompression (BROD) and fat removal orbital decompression (FROD) which can be performed in isolation or in combination. The design of BROD is by the choice of surfaces and incisions for bone removal, e.g. medial (via transcaruncular, transcutaneous Lynch or endonasal), inferior (tranconjunctival forniceal/swinging eyelid, transcutaneous subciliary or transantral) and lateral walls (transcutaneous upper lid crease/swinging eyelid, coronal). Complications include diplopia, globe dystopia, periorbital sensory changes, orbital haemorrhage, infection, lid malposition, lacrimal gland or lacrimal drainage injury, cerebrospinal fluid (CSF) leak and rarely subarachnoid/cerebral haemorrhage.

FROD involves removing intraconal and sometimes extraconal orbital fat via a transcutaneous (upper lid crease or lower lid subciliary) or transconjunctival approach. Fat pockets are usually debulked in the following sequence: inferolateral, superonasal, inferomedial, and superotemporal (to avoid lacrimal gland and its neurovascular structures). FROD may cause fewer cases of new-onset diplopia or worsening of pre-existing diplopia compared to pure BROD (for similar amount of proptosis reduction). Anecdotal experience reports occasional improvement of motility restriction after FROD. Orbital haemorrhages and periorbital sensory loss may develop.

Strabismus correction in TED is typically challenging due to the fibrotic nature of EOM involvement. Usually EOM recession instead of resection is performed to correct the limitation of movement rather than the amount of ocular deviation at primary gaze. For example bilateral asymmetric inferior rectus (IR) muscles recession is performed to correct vertical diplopia, to improve upgaze and to avoid late progressive overcorrection. Detachment of capsulopalpebral fascia (lower lid retractor) has been proven to minimise postoperative lower lid retraction/ scleral show. Different techniques have been proposed to improve the outcome in strabismus surgery including an intraoperative relaxed muscle positioning technique, the use of adjustable sutures and operating under monitored aesthetic care or topical anaesthesia.

To correct upper lid retraction, mullerotomy, Muller's' muscle extirpation, levator aponeurosis disinsertion/ recession, levator muscle myotomy with/without the use of adjustable or hangback sutures can be used. Recently full thickness blepharotomy has gained much popularity because of technical simplicity. Spacers are often required for lower lid retraction (against gravity) after retractor disinsertion / lysis. Available materials include hard palate graft, dermis fat/strip graft, auricular/nasal septal cartilage, donor sclera and synthetic materials such as Medpor, Alloderm, aluminium foil, and Polytetrafluoroethylene with or without intraoperative anti-metabolites including mitomycin C (MMC) and 5-fluorouracil (5FU). Transcutaneous/transconjunctival Botulinum toxin A (BTA), steroid (triamcinolone acetonide) or filler (Restylane) injections have been used to alleviate lagophthalmos and exposure keratopathy before surgical intervention.

Prognosis of Thyroid Eye Disease[25]

Upon achieving euthyroidism, up to 90% of lid retraction, 30% of restrictive myopathy but rarely does proptosis improve. For patients having clinically evident TED (NOSPECS class 3 or above) the typical disease course usually runs for 12 to 24 months before it quiets down.

Prevention of Thyroid Eye Diseasen[25]

While the aetiology/pathogenesis of GD/TED is still unknown, primary prevention of TED (avoid occurrence) is not impossible by encouraging subjects at risk (family or personal history of autoimmune thyroid diseases, autoantibodies or biochemical dysthyroidism) to quit active and to avoid passive smoking. Secondary prevention (avoid progression of subclinical TED) involves early and accurate control of dysthyroidism (particularly post ablative treatment). Tertiary prevention (avoid development of visual threatening complications) requires early and judicious use of immunosuppressive therapies, orbital irradiation and timely surgical rehabilitation.

Conclusions

TED is the most common cause of proptosis or lid retraction in adults and can be markedly asymmetric. One in five patients with TED has normal thyroid functions on presentation. In some cases, visual threatening TED can present as subacute blurring of vision with minimal proptosis but upper lid fullness (DON) or severe eye pain, redness with incomplete lid closure (exposure keratopathy). Most thyroid patients do not require eye surgery but smokers and those who present with ocular symptoms or findings should be referred for assessment. Smoking cessation and early stabilisation of thyroid functions are the most important primary or secondary measures to prevent TED. More than one operation may be required to correct established or iatrogenic deformities in TED patients.

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MCHK CME Programme Self-assessment Questions

Please read the article entitled "Thyroid Eye Disease: a Comprehensive Review" by Dr. Kelvin KL CHONG 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 31 October 2010. Answers to questions will be provided in the next issue of The Hong Kong Medical Diary.

Questions 1-10: Please answer T (true) or F (false)

1. **Thyroid eye disease (TED) is a spot diagnosis.**
2. **Disease process in TED is always symmetrical.**
3. **TED is usually concurrent with thyroid dysfunction.**
4. **TED can be a feature of hypothyroidism.**
5. **TED does not present to non-ophthalmologists.**
6. **Non surgical treatments in TED equal to artificial tear.**
7. **Surgery for TED is only reserved for patients with poor vision.**
8. **RAI can be used in patients with TED.**
9. **TED is preventable.**
10. **Most patients with TED require ophthalmologists' care.**

ANSWER SHEET FOR OCTOBER 2010

Please return the completed answer sheet to the Federation Secretariat on or before 31 October 2010 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Thyroid Eye Disease: a Comprehensive Review

Dr. Kelvin KL CHONG

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1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9 ☐ 10 ☐

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

HKID No.: __ __ - __ __ __ __ X X (X) HKDU No.: _____

Contact Tel No.:_____ DCHK No.: _____

Answers to September 2010 Issue

The Current Status of Breast Augmentation

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

Dry Eye Syndrome – Diagnosis and Management

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Introduction

Dry eye is a common eye disease. In the US, as many as 6% of the population over the age of 40 and more than 15% of the population over the age of 65 suffer from dry eye.[1,2] According to a survey locally conducted, there is nearly 20% of the population with some dry eye symptoms.[3] According to the National Eye Institute's definition, dry eye is a disorder of the tear film due to tear deficiency or excessive tear evaporation which causes damage to the intraepalbebral ocular surface and is associated with symptoms of discomfort.[4] The dry eye syndrome (keratoconjunctivitis sicca) can be divided into the non-Sjogren syndrome, Sjogren syndrome and meibomian gland diseases. Clinically, symptoms associated with dry eyes can include ocular burning, foreign body sensation, stinging sensation, pain, photophobia or blurred vision.

Clinical Types of Dry Eye

The precorneal tear film is an essential structure of the ocular surface. This tear film can be divided into the anterior lipid layer, the middle aqueous layer and the innermost mucin layer which is secreted by the meibomian glands, the lacrimal gland and the goblet cells of the conjunctival epithelium respectively. Its use is to lubricate the eye, maintain nutrients and oxygenation of the ocular structures, act as a part of the refractive surface and help to remove debris from the ocular surface. In terms of tear production, dry eyes can be divided into the tear deficiency type and evaporative type. Tear deficiency dry eyes can be further divided into the non-Sjogren syndrome and Sjogren syndrome, which is an autoimmune disease associated with lacrimal gland and salivary gland lymphocytic infiltration. Evaporative dry eyes can be divided into the meibomian gland disease (MGD), exposure-related dry eyes and mucin deficiencies such as the Steven-Johnson syndrome and ocular cicatricial pemphigoid.

Causes of Dry Eyes

The dry eye syndrome is associated with a long list of causes. Essentially, causes of dry eyes can be divided into primary and secondary ones. Dry eye diseases can be secondary to environmental, hormonal, physiological, contact lens wear and pathological causes. With pathological causes, both the tear deficiency type and evaporative type can lead to the dry eye syndrome. Systemic diseases such as diabetes, thyroid disease, rheumatoid arthritis, systemic lupus etc. can also lead to dry eyes. In addition, patients with previous eye surgeries or regular use of eye medications or systemic medications can predispose to dry eyes. Many systemic medications, such as antihistamines, antidepressants, beta-blockers and oral contraceptives can also be associated with dry eyes.

Diagnostic Criteria

In terms of diagnostic criteria, Ohashi reported that (1) Symptoms of dry eyes, (2) Schirmer tests (< 5 mm after 5 mins) and clearance test (< 8x) (3) Fluorescein stain and Rose Bengal staining (>3+) are qualified as clinical dry eyes.[5] Other authors have devised other diagnostic criteria and there is so far no consensus. In many cases symptoms and signs do not correlate well with each other.

Essentially, to confirm the diagnosis of dry eyes, certain tests are necessary to be performed in the clinical setting. The tear film stability can be assessed with the fluorescein tear break-up time test (TBUT), measuring the interval in seconds between a complete blink and the first appearing dry spot or discontinuity in the precorneal film. Patients with TBUT less than 3 seconds are classified as clinical dry eyes. The tear meniscus is the tear pooling on the edge of the lower lid. If there is aqueous deficiency, the tear meniscus will appear to be thin and less than 1 mm in height. Another clinical method for assessing the severity of dry eye is the ocular surface dye staining. Fluorescein and Rose Bengal stain can both be used as diagnostic dyes for evaluating the staining. Fluorescein staining occurs when the epithelial barrier is disrupted, due to the loss of epithelial cells and serves as a good test for evaluation of dry eyes. Rose Bengal stains the devitalised epithelial cells of the conjunctiva and serves a similar purpose. However, Rose Bengal stain causes transient irritation after instillation and can be a problem for some patients. Patients with the dry eye syndrome can show signs of punctate epitheliopathy and even corneal abrasions.

Another important clinical test is the Schirmer test. It is a useful and robust test for measuring aqueous tear production. It is also easy to be performed in a simple clinical setting but it can be subjected to errors. Essentially, filter paper strips called Schirmer strips are placed on the lower lid inside the tarsal conjunctiva area. The patient is then allowed to blink as normally and the tear strip is reassessed according to the degree

it wets in 5 minutes. There are two ways to perform this test: a) Schirmer test I is performed without topical anaesthesia, which evaluates better the ability of the ocular surface to respond to ocular surface stimulation; b) Schirmer test II (or Basic Secretion test) which is performed under topical anaesthesia, evaluating better the basal tear secretion. Patients with tear soaked less than 10mm is said to have clinical dry eyes and those with less than 5 mm is said to be severely dry. However, it is important to note that Schirmer tests are subjected to environmental and physiological changes and results can vary with time.

There are other useful tests that can be done in dry eye diseases. Some of them are however experimental and used only in clinical studies. Functional vision is a test whereby vision is continuously tested for around 30 seconds without the patient blinking. The definition of FVA testing has been suggested to be an important indication of an individual's performance in relation to certain daily activities involving visual performance. The concept of FVA was first introduced by Goto et al6 who previously reported abnormalities of FVA in subjects with dry eyes. This test is however still limited by the subjectivity of the method of measurement and uncertainty of the timing of FVA measurements. Studies on functional VA have claimed advantages in diagnosing a range of dry eye diseases such as the Steven-Johnson syndrome.[7]

Another method to visualise the lucent tear meniscus is to utilise the principles of interference phenomena. Tear interferometry is a non-invasive visualisation method of the transparent tear film. The optical path difference from the reflectance at the surface of the tear lipid layer and at the interface of the tear lipid-aqueous layer causes a tear interference image. With a tear interference device (Tearscope Plus, Keeler, Windsor, United Kingdom) not only can the precorneal tear film be observed clearly and non-invasively, but the tear film at the tear meniscus can also be assessed as well. The tear interference therefore helps the clinician in diagnosing dry eyes and to assess the status of precorneal tear film post treatment.[8] Thus, with the help of different measurement devices and clinical tests, making a diagnosis of the dry eye syndrome now becomes more accurate.

Pathogenesis of Dry Eyes

Studies have been performed in looking at the proteomic profiles of the ocular surface. Protein analysis comparing dry eyes and normal eyes found decreases in lactoferin and Epidermal growth factor in the dry eye syndrome using enzyme-linked immunosorbant assay (ELISA). A protein found in acinar cells of the lacrimal gland, AQP-5 was shown to have increased in the Sjogren type of dry eye syndrome, indicating the possible leakage of such proteins into the tear via lymphocytic infiltration of the lacrimal gland.[5] Solomon et al found an increase in inflammatory cytokines of interleukin 1 (IL-1) alpha and IL-1 beta in both MGD and the Sjogren type of dry eye syndrome, indicating increased protease activity on the ocular surface, mainly on the conjunctival epithelium.[9] Apart from IL-1, IL-6 in the tear was also increased in the Sjogren syndrome,[10] indicating an inflammatory process of the Sjogren dry eye disease. Another study to look at sialic acid, a component of mucin in tear has found a lower level in dry eye patients compared to controls, indicating a change in quantity and quality of glycoproteins in the tear in dry eye diseases?.[11] The change in tear protein profile in the dry eye syndrome, especially the Sjogren disease, has shed some light on the mechanism of the dry eye syndrome.

Workup for the Dry Eye Syndrome

As the dry eye syndrome can be associated with a large range of causes, it is important for clinicians to perform a careful clinical examination of the eyes before proceeding to treatment. A careful history taking to illicit the patient's medical history such as diabetes, thyroid disease and other connective tissue diseases is necessary. A thorough contact lens history is also useful. Previous eye diseases and eye surgeries such as Laser refractive surgeries like LASIK are important considerations for the cause of the dry eye syndrome. Many medications can affect the tear secretion and it is important to illicit that in the history. A careful clinical examination involves a slit lamp examination to look at the ocular surface and to look for any possible meibomian gland dysfunction such as blepharitis, meibomian seborrhoea which can interfere with lipid production and therefore the tear film condition. A look at the fornices and tarsus for any scars and symblepharon is important to exclude any pre-existing Steven-Johnson syndrome and ocular surface inflammatory diseases or previous infections. A careful look at the conjunctiva and the corneal status will be helpful to assess the severity of dry eyes, with an increase in staining frequency and staining area in the more severe cases. Occasional corneal filaments and corneal oedema can be seen with extreme dry eyes. Some of the systemic causes of dry eyes such as rheumatoid arthritis, systemic lupus etc. involve not only the ocular surface and can give rise to inflammation of the episclera, sclera and even vitreo-retinal involvements. It is important to be thorough in the examination to look for any correlated diseases.

Management of Dry Eye Diseases

Management of dry eye diseases depends on the causes and severity of the dry eye syndrome. Essentially, artificial tear used to replenish the deficient aqueous layer of the tear film, and to dilute the cytokines necessary to substantiate the disease. Artificial tear comes in different viscosities and can be divided into preserved or non-preserved forms. If the tear deficiency is severe, then more viscous forms such as eye gel or even ointment can be used to maintain a better and longer ocular protection. Since dry eye diseases, such as the Sjogren syndrome is associated with inflammation, the use of topical steroids or non-steroidal anti-inflammatory medications are sometimes useful. Topical antibiotics may be necessary if the dry eye syndrome is associated with corneal complications. Meibomian gland diseases may warrant vigorous lid hygiene and warm compresses of the lid, together with topical or even systemic antibiotics such as doxycycline.[12,13] For more severe disease, topical immunomodulating

drugs such as cyclosporine-A drops (Restasis) may be necessary. Studies have demonstrated an improvement of symptoms and signs of the dry eye syndrome, together with improvement of T cell infiltration in conjunctiva and cytokines level in the tear with the use of cyclosporine-A drops.[14, 15]

In very severe dry eye cases, frequent topical lubricants may not suffice. Studies have looked into the use of autologous serum as topical eye drops for severe dry eyes and clinical improvement was evident with prolonged treatment of 4-6 weeks. Its additional growth factors compared to artificial tear are cited as important components necessary for epithelial healing. Autologous serum can be produced from a designated period of centrifuging venous blood and diluting it with balanced salt solution to around 20%.[16]

Bandage contact lens is sometimes useful in dry eyes to prevent and minimise the extent of exposure keratopathy. Severe dry eye diseases with corneal complications may warrant surgical intervention such as punctal occlusion. Lacrimal puncta can be plugged either temporarily with collagen plugs which are absorbable, or for a longer period with non-absorbable plugs which needs to be removed if problems arise. Permanent punctal occlusion can also be performed using local anaesthetics to permanently save the tear from draining through the tear ducts and canaliculi. For patients with dry eyes secondary to connective tissue diseases, it is important to work with medical physicians to optimise treatment for their systemic diseases. In very severe dry eyes diseases secondary to ocular surface diseases such as chemical injury, Steven-Johnson syndrome or ocular circatricial pemphigoid, amniotic membrane transplantation, tarsorrhaphy, keratoplasty, limbal stem cells transplantation or even other ocular prosthesis is necessary to restore vision.[17]

Accupuncture has been recently cited as a treatment option in the dry eye syndrome. Meta-analyses of studies have revealed clinical improvements in fluorescein staining, tear break up time and Schirmer tests after accupuncture. It is yet to see if the alternative medical practice does have a role in this common disease.[18]

Conclusions

The dry eye syndrome consists of a wide spectrum of diseases with different causes. Useful clinical tests include Schirmer tests, fluorescein dye tests and the tear break up time for the assessment of severity of the syndrome. More advanced tests such as tear interferometry, functional Vision test and other tear proteomics studies can be used to distinguish clinical dry eyes and their severity at the experimental level. Treatment depends on an accurate diagnosis and the severity of the dry eyes. Treatments that can replenish deficient tear include artificial tears, gel and ointment in mild to moderate dry eyes. In severe dry eyes, surgical approaches such as punctal occlusion can be used to save the tear. Other treatments such as topical steroids, topical immuno-modulating drugs, topical antibiotics, bandage contact lens, autologous serum and amniotic membrane can be useful in very severe cases. Certain conjunctival and lid surgeries can also be performed to treat specific causes. Clinicians should be aware of the extent of the dry eye symptoms and do not overlook lightly. A thorough history taking and investigation is necessary to identify the cause of the dry eyes.

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Diploma in Child Health Examination (DCH) 2011

The Hong Kong College of Paediatricians (HKCPaed) and the Royal College of Paediatrics and Child Health (RCPCH) will hold a Joint Diploma in Child Health Examination in Hong Kong in 2011 awarding DCH (HK) and DCH (International) to successful candidates.

The Examination is divided into two parts, Written (MRCPCH Pt IA) and Clinical. The MRCPCH Part 1A Examination is held three times a year in Hong Kong. The next MRCPCH Part 1A Examination will be held on **Tuesday, 8 February 2011.** The examination fee is **HK$4,250** for Part IA. Candidates who wish to enter the examination must hold a recognized medical qualification in Hong Kong.

Application: Candidates who wish to sit the examination in Hong Kong **MUST** apply through the Hong Kong College of Paediatricians (HKCPaed). For application details, please visit the HKCPaed website at www.paediatrician.org.hk/entcnews.htm or call the College Secretariat at 28718871.

Deadline for Application: Tuesday, 9 November 2010

Important Notice

New Clinical Examination for DCH from March 2006

A new format of the DCH clinical examination has been adopted since March 2006. Details of the new format and other relevant information can be viewed on the RCPCH website at: www.rcpch.ac.uk

Management of Strabismus

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Introduction

The term "strabismus" came from Greek, which means to squint or to look obliquely. Strabismus means ocular misalignment. One eye is misaligned in relation to the other when focusing on an object. It can be due to abnormalities in binocular vision or anomalies of neuromuscular control of ocular motility[1].

Classification of Strabismus

There is no single classification which is perfect or all-inclusive. The following are a few of the useful classifications[1]:

According to the age of onset:

- Congenital/ infantile: a deviation documented prior to the age of 6 months;
- Acquired: an ocular deviation with onset documented after the age of 6 months.

According to the direction of deviation:

- Horizontal: esodeviation (convergent squint), or exodeviation (divergent squint);
- Vertical: hyperdeviation (upward) or hypodeviation (downward);
- Torsional: incyclotorsion or excyclotorsion;
- Combined

According to fusional status

- Phoria: a latent deviation that is controlled by the fusional mechanism so that under normal binocular vision the eyes remain aligned;
- Tropia: a manifest deviation in which fusional control is not present;
- Intermittent tropia: fusional control is present part of the time.

According to fixation/ laterality

- Monocular: definite preference for fixation with one eye;
- Alternating: spontaneous alternation of fixation from one eye to the other.

According to variation of the deviation with gaze position or fixation eye

- Comitant (concomitant): the deviation does not vary with the direction of gaze or fixating eye;
- Incomitant (non-comitant): the deviation varies with the direction of gaze or fixating eye. Most incomitant strabismus are paralytic or restrictive. If acquired, incomitant strabismus may indicate neurologic or orbital diseases.

Assessment of Strabismus[1,2]

The assessment of strabismus is no different to other diseases. It starts with a good history taking, physical examination and then ordering appropriate investigations if needed.

History

Some special points to note in assessing strabismus include:

- The age of onset/ duration of the deviation or symptoms (e.g. diplopia). Old photographs are invaluable for this purpose;
- Is the deviation/ symptom associated with trauma or physical stress?
- Is the deviation/ symptom constant or intermittent?
- The past treatment should be reviewed.

Assessment of the visual acuity of each eye

Numerous tests are available to check for distance visual acuity, e.g. Snellen letters chart, illiterate E chart, Sheriden-Gardiner chart (for children). If the patient does not have corrective lenses, a pinhole may be used to try to ascertain the best visual acuity potential.

For pre-verbal children, preferential looking tests e.g. Cardiff picture cards can be used to estimate visual acuity. For uncooperative children/ patients, each eye can be occluded with hand or occlude to test if any objection is demonstrated when one eye is occluded. The patient may attempt to manoeuvre around the occluder when the good eye is covered but not when the poorly seeing eye is covered.

If these tests fail, simple observation on fixation and following ability can give an idea on the visual function, e.g. "CSM method". Corneal light reflex is "Central"; fixation on the examiner's torch when held motionless and when slowly moved about should be "Steady"; the alignment of the two eyes is "Maintained".

Assessment of stereo acuity

"Stereo acuity" is a sense of depth. Stereo acuity is appreciated when two simultaneous but slightly different images are fused and integrated by the brain.

Special charts e.g. Titmus stereotest fly test, Lang cards can be used to test and grade stereo acuity.

Assessment of ocular alignment

Abnormal head posture: Before concentrating on examining the eyes, one should note if the patient has any "abnormal head posture". Abnormal head postures may indicate restrictive or paralytic strabismus. Usually the patient places the head in a position that provides comfortable single binocular vision for the straightened view. Occasionally the head is placed to separate diplopic images maximally. Common "abnormal head postures" include face-turn, head tilt, chin-up or chin down, or any combination of the above.

Corneal light reflex tests: The patient fixates on a pen torch in front of him/ her. In normal circumstances, the corneal light reflexes will be central and equal between two eyes. The corneal light reflex will be displaced nasally (inner-side) from an exotropic eye; displaced temporally (outer-side) from an esotropic eye; displaced upward from a hypotropic eye and vice-versa from a hypertropic eye.

The amount of deviation can be estimated by the "Hirschberg method". Based on the assumption of a 4mm-pupil, the Hirschberg method assumes 1mm of displacement of light reflex across cornea corresponds to 7^0 of decentration or 15Δ (prism-dioptre), of ocular deviation of the visual axis. Therefore, a light reflex at the papillary margin is about 2mm from the papillary centre, which corresponds to 15^0, or 30Δ of deviation. Similarly, a reflex in the midpoint of the iris is about 4mm from the papillary centre, which is roughly 30^0 or 60Δ of deviation; a reflex at the limbus is about 45^0 or 90Δ of deviation.

The "Krimsky method" or "Modified Krimsky method" quantifies the light reflex displacement using appropriately held prisms (**Fig. 1**). The original description suggested placing the prism before the fixating eye, but it can be modified to hold the prism before the deviating eye, which is easier. [3]

Figure 1. Krimsky method to quantify the light reflex displacement with prism.

Cover tests: Cover tests are objective tests that measure horizontal and vertical strabismus (**Fig. 2**). Cover tests require the patient to be attentive and cooperative. Each of the patient's eye needs to be able to see the target and moves to take up fixation upon that target. If any of these requirements is lacking, the results of cover tests may not be valid. There are 3 types of cover tests: the "cover-uncover test", the "alternate cover test" and the "simultaneous prism-cover test".

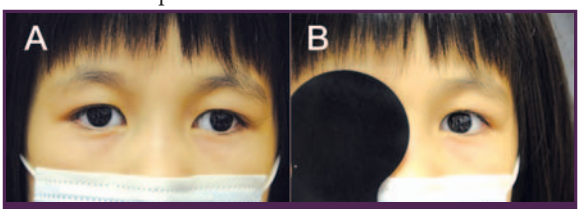

Figure. 2. Cover test in a patient with left exotropia. (A) Before covering the eye, the right eye was fixating and the left eye was deviated out. (B) After covering the right eye, the left eye took up fixation, indicating there was left eye exotropia.

The "cover-uncover test" detects tropia/ manifest squint. The examiner observes the uncovered eye for movement to take up fixation as the fellow eye is covered with an occluder or the examiner's hand. A movement towards the nose implies exotropia; a movement temporally, esotropia; an upward movement, hypotropia; a downward movement, hypertropia.

For the covered eye, the examiner should observe if it is deviated or moves when the cover is removed. The former implies that the tropia fixation preference shiftes to the original deviated eye; for the latter, the eyes become straight again when not occluded and it implies that there is phoria (latent squint).

Phoria is better detected by the "alternate cover test". Each eye is occluded alternately several times to dissociate the eyes. It is important to transfer the cover quickly from one eye to the other to prevent fusion. The direction of eye movement is noted when the occluder is swung between the two eyes. If no tropia was noted previously by the cover-uncover test, the eye movement elicited by the alternate cover test signifies phoria. If tropia was already present on the cover-uncover test, the alternate cover test measures the total deviation, both latent and manifest. When movement is detected, it can be quantified using prisms. Different powers of hand-held prisms are placed in front of an eye while doing the alternate-cover test until the eye movement is neutralised.

Tropia, when co-exists with phoria, can be measured using the "simultaneous prism-cover test". This test is done by placing prisms in front of the deviated eye at the same time when the fixating eye is covered.

Assessment of ocular motility

After assessing ocular alignment, the eye movement needs to be thoroughly checked. Three cranial nerves innervate six extraocular muscles (some would consider the levator as the seventh extraocular muscle). The fourth cranial nerve (trochlear nerve) innervates the superior oblique muscle, the sixth cranial nerve (abducens nerve) innervates the lateral rectus muscle and the third cranial nerve (oculomotor nerve) innervates the rest. Each extraocular muscle has different actions in different gaze positions. In the primary position (looking straight ahead), each muscle has its primary action, secondary action and tertiary action. (**Table 1**).

Table 1 Action of extraocular muscles			
Muscle	Action in primary gaze		
	Primary	Secondary	Tertiary
Medial rectus	Adduction	-	-
Lateral rectus	Abduction	-	-
Inferior rectus	Depression	Extorsion	Adduction
Superior rectus	Elevation	Intorsion	Adduction
Inferior oblique	Extorsion	Elevation	Abduction
Superior oblique	Intorsion	Depression	Abduction

There are six positions of gaze in which one muscle is the prime mover of the eye. These are called the six cardinal positions. The six cardinal gaze positions together with the primary gaze, straight up and straight down positions, a total of 9 gaze positions need to be examined.

Investigations for Strabismus

If the visual acuity is subnormal, the reason must be sorted. It may be as simple as a refractive error but it could be due to more sinister causes, such as retinoblastoma, congenital malformations, cataract, optic neuropathy or cortical blindness.

If the eye movement is impaired, it can be caused by numerous reasons. The site of pathology can be at various levels including:

- Central nervous system, e.g. stroke
- Cranial nerves, e.g. diabetic mononeuritis
- Neuromuscular junction, e.g. Myasthenia gravis
- Extraocular muscles, e.g. thyroid eye disease

Appropriate blood tests or imaging are indicated to establish the cause for the impaired eye movement.

Treatment of Strabismus

The treatment of strabismus must be tailored to the patient's own functional and cosmetic needs. It can be non-surgical or surgical. Non-surgical treatments include simple observation, wearing appropriate corrective lenses or prismatic glasses, doing orthoptic exercise and fogging or partial occlusion of one eye. Each form of non-surgical treatment is discussed in more details below.

Non-surgical treatments

Simple observation: Some forms of strabismus will go away with time or when the root cause is treated. Examples are Myasthenia gravis, diabetic mononeuritis, temporary limited eye movement related to post-trauma periorbital soft tissue oedema. In cases of small angle strabismus, patients will try to avoid diplopia by a slight abnormal head posture. If this abnormal head posture is not bothering the patient, then conservative treatment can be adopted. Interestingly in cases of very large angle strabismus, patients may not experience any symptoms of diplopia. The second image from the deviated eye is so far away from the main image that the patient is able to ignore it in daily life.

Correction of refractive error: This is especially important for children with accommodative esotropia. In these patients, esotropia may be fully or partially corrected when hypermetropic (plus-lenses) glasses are worn (**Fig. 3**). Whereas for children with intermittent exotropia, control of exotropia is often improved when full myopic (minus-lenses) correction is prescribed. It has also been suggested that even over-minus lenses can be used to induce more convergence associated with accommodation. However the prescription of over-minus lenses is not widely practised locally.

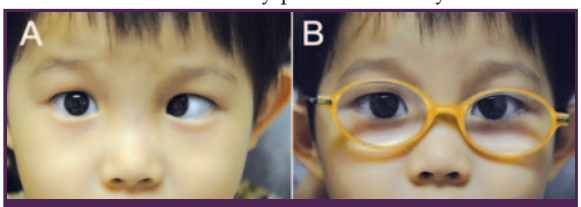

Figure 3. Accommodative esotropia. (A) Without glasses, there was left esotropia due to excessive accommodation. (B) With full correction of hypermetropia (plus-lenses), the left esotropia was corrected.

Orthoptic exercise: A commonly prescribed orthoptic exercise is the "pencil push-ups" to train up the control of intermittent exotropia or exophoria. A fixation target is placed at an arm's length and moved towards the nose. The patient needs to keep focusing on the target. Increasing accommodation is induced when focusing on progressively near target, thus convergence of the two eyes is also induced.

Prismatic glasses: Hand-held prisms are used to measure the angle of deviation clinically. Small angle prisms can be fitted into spectacle lenses for the patient to wear to neutralise the deviation (**Fig. 4**).

Fogging / partial occlusion: Especially in patients who are not willing to undergo surgery or cannot tolerate wearing prisms, fogging/ partial occlusion of one eye can be used to relieve diplopia. This method can also be used temporarily while waiting for surgery or to buy time for some forms of strabismus to resolve/ stabilise.

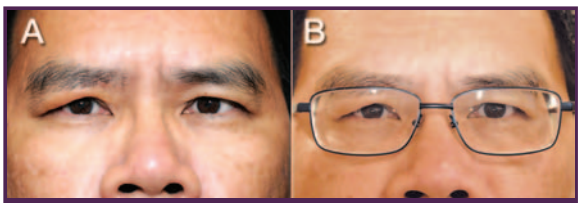

Figure 4. Primatic glasses for strabismus. (A) Without glasses, corneal reflex on the left eye demonstrated left esotropia. (B) After wearing prismatic glasses, the left esotropia was corrected with central corneal reflex.

Surgical treatments

Surgical treatments of strabismus include operating on the extraocular muscles or paralysing the extraocular muscles with Botulinum toxin injection.

Muscle surgery: Extraocular muscles are inserted into the sclera. The action of extraocular muscles can be manipulated by: 1. recession (weakening), 2. resection/ plication (strengthening), or 3. transposition (changing the vector of force). The extraocular muscles are accessed via the conjunctiva (i.e. no skin wound on the lids) (**Fig. 5A**). International tables are used as references to guide the amount of recession/ resection/ transposition to correct the measured deviation. Absorbable sutures are used in most cases

of strabismus surgery for both muscles (6/O Vicryl) and conjunctival wound closure (8/O Vicryl) (**Fig. 5B**). The risks of strabismus surgery include residual/ recurrent/ consecutive strabismus. Rare but important complications include globe penetration (through-and-through sutures), ocular ischaemia (especially after multiple muscles surgeries).

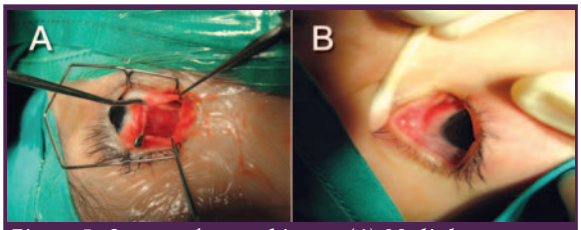

Figure 5. Surgery for strabismus (A) Medial rectus was identified for surgery after gaining access via the conjunctiva. (B) Conjunctiva wound with Vicryl sutures after strabismus surgery.

Botulinum injection: Botulinum toxin can be injected into the extraocular muscle(s) under electromyography guidance to temporarily paralyse the antagonist of a weak muscle, such as in the case of a weak lateral rectus in sixth cranial nerve palsy, Botulinum toxin can be injected into the medial rectus as a form of treatment.

Some Examples of Different Types of Strabismus

Congenital/ infantile esotropia

"Congenital" esotropia is rarely truly congenital. In fact, many babies have a moderate exodeviation at birth and this would go away by 6 months. Esodeviation can be occasionally seen and these babies may become orthophoric (straight eyes), usually by about 2 months[4]. One cannot predict if a baby will have a deviation at 2 to 4 months. So the documented presence of esotropia by 6 months of age has been arbitrarily defined as "congenital" or "infantile" esotropia. This type of esotropia is typically of a large angle, > 30Δ. Cross-fixation is frequent. Usually there is no significant refractive error. Associated vertical deviations e.g. inferior oblique overaction and dissociated vertical deviation are common. Nystagmus may be present. Monocular smooth pursuit is often asymmetrical. Treatment of congenital esotropia is essentially by surgery. Early surgery before 2 years of age is advocated[5,6].

Acquired accommodative esotropia

Accommodative esotropia is always acquired. The onset of accommodative esotropia is generally between 6 months and 7 years. It usually begins as intermittent esotropia and then becomes constant. Most of these children have high hyperopia (long-sighted) of at least +3.0 dioptres. Atropine drops or ointment is often needed to relieve all accommodation before the true refractive status can be fully revealed. One other feature is that the esodeviation is more at near than at distance fixation. The treatment for accommodative esotropia is to prescribe maximal plus-lenses for constant wear as a start and then reassess for any residual strabismus. If that is the case, atropinised refraction is to be repeated to see if there is anymore uncorrected hyperopia. If full-plus correction has been given and the child is compliant to wearing glasses, significant residual esotropia may be treated by surgery.

Intermittent exotropia

Exotropia is more common than esotropia in Hong Kong[7]. Intermittent exotropia is the commonest among all types. Intermittent exotropia typically presents between the ages of 18 months and 4 years. These children's eyes are mostly straight during the day. But when they get tired or are looking at distant objects, the exophoria breaks down to exotropia and one eye will deviate out. Often the deviation corrects itself after one or two blinks. Some would progress and the frequency of exotropia increases with time. The initial treatment of intermittent exotropia is to correct all myopia with glasses. Often the frequency of exotropia would reduce as the visual acuity at distance after wearing glasses improves. At near fixation, accommodative convergence is also induced. Accommodative convergence can also be enhanced by orthopic exercises, such as "pencil push-ups". To ensure this test is done correctly, an observer should watch for the convergence movements of both eyes. Apart from "pencil push-ups", sometimes monocular occlusion is used to prevent suppression. If non-surgical treatments have failed and the angle of exotropia is significant (>20Δ), muscle surgery may be indicated.

Graves' ophthalmopathy

Graves' ophthalmopathy is an immunological disorder that affects the orbital muscles and fat. Patients with Graves' ophthalmopathy are not necessarily hyperthyroid. A minority of patients (< 10%) are euthyroid or even hypothyroid.[8] Smoking is a known risk factor that increases the severity of Graves' ophthalmopathy.[9] Lid retraction and proptosis cause corneal exposure symptoms of grittiness, tearing and photophobia; enlarged or fibrotic extraocular muscles can cause diplopia. The inferior rectus and the medial rectus muscles are most commonly affected. Graves' ophthalmopathy can be a blinding condition when there is severe corneal complication or compressive optic neuropathy. Overall, there is also a cosmetic issue apart from functional eye problems. When troublesome diplopia due to restrictive strabismus is present, non-surgical treatments can be used first. The thyroid status needs to be controlled and smokers are strongly advised to quit smoking. Strabismus surgeries should be delayed until the overall ophthalmopathy is stable for at least 6 months and after orbital decompression surgery if both procedures are required.

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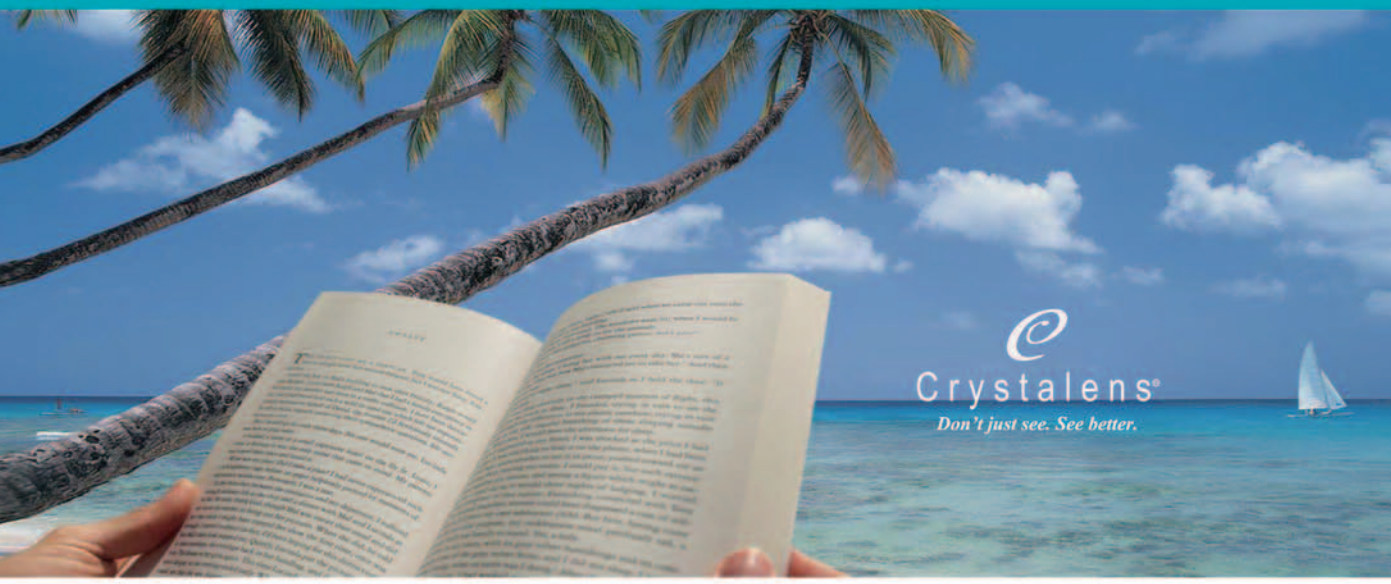

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The Role of Exercise in Glaucoma Management

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Introduction

Glaucoma is a disease of the optic nerve, with progressive and irreversible loss of optic nerve fibres. Risk factors for glaucoma include intraocular pressure (IOP), age, race, family history, refractive error and vascular factors. Exercise has both short- and long-term effects on IOP and vascular factors, such as ocular blood flow (OBF). Exercise may, therefore, influence the pathogenesis and / or progression of glaucoma.

Potentially Beneficial Effects of Exercise in Glaucoma Patients

Intraocular pressure-lowering effects

Isometric exercise is defined as work performed by a muscle with no change in the length of that muscle. In general, acute isometric exercise results in acute but transient IOP reduction,[1] which correlates with hyperventilation and hypocapnia.[2]

Dynamic (isokinetic) exercise is defined as work performed by a muscle with changes in the length of that muscle. Walking and swimming are examples of dynamic exercises. Acute dynamic exercise results in acute but transient IOP lowering in the post-exercise period.[3] The magnitude of IOP lowering can be up to 12.8 mmHg in glaucoma patients. IOP lowering induced by dynamic exercise appears to correlate with the intensity of the exertion,[1,4] and is more pronounced in glaucoma patients than in the normal population.[5] It has no significant correlation with blood pressure,[6] heart rate[7] or hypocapnia.[8] The IOP-lowering effect appears to be addictive to the effects of glaucoma drugs.[9] There is no significant difference in IOP lowering between aerobic and anaerobic exercises.[10] Dynamic exercise results in greater IOP reduction than isometric exercise, but of shorter durations.[11]

The mechanisms underlying exercise-induced IOP reduction are not well delineated. Three mechanisms have been proposed: osmotic dehydration of the globe, reduced aqueous production due to reduced ultrafiltration, and a hypothalamic reflex.[12] The above exercise-induced IOP lowerings were all short-lived, and their relevance in the long-term management of chronic glaucoma is uncertain. Long-term regular exercise is associated with overall improvement in physical fitness. Physical fitness appears to be associated with lower baseline IOP,[13] but diminished acute IOP-lowering response to exercise.[4] On termination of the exercise regimen, values return to pre-training levels within 3 weeks.[14] Such sustained reduction of IOP associated with regular exercise and improved physical fitness may be more relevant to the halting of glaucoma progression, but controlled studies are needed to confirm such potential therapeutic benefits.

Effects of Exercise on Ocular Blood Flow

Reduced ocular blood flow (OBF) is a potential risk factor for glaucoma.[15] In healthy subjects, OBF is unchanged during exercise due to vascular autoregulation.[16] This autoregulation fails at ocular perfusion pressures greater than 67% above baseline.[16] The relevance of these findings to the pathogenesis and progression of glaucoma is uncertain. The effects of exercise on OBF in glaucoma patients have not been studied.

Potential Deleterious Effects of Exercise in Glaucoma Patients

Certain isometric exercises, such as weightlifting and exercise at maximal exertion, may paradoxically increase IOP,[17,18] and the increase may be even more significant when the subjects are holding their breath.[19] Raised intracranial pressure may contribute to the IOP increase.[20] Exercise may also provoke increased IOP in patients with pigmentary glaucoma.[21] In these patients, the potentially harmful effects of exercise on IOP should be carefully weighed against the beneficial effects of exercise on general health. Young adults with advanced glaucoma may sometimes experience a temporary loss of vision during vigorous exercise. This was attributed to a 'vascular steal' phenomenon.[22] The relevance of this phenomenon to glaucoma progression is uncertain.

Conclusions

In general, acute exercise results in an acute but transient IOP reduction in the post-exercise period. Physical fitness secondary to a long-term regular exercise regimen is associated with lower long-term baseline IOP. Certain types of exercise, e.g. weight lifting, may increase IOP. Certain subtypes of glaucoma, e.g. pigmentary glaucoma, may have IOP increased after exercise. However, it remains uncertain whether such exercise-induced IOP changes correlate with glaucoma pathogenesis and / or progression. Taking also into consideration the beneficial effects of exercise on general health and well being, the author believes glaucoma patients should not be discouraged from regular and moderate exercises.

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Advances in Vitreo-retinal Surgery: 23-gauge Sutureless Pars Plana Vitrectomy

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Introduction

Pars plana vitrectomy is one of the most common surgical procedures performed for the treatment of various vitreo-retinal diseases such as retinal detachment, vitreous haemorrhage, proliferative diabetic retinopathy, epiretinal membrane and macular hole. Traditionally, most vitrectomy surgical systems utilise the 20-gauge instruments pars plana vitrectomy includes multiple incisions including periotomy (opening of the conjunctiva) and sclerotomy. In the past few years, advances in the development of surgical instruments have enabled the performance of vitrectomies using smaller gauge instruments such as 23 and 25-gauge, and thus allowing the performance of sutureless vitrectomies. The 23-gauge sutureless vitrectomy system has the advantages to overcome the excessive flexibility of instruments associated with a 25-gauge pars plana transconjunctival sutureless vitrectomy system which might cause difficulties in manoeuvring the globe during surgery. Most major vitreo-retinal surgical instrument manufacturers have already made a 23-gauge vitrectomy system available. With the increasing availability and better designed instruments, the 23-gauge sutureless system has become one of the preferred vitrectomy systems in the vitreo-retinal community in recent years.

Operative Techniques

Anaesthesia

As with conventional 20-gauge pars plana vitrectomy, 23-gauge pars plana vitrectomy can be performed under general anaesthesia or retrobulbar anaesthesia. In cases of using retrobulbar anaesthesia, proper techniques should be taken to avoid subconjunctival accumulation of the local anaesthetic agent. Any ballooning of the bulbar conjunctiva would render subsequent insertions of the transconjunctival cannulas more difficult to perform and might potentially increase the risk of postoperative wound leakage due to poor wound construction.

Two-step Sclerotomy Technique

The original method of inserting the transconjunctival microcannulas was described by Eckardt.[1] This method involves a two-step technique with the use of a specially designed pressure plate and a 23-gauge stiletto blade for entering into the vitreous cavity. The first step involves displacing the conjunctiva over the intended sclerotomy site with a pair of forceps or a cotton applicator. This displacement of the conjunctiva over the scleral incision allows coverage of the sclerotomy by keeping an intact conjunctiva at the end of the operation and thereby decreases the chance of wound leakage and potential entry of microorganisms. After displacing the conjunctiva, a pressure plate can be applied firmly on the displaced conjunctiva against the sclera (**Fig. 1A**). The edge of the pressure plate should be placed at the corneoscleral limbus. At this position, the central opening of the plate is located 3.5mm posterior to the limbus. Apart from acting as a tool for measurement, the pressure plate also prevents slipping of the displaced conjunctiva over the sclerotomy site and allows the surgeon to rotate the globe during the insertions of the cannulas. Moreover, flattening the sclera during the incision with the stiletto blade would increase the length of the scleral wound and enhance the self-sealing effect.

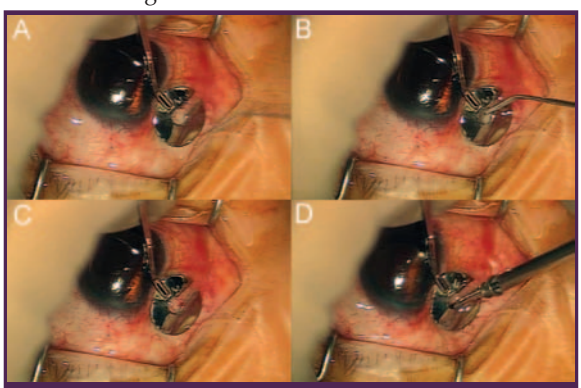

Figure 1. Steps for insertion of the 23-gauge vitrectomy microcannulas using the two-step technique. (A) Using a pressure plate, the conjunctiva over the intended sclerotomy site is displaced and pressed firmly against the sclera. (B) The transconjunctival scleral tunnel incision is performed using a 45°-angled-stiletto blade through the central opening of the pressure plate at an angle of 30°. (C) The pressure plate is kept in place against the sclera after creation of the scleral tunnel so that the conjunctival opening is still in line with the tunnel. (D) The microcannula is inserted into the scleral tunnel through the conjunctival opening using a blunt trocar inserter. The direction of insertion is shifted from the initial tangential path to a more perpendicular course towards the center of the globe.

With the pressure plate held firmly in place, a 45°-angled-stiletto blade is used to create a 30° blique scleral tunnel (**Fig. 1B**). The scleral tunnel should be made parallel to the corneoscleral limbus in order to ensure that the full length of the wound is in equal distance from the limbus and within the pars plana. During the passage of the stiletto blade, the surgeon may experience some rotation of the globe in the direction of the incision. This can be avoided by

using a new sharp blade and by stabilising the globe with the pressure plate. Some surgeons advocate an antero-posterior scleral tunnel with the blade aiming towards the posterior pole. It is thought that this technique would allow splitting rather than cutting of the concentrically arrange scleral fibres, allowing faster postoperative wound healing. However, this benefit is offset by the potential risk of retinal damage if the internal opening is created too close to the ora serrata.

The pressure plate should be kept in place after withdrawal of the blade (**Fig. 1C**). With the DORC system, the microcannula is inserted through the conjunctival incision into the scleral tunnel with a blunt inserter. During the insertion of the microcannula, the direction of insertion should be shifted from the original tangential path to a more perpendicular direction towards the centre of the globe (**Fig. 1D**). This manoeuvre helps exert the necessary pressure on the globe to minimise any rotational movements. The pressure plate can be slowly withdrawn before the complete insertion of the trocar. During removal of the trocar, the microcannula should be held in place with forceps to avoid dislodgement. The microcannula is then left in place and plugged. Normally, the first microcannula is inserted at the inferotemporal quadrant for the infusion line. Two more microcannulas are then placed at the superotemporal and superonasal quadrants for the microsurgical instruments. In case of combined phaco-vitrectomy, the micocannulas should be inserted before the phacoemulsification procedures as this allows better control of vitreous pressure during phacoemulsification.

Single-step Sclerotomy Technique

The main limitation of the previously described two-step sclerotomy technique is the difficulty in identifying and tracing the original conjunctival incision after the first stiletto cut. This might cause problems during insertion of the blunt inserter and result in double-incision through the conjunctiva. To tackle this limitation, major manufacturers such as Alcon and Bausch & Lomb have introduced one-step trocar/cannula systems (**Fig. 2**). With the one-step technique, the blunt inserter is replaced with a sharp needle trocar for the creation of the conjunctival incision and scleral tunnel without the use of a stiletto blade. Similar to the two-step system, a scleral tunnel at a 30° angle parallel to the limbus is created after the conjunctiva is displaced. Compared with the stiletto blade, the needle trocar has slightly greater tissue resistance during incision and this might occasionally result in a more significant globe rotation. However, newly designed needle trocar systems have greatly enhanced the blade design and the increased sharpness has allowed less resistance during wound construction with a more water tight wound.

Vitrectomy

Techniques of vitrectomy using the 23-gauge system are similar to the conventional 20-gauge system due to the similar stiffness of the instruments. Nevertheless, forceful insertion and manipulation of instruments can lead to bending within the metal cannula and later difficulties during removal of instruments. Careful handling of instruments is important to prevent this complication. Dislodgement of microcannula during vitrectomy is another potential problem for the 23-gauge system. It is usually related to incomplete insertion of the microcannula at the start of the operation. With the help of a blunt inserter, the dislodged microcannula can usually be re-inserted smoothly into the original sclerotomy. Gentle and slow removal of microsurgical instruments through the lumen of the cannula will also be useful in preventing dislodgement of the microannula. Since no suture is used to hold the infusion line, dislodgement of the infusion line may sometimes occur and can lead to sudden hypotony and collapse of the globe. Extra caution is therefore needed when securing the connection of the infusion line before starting vitrectomy.

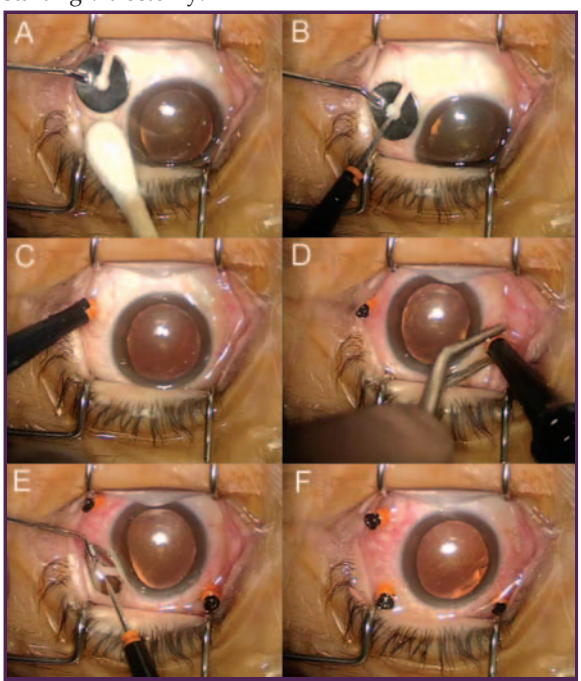

Figure 2. Steps for insertion of the 23-gauge vitrectomy microcannulas using the one-step technique. (A) The conjunctiva is displaced over the intended sclerotomy site and pressed firmly against the sclera using the pressure plate. (B, C) A sharp trocar needle is used for the transconjunctival scleral tunnel incision through the central opening of the pressure plate at an angle of 30°. (D) A forceps can be used to provide a counterforce and to hold the cannula in place while the needle is being withdrawn. (E) A third transconjunctival sclerotomy is being created by the sharp needle at a 30° angle. (F) All three sclerotomy microcannulas with plugs in place.

Removal of Microcannulas

Removal of the microcannulas should be started by lowering the intra-ocular pressure to about 20mmHg. The two superior cannulas for the surgical instruments should be plugged before removal and the infusion cannula is removed last. The microcannulas are held by forceps and withdrawn along the direction of the scleral tunnel (**Fig. 3**). A cotton wool applicator is used to press on the conjunctiva and rotate back the conjunctiva over the sclerotomy immediately after removing the microcannulas. Sustained pressure for 30 to 60 seconds is generally adequate to allow self opposition of the sclerotomy wounds and to stop any bleeding from the wound area. Globe pressure and the external opening of the scleral tunnels should be checked carefully. In case of persistent leakage from the sclerotomy and overt hypotony, the scleral wound should be sutured and the

globe reinflated with air, gas or balanced salt solution as required.

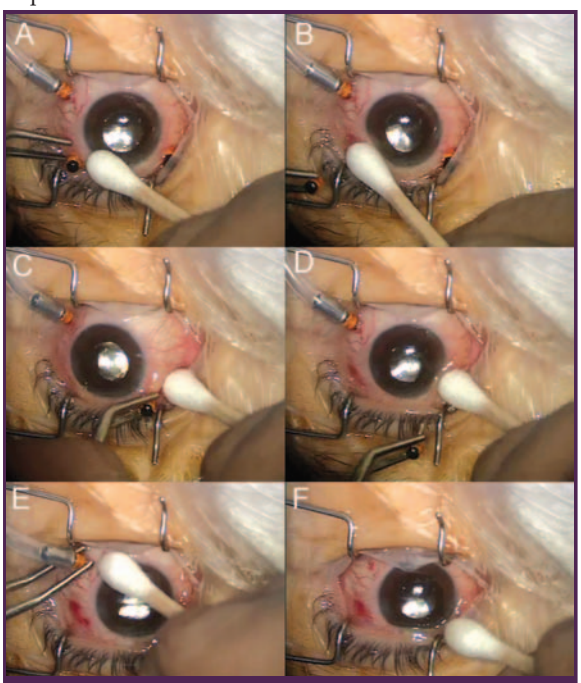

Figure 3. Steps for removal of the 23-gauge vitrectomy microcannulas (A) The microcannula is grasped with a forceps and removed along the path of the scleral tunnel. (B) A cotton wool applicator is applied on the wound after removal of the microcannula to oppose the scleral wound edges and to stop any bleeding. (C, D) The procedure is repeated for removal of the superonasal microcannula. (E) After stopping the infusion of balanced salt solution or the gas infusion is clamped, the microcannula with the infusion line is removed last. (F) All three microcannulas removed.

Indications of 23-gauge Sutureless Vitrectomy

There has been a continuous expansion in the clinical indications for the 23-gauge vitrectomy system. With the increase in the spectrum of various 23-gauge instruments and improved surgical techniques, the indications for any 23-gauge system are now almost identical to the conventional 20-gauge system. These include macular conditions such as epiretinal membrane and macular hole, vitreous haemorrhage, retinal detachment, proliferative diabetic retinopathy and removal of low viscocity silicone oil.

The 23-gauge vitrectomy system offers a clear advantage over the conventional 20-gauge system in less complicated surgical conditions, such as for removal of vitreous haemorrhage followed by endolaser panretinal photocoagulation. Macular pathologies, including macular hole, epiretinal membrane and vitreomacular traction syndrome are also amongst the most suitable indications for 23-gauge surgery. In general, most studies have demonstrated good success rates in using 23-gauge vitrectomy for macular hole and epiretinal membrane peeling.[2,3] 23-gauge vitrectomy has also been successfully applied in the treatment of retinal detachment and the primary anatomical success rate appeared to be comparable with conventional 20-gauge pars plana vitrectomy.[4] Moreover, 23-gauge vitrectomy is also feasible in complex retinal detachment surgery with silicone oil tamponade for retinal detachments associated with proliferative vitreoretinopathy, diabetic tractional retinal detachment, and giant retinal tear.[5] Removal of silicone oil of low viscosity is also possible with 23-gauge system by expelling the oil passively through the microcannula under the hydrostatic pressure from the infusion line. In cases with high viscosity silicone oil, such passive removal would be excessively time-consuming and impractical. In this instance, one sclerotomy can be enlarged to 20-gauge in order to accommodation the use of conventional 20-gauge instruments.

Contraindications of 23-gauge Sutureless Vitrectomy

Conditions that involve ultrasonic endo-fragmentation of retained lens fragments are amongst the few contraindications of 23-gauge vitrectomy. At the time of writing, a 23-gauge phacofragmatome is still not yet commercially available. The design of the microcannulas also prohibits the passage of various angled tools. For conditions that require the use of such special instruments, 23-gauge vitrectomy may not be the preferable choice. Nonetheless, some 23-gauge instruments with retractable and bendable materials are now available and these instruments can facilitate intraoperative procedures by allowing improved accessibility.

Advantages of 23-gauge Sutureless Vitrectomy

One of the most obvious advantages of using 23-gauge vitrectomy is the shortening of operation time during creation and closure of sclerotomies. The wound opening and closure time was significantly shorter for the 23-gauge system when compared with the conventional 20-gauge system. In addition to shortening the operation time, patients who had 23-gauge pars plana vitrectomy also had significantly less pain compared with the 20-gauge system.[6] The increased level of patients' comfort can be attributable to the absence of sutures and conjunctival peritomy, thereby allowing a faster wound healing and less inflammatory reaction (**Fig. 4**). The smaller wound sizes in 23-gauge surgery also allow a shorter recovery time and minimise postoperative discomfort.

Reoperations in patients who have previously undergone multiple conventional 20-gauge vitrectomies can be challenging not only due to the extensive conjunctival scarring, but also because of the difficulty in finding a new site for sclerotomy. The sutureless transconjunctival approach of 23-gauge surgery enables the surgeon to preserve the mobility of the conjunctiva even after repeated operations. This is especially important in glaucoma patients who may later require filtering surgeries. The smaller diameter of the scleral wound in 23-gauge vitrectomy also leaves more fresh sites for future sclerotomies. Compared

with conventional 20-gauge vitrectomy, there is less surgically induced astigmatism following 23-gauge vitrectomy.[7] This allows for faster stabilisation of postoperative refraction and faster visual rehabilitation of the patients.

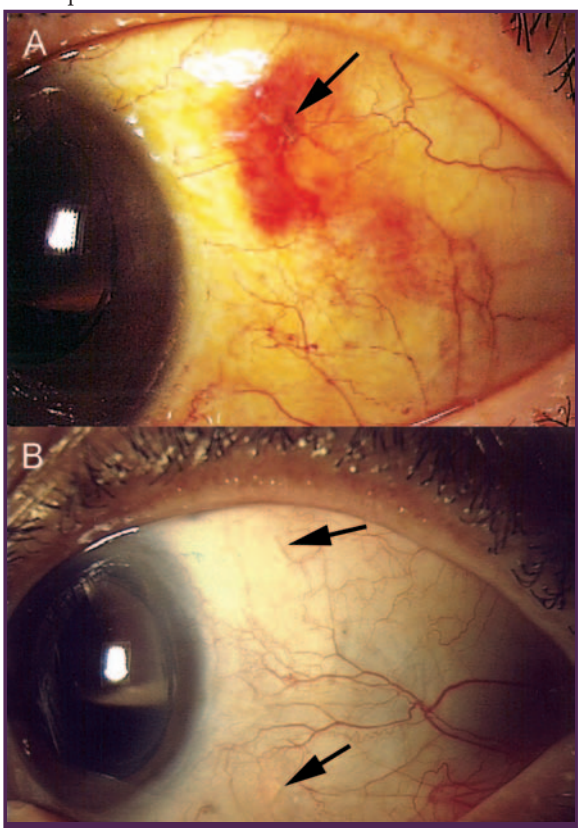

Figure 4. Postoperative slit-lamp photographs of patients after 23-gauge transconjunctival vitrectomy. (A) At day 1 postoperation, there was mild subconjunctival hemorrhage associated with a well-sealed sclerotomy wound (arrow). No leakage was seen at the sclerotomy site. (B) At week 1 post-operation, the sclerotomies (arrows) were barely visible under the conjunctiva.

The cutting port of a 23-gauge vitreous cutter is located closer to the tip when compared with a conventional 20-gauge cutter. This feature enables the surgeon to cut the vitreous closer to the retina more efficiently. The smaller calibre of the cutting probe also enables access into narrower tissue planes. Together with the newer vitrectomy machines with a cut-rate of up to 5000 cuts per minute (cpm), dissection of tractional membranes is made much easier with a 23-gauge probe and the need for microscissors is greatly reduced.

Compared with the 25-gauge system, 23-gauge instruments are much stiffer and less flexible. Manoeuvring of surgical instruments and control of globe movements are much more efficient with 23-gauge instruments. This is especially important when performing endolaser and accessing the peripheral retina. The closer resemblance to the conventional 20-gauge system also shortens the learning process for changing from a 20-gauge to a 23-gauge system. Reusable 23-gauge microsurgical instruments are also more durable and easier to clean than 25-gauge instruments.

In paediatric patients, post-operative inflammation after intra-ocular surgery is known to be more severe compared with their adult counterparts. The smaller wound size of the 23-gauge vitrectomy system can help to minimise surgical trauma and decrease the post-operative reaction in this age group. The faster wound healing also allows an early rehabilitation for the developing visual system.

Disadvantages of 23-gauge Sutureless Vitrectomy

Eckardt noted in his original series of 23-gauge vitrectomies that the vitreous cutter was 'somewhat slower' for extensive vitrectomies when compared with using the conventional 20-gauge system.[1] This slower cutting is due to the limitation in flow rate by the smaller lumen of the 23-gauge cutter. A simple vitrectomy using a 23-gauge system may need up to 30% more time than using a 20-gauge system. The decrease in aspiration efficiency is also obvious when using a 23-gauge silicone tube for removal of non-clotting blood in the vitreous cavity and during gas/fluid exchange. The cutting performance of some newer 23-gauge cutters has been improved by the thin-wall design that increases the lumen diameter. When a vitrectomy is performed under a high flow rate, the difference in cutting efficiency between a 23-guage and 20-guage is now minimal.

In conventional 20-gauge vitrectomy, examination and assessment of the peripheral retina by scleral indentation is facilitated by opening the conjunctiva and exposing the anterior sclera. In 23-gague vitrectomy, due to the intact conjunctiva, scleral indentation might be less efficient and more difficult to perform. This limitation in scleral indentation is especially obvious over the nasal anterior retina and near the equator. Moreover, scleral indentation should be performed more cautiously due to the potentially easier dislodgement of the infusion line. Another difficulty with 23-gauge vitrectomy might develop during fluid-air exchange. Due to the potential space between the lumen of the cannula and the microsurgical instrument, bubbles might foam up due to air leak around the instrument and this can obscure the surgical view. This can be prevented by avoiding the use of an excessively viscous material to lubricate the ocular surface during the surgery.

Complications of 23-gauge Sutureless Vitrectomy

Subconjunctival haemorrhage

Subconjunctival haemorrhage is usually the consequence of damaging conjunctival or episcleral vessels during insertion or removal of the microcannulas. Apart from cosmetic concerns, inspection of the sclerotomy for leakage at the end of the operation can be made difficult by the overlying blood. In patients taking aspirin or anti-coagulants, withholding the medications before the operation might help to decrease the risk of this complication.

Wound leak and hypotony

Postoperative hypotony due to leakage from sclerotomies, though not common, may sometimes result in choroidal detachment and warrant reoperations for suturing of the scleral wound. Such sclerotomy leakage is most commonly related to poor techniques during wound construction and removal of the microcannulas. A leaking sclerotomy can be detected by observing the formation of a subconjunctival bleb over the sclerotomy site. It is imperative for the surgeon to examine for any leakage at the end of the operation and to close any leaking sclerotomy with suture. The incidences of postoperative hypotony have been estimated to be 11.3% at 2 hours and 3.8% at 1 day.[8] It has been shown that up to 11% of cases which had 23-guage pars plana vitrectomy required intraoperative suture placement for leaking sclerotomies.[10]. Risk factors of leaking sclerotomies include prior vitrectomy, young patients, high myopia and vitreous base dissection.

Endophthalmitis

Endophthalmitis or intraocular inflammation of the eye is one of the most serious complications of intraocular surgery. In a large retrospective case series, the incidence of endophthalmitis after 25-gauge vitrectomy has been shown to be 12 times higher than conventional 20-gauge vitrectomy.[9] The sutureless nature of the scleral wound poses a definite risk for this devastating complication. However, subsequent series using 25- and 23-gauge transconjunctival vitrectomy have shown that there were no obvious increased incidence of endophthalmitis compared with 20-gauge vitrectomy.[10,11] Wound leakage and postoperative hypotony should be avoided in order to minimise the chance of bacterial entry and hence the risk of endophthalmitis.

Conclusions

The safety and efficacy of 23-gauge transconjuntival sutureless vitrectomy in the management of various vitreo-retinal conditions have been demonstrated in multiple studies in recent years. The use of 23-gauge sutureless vitrectomy allows faster postoperative recovery and less surgical discomfort after the surgery. Unlike its 25-gauge counterpart, the 23-gauge sutureless vitrectomy system has the benefits of allowing microincisional sutureless procedures while working with instruments with greater stiffness and higher flow dynamics similar to the conventional 20-gauge system. With the increasing availability of different 23-gauge instruments, the 23-gauge vitrectomy system has good potential to replace the 20-gauge system as the standard of vitreo-retinal surgery.

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Seminar on Doping Control in Sports

Jointly organised by

The Federation of Medical Societies of Hong Kong

Sports Federation & Olympic Committee of Hong Kong, China

Hong Kong Anti-Doping Committee

Objectives:

This one-day seminar aims to provide anti-doping knowledge to local physicians and to increase their awareness of the roles and responsibilities they have in the fight against doping. After attending the course, the attendees will be equipped to help their patients who happen to be an athlete. The course will also update the participants on the latest development in the fight against doping, both in terms of international coordinated efforts and testing technology.

Time	Programme	Speaker
9:00 am	Registration	
9:30 am	Opening ceremony	
9:45 am	The international efforts in the fight against doping in sport *	Ms. Michele VERROKEN, (Former Director of Ethics and Anti-Doping at UK Sport)
10:45 am	Tea break	
11:00 am	The prohibited list and the role of physician in doping control #	Dr. Julian CHANG, MH (Hon. Medical Advisor of SF&OC)
12:30 pm	Q&A	
12:45 pm	Lunch break	
2:00 pm	Therapeutic use exemption (TUE) and doping control procedure #	Dr. James LAM (Member of TUE Panel, HKADC)
3:30 pm	Tea break	
3:45 pm	An overview on the testing technology for doping control #	Dr. Terrence WAN, PhD (Head of Racing Laboratory, Hong Kong Jockey Club)
4:45 pm	Q&A	
5:00 pm	Closing ceremony	

Date: 5 December 2010
Time: 9:30 am - 5:30 pm
Venue: Lecture Theatre, Olympic House, 1 Stadium Path, So Kon Po, Causeway Bay, Hong Kong
Language: * English; # Cantonese (Supplemented with English)
Parking Fee: HK$180 ; **Reservation Deadline:** 5 November 2010 (Optional)
Registration Fee: HK$300
Certificate: Award to participants with a minimum attendance of 70%
Enquiries: The Secretariat of The Federation of Medical Societies of Hong Kong
Tel : 2527 8898 Fax : 2865 0345 Email : info@fmshk.org

CME / CPD Accreditation in application

A total of **9 CNE** points for the whole course and the points will be awarded according to the number of hours attended.
Application form can be downloaded from websites: http://www.fmshk.org

Drug-induced Glaucoma

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Dr. Nafees BAIG

Introduction

Glaucoma is a form of optic neuropathy with specific visual field loss. It is usually associated with raised intraocular pressure (IOP). Several drugs have the potential to cause raised IOP; this can occur via an open-angle or a angle-closure mechanism.[1] One of the most important drugs is steroid.[2] Steroid-induced glaucoma is a form of open-angle glaucoma. It is usually associated with topical steroids. However other forms of administration such as inhaled, oral, intravenous, periocular and intravitreal, can also lead to raised IOP. Medications for treating a variety of systemic conditions including depression, allergies, Parkinson disease etc., can produce pupillary dilation and precipitate an attack of acute angle-closure glaucoma in anatomically predisposed eyes that have narrow angles.

Mechanisms of IOP Elevation in Drug-induced Glaucoma

Open-angle

Steroid is a group of drugs that may produce IOP elevation by open-angle mechanism. Not all patients taking steroid will develop glaucoma. Risk factors include preexisting primary open-angle glaucoma, a family history of glaucoma, high myopia, diabetes mellitus and young age.[3] It has been shown that 18-36% of the general population and 46–92% of patients with primary open-angle glaucoma respond to topical ocular administration of corticosteroids with an elevation of IOP, usually within 2–4 weeks after therapy has been instituted.[1]

Topically applied eyedrops, topically applied creams to the periorbital area and intravitreal injections are more likely to cause IOP elevation. The incidence of elevated IOP is less with intravenous, parenteral and inhaled routes of administration. Since IOP elevation can be gradual and asymptomatic, patients on chronic corticosteroid therapy can remain undiagnosed, which can result in glaucomatous optic nerve damage.

Steroid-induced IOP elevation typically occurs within a few weeks after commencing steroid therapy. In most cases, IOP returns spontaneously to the baseline within a few weeks to months upon stopping the steroid. In rare situations, the IOP remains high that requires prolonged glaucoma medication or even surgery.

Closed-angle

Some drugs have contraindications or adverse effects concerning with acute angle-closure glaucoma. These drugs will incite an attack in those individuals with very narrow anterior chamber angles that are prone to occlusion especially when the pupils are dilated. The classes of medications that have the potential to induce angle-closure are topical anticholinergic or sympathomimetic pupil dilating drops, tricyclic antidepressants, monoamine oxidase inhibitors, antihistamines, antiparkinsonism drugs, antipsychotic medications and antispasmolytic agents.

Sulfonamide-containing medications may induce angle-closure glaucoma by a different mechanism, involving the anterior rotation of the ciliary body. Typically, the angle-closure is bilateral and occurs within the first few doses. Patients with narrow or wide open angles are potentially susceptible to this rare and idiosyncratic reaction.

Pathophysiology of Drug-induced Glaucoma

Open-angle

The exact pathophysiology of steroid-induced glaucoma is unknown. It is known that steroid-induced IOP elevation is secondary to increased resistance to aqueous outflow. Some evidence shows that there could be increased accumulation of glycosaminoglycans or increased production of trabecular meshwork-inducible glucocorticoid response (TIGR) protein, which could mechanically obstruct the aqueous outflow. Other evidence suggests that the corticosteroid-induced cytoskeletal changes could inhibit pinocytosis of aqueous humour or inhibit the clearing of glycosaminoglycans, resulting in the accumulation of this substance and blockage of the aqueous outflow.

Closed-angle

Aqueous humour is secreted by the ciliary body and circulates through the pupil to the anterior chamber angle. (**Fig. 1**) The pathophysiology of angle-closure glaucoma is usually due to pupillary blockage, i.e. iris-lens contact at the pupillary border resulting from pupillary dilation. Medications have a direct or secondary effect, either in stimulating sympathetic or inhibiting parasympathetic activation causing pupillary dilation, which can precipitate acute angle-closures in patients with occludable anterior chamber angles. These include adrenergic agonists (e.g.

phenylephrine), β2-specific adrenergic agonists (e.g. salbutamol), noncatecholamine adrenergic agonists (e.g. amphetamine, dextroamphetamine, methamphetamine and phendimetrazine) and anticholinergics (e.g. tropicamide). Histamine H1receptor antagonists (antihistamines) and histamine H2 receptor antagonists (e.g. cimetidine and ranitidine) have weak anticholinergic adverse effects. Antidepressants such as fluoxetine, paroxetine, fluvoxamine and venlafaxine have been associated with acute angle-closures. It is believed to be induced by either the anticholinergic adverse effects or the increased levels of serotonin that cause mydriasis.

Sulfa-containing medications result in acute angle-closures in a different mechanism. This involves the anterior rotation of the ciliary body with or without choroidal effusions, resulting in a shallow anterior chamber and blockage of the trabecular meshwork by the iris. Pupillary dilation and a preexisting shallow anterior chamber angle are not necessary. The exact reason causing ciliary body swelling is unknown in susceptible individuals.

Topiramate is a sulfa-containing anticonvulsant. There were reports about patients on topiramate developing acute angle-closures. However, a pilot study was conducted in the Hong Kong Eye Hospital and the Prince of Wales Hospital recently which showed that short-term use of topiramate did not induce asymptomatic angle narrowing.[4] Therefore it was suggested that topiramate-induced secondary angle-closure glaucoma may be an all-or-none phenomenon.

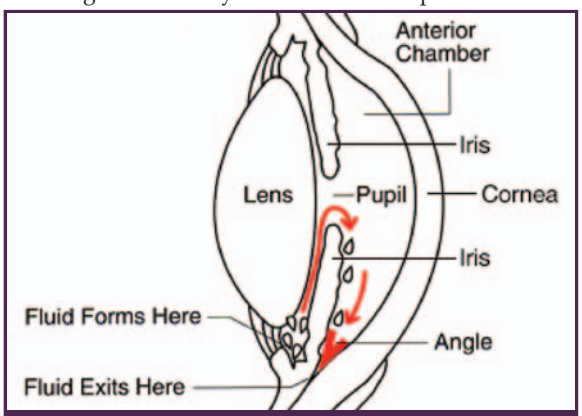

Fig. 1. Flow of aqueous humor indicated in red.

Clinical Assessment for Drug-induced Glaucoma

History
The patient's current medications should be carefully elicited.

Symptoms
With steroid-induced glaucoma, the pressure elevation is gradual. Therefore, there are very few symptoms during the early stage of disease. At a later stage, patients may complain of loss of the peripheral visual field. At the more advanced stage, when the central vision is also affected, patients may complain of blurring of vision.

In drug-induced acute angle-closure glaucoma, the symptoms are the same as in primary acute angle-closure glaucoma. These include sudden eye pain, headache associated with nausea and/ or vomiting, blurring of vision and halos around bright objects.

Past Ocular History/Past Medical History
History of systemic medical disease, which could require chronic corticosteroid use (e.g., uveitis, collagen vascular disease, asthma, dermatitis) should be elicited.

Patients with preexisting primary open-angle glaucoma, a family history of primary open-angle glaucoma, diabetes mellitus, high myopia, or connective tissue diseases are at greater risk to be steroid responders.

Physical Examination
A complete ophthalmic examination should be performed including the followings:

Visual acuity and refraction
Patients with acute angle-closures have significant drops in visual acuity. Patients with hyperopia are at higher risks for narrow anterior chamber angles.

Pupil reflex
Acute angle-closure presents with a fixed, mid-dilated pupil (**Fig. 2**) while an afferent pupillary defect indicates unilateral optic nerve damage.

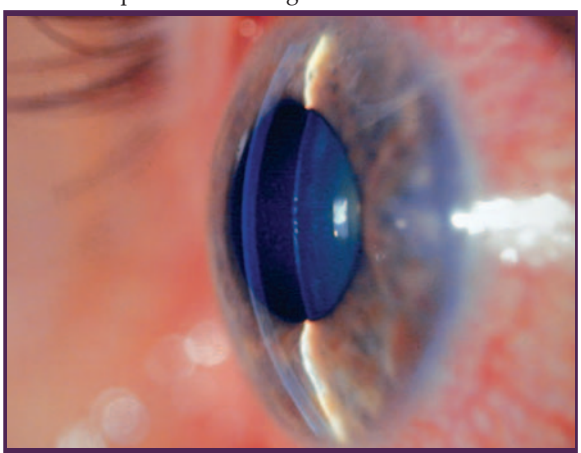

Fig. 2. Slit lamp photo of an eye with acute angle closure showing shallow anterior chamber, mid dilated pupil and ciliary injection

Intraocular pressure
Acute angle-closure usually presents with a much higher IOP than steroid-induced glaucoma which presents with a gradual IOP elevation.

Slit lamp examination
Examination of the anterior chamber is essential to look for signs of other secondary glaucomas such as uveitic, pigment dispersion and pseudoexfoliation glaucoma. It can also assess the depth of the anterior chamber and to exclude pupillary block. **Fig. 2** shows a shallow anterior chamber in an acute angle-closure. Cataract is also associated with chronic steroid use.

Gonioscopic examination
Gonioscopy can evaluate the angle anatomy (i.e. open

or narrow) and to determine whether the angle is occludable during pupil dilation.

Optic disc evaluation

Stereoscopic examination of the optic disc is necessary to exclude glaucomatous damage. The signs of glaucoma optic nerve damage include increased cup-to-disc ratio in horizontal and vertical meridians; progressive enlargement of the cup; evidence of nerve fibre layer damage with red-free filter; notching or thinning of disc rim; pallor; presence of haemorrhage; asymmetry between discs; and peripapillary atrophy. **Fig. 3** shows a pink optic disc with normal cup-disc ratio while **Fig. 4** shows a pale glaucomatous disc with increased cup-disc ratio.

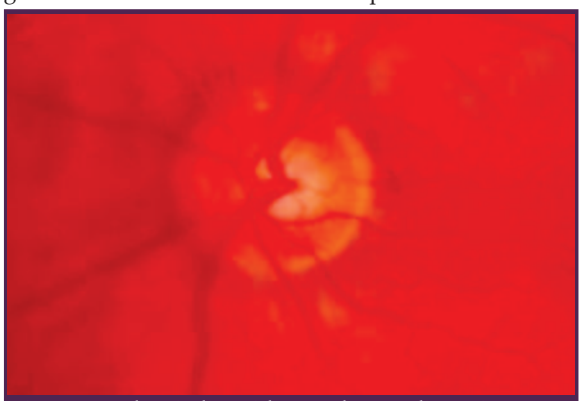

Fig. 3. Normal optic disc with normal cup to disc ratio

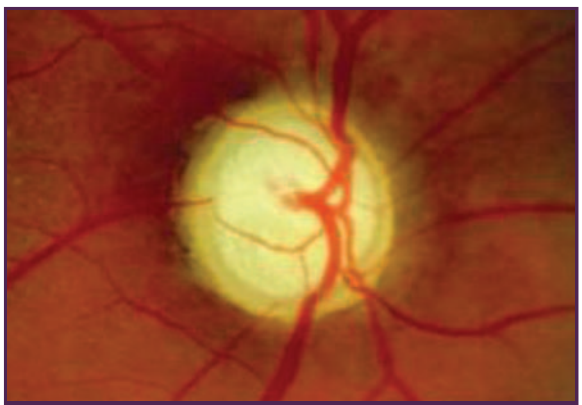

Fig. 4. Pale glaucomatous optic disc with increased cup to disc ratio

Investigations

Perimetry

Visual Field testing such as Humphrey or Goldman is used to evaluate the severity of optic neuropathy.

Optical Coherence Tomography (OCT)

OCT is an optical signal acquisition and processing method. It captures micrometer-resolution, three-dimensional images from within the optical scattering media (e.g., biological tissue). OCT is an interferometric technique, typically employing near-infrared light. It is used to evaluate the retinal nerve fibre thickness around the optic disc in glaucoma patients. Serial scans can be used to demonstrate any progression of disease.

Ultrasound Biomicroscopy (UBM)

UBM is an imaging technique that uses high frequency ultrasound to produce images of the eye at near microscopic resolution. This technique is used to evaluate the anterior chamber angle configuration (i.e. open or closed) and the position of the ciliary body (any anterior rotation). **Fig. 5** shows a narrow anterior chamber angle on UBM.

Anterior Segment OCT (ASOCT)

It applies the same principle as OCT but it provides images of the anterior chamber including the angle and the lens.

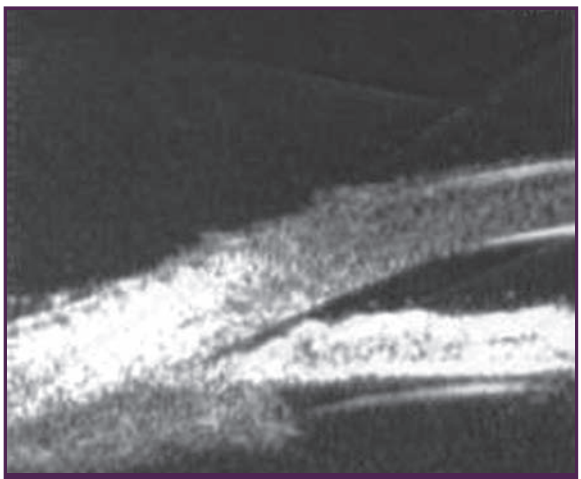

Fig. 5. Ultrasound biomicroscopy (UBM) showing a narrow anterior chamber angle

Treatment of Drug-induced Glaucoma

Medical: Open-angle

If the patient's underlying medical condition can tolerate discontinuation of corticosteroids, then cessation of the medication will usually result in normalisation of IOP.

In the case of topical corticosteroid drops, using a lower potency steroid medication, such as the phosphate forms of prednisolone and dexamethasone, loteprednol etabonate or fluorometholone should be considered. These drugs have a lesser chance of IOP rise, but they are usually not as effective as an anti-inflammatory drug. Topical nonsteroidal anti-inflammatory medications (e.g., diclofenac, ketorolac) are other alternatives that have no potential to elevate IOP, but they may not have enough anti-inflammatory activity to treat the patient's underlying condition.

In the occasional cases in which the patient's IOP does not normalise upon cessation of the steroid or in those patients who must continue to be on corticosteroid medications, topical antiglaucoma medications are considered.

Medical: Closed-angle

If the aetiology is because of sulfa-containing medications, the increase in IOP generally will resolve upon stopping the medication. However, severe cases of sulfonamide-induced angle-closure (i.e. IOP >45 mm Hg) may not respond to simply discontinuing the offending medication. These cases may respond to intravenous mannitol.

For other aetiologies of drug-induced angle-closure, they are treated similar to primary acute angle-closure glaucoma by using antiglaucoma medications including topical beta blockers, prostaglandin analogues, cholinergic agonists and often oral acetazolamide.[5]

Laser treatment

For open-angle steroid-induced glaucoma, Argon laser trabeculoplasty or selective laser trabeculoplasty can be applied in the absence of ocular inflammation if the IOP is suboptimal with medication.

In closed-angle glaucoma, argon laser peripheral iridoplasty (ALPI) may be applied to deepen the anterior chamber and widen the angle. Laser iridotomy (LI) can be performed to reverse pupillary block or to prevent further pupillary block. **Fig. 6** shows evidence of argon laser peripheral iridoplasty (in green) and laser iridotomy (in red).

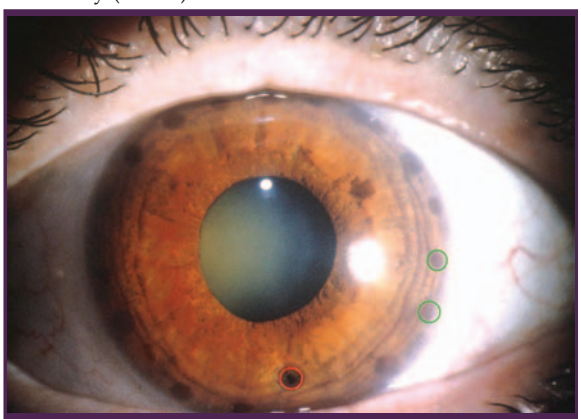

Fig. 6. Slit-lamp photo showing argon laser irioplasty marks (green circles) and an inferior laser iridotomy (red circle)

Surgical: Open-angle

When medical therapy is ineffective in lowering the IOP to target pressure or the patient is intolerant of medical therapy, then surgical therapy is indicated.

In patients whom both medical and laser therapy have failed to lower the IOP adequately, surgical treatment is warranted. Usually, trabeculectomy, a guarded filtration procedure, with or without intraoperative antimetabolites, is the primary procedure. In cases of eyes with active neovascularisation or inflammation, a glaucoma drainage implant may be used as the primary procedure.

Surgical: Closed-angle

Trabeculectomy can also be performed with similar indications as open-angle glaucoma. However the surgery is more difficult since the anterior chamber is shallower and the cornea is usually hazier due to the acute IOP rise.

Prevention of Drug-induced Glaucoma

Open-angle

Unnecessary prolonged use of steroid should be avoided. Ophthalmic evaluation is recommended for patients treated with long-term steroids especially with risk factors such as family history of primary open-angle glaucoma.

Closed-angle

Prophylactic laser iridotomy may be performed in patients requiring frequent mydriasis such as frequent fundus examinations for diabetic retinopathy. Medications causing secondary angle-closure are avoided in susceptible individuals as far as possible.

Conclusion

The prognosis of steroid-induced glaucoma depends on the duration of the IOP elevation and the control of IOP after diagnosis. Uncontrolled increase in IOP can lead to permanent optic nerve damage and hence permanent blindness. In patients with controlled IOP, the prognosis can be favourable depending on the severity of disease on presentation. Drug-induced IOP rise can be asymptomatic initially especially in open-angle type. General practitioners should be aware of the risk factors for glaucoma before prescribing a drug that has the potential to cause, precipitate or exacerbate glaucoma. Topical steroids can cause IOP rise in susceptible individuals in a fairly short period of time and therefore it is advised that topical steroids should be prescribed by doctors capable of measuring IOP. Whenever in doubt, an ophthalmologist should be consulted.

References

1. Tripathi RC, Tripathi BJ, Haggerty C, et al. Drug-induced glaucomas: mechanism and management. Drug Safety 2003;26:749-767.
2. Tripathi RC, Parapuram SK, Tripathi BJ, et al. Corticosteroids and glaucoma risk. Drugs Aging 1999;15:439-450.
3. Shukla D, Vidhya N, Prasad NM, et al. Evaluation of patient age as a risk factor for intraocular pressure elevation after intravitreal triamcinolone. Am J Ophthalmol 2007;144:453-454.
4. Leung DY, Leung H, Baig N, et al. Topiramate and asymptomatic ocular angle narrowing: a prospective pilot study. Eye 2009;23:2079-2081.
5. Lam DS, Tham CC, Lai JS, Leung DY. Current approaches to the management of acute primary angle closure. Curr Opin Ophthalmol 2007;18:146-151.

News from Member Societies

1. **British Medical Association (Hong Kong Branch)**
   Updated office-bearers are as follows: President: Dr. Raymond See-kit LO; Honorary Secretary: Dr. Terry Che-wai HUNG; Honorary Treasurer: Dr. Clarence LEUNG

The FMSHK would like to send its congratulations to the new office-bearers and look forward to working together with the society.

The Science Behind Medical Conferences

Meetings & Exhibitions Hong Kong (MEHK) office of the Hong Kong Tourism Board welcomed about 100 medical professionals, event planning and facilities experts to the inaugural 'MEHK Association Forum for the Medical Industry in Hong Kong' on September 13.

Held at the Kowloon Shangri-La Hotel, this forum was supported by the Federation of Medical Societies in Hong Kong and the Hong Kong Association of the Pharmaceutical Industry.

Welcoming remarks from Mr Philip Yung, Commissioner for Tourism, and Mr Anthony Lau, Executive Director of the Hong Kong Tourism Board, drew reference to Hong Kong being the destination enabling "convergence of possibilities".

Industry experts such as international professional conference organisers (PCOs), association management companies (AMCs) and MEHK touched upon topics from financial planning, medical codes of practice, trends and insights, to the running of a successful conference. Ms Gilly Wong, General Manager, MICE and Cruise of the Hong Kong Tourism Board concluded the programme outlining MEHK's proven track record in enabling one-stop-shop solutions to medical conferences.

"Trends and Developments Relevant to Associations in Asia"

Ms Quirine Laman Trip, Kenes Group Director for Business Development, cited three major conference trends – Asia's growth, broader distribution in Asia and professionalization within industry requiring increased need for year round learning and networking, and elaborated further on their impact.

"Effective Financial Planning"

Mr Stephan Wurzinger, Director of Association Relations of MCI Asia Pacific, listed two major considerations for effective budget planning. He suggested learning from best practices, benchmarking against own conference's financial model to close possible gaps. Also, be creative and pro-active when soliciting sufficient sponsorships from decision makers – with a solid financial proposal that meet the needs of pharmaceutical companies. "They want to be considered as partners, not vendors", Wurzinger highlighted. These would lead to increased profit by revenue maximization and cost reduction. Nowadays, experienced PCOs can provide risk sharing or profit sharing financial models facilitating the legwork.

"Application of Pharmaceutical Codes"

Dr Anthony Chan, President of the Hong Kong Association of Pharmaceutical Industry (HKAPI) introduced the HKAPI Code of Marketing Practice. He also presented on code updates, quoting, "The HKAPI Code of Marketing Practice is updated consistently to be responsive to the changing society. The recent 14th edition was updated to specifically address festive gifts as a part of Chinese customs. The Code also provides a concise guideline on sponsoring symposiums and conferences." Future trends on pharmaceutical industry support to medical conferences were provided, citing current practices in Denmark and India. All in all, "Transparency is the Key".

"Preparing for Growing Demands of Medical Association Management"

Ms Isabel Mortara, Executive Director of Kenes Associations Worldwide, shared on association management solutions to tackling market trends. "Medical Associations have a unique role in communities as engines of change. Challenges include changes in socio-demographics, technology, economy, environment and political/legal. These are all leading to new areas of debate, in addition to a world which is becoming more global and interconnected, associations increasingly need to operate in a businesslike way, to be sustainable and efficient in these challenging times." Medical associations could leverage on the professionalism of AMCs to increase association's capabilities, thus reducing their costs.

Prescribing Success:
Planning & Adherence

When planning a conference there are three major steps: planning, programme design and venue choice. From the medical professional panel, the key advice from three world-renowned speakers is to define target audience, scale and budget. A balanced scientific programme consisting of famous and excellent speakers is of vital importance, as with venue choice.

Dr Lilian L.Y. Leong, Founding President and Immediate Past President of the Hong Kong College of Radiologists, and a 40-year conference veteran herself, summed up in two words – forward planning.

The night ended with a networking session where Medical Professionals took the oppourtunity to speak with various AMCs, PCOs and venue partners.

"Conferences need at least two to three years in advance to plan especially in securing eminent speakers and for early announcement to potential participants for their planning," said Dr Leong. "To attract attendance, it's always good to have a key visual theme with the design of the conference programme evolving around it." Supplemented by Dr Cheuk-Man Yu, Assistant Dean (External Affairs) of the Faculty of Medicine at the Chinese University of Hong Kong, recent discoveries and advancement in the field should be explored and included in the scientific programme to maximize programme attractiveness.

According to Dr Chris Wong, President-Elect of the Hong Kong College of Cardiology, Hong Kong as the gateway to China, associations can leverage on their ability to run English-speaking conferences with their China counterparts at an international scale, and Hong Kong is an ideal platform for professional bodies to work with China and other Asian counterparts.

(top picture) MEHK Association Forum presenting its speakers, panel members and officiating guests with souvenirs: (left to right) Ms Gilly Wong, Mr. Anthony Lau, Ms Quirine Laman Trip, Ms Isabel Mortara, Mr Philip Yung, Dr Lilian Leong, Dr Anthony Chan and Mr Stephan Wurzinger.

Partnering MEHK:
Your Conference Expert

A panel discussion on 'How to Run a Successful Conference' with (left to right), Ms Gilly Wong as moderator and Dr Lilian Leong, Dr Cheuk-Man Yu and Dr Chris Wong as speakers.

MEHK is aspired to partner with medical associations in Hong Kong to introduce more conferences to the city. From planning and bidding, pre-event marketing, official invitations and hospitality, MEHK has a dedicated team to give the best possible advice and support.

As Dr Lilian Leong concluded, "MEHK has many experts who have great experience and can help. Seek advice from professionals, have a good association team and aim for success!"

Mr Stephan Wurzinger, Director of the Association Relations MCI Asia Pacific also commented, "I was impressed with the professionalism of the Association Forum, which ticked all the right boxes as regards speakers, content, audience and atmosphere. I applaud the MEHK for their efforts in facilitating dialogue amongst medical associations that will hopefully excite them to bring more international congresses to Hong Kong."

Website: mehongkong.com

HONG KONG TOURISM BOARD

HONG KONG

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
					* Trailwalker 2010 Training 1	* PALS Course 2010 2
* PALS Course 2010 * HKMA Tennis Tournament 3	* PALS Course 2010 * A "Hard" Scrotal Abscess * HKMA Choir Voice Training Course 2010 4	* PALS Course 2010 * HKMA YTM Community Network - An Update on Management of Rhinosinusitis * FMSHK Officers' Meeting * HKMA Council Meeting 5	* MPS - Mastering Adverse Outcomes 6	* HKMA NT West Community Network Annual Meeting cum Certificate Course on Allergic Diseases (I) * HKMA YTM Community Network - Lecture Series on Hypertension, Diabetes Mellitus & Chronic Kidney Disease 7	8	* Federation President Cup Soccer Five Tournament 2010 * Refresher Course for Health Care Providers 2010/2011 * Dr. WONG Kwai Kuen, BBS Inauguration Seminar of HKMAPS "Reminiscence through light and shadow" cum HKMA 90th Anniversary 9
* HKMA Tennis Tournament * MPS - Mastering Adverse Outcomes * HKMA Swimming Gala * HKMA Certificate Course on Family Medicine 2010 10	* HKMA Choir Voice Training Course 2010 11	* HKMA Kowloon West Community Network - "Certificate Course on Dermatology" 12	* MPS - Mastering Adverse Outcomes * Continuous tug of war in Haemostasis: Thrombosis and Haemorrage in Neurosurgical * HKMA Central, Western & Southern Community Network - Certificate Course on Orthopaedics (I) 13	* HKMA YTM Community Network - Lecture Series on Hyperfension, Diabetes Mellitus & Chronic Kidney Disease * HKMA Hong Kong East Community Network - Menopause 2010 - Myth & Legend * HKMA NT West Community Network - Certificate Course on Allergic Diseases (II) * HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2010 - Recent Advances in Interventional Cardiology 14	15	* 12th BHME * The 1st Global Drug Safety Conference and Exposition Hong Kong 2010 16
* Trailwalker 2010 Training * HKMA Tennis Tournament * 12th BHME * The 1st Global Drug Safety Conference and Exposition Hong Kong 2010 * Federation President Cup Soccer Five Tournament 2010 17	* HKMA Choir Voice Training Course 2010 * The 1st Global Drug Safety Conference and Exposition Hong Kong 2010 18	* FMSHK Executive Committee Meeting 19	* MPS - Mastering Adverse Outcomes * HKMA Shatin Doctors Network - Approach to Patient with Chest Pain 20	* MPS - Mastering Adverse Outcomes * HKMA NT West Community Network - Certificate Course on Allergic Diseases (III) * HKMA YTM Community Network - Lecture Series on Hypertension, Diabetes Mellitus & Chronic Kidney Disease (cum Annual Meeting) 21	* HKMA Kowloon City Community Network - The Management of Common Nasal Symptoms 22	* CNHK - Understanding of Cord Blood Transplantation & Donation (Code no: MFC-10-02) 23
* HKMA Tennis Tournament * HKMA YTM Community Network - KDPTG - Public Health Day * MPS - Mastering Your Risk * Federation President Cup Soccer Five Tournament 2010 24	* HKMA Choir Voice Training Course 2010 25	* HKMA Kowloon West Community Network - "Certificate Course on Dermatology" 26	* MPS - Mastering Adverse Outcomes * HKMA Central, Western & Southern Community Network - Certificate Course on Orthopaedics (II) 27	* HKFMS Foundation Meeting 28	29	* HKMA CME - Seminar In Infectious Disease - Infectious Diseases Update * C-Symposium - Collapsed Spine: Clinical Approach & Contemporary Management * Federation President Cup Soccer Five Tournament 2010 30
* Trailwalker 2010 Training * HKMA Tennis Tournament * MPS - Mastering Adverse Outcomes 31						

Medical Diary of October

Date / Time	Function	Enquiry / Remarks
1 FRI 7:00 am *(17, 31)*	**Trailwalker 2010 Training** Organiser: The Hong Kong Medical Association, Chairman: Dr. CHOW Yuen Hon Francis, Venue: MacLehose Trail	Miss Peony CHAN Tel: 2527 8285
2 SAT *(3, 4, 5)*	**PALS Course 2010** Organiser: Hong Kong College of Paediatricians, the Heart Institute for Children, Hope Children's Hospital, Illinois, USA & Hong Kong Paediatric Nurses Association, Speakers: Various, Venue: A & E Training Centre, Tang Shiu Kin Hospital	Ms. Prudence TANG / Ms. Vanessa WONG Tel No.: 28718 871 Fax No. 27851 850 Email: enquiry@paediatrician.org.hk, Website: http://www.paediatrician.org.hk/entcnews.htm 12 CME points
3 SUN 7:30 pm *(10, 17, 24, 31)*	**HKMA Tennis Tournament** Organiser: The Hong Kong Medical Association, Chairman: Dr. CHIN Chu Wah, Venue: Kowloon Tong Club	Miss Peony CHAN Tel: 2527 8285
4 MON 7:30 pm - 8:30 pm	**A "Hard" Scrotal Abscess** Organiser: Hong Kong Urological Association, Chairman: Dr. LAW Yin Chak, Speaker: Dr. CHU Wing Hong Ringo, Venue: Seminar Room, G/F, Block A, Queen Elizabeth Hospital, Kowloon	Dr. HUNG Hing Hoi / Ms. Tammy HUNG Tel: 2598 6006 / 9609 6064 Fax: 2958 6076 / 8344 5115 1 CME Point
8:00 pm *(11, 18, 25)*	**HKMA Choir Voice Training Course 2010** Organiser: The Hong Kong Medical Association, Venue: GP1, HKCC	Ms. Candy YUEN Tel: 2527 8285
5 TUE 1:30 pm	**HKMA YTM Community Network - An Update on Management of Rhinosinusitis** Organiser: HKMA YTM Community Network, Chairman: Dr. Ricky W.H.HO, Speaker: Dr. CHOW Chun Kuen, Venue: Mongkok	Ms. Queenie HO Tel: 2839 4320 1.5 CME Points
8:00 pm - 10:00pm	**FMSHK Officers' Meeting** Organiser: The Federation of Medical Societies of Hong Kong, Venue: Gallop, 2/F., Hong Kong Jockey Club Club House, Shan Kwong Road, Happy Valley, Hong Kong	Ms. Sonia CHEUNG Tel: 2527 8898 Fax: 2865 0345
8:00 pm	**HKMA Council Meeting** Organiser: The Hong Kong Medical Association, Chairman: Dr. K CHOI, Venue: HKMA Head Office, 5/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong	Ms. Christine WONG Tel: 2527 8285
6 WED *(10, 13, 20, 21, 27, 31)*	**MPS - Mastering Adverse Outcomes** Organiser: The Hong Kong Medical Association, Speakers: Various, Venue: The HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong or Mongkok	Miss Viviane LAM Tel: 2527 8452 2.5 CME Points
7 THU 1:00 pm	**HKMA NT West Community Network Annual Meeting cum Certificate Course on Allergic Diseases (I)** Organiser: HKMA NT West Community Network, Speaker: Dr. Edwin POON, Venue: Maxim's Palace Chinese Restaurant, Tuen Mun Town Hall, 3 Tuen Hi Road, Tuen Mun, N.T.	Miss Alice TANG Tel: 2527 8285
1:00 pm *(14)*	**HKMA YTM Community Network - Lecture Series on Hypertension, Diabetes Mellitus & Chronic Kidney Disease** Organiser: HKMA YTM Community Network, Chairmen: Dr. MAK Chun Kee & Dr. NG Kwok Keung, Speakers: Various, Venue: Jade Ballroom, 2/F., Eaton Hotel Hong Kong, 380 Nathan Road, Kowloon, Hong Kong	Miss Carman WONG Tel: 2527 8285
9 SAT 2:00 pm - 8:00 pm *(17, 24, 30)*	**Federation President Cup Soccer Five Tournament 2010** Organiser: The Federation of Medical Societies of Hong Kong, Chairman: Dr. CHAN Hau Ngai Kingsley; Dr. CHAN Chi Wing Timmy & Dr. LIU Wing Hong, Venue: Ying Wa College	Ms. Karen CHU Tel: 2527 8898 Fax: 2865 0345
2:30 pm	**Refresher Course for Health Care Providers 2010/2011** Organiser: The Hong Kong Medical Association, Speaker: Dr. CHOW Hung Lit, Venue: OLMH	Miss Viviane LAM Tel: 2527 8452 2 CME Points
7:30 pm	**Dr. WONG Kwai Kuen, BBS Inauguration Seminar of HKMAPS "Reminiscence through light and shadow" cum HKMA 90th Anniversary** Organiser: The Hong Kong Medical Association, Chairman: Dr. PANG Lai Man Amy, Venue: HK Central Library	Miss Peony CHAN Tel: 2527 8285
10 SUN 1:30 pm	**HKMA Swimming Gala** Organiser: The Hong Kong Medical Association, Chairman: Dr. IP Man Ho, Venue: La Salle College	Miss Peony CHAN Tel: 2527 8285
2:00 pm	**HKMA Certificate Course on Family Medicine 2010** Organiser: The Hong Kong Medical Association, Speakers: Dr. FONG Yeung Francois & Dr. HO Chung Ping, Venue: Queen Elizabeth Hospital, Kowloon	Miss Viviane LAM Tel: 2527 8452 3 CME Points
12 TUE 1:00 pm *(26)*	**HKMA Kowloon West Community Network - "Certificate Course on Dermatology"** Organiser: HKMA Kowloon West Community Network, Chairmen: Dr. TONG Kai Sing & Dr. CHAN Siu Man Bernard, Speakers: Dr. TANG Yuk Ming William & Dr. HO Ka Keung, Venue: Crystal Room I-III, 30/F., Panda Hotel, Tsuen Wan, N.T.	Miss Carman WONG Tel: 2527 8285 1 CME Point
13 WED 7:30 am	**Continuous tug of war in Haemostasis: Thrombosis and Haemorrage in Neurosurgery** Organiser: Hong Kong Neurosurgical Society, Chairman: Dr. Kin-Ming CHENG, Speaker: Dr. Jane LAU, Venue: Seminar Room, G/F, Block A, Queen Elizabeth Hospital, Kowloon	Dr. Gilberto LEUNG Tel: 2255 3368 Fax: 2818 4350 2 CME Points
1:00 pm *(27)*	**HKMA Central, Western & Southern Community Network - Certificate Course on Orthopaedics (I) & (II)** Organiser: HKMA Central, Western & Southern Community Network, Speaker: Dr. CHENG Ngai Shing Justin & Dr.YEUNG Yeung, Venue: The HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Miss Alice TANG Tel: 2527 8285
14 THU 1:00 pm	**HKMA Hong Kong East Community Network - Menopause 2010 - Myth & Legend** Organiser: HKMA Hong Kong East Community Network, Speakers: Dr. CHAN Leung Kwok Clement, Venue: HKMA Head Office, 5/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong	Miss Alice TANG Tel: 2527 8285
1:00 pm	**HKMA NT West Community Network - Certificate Course on Allergic Diseases (II)** Organiser: HKMA NT West Community Network, Venue: Plentiful Delight Banquet (元朗喜尚嘉喜酒家), 1/F., Ho Shun Tai Building,10 Sai Ching Street, Yuen Long, N.T.	Miss Alice TANG Tel: 2527 8285
2:00 pm	**HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2010 - Recent Advances in Interventional Cardiology** Organiser: The Hong Kong Medical Association, Speaker: Dr. Vincent O.H. KWOK, Venue: The 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 1 CME Point

Date / Time	Function	Enquiry / Remarks
16 SAT (17) (17, 18)	**12th BHME** Organiser: The Hong Kong Medical Association, Chairman: Dr. CHOW Pak Chin & Dr. LI Sum Wo, Speakers: Various, Venue: The Mira, Tsim Sha Tsui, Kowloon **The 1st Global Drug Safety Conference and Exposition Hong Kong 2010** Organiser: Global Drug Safety Development Center, Chairperson: Ms. Iris CHANG, Speakers: Various, Venue: The Regal Kowloon Hotel, Tsim Sha Tsui, Kowloon	Ms. Candy YUEN Tel: 2527 8285 Max. 8 CME Points Conference Secretary, Tel: 3151 8900, Website: http://www.globaldrugsafety.org (Special Rate at HK$1800 for PPAHK Member)
19 TUE 8:00 pm - 10:00 pm	**FMSHK Executive Committee Meeting** Organiser: The Federation of Medical Societies of Hong Kong, Venue: Council Chambers, 4/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Sonia CHEUNG Tel: 2527 8898 Fax: 2865 0345
20 WED 1:00 pm	**HKMA Shatin Doctors Network - Approach to Patient with Chest Pain** Organiser: HKMA Shatin Doctors Network, Speaker: Dr. CHAN Wai Kwong Andy, Venue: Shatin	Miss Alice TANG Tel: 2527 8285 1.5 CME Points
21 THU 1:00 pm 1:00 pm	**HKMA NT West Community Network - Certificate Course on Allergic Diseases (III)** Organiser: HKMA NT West Community Network, Venue: Maxim's Palace Chinese Restaurant, Tuen Mun Town Hall, 3 Tuen Hi Road, Tuen Mun, N.T. **HKMA YTM Community Network - Lecture Series on Hypertension, Diabetes Mellitus & Chronic Kidney Disease (cum Annual Meeting)** Organiser: HKMA YTM Community Network, Chairman: Dr. CHAN Yee Shing Alvin, Speakers: Dr. CHOI Kin & Dr. HO Chung Ping, MH, Venue: Jade Ballroom, 2/F., Eaton Hotel Hong Kong, 380 Nathan Road, Kowloon, Hong Kong	Miss Alice TANG Tel: 2527 8285 Miss Carman WONG Tel: 2527 8285
22 FRI 1:00 pm	**HKMA Kowloon City Community Network - The Management of Common Nasal Symptoms** Organiser: HKMA Kowloon City Community Network, Speaker: Dr. LAU Hin Wang	Miss Alice TANG Tel: 2527 8285
23 SAT 10:00am - 12:00pm	**CNHK - Understanding of Cord Blood Transplantation & Donation (Code no: MFC-10-02)** Organiser: College of Nursing, Hong Kong	Secretariat Tel: 2572 9255 Fax: 2838 6280 2 CNE/PEM Points
24 SUN 1:00 pm 2:30 pm	**HKMA YTM Community Network - KDPTG - Public Health Day** Organiser: The Hong Kong Medical Association, Venue: HA **MPS - Mastering Your Risk** Organiser: The Hong Kong Medical Association, Speaker: Dr. Andy CHEUNG, Venue: The HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Miss Alice TANG Tel: 2527 8285 Miss Viviane LAM Tel: 2527 8452 2.5 CME Points
28 MON 8:00 pm - 10:00 pm	**HKFMS Foundation Meeting** Organiser: The Federation of Medical Societies of Hong Kong, Venue: Council Chambers, 4/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Sonia CHEUNG Tel: 2527 8898 Fax: 2865 0345
30 SAT 1:30 pm	**HKMA CME - Seminar In Infectious Disease - Infectious Diseases Update** Organiser: The Hong Kong Medical Association, Chairman: Dr. ST LAI, Speakers: Various, Venue: Princess Margaret Hospital **C-Symposium - Collapsed Spine: Clinical Approach & Contemporary Management** Organiser: Osteoporosis Society of Hong Kong, Chairman: Prof. Annie KUNG & Dr. CHAN Ying Ki, Speakers: Dr. William CHEUNG; Dr. LEE Ka Kui & Dr. WONG Yat Wah, Venue: The Langham Place Hotel, Mongkok	Miss Viviane LAM Tel: 2527 8452 2.5 CME Points Ms. Sandy CHUNG Tel: 3971 2929 Fax: 2834 0821

Courses / Meetings

27-28/11/2010	**The 3rd Hong Kong International Burns and Wound Healing Symposium** Organiser: HK Society of Burns and Wounds Healing, Chairman: Prof. Andrew BURD & Prof. Shekhar KUMTA, Speakers: Various, Venue: Prince of Wales Hospital, Enquiry: Ms. Vicky CHUNG / Ms. Ruby LAM, Tel: 3151 8900, Fax: 2590 0099
30/11/2010 - 3/12/2010	**9th Asia-Pacific Conference Human Genetics** Organiser: Hong Kong Society of Medical Genetics & The Asia Pacific Society of Human Genetics, Chairman: Dr. Stephen LAM, Speaker: Various, Venue: Hong Kong Academy of Medicine, 99 wong Chuk Hang Road, Aberdeen, Hong Kong, Enquiry: Conference Secretariat, Email: apchg2010@ctshk.com
17-19/12/2010	**2010 Asian Chinese Quality of Life Conference** Organiser: International Society for Quality of Life Research - Asian Chinese Chapter; Family Medicine Unit, Department of Medicine, Li Ka Shing Faculty of Medicine, The University of Hong Kong & Hong Kong Society for Quality of Life, Co-Chairmen: Prof. LIU Feng Bin, Prof; Cindy LAM & Mr. LEUNG Kwok Fai, Speakers: Various, Venue: Li Ka Shing Faculty of Medicine, The University of Hong Kong, 21 Sassoon Road, Pokfulam, Hong Kong, Enquiry: Ms. Candy LAW, Tel: 6509 6582, Fax: 3528 5727, Email: candy@hksoqol.org, Website: http://www.hksoqol.org/conf2010
14-16/1/2011	**Hong Kong International Acupuncture Conference - Neurological and Mental Illness** Organiser: Hong Kong Association for Integration of Chinese-Western Medicine & Hospital Authority, Chairman: Dr. WONG Taam Chi Woon Vivian, Speakers: Various, Venue: Hong Kong Academy of Medicine Jockey Club Building, Enquiry: Ms. Jessie CHOW & Ms. Y.C. YEUNG, Tel: 2871 8787, 2871 8897 / 3119 1850, Fax: 2871 8898
22/1/2011	**Hepatobiliary & Pancreatic Surgery and Liver Transplantation** Organiser: Department of Surgery, The University of Hong Kong, Queen Mary Hospital & Hong Kong Chapter of American College of Surgeons, Venue: Underground Lecture Theatre, New Clinical Building, Queen Mary Hospital, Pokfulam, Hong Kong, Enquiry: Forum Secretary, Hong Kong Surgical Forum, Tel: (852) 2255 4885 / (852) 2255 4886, Fax: (852) 2819 3416, E-mail: hksf@hku.hk, Web-site: http://www3.hku.hk/surgery/forum.php
12-14/5/2011	**18th Asian Congress of Surgery & 37th Philippine College of Surgeons Mid-year Convention** Organiser: Asian Surgical Association, Venue: Waterfront Cebu City Hotel & Casino, Lahug, Cebu City, Philippines, Enquiry: Congress Secretariat, Tel: (632) 9274973-74; (632) 9281083; (632) 9292359, Fax: (632) 9292297, E-mail: secretariat@acs2011.org, Website: www.acs2011.org

Course No. C169 Certificate Course For All Health Care Professionals CME/CNE Course

Certificate Course on Assessing and Managing Violent Patients/People in the General Health Care Settings

Jointly organised by

The Federation of Medical Societies of Hong Kong

Hong Kong Society of Nursing Education

Objectives:

1. To increase clinical awareness, competency and psychological readiness of hospital staff in facing potential and imminent threats of critical incidents and/or diasters.
2. To manage and minimize the adverse potential consequences of violence and critical incidents.
3. To prevent and minimize traumatic consequence in facing and during the onset of a potential violent event.
4. To learn to facilitate post critical event growth and return to normal operation.

Date	Topics
12 Nov 2010	Assessment : mental health status (mental health first aid) assessment & essentials in psychosocial assessment
19 Nov 2010	Managing acute mental health (schizophrenia) patients: social network approach; multidisciplinary approach; psycho-educational approach & psycho-pharmacology
26 Nov 2010	Basic crisis intervention: therapeutic directions and skills
3 Dec 2010	Post crisis counselling: cognitive behavioral therapy; family therapy & intergrative approach

Speaker

Dr. Albert Tsun-Hung CHAN
Psychologist (Neo-Health Care), HKU, CUHK & HKBU Faculty
Visiting Scholar of Lingnan University

Time	7:00 p.m. – 9:30 p.m.
Venue	Lecture Hall, 4/F., Duke of Windsor Social Service Building 15 Hennessy Road, Wanchai, Hong Kong
Language Media	Cantonese (Supplemented with English)
Course Fee	HK$850 (4 sessions)
Certificate	Awarded to participants with a minimum attendance of 70%
Enquiry	The Secretariat of The Federation of Medical Societies of Hong Kong **Tel :** 2527 8898 **Fax :** 2865 0345 **Email :** info@fmshk.org

CME / CPD Accreditation in application
A total of **10 CNE** points for the whole course and the points will be awarded according to the number of hours attended.
Application form can be downloaded from website: http://www.fmshk.org

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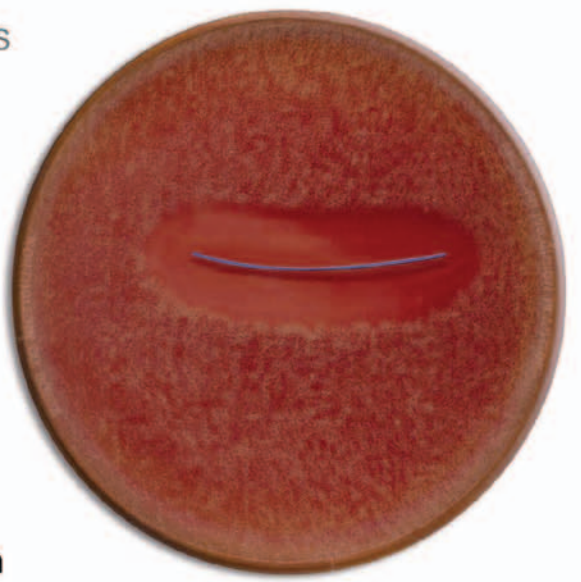

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References: 1. Storch ML, Rothenburger SJ, Jacinto G. Experimental efficacy study of coated VICRYL plus antibacterial suture in guinea pigs challenged with *Staphylococcus aureus*. *Surg Infect (Larchmt)*. 2004;5:281-288. **2.** Ming X, Nichols M, Rothenburger S. In vivo antibacterial efficacy of MONOCRYL plus antibacterial suture (poliglecaprone 25 with triclosan). *Surg Infect (Larchmt)*. 2007;8:209-213. **3.** Ming X, Rothenburger S, Nichols M. In vivo and in vitro antibacterial efficacy of PDS* plus (polidioxanone 25 with triclosan) suture. *Surg Infect (Larchmt)*. 2008;9:451-457.