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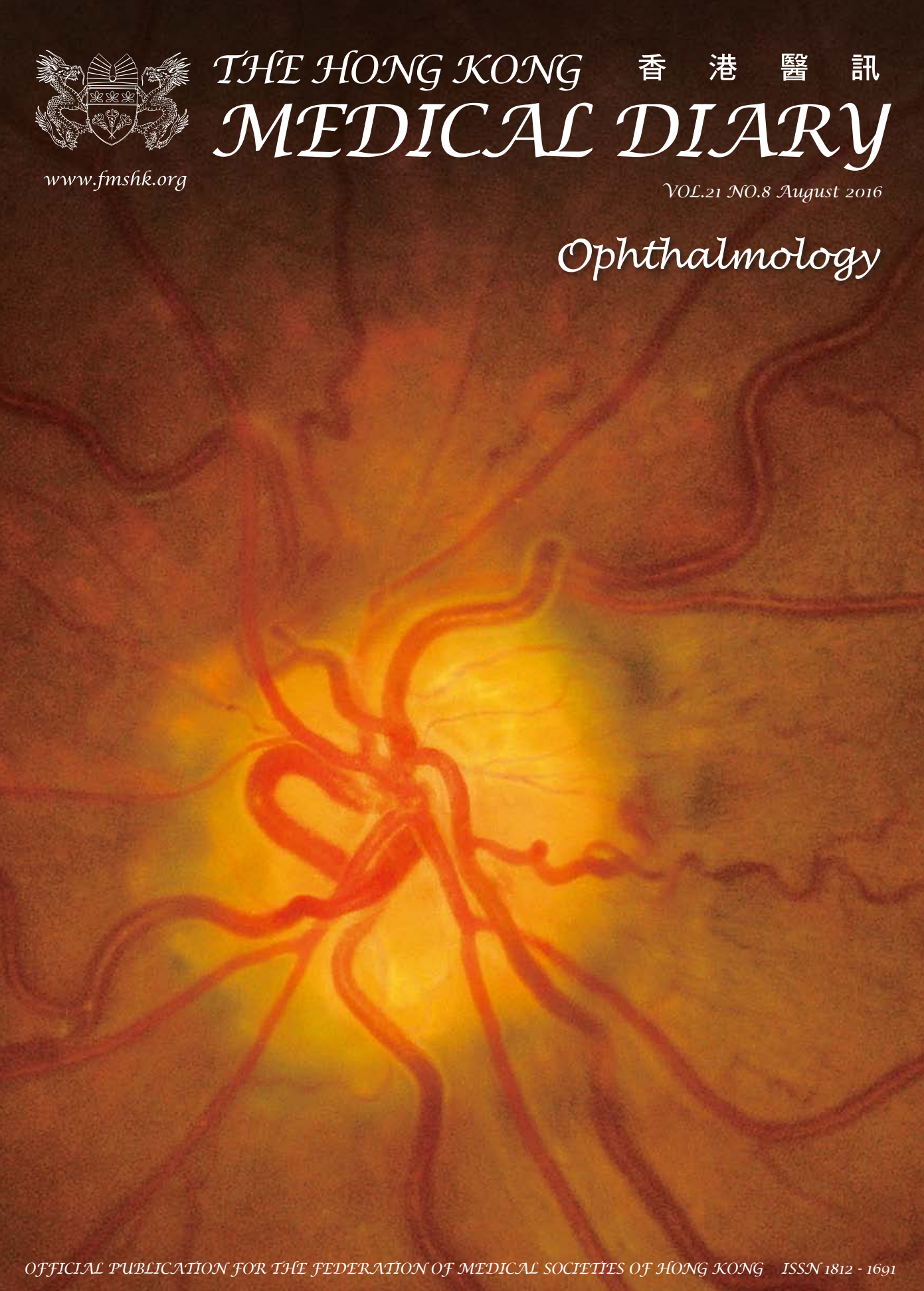
Ophthalmology



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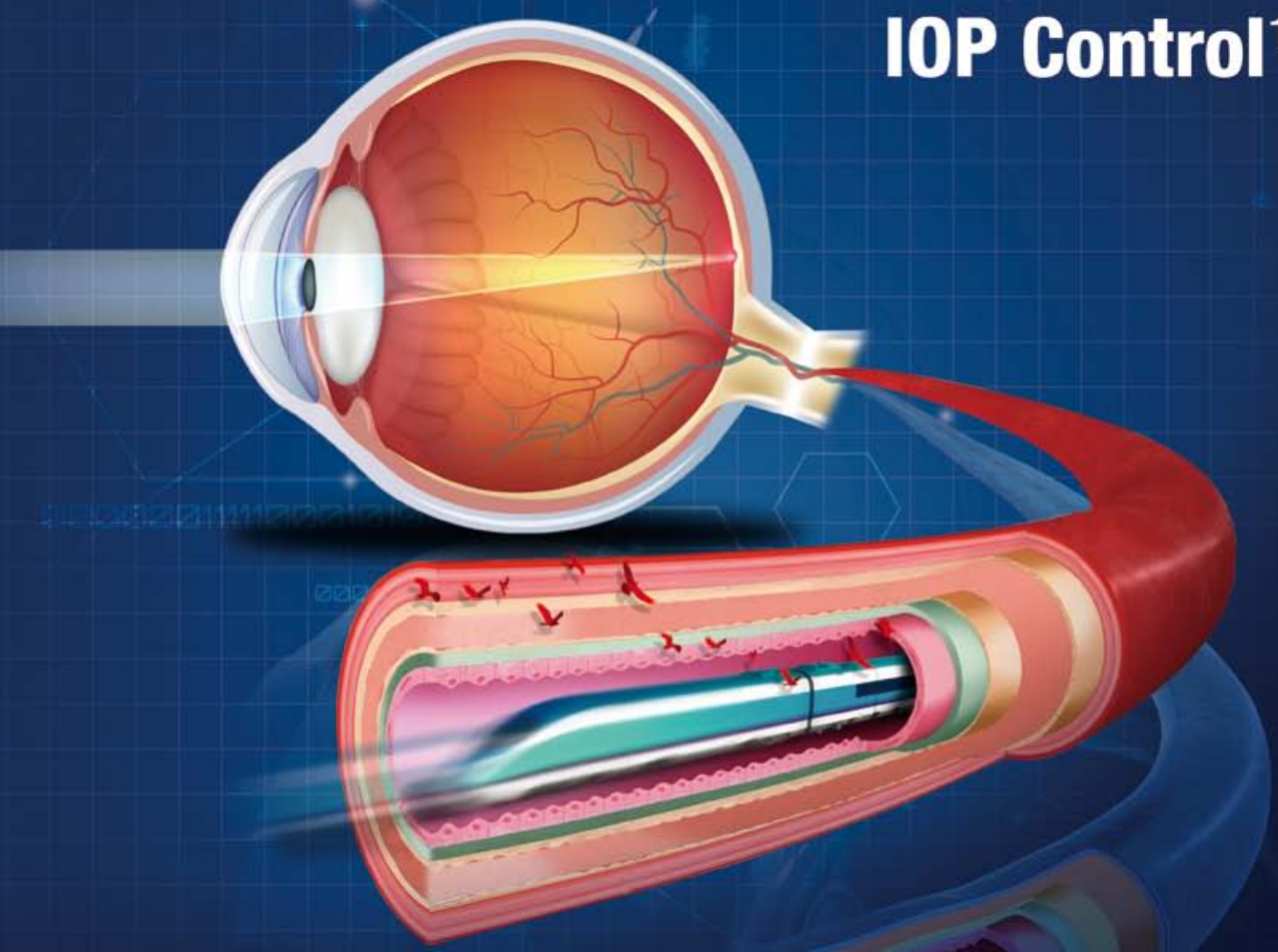

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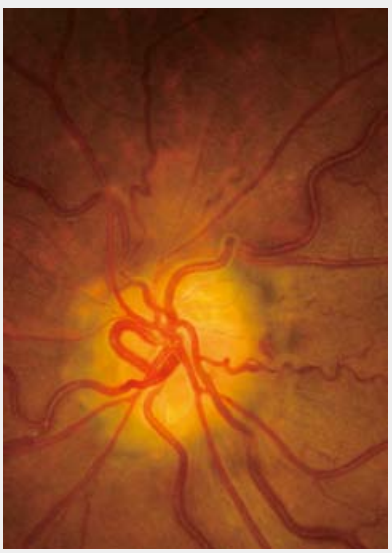

This photograph shows an optic disc with optic disc drusen. Optic disc drusen consist of deposits of mucoproteins and mucopolysaccharides that may eventually calcify in the optic disc. Optic disc drusen are also sometimes known as optic nerve head drusen, congenitally elevated or anomalous discs, pseudopapilloedema, pseudoneuritis, buried disc drusen, and disc hyaline bodies. Optic disc drusen are usually benign, but may rarely be associated with visual loss.

(Fundus photograph captured by Ms Yolanda YIP. Cover design and photograph digitally processed by Prof Clement CY Tham.)

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Editorial

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Editor

Prof Clement CY THAM

Ophthalmology remains a hugely desirable and 'sexy' specialty.

Each year, the few ophthalmology resident trainee vacancies are intensely sought after by the cream of medical graduates from our two medical schools. If you ask the applicants why they choose ophthalmology in interviews, they will usually tell you how important vision is to an individual's quality of life, especially in this day and age when most of our daily activities are visually-oriented or dependent. They will tell you with modern advanced technologies, ophthalmology can work miracles to restore sight. They will tell you ophthalmology is never dull, as new diagnostic and therapeutic 'gadgets' appear on the horizon ever so frequently. The phenomenal advances in technologies never stop to bewilder and fascinate. They may tell you the relatively more family-friendly lifestyle of ophthalmologists, but they do not usually mention financial rewards. After more than twenty years in ophthalmology, I can testify that they are actually right, after all.

In this Ophthalmology Issue, we aim to share with you some of the most exciting developments in ophthalmology that are happening right now. These advances will re-shape ophthalmic care in the coming decades.

Starting from the front of the eye, the public is already familiar with corneal transplantations, but few may know that specific layers, or 'lamella', of the cornea can now be transplanted, and thus allowing better preservation of host tissues and maximising the usage of much needed donated graft materials. Surgical outcomes and risk profiles are both enhanced. Dr Alvin Young, Chairman of the Hospital Authority Coordinating Committee in Ophthalmology, will present to you some of the landmark advances in the field of lamellar keratoplasty.

With an ageing population in a world increasingly dependent on near vision, presbyopia has never been a more serious problem. Heavy investments have gone into developing advanced technologies to correct, or at least alleviate, presbyopia. These include PresbyLASIK, corneal inlays, monovision, and lens implantation, which will be reviewed by Dr George Cheng, Clinical Assistant Professor (Honorary) of The Chinese University of Hong Kong (CUHK) and a much respected local ophthalmologist.

In Hong Kong and many other urban Chinese communities, myopia, or short-sightedness, is endemic. LASIK revolutionised myopic correction, and replaced spectacles correction, for a whole generation. Following in the footsteps of LASIK, a whole new spectrum of advanced technologies have emerged which could possibly cover every clinical scenario. Technologies such as advanced surface ablation, refractive lens / lenticule exchange, and phakic intraocular lens implantation, are reviewed in an article by Dr Vishal Jhanji, Director of the CUHK Refractive Surgery Centre. Apart from treating established myopia, recent advances in both Singapore and Hong Kong have also enabled the prevention of myopic progression in school children through effective and safe drug therapy. Though the optimal therapeutic regime

has yet to be clearly defined through further research, prevention of myopia could be the next major medical revolution that could change the destiny of future generations. Dr Jason Yam, Assistant Professor of the CUHK, will share with you evidence-based therapies to help your children, if they have been moved forward in their classrooms again lately.

Paediatric glaucoma used to be relatively unknown to our unsuspecting public. In the past, this had often resulted in late diagnosis and irreversible visual loss. Medical treatment is generally suboptimal, while surgical treatment requires highly subspecialised expertise and often repeated surgery, and yet visual outcomes may remain undesirable. It is extremely important to raise the awareness of this blinding disease amongst family physicians and the public. Dr Nafees Baig, Associate Consultant and Coordinator of the Glaucoma Service at the Hong Kong Eye Hospital, aims to share with you some of the latest, promising advances in the treatment of this serious eye disease in children.

Age-related macular degeneration is the commonest cause of irreversible elderly blindness in the developed world. It used to be an untreatable disease, until the avalanche of advanced therapies, such as photodynamic therapy and intravitreal anti-vascular endothelial growth factor injections, that emerged in the past decade for wet-type age-related macular degeneration. Lifestyle measures also prove to be useful in improving outcomes. These latest advances will be presented by Dr Ian Wong, Assistant Professor from the The University of Hong Kong (HKU).

For those who know a few ophthalmologists, you would probably have discovered that ophthalmologists are generally very interesting people, with broad and intense interests in many subjects and hobbies, outside ophthalmology. The final article in the lifestyle section, in which Prof Jimmy Lai from HKU shares with us all his expertise in premium shoes and their collection, should testify this. Most ophthalmologists generally do not suffer from tunnel vision!

Happy reading!!

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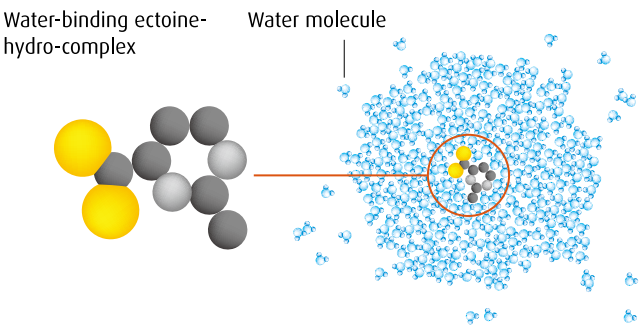

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1. Graf, R.et al., 2008
2. Bunger, J. and Driller, H., 2004
3. N.N., 2010
4. Harishchandra, R.K. et al., 2010

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Advances in corneal transplantation: lamellar keratoplasty

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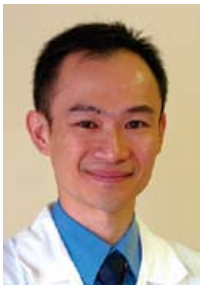

Dr Alvin L. YOUNG Dr Ka-wai KAM

Introduction

Corneal transplantation (keratoplasty) is the most commonly performed transplantation in humans.[1] Attempts were made as early as the 1800s to transplant animal eyes to humans but with very limited success.[2,3] It was only a century later in 1905 that Dr Eduard Zirm performed the first ever successful corneal transplant on a young labourer with bilateral scarred corneae resultant from chemical injury.[4] The donor was a 14 year old boy who suffered from severe penetrating injuries to both eyes that were beyond salvage. Permission was granted by the boy's father for Dr Zirm to enucleate his eyes, preserving the two cornea buttons to be transplanted onto the labourer's eyes. Despite the absence of microsurgical instruments, modern sutures and medications such as antibiotics and steroid, one of the two transplanted corneae survived and remained clear, allowing the young labourer to return to work.

In 2014, more than 70,000 corneal transplantations were performed in the United States alone.[5] The majority of the transplants were full thickness replacement, also known as penetrating keratoplasty (PK), which in principle is similar to what Dr Zirm had performed more than a century ago. However, we are now armed with better operating theatres, surgical microscopes, microsurgical equipment, sutures, antisepsis, topical steroid, antibiotics and immunosuppressants[6]. Most corneal grafts can now survive well beyond the early postoperative period.[7,8] In the modern era, minimising long term graft rejection is the primary focus in order to achieve a better long-term graft survival rate.

However, even with optimal medical care & the absence of rejection, endothelial cell loss & hence graft failure[9] over time remains a significant and an important issue to be addressed. Therefore selective replacement of only specific diseased layer(s) of the cornea gained popularity over the past two decades.[10-12] In this article, we will review the different types of more commonly performed lamellar keratoplasty (LK), indications & their advantages and disadvantages in comparison to conventional PK.

Types of Lamellar Keratoplasty

The human cornea is basically a five-layered structure. It consists of the epithelium, the Bowman's membrane, the stroma, the Descemet's membrane and the endothelium antero-posteriorly. (Fig. 1)

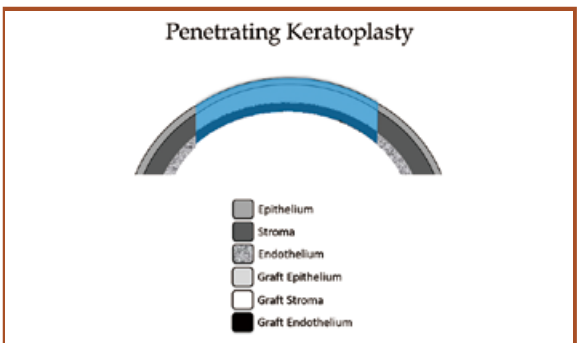

Fig. 1. Penetrating keratoplasty schematic diagram

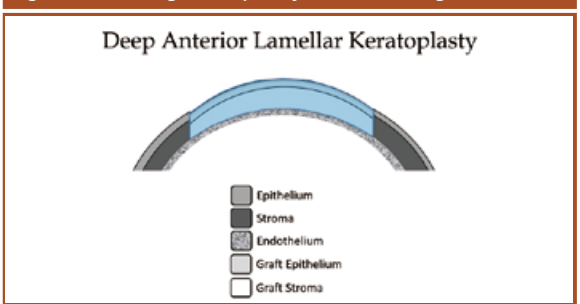

Fig. 2. Deep anterior lamellar keratoplasty schematic diagram

Lamellar keratoplasty is defined as partial thickness corneal tissue replacement, and can be broadly divided into anterior and posterior layer replacements. Anterior lamellar keratoplasty (ALK) refers to the replacement of the epithelium, the Bowman's layer and the stroma up to the Descemet's membrane (DM). Posterior layer replacement, better known as, endothelial keratoplasty (EK), aims to replace the diseased endothelial cells, the Descemet's membrane with or without adjacent stroma.

Anterior lamellar keratoplasty

Anterior Lamellar Keratoplasty (ALK) was commonly performed in the nineteenth century, when little was known about transplant immunology and the significance of corneal endothelium in allograft rejection, which was the prime reason for PK failures. ALK replaces only the anterior components of the cornea without disturbing (or treating) the corneal endothelium, thus circumventing the risk of immunological endothelial graft rejection.

ALK is further defined by the depth of anterior corneal tissue being replaced, with the retention of a variable thickness of the residual stroma, Descemet's membrane and endothelium of the recipient.

According to the John-Malbran classification for optical LK: Superficial-ALK (SALK) replaces the superficial 30% (160um) of the anterior cornea, Mid-ALK (MALK) - 30-70% (160-400um), Deep ALK (DALK)- 90-95% (470-495um) and Total ALK (TALK)- 100% (500-520um) of the stroma, excluding the Descemet's membrane and endothelium. Since each cornea differs in thickness, these figures are often arbitrary. After careful excision of the anterior diseased layer, either manually or with the assistance of air[13], femtosecond laser[14] medical gases or viscoelastic material, a donor button of matching thickness and suitable diameter would be sutured to the recipient bed.

Table 1. Abbreviations for commonly performed corneal transplantations.

PK	Penetrating keratoplasty (full thickness)
ALK	Anterior lamellar keratoplasty
DALK	Deep anterior lamellar keratoplasty
EK	Endothelial keratoplasty
DSEK	Descemet's stripping endothelial keratoplasty
DSAEK	Descemet's stripping automated endothelial keratoplasty
DMEK	Descemet's membrane endothelial keratoplasty

Posterior lamellar keratoplasty

Posterior lamellar keratoplasty (PLK), now better known as endothelial keratoplasty (EK) is a lamellar procedure that involves selective replacement of the corneal endothelium without disturbing the epithelium with preservation of various amounts of stroma (Fig. 3 & Fig. 4).[15] Selective replacement of corneal endothelium can be performed via much smaller wounds leaving the anterior part of the host cornea intact and hence better integrity of the globe.

EK can be further classified by the thickness/contents of the endothelial button, and by how the graft is prepared. Commonly performed EKs include Descemet's Stripping Endothelial Keratoplasty (DSEK), or Descemet's Stripping Automated Endothelial Keratoplasty, whereby the endothelial button is prepared by special keratomes, Descemet's Membrane Endothelial Keratoplasty (DMEK), which offers the thinnest button consisting of only the endothelial cells and the Descemet's membrane. In the future, it may even be possible to only transplant endothelial cells by injection.[16]

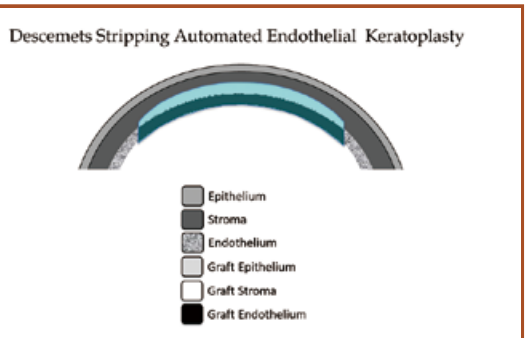

Fig. 3. Descemet's stripping automated endothelial keratoplasty

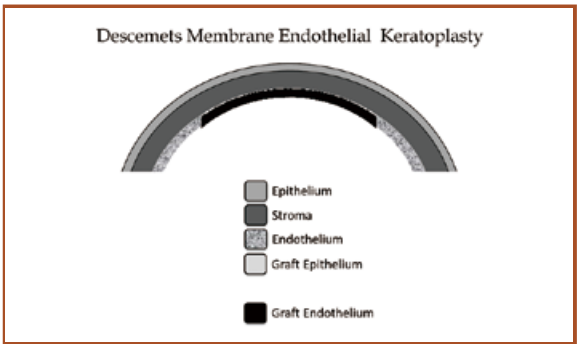

Fig. 4. Descemet's membrane endothelial keratoplasty

Indications for Lamellar Keratoplasty

Anterior lamellar keratoplasty

Indications for anterior lamellar keratoplasty include pathologies affecting the anterior layer of the cornea from the epithelium, the Bowman's membrane to the stroma up to the level of the Descemet's membrane.

Common causes in our locality would include scars from infective keratitis such as herpetic keratitis[17-19], trauma, ruptures, immune disorders[20], keratoconus with or without history of hydrops, or occasionally as a therapeutic option for medically uncontrolled infection of the cornea.[21-23]

Keratoconus (KCN) is a relatively uncommon condition in which the stroma of the cornea becomes progressively thin and bulges forward to yield a conical cornea. This may result in severe refractive changes of the eye, astigmatism, scarring of the cornea and occasionally a spontaneous rupture in the Descemet's membrane (leading to influx of aqueous into the stroma) as acute hydrops. Replacement of the anterior lamellar scars by DALK can yield a favourable outcome in restoring a more regular surface of the cornea and better refractive function.[24]

Posterior lamellar keratoplasty

Indications for EK include primary or secondary dysfunction of the corneal endothelium.[25]Primary dysfunctions of endothelium include various corneal dystrophies affecting the posterior layer of the cornea – more commonly seen is Fuchs endothelial dystrophy where loss of endothelial cells is imaged by specular microscopy. Other rarer forms of endothelial dystrophy include posterior polymorphous dystrophy (PPMD) and congenital hereditary endothelial dystrophy (CHED). Iridocorneal endothelial syndrome is another rarer cause of unilateral primary corneal dysfunction.

Secondary endothelial dysfunction of the cornea refers to corneal decompensation following acute or chronic rise in intraocular pressure, glaucoma, infection and/or intraocular inflammation, multiple ocular operations or lasers, trauma or epithelial downgrowth.

EK can also be performed in selected eyes with a history of failed corneal transplant if the anterior part of the eye remains reasonably clear.

Advantages and Disadvantages of Lamellar Keratoplasty

One major advantage of LK is that one cornea graft can benefit more than one patient.[10] Up to this moment, graft scarcity is still a major issue both globally and locally.[26-28] On the other hand, one disadvantage common to both anterior & posterior lamellar keratoplasty is the relatively steeper learning curve.[29-31]

Anterior lamellar keratoplasty

The major advantage of anterior lamellar keratoplasty is to preserve the endothelium of the recipient and thus eliminating the cumulative risk of endothelial rejection in the long run.[32] This is especially important for younger patients such as those with corneal scars and or keratoconus.

Two recent meta-analyses of randomised controlled trials comparing DALK to PK in treatment of non-endothelial pathologies showed that DALK is associated with significantly fewer endothelial graft rejections, a lower percentage of endothelial cell loss[33], and lower rates of postoperative complications.[34]

A second advantage of DALK is that the whole procedure is extraocular and hence the risk of intraocular infection is much less.[34] Unlike PK during which the eye is opened thus deflated after removal of the diseased cornea, the globe remains formed and pressurised throughout DALK. This will minimise the risk of massive haemorrhage or expulsion of intraocular contents during the operation.[35] Postoperatively, DALK also gives a better integrity to the globe in comparison to PK where a circumferential full-thickness wound is created.[36]

Perhaps the biggest challenge of LK is the technical demands in order to achieve a good lamellar dissection.[31,37] Different surgeons have proposed different approaches either manually or with the assistance of air, balanced salt solution, viscoelastic material, or even intraoperative pachymetry or optical coherence tomography guidance.[38,39]

Intraoperative inadvertent macro-perforations of the Descemet's membrane (ranging from at least 2.3%-32%) would necessitate conversion to PK. It is therefore mandatory for all patients planned for DALK to be properly informed of the potential risk of conversion to PK prior to the operation.

There has been inconsistency in reports on visual outcome post DALK. A recent meta-analysis demonstrated that the number of eyes achieving vision better than 20/40 is comparable to PK. However, PK eyes achieved a better uncorrected and best-corrected visual acuity overall. This may be related to residual scarring and irregularities at the interface of DALK. Refractive outcomes for spherical equivalence and astigmatism are comparable between the two surgical techniques.[33,34]

Posterior lamellar keratoplasty

Descemet's stripping endothelial keratoplasty (DSEK) requires the surgeon to strip off the diseased endothelium and the Descemet's membrane of the patient, then manually dissect a thin posterior layer from a full thickness cornea graft. The surgeon will then transfer the posterior graft (EK button) to the recipient's anterior chamber, and attach the endothelial graft to the posterior stroma of the recipient by means of an air bubble.[15,40]

Smaller wounds and fewer sutures are required for the procedure, and the risks associated with an open deflated globe are thus minimised. A smaller wound would allow for stronger structural integrity postoperatively. As fewer (if any) sutures are required and often placed peripherally, there is thus relatively much less astigmatism or refractive error following EK. However, similar to ALK, EK also requires a much steeper learning curve, in both the preparation of a thin donor button and in the management of the EK button in the recipient's anterior chamber.

Potential intraoperative complications may include inadvertent buttonhole of the posterior graft, eccentricity of the graft and even inadvertent reversal of the graft button. Early postoperative complications include slippage or dislocation of graft, requiring a second surgery to reposition the graft, interface fluid, primary graft failure (especially in the case of reversal) & elevated intraocular pressure.

Some eye banks may prepare a pre-cut cornea, and hence shorten the operative time and avoid the associated complications in manual preparation. If the graft is prepared with an automated keratome, the procedure is then termed 'Descemet's stripping automated endothelial keratoplasty' (DSAEK).

Fig. 5. Postoperative photo showing circumferential wound with interrupted 10-O nylon sutures securing the anterior lamellar graft. The arrow is pointing at blood vessels within the graft-host interface.

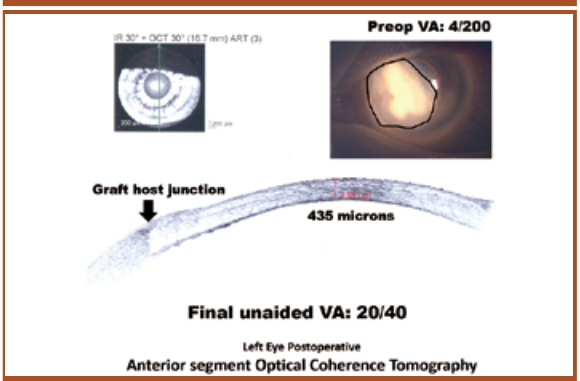

Fig. 6. Postoperative optical coherence tomography (OCT) image showing the graft host junction and a central corneal thickness of 435 microns.

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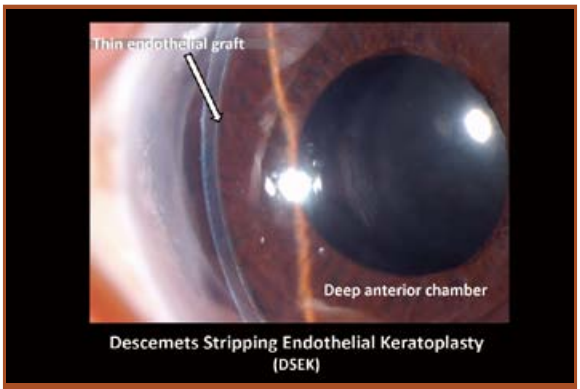

Fig. 7. Postoperative appearance of a Descemet's stripping endothelial keratoplasty.

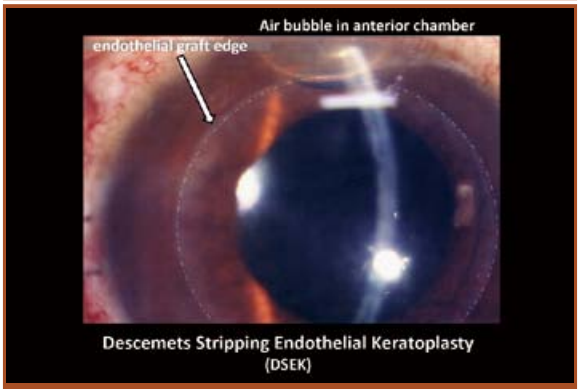

Fig. 8. Diffuse illumination showing the edge of the posterior endothelial graft.

In recent years, thinner & thinner grafts were tried. A procedure known as Descemet's membrane endothelial keratoplasty (DMEK) of which only the Descemet's membrane containing the endothelial cells was transplanted. DMEK has a lower reported rate of rejection, but it is also associated with higher rates of graft wastage in preparation & endothelial cell loss, as the graft is extremely thin and difficult to manipulate throughout.[41]

In conclusion, LK now offers an alternative to patients with various corneal disorders in whom a penetrating keratoplasty would have been performed in the past. LK bypasses many potential intraoperative and postoperative risks associated with conventional full-thickness transplants and ultimately aims to achieve better graft survival and visual outcomes in the long run.

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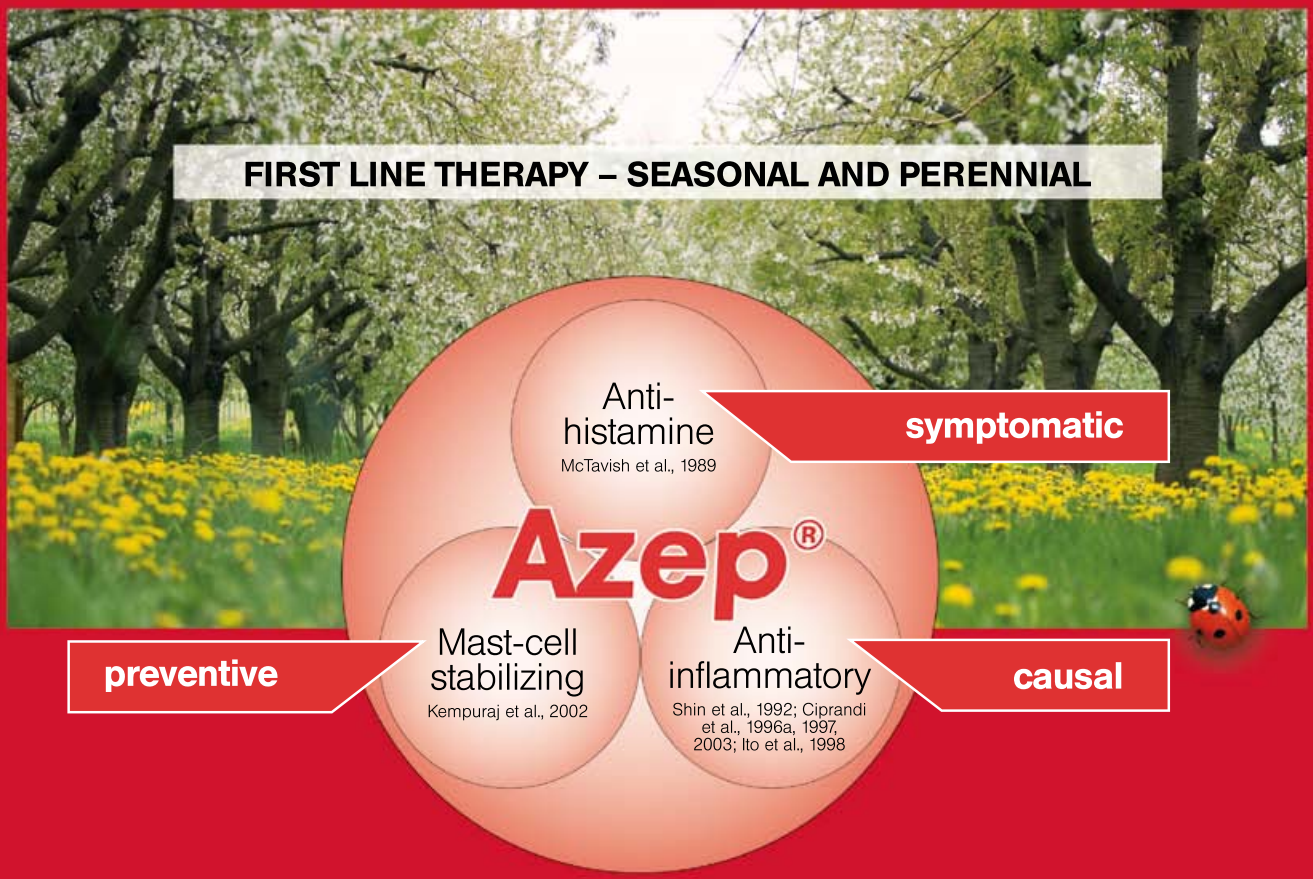

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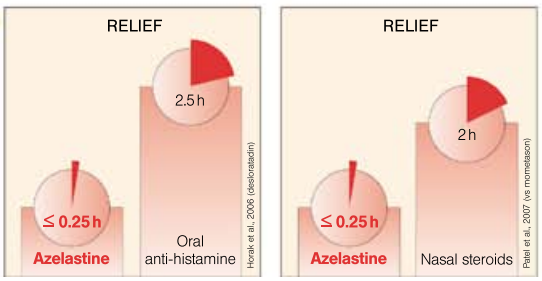

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Presbyopic correction: the time has come?

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Hong Kong Laser Eye Centre
Dr Alex NG
The University of Hong Kong


Dr George CHENG Dr Alex NG

Introduction

Presbyopia refers to the age-related loss of accommodation and thus the inability of the crystalline lens to change its focus from far to near in order to achieve a clear near vision. It affects everyone starting at the age of 40 to 45, and is affecting billions of people worldwide. Presbyopia causes considerable inconvenience when one switches between common daily visual tasks, such as from distance objects to a computer screen (intermediate vision), or to the smartphone screen (near vision).

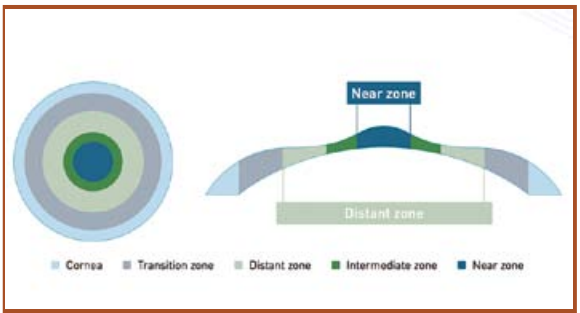
Using spectacles (bifocal or progressive additional lenses) or contact lenses are the traditional ways of correcting presbyopia. In recent years, more and more patients wish to be spectacle free, both for convenience and life-style considerations. There have been major advances in both corneal procedures and intraocular lens technologies in presbyopia correction, and this have provided a wide range of new options for the presbyopes. This review aims to give an overview of the current mainstream surgical options for presbyopic correction.

1. Corneal approach

The human cornea is responsible for two-thirds of the refractive power of the eyeball. The main advantage of these corneal surgical options is that intraocular surgeries (lens-based procedures), which have higher surgical risks, are avoided.

A. PresbyLASIK

In the past twenty years, laser vision correction using laser-assisted in situ keratomileusis (LASIK) has been proven to be safe and accurate in correcting myopia, hyperopia and astigmatism. With continued advancement in the laser platforms, correction for presbyopia is now possible by special ablation profiles that would create a multifocal cornea, and these are referred to as PresbyLASIK. Multiple laser platforms exist (such as PresbyMAX), but most of them work by modifying the aberration profiles of the cornea in order to increase its depth of focus. There are broadly two main types of PresbyLASIK, namely central presbyLASIK, where the centre of the cornea is responsible of focusing near objects and the peripheral cornea for distance objects, and vice versa (centre for distance, peripheral for near) which is peripheral presbyLASIK. Studies have shown a relatively high degree of patient satisfaction and spectacle independence with this procedure. The main advantage of presbyLASIK is that it can be combined with the usual LASIK procedure to correct for other refractive errors at the same surgery. The technique is almost identical to LASIK except the computer programme would change the ablation profile, and most refractive surgeons are very familiar with this technique. The procedure is completely reversible and very safe, with similar safety profile as the usual LASIK. PresbyLASIK is best for young presbyopes aged around 40 to 55 years old where there are still some residual accommodation. However, in older presbyopes, presbyLASIK might not be adequate to fully compensate for the complete loss of accommodation. Other side effects are halos and glares, which is common among most presbyopic correcting procedures. When these patients with a multifocal cornea require cataract surgery in the future, issues concerning an accurate intraocular lens power calculation may arise too.

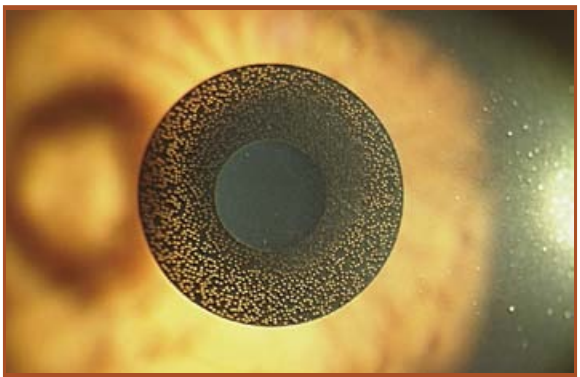


A schematic diagram and cross-section of the cornea as an example of PresbyLASIK treatment using the PresbyMax platform. Source: http://www.schwind-amaris.com

B. Corneal inlays

Corneal inlays are intrastromal implants for the correction of presbyopia. They could be placed underneath a LASIK flap, or into a femtosecond laser created corneal pocket. They are usually implanted in the non-dominant eye to aid near vision, while the dominant eye remains emmetropia for clear distance vision. Currently there are 4 types on the market and they work through different principles. The first type is a small-aperture inlay (Kamra, AcuFocus) which increases the depth of focus. The other type is a reshaping inlay (Raindrop, ReVision Optics) and changes the corneal curvature at the implanted area for near vision. The remaining two types are refractive optics inlay (Flexivue Microlens, Presbia; Icolens, Neoptics AG) which alter the refractive index with a bi-focal optics. The advantage of a corneal inlay is that it is

a fully reversible procedure. No corneal tissue is ablated and thus the patient could theoretically receive other forms of laser vision correction if the inlay is explanted. They can also be implanted in combination with a usual LASIK procedure. By far, up to 5 years follow up data are available in the literature, and the outcomes are promising. There may be unilateral loss of distance visual acuity in the implanted eye, but the binocular distance visual acuity is not affected. However, some studies have reported up to 20% explantation rate due to the reduced unaided distance visual acuity. Other disadvantages of the procedure include reduced contrast sensitivity, halos and glares, which are common among most presbyopia correcting procedures. These side effects are more pronounced if the centration of the inlay is not ideal. Low-grade inflammation and corneal haze has also been reported after corneal inlays, and there could also be a change of corneal topography leading to a later hyperopic shift.



The Kamra intracorneal inlay in a patient's eye with visible small nutritional holes. Source: Dexl et al, J Cataract Refract Surg. 2011;37(3):525-31.

C. Monovision

Despite the above emerging corneal procedures, monovision remains a very popular and viable technique to correct presbyopia. In monovision, one eye is corrected for clear distance vision (usually the dominant eye), while the other eye is corrected for near or intermediate vision by aiming for around 1.5 to 2 dioptres of myopia, depending on the functioning need of the patient. Monovision is commonly done during laser vision correction procedures, such as LASIK or the newer small incision lenticule extraction (SMILE), but can also be employed during spectacle correction, contact lens correction or intraocular lens implantation. It works by the principle of inter-ocular blur suppression, and most patients can adapt to this intentional anisometropia with good outcomes. Studies have reported up to 93% acceptance rate. Limitations of monovision include reduced stereopsis and contrast sensitivity. In the occasional cases where patients could not adapt to the monovision, further enhancements could be performed on the near eye to achieve clear bilateral clear distance vision.

Other than using monovision alone, very often a mini-monovision approach (leaving the non-dominant eye slightly myopic at -0.5 to 1.0 dioptre for enhanced intermediate vision) is combined with other corneal or intraocular lens implantation procedures to further enhance the presbyopia correction effect.

2. Lenticular approach with intraocular lens implantation

In traditional cataract surgery where an intraocular lens (IOL) is implanted, the IOL is monofocal, and thus the patient could either have a clear distance or near vision, but not both. With the advances in IOL technology, presbyopia-correcting IOL is becoming an alternative choice for patients wishing to become spectacle independent. They are most suitable for presbyopes with co-existing cataracts, since both conditions can be treated through one surgery. In patients without cataract, a refractive lens exchange (RLE) procedure can also be performed, where their own clear crystalline lens is removed and 'exchanged' with a presbyopia-correcting IOL. RLE is becoming more and more acceptable in younger presbyopic patients (40 years and over), especially for those with very high refractive errors where traditional laser vision correction cannot be performed. The main disadvantage of a refractive lens exchange is its intraocular nature. Although very low, there are risks of infection, retinal detachment and cystoid macular oedema associated with this intraocular surgery, and this must be discussed with the patient.

As for the presbyopia-correcting IOL, the Holy Grail is an IOL that could fully mimic the natural lens with the ability to accommodate. Currently, hinge-based 'accommodating' IOLs could only achieve a very small degree of pseudo-accommodation. True accommodating IOLs are still underway, and multifocal IOL (MFIOL) is the mainstay of choice for most refractive surgeons. A MFIOL either employs a refractive or diffractive design to offer more than one focal point and utilises the principle of simultaneous vision, where the patient's brain needs to 'select' the correct focused image when undertaking visual tasks at different distances. This multifocality requires some neural adaptation, and although this adaptation may vary among patients, most patients could achieve spectacle independence. Bi-focal MFIOLs with a distance and near focal points have been on the market for a long time and patients are able to achieve clear unaided distance and near vision. However, the intermediate vision (such as for viewing a computer screen) is still suboptimal. In recent years, trifocal IOLs with 3 focal points, or extended-depth of focus IOLs, have become available to provide improved intermediate vision. The main limitation of MFIOL is the compromised optical quality due to multiple focal planes, where the incoming light rays are divided between the focal points. This results in reduced contrast sensitivity. Nevertheless, a recent meta-analysis indicated that the unaided near vision improvement with MFIOLs outweighs the reduction in contrast sensitivity in patients who wish to achieve spectacle independence. The other common problems with MFIOL are glares and halos which are common among almost all presbyopia correcting procedures. Fortunately, these tend to subside with time. However, for the small proportion of patients with persistent symptoms and low satisfaction, surgical explantation of the MFIOL may be required.

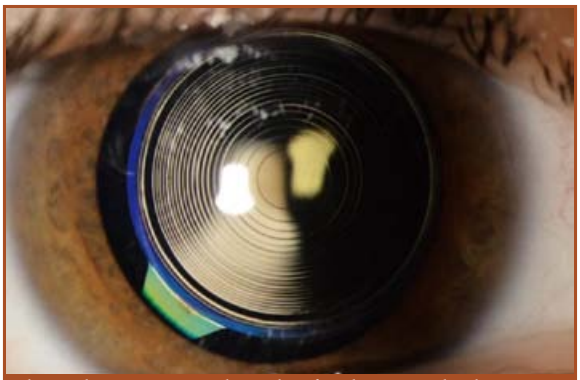


Photo showing an implanted trifocal intraocular lens. Multiple concentric rings on the IOL can be seen. Source: http://ophthalmologytimes.modernmedicine.com

Conclusion

In conclusion, there are many exciting new procedures for presbyopic correction. Techniques to truly restoring the natural accommodative power are still lacking. The current methods described above are all 'static' methods which aim to create multiple foci for the patients, but with a small compromise in the visual quality and cause symptoms such as glares and halos. Nevertheless, these procedures are already providing significant improvements to the quality of life of the presbyopes. The surgeon should understand the limitations of each procedure, and giving the patient realistic expectations is the key to a successful outcome and a happy patient.

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Myopic correction: are there options other than LASIK?

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Introduction

Myopia is the most common refractive error worldwide. The prevalence of myopic refractive error has been increasingly progressively especially in Asia. With over 80 million reported myopic children worldwide there are considerable public health concerns. Recent evidence on the use of atropine eye drops for prevention of myopia progression is compelling but it has not been put to actual use. In the face of increasing number of patients with myopia worldwide, more emphasis is being put on the betterment of existing techniques for management of myopic refractive errors.

Laser in situ keratomileusis (LASIK)

LASIK is by far the most popular refractive surgery for myopia. It involves creating a corneal flap using a microkeratome blade or femtosecond laser, reshaping the cornea using an excimer laser to remove tissue from the underlying stromal bed and then replacing the flap. The currently available data on the safety and efficacy data on LASIK are very robust. It is considered to be an extremely safe procedure for management of myopia, hyperopia and astigmatism. However, potential problems with LASIK include flap-related complications and post-refractive surgery ectasia. Progressive corneal steepening and thinning is associated with irregular corneal topography, age, higher myopia, thinner central corneal thickness and low residual stromal bed thickness.

Alternatives to LASIK include a wide range of surgeries including advanced surface ablation, refractive lens exchange (RLE), phakic intraocular lens and refractive lenticule extraction.

Advanced Surface Ablation

Photorefrative keratectomy (PRK) is a viable alternative especially in cases with high myopia and inadequate residual stromal bed. The surgery involves removal of corneal epithelium followed by ablation of anterior corneal stroma. It has been shown that corneal stability is higher in the postoperative period after PRK as compared to LASIK in highly myopic eyes. Since PRK is a non-incisional surgery, it maintains the overall biomechanical strength of the cornea in the long-term. Postoperative corneal stromal haze is the most worrisome issue after PRK. The surgeon applies mitomycin C 0.02% for 12 -60 seconds after the laser ablation to mitigate corneal haze in eyes where the ablation depth exceeds 75 microns.

Other important consideration unique to advanced surface ablation is a delayed visual recovery. Whereas the patients can resume work shortly after LASIK, PRK is associated with pain and discomfort for the first few days after surgery. The overall visual recovery can take up to 4 weeks after PRK.

Refractive Lens Exchange

Refractive lens exchange involves phacoemulsification of a clear lens and replacing it with an artificial intraocular lens of desirable power in order to correct the refractive error. It is an effective surgical alternative when corneal laser refractive surgery may not be indicated, such as for very high myopia greater than 10.00 D, irregular or higher levels of astigmatism, and thin corneas. RLE is often the treatment of choice for hyperopic presbyopes aged 40 years and older. It is also a good option for patients who are contact lens-intolerant and are otherwise not suitable for corneal refractive surgery.

RLE is associated with an increased risk of retinal detachments especially in young patients with posterior vitreous detachments. In young patients, RLE can lead to dissatisfaction that is mainly related to the loss of accommodation.

Phakic Intraocular Lens Implantation

Phakic IOLs (PIOLs) represent an excellent surgical treatment option for all ranges of myopia from 3.00 D to 20.00 D. It is suitable for patients who are unsuitable for corneal excimer laser surgery mainly the ones with inadequate corneal thickness and ocular surface problems. The main advantages of PIOLs include its potential reversibility and preservation of accommodation. The level of satisfaction in patients with high myopia is higher as compared to excimer laser surgery. This could be partially a better contrast sensitivity after PIOL implantation than corneal excimer laser surgery.

The major difference between PIOL implantation and corneal excimer laser surgery is that the former is an intraocular surgery. It can be associated with devastating complications including intraoperative bleeding and lens damage. The most concerning long-term safety issues for PIOLs include endothelial cell loss and cataract formation. Secondary glaucoma, intraocular inflammation, and traumatic lens dislocation are potential long-term risks that have been associated with PIOLs.

PIOL is a relatively expensive option for patients compared to LASIK and advanced surface ablation. Only one eye can be operated at one time therefore increasing the postoperative recovery time. PIOLs are currently reserved for patients who are not suitable for corneal refractive surgery.

Refractive Lenticule Exchange

The concept of refractive lenticule extraction (ReLEx) involves the creation of an intrastromal corneal lenticule that can then be removed as a single piece through a small incision thereby circumventing the need for incremental photoablation by an excimer laser. The VisuMax femtosecond laser was introduced in 2007 for performing a procedure called Femtosecond Lenticule Extraction (FLEx). Although the refractive results were similar to those observed in LASIK, the visual recovery time was longer.

Subsequently, a new procedure called Small Incision Lenticule Extraction (SMILE) was developed. This procedure involves separation of corneal lenticular surfaces using a dissector through a small 2–3 mm. The lenticule is then removed from the incision. The initial results of SMILE procedure are encouraging with outcomes comparable to LAISK.

The SMILE procedure is a femtosecond-laser assisted refractive surgery that does not involve creation of a large flap. The corneal biomechanics are better preserved after SMILE as compared to LASIK. It has also been shown that the incidence of dry eyes is less with SMILE when compared to LASIK. The potential disadvantages include slightly delayed visual recovery compared to LASIK and limited options for enhancement. However, with improvements in laser platforms, SMILE surgery is gradually gaining popularity amongst refractive surgeons.

Conclusions

LASIK has long enjoyed the status of a popular refractive surgery option for management of myopia. Its long-term safety and efficacy are clear advantages over other newer surgeries. However, not all patients are suitable for LASIK. The need to have reliable surgical alternatives is increasing mainly due to an increase in myopia prevalence. Advanced surface ablation is an excellent and economical option for patients with high myopia. PIOLs are indicated in patients with extremely high myopia. Refractive lens exchange is suitable for patients in the presbyopic age group who would otherwise need a cataract surgery at a later date. Advancements in laser platforms have made surgeries like SMILE possible, which have the potential advantages of both LASIK and advanced surface ablation. Instead of adopting a 'one size fits all' policy, it is prudent to customizse each treatment according to the needs of the patients.

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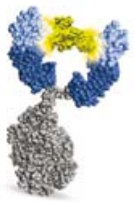

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- Unsurpassed vision gains in wAMD, 33% of patients gained ≥3 lines at 96 weeks[1]
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Eylea® 40 mg/ml solution for injection in a vial (aflibercept) Prescribing Information (Refer to full Prescribing Information before prescribing) **Presentation:** 1 ml solution for injection contains 40 mg aflibercept. Each vial contains 100 microlitres, equivalent to 4 mg aflibercept. **Indication(s):** Treatment of neovascular (wet) age-related macular degeneration (AMD),macular oedema secondary to central retinal vein occlusion (CRVO) and visual impairment due to diabetic macular edema (DME) in adults. **Posology & method of administration:** For intravitreal injection only. Must be administered according to medical standards and applicable guidelines by a qualified physician experienced in administering intravitreal injections. Each vial should only be used for the treatment of a single eye. The vial contains more than the recommended dose of 2 mg. The extractable volume of the vial (100 microlitres) is not to be used in total. The excess volume should be expelled before injecting. Refer to full prescribing information for full details. ***Adults:*** The recommended dose is 2 mg aflibercept, equivalent to 50 microlitres. For wAMD treatment is initiated with one injection per month for three consecutive doses, followed by one injection every two months. No requirement for monitoring between injections. After the first 12 months of treatment, treatment interval may be extended based on visual and anatomic outcomes. In this case the schedule for monitoring may be more frequent than the schedule of injections. For CRVO, after the initial injection, treatment is given monthly at intervals not shorter than one month, and continues until visual and anatomic outcomes are stable for three monthly assessments. Thereafter the need for continued treatment should be reconsidered. Treatment may be continued with gradually increasing treatment intervals to maintain a stable visual and anatomic outcome. Continued treatment is not recommended if no improvement in visual and anatomic outcomes over the first three injections. If treatment is discontinued, monitor visual and anatomic outcomes and resume treatment if these deteriorate. Usually, monitoring should be done at the injection visits. During treatment interval extension until therapy completion, the monitoring schedule should be determined by the treating physician based on the individual patient's response and may be more frequent than the schedule of injections. For DME, initiate treatment with one injection/month for 5 consecutive doses, followed by one injection every two months. No requirement for monitoring between injections. After the first 12 months of treatment, the treatment interval may be extended based on visual and anatomic outcomes. The schedule for monitoring should be determined by the treating physician. If visual and anatomic outcomes indicate that the patient is not benefiting from continued treatment, treatment should be discontinued. ***Hepatic and/or renal impairment:*** No specific studies have been conducted. Available data do not suggest a need for a dose adjustment. ***Elderly population:*** No special considerations are needed. Limited experience in those with DME over 75 years old. ***Paediatric population:*** No data available. **Contra-indications:** Hypersensitivity to active substance or any excipient; active or suspected ocular or periocular infection; active severe intraocular inflammation. **Warnings & precautions:** As with other intravitreal therapies endophthalmitis has been reported. Aseptic injection technique essential. Patients must report any symptoms of endophthalmitis without delay. Increases in intraocular pressure have been seen within 60 minutes of intravitreal injection; special precaution is needed in patients with poorly controlled glaucoma (do not inject while the intraocular pressure is ≥ 30 mmHg). Immediately after injection, monitor intraocular pressure and perfusion of optic nerve head and manage appropriately. There is a potential for immunogenicity as with other therapeutic proteins; patients should report any signs or symptoms of intraocular inflammation e.g pain, photophobia or redness, which may be a clinical sign of hypersensitivity. Reports of systemic adverse events including non-ocular haemorrhages and arterial thromboembolic events following intravitreal injection of VEGF inhibitors. Safety and efficacy of concurrent use in both eyes have not been systemically studied. Caution in patients with risk factors for development of retinal pigment epithelial tears including large and/or high pigment epithelial retinal detachment. Withhold treatment in patients: with rhegmatogenous retinal detachment or stage 3 or 4 macular holes; with retinal break and do not resume treatment until the break is adequately repaired. Withhold treatment and do not resume before next scheduled treatment if there is: decrease in best-corrected visual acuity of ≥30 letters compared with the last assessment; central foveal subretinal haemorrhage, or haemorrhage ≥50%, of total lesion area. Do not treat in the 28 days prior to or following performed or planned intraocular surgery. Eylea should not be used in pregnancy unless the potential benefit outweighs the potential risk to the foetus. Women of childbearing potential have to use effective contraception during treatment and for at least 3 months after the last intravitreal injection. Populations with limited data: There is limited experience of treatment with Eylea in patients with ischaemic, chronic CRVO. In patients presenting with clinical signs of irreversible ischaemic visual function loss, the treatment is not recommended. There is limited experience in DME due to type I diabetes or in diabetic patients with an HbA1c over 12% or with proliferative diabetic retinopathy. Eylea has not been studied in patients with active systemic infections, concurrent eye conditions such as retinal detachment or macular hole, or in diabetic patients with uncontrolled hypertension. This lack of information should be considered when treating such patients. **Interactions:** No available data. **Fertility, pregnancy & lactation:** Not recommended during pregnancy unless potential benefit outweighs potential risk to the foetus. No data available in pregnant women. Studies in animals have shown embryo-foetal toxicity. Women of childbearing potential have to use effective contraception during treatment and for at least 3 months after the last injection. Not recommended during breastfeeding. Excretion in human milk: unknown. Male and female fertility impairment seen in animal studies with high systemic exposure not expected after ocular administration with very low systemic exposure. **Effects on ability to drive and use machines:** Possible temporary visual disturbances. Patients should not drive or use machines if vision inadequate. **Undesirable effects:** *Very common:* conjunctival haemorrhage (phase III studies: increased incidence in patients receiving anti-thrombotic agents), eye pain, visual acuity reduced. *Common:* retinal pigment epithelium tear, detachment of the retinal pigment epithelium, retinal degeneration, vitreous haemorrhage, cataract (nuclear or subcapsular), corneal abrasion or erosion, corneal oedema, increased intraocular pressure, blurred vision, vitreous floaters, vitreous detachment, injection site pain, foreign body sensation in eyes, increased lacrimation, eyelid oedema, injection site haemorrhage, punctate keratitis, conjunctival or ocular hyperaemia. *Uncommon:* Injection site irritation, abnormal sensation in eye, eyelid irritation. *Serious:* Cataract, retinal detachment, vitreous detachment, endophthalmitis, and intraocular pressure increased. Consult the full prescribing information in relation to other side effects. **Overdose:** Monitor intraocular pressure and treat if required. **Incompatibilities:** Do not mix with other medicinal products. **Special Precautions for Storage:** Store in a refrigerator (2°C to 8°C). Do not freeze. Unopened vials may be kept at room temperature (below 25°C) for up to 24 hours before use. **Date of preparation: August 2014**

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VEGF-A = vascular endothelial growth factor, PlGF = placental growth factor
*Neovascular (wet) age-related macular degeneration (wAMD), visual impairment due to macular edema secondary to central retinal vein occlusion (CRVO), visual impairment due to diabetic macular edema (DME).

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1. EYLEA® full Prescribing Information, Hong Kong, August 2014.
2. Papadopoulos N, Martin J, Ruan Q, et al. Binding and neutralization of vascular endothelial growth factor (VEGF) and related ligands by VEGF Trap, ranibizumab and bevacizumab. Angiogenesis. 2012;15(2):171-185.

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Paediatric glaucoma: update on diagnosis and treatment options

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Introduction

Paediatric glaucoma, characterised by elevated intraocular pressure (IOP), affects infants and children, and can lead to irreversible blindness. The prognosis is largely dependent on early and accurate diagnosis with timely treatment, involving rigorous IOP reduction to a level at which further progression is unlikely, along with the prevention of amblyopia.[1] It is classified as 'primary' when an isolated idiopathic developmental abnormality of the anterior chamber angle exists, and 'secondary' when aqueous outflow is reduced due to either a congenital or an acquired ocular disease or systemic disorder.[2]

Primary paediatric glaucoma

Primary paediatric glaucoma includes primary congenital glaucoma (PCG) and juvenile open-angle glaucoma (JOAG). PCG is the commonest glaucoma in infancy,[3,4] but it has a variable reported incidence worldwide, the average incidence is about 1 in 10,000 live births. A higher prevalence (up to 1:1,250) has been observed in genetically inbred populations and in which parental consanguinity is common.[5,6,7] PCG has been reported to occur more frequently in males than in females[8,9,10] and is reported to be bilateral in 70% to 80% cases.[11,12] Familial cases tend to have an equal sex distribution with autosomal recessive inheritance and variable penetrance.[10,11,13]

Secondary paediatric glaucoma

Secondary paediatric glaucoma is commonly associated with anterior segment dysgenesis, developing in 50% of cases.[14] Glaucoma associated with aniridia is usually due to progressive angle closure, presenting often in childhood with an incidence ranging from 6% to 75%.[15] Aphakic glaucoma can occur early or years after initial uneventful cataract extraction surgery in children with congenital cataract, and has a variable incidence from 5% to 41%, depending on the age of surgery, corneal diameter, and surgical techniques.[16,17,18,19] Phacomatoses that are commonly associated with glaucoma, include the Sturge-Weber syndrome and Klippel-Trenaunay-Weber syndrome.[20] The glaucoma seen in inflammatory disorders is multifactorial, with the reported incidence as high as 38% in children with juvenile idiopathic arthritis.[21]

As paediatric glaucoma is a relatively uncommon disease, it was estimated that a consultant ophthalmologist in a non-specialist centre in the Western world would expect to see a new case of primary congenital glaucoma (PCG) approximately every 5 years.[22] As a result of its relative rarity, PCG is sometimes misdiagnosed or sub-optimally treated, especially in non-specialist centres, leading to irreversible corneal and optic nerve damage, and unnecessary irreversible visual loss. Consequently, it accounts for a disproportionate percentage (up to 18%) of children in blind institutions around the world.[23,24] Congenital glaucoma accounts for 30% of paediatric patients attending a university's low vision service.[25] Overall, glaucoma is responsible for 5% of irreversible blindness in children worldwide.[26]

Clinical presentation

The classical triad of symptoms is epiphora, photophobia and blepharospasm but these can be absent in some cases. Patients can also present with cloudy large cornea, buphthalmos, strabismus, lack of eye contact and nystagmus. Clinical examinations involve the measurement of IOP and ocular dimensions, refraction, examination of the cornea and anterior segment with the slit lamp, examination of the anterior chamber angle using a gonioscopy and fundal examination for optic disc appearance and cup-disc ratio. These can be performed under sedation or general anaesthesia for younger/ uncooperative patients. Visual acuity test can be performed in older children. Diagnosis is made when there are signs of ocular enlargement i.e. increase in corneal diameters and/ or axial length and myopic shift, or an elevated IOP. Clinicians should note that since younger babies' eyes are 'inflatable', IOP sometimes could be falsely normal while the diseased eye grows significantly bigger than the normal eye. Other signs include Haabs striae (breaks in the descemet membrane of the cornea due to a fast growing eyeball), corneal oedema (due to corneal endothelial dysfunction), increase in cup-disc ratio, and signs of associated disorders.

Treatment

Medical

Medical treatment is often the first-line treatment in paediatric glaucoma, because it is often useful in reducing IOP in the short term. However, in the longer term, surgery is the definitive treatment modality for IOP control. Topical anti-glaucomatous medications include beta-blocker, prostaglandin analogue, and carbonic anhydrase inhibitor. Topical alpha agonist should be avoided in children under the age of 3 due to its adverse effect causing apnoea. Oral carbonic anhydrase inhibitors can be used in severe cases when topical medications are not sufficient to control IOP and surgery cannot be arranged early enough due to some reasons. However, its use should not be prolonged due to various side effects such as gastrointestinal upset, hypokalaemia, renal stones etc.

Surgical

Surgery is often the definitive treatment modality in PCG and in most cases of secondary glaucoma. The basic principle of the surgery is to either open up the original drainage of the eye or to create a new drainage. Goniotomy and trabeculotomy involve the opening up of the original drainage angle so that aqueous can be adequately drained. Goniotomy is performed at an internal approach where a needle is used to enter the anterior chamber at the opposite limbus of the cornea and an incision is made along the drainage angle to open it up and facilitate drainage. This procedure requires a reasonably clear view of the anterior chamber. In patients with cloudy cornea and suboptimal view of the anterior chamber, external approach i.e. trabeculotomy can be performed.

In conditions where the patient's own drainage is not functioning properly, a new drainage should be created to facilitate aqueous outflow. These can be performed through trabeculectomy with anti-metabolite (e.g. mitomycin-C) or implantation of glaucoma drainage device. In these procedures, aqueous will be bypassed into the subconjunctival space where it will be absorbed. Complications are not uncommon especially among younger children, these include overfiltration, underfiltration, filtering bleb-related complications such as bleb leakage, astigmatism and blebitis, implant-related complications such as displacement, exposure and leakage. Post-operative management/ handling is an important part of patient/ parent education.

Laser

Diode laser cyclophotocoagulation performed either transsclerally or endoscopically, can be used in refractory cases. The laser energy is delivered to the ciliary processes causing coagulation that in turn reduces aqueous production and thus lowers the IOP. Conventionally, laser is performed through a transcleral approach, since there was no direct visualisation of the ciliary processes, the response was not easily predicted. With the introduction of endoscopic laser, the ciliary processes can be visualised directly and therefore the energy level can be titrated more effectively. However, the endoscopic laser is more safely applied in aphakic (absence of lens) or pseudophakic (presence of intraocular lens) eyes to avoid damage of the crystalline lens.

Treatment of amblyopia

Amblyopia or "lazy eye" is characterised by a decreased vision in one or both eyes due to abnormal development of vision in infancy or childhood. The brain then "learns" to see only blurry images with the amblyopic eye and as a result, the brain favours the better eye. It is the leading cause of vision loss amongst children. The risk factors of amblyopia among patients with paediatric glaucoma are refractive error (myopia resulted from long axial length; astigmatism resulted from previous surgery or corneal condition), corneal clarity (due to Haabs striae, corneal oedema etc) and glaucomatous nerve damage. The treatment of amblyopia involves the appropriate spectacle prescription as well as patching of the better eye or using mydriatic eye drops to blur the better eye so as to force the child to use the amblyopic eye.

Conclusion

Paediatric glaucoma is a rare but potentially blinding disease in infants and children. Early detection of signs and symptoms followed by a prompt treatment plan is essential in preventing irreversible visual loss. Since it is a life long disease, parents are advised that their children should have regular follow-ups and good compliant to medications in order to prevent disease progression. A close liaison with paediatricians, family physicians and clinical geneticists will often optimise patient care.

Fig. 1: A baby underwent examination under anaesthesia, showing bilateral corneal oedema and buphthalmos. (Source: American Association for Pediatric Ophthamology and Strabismus)

Fig. 2: A child with unilateral glaucoma presenting with right eye enlarged cornea and pseudo-proptosis due to longer axial length. (Source: Review of Ophthalmology)

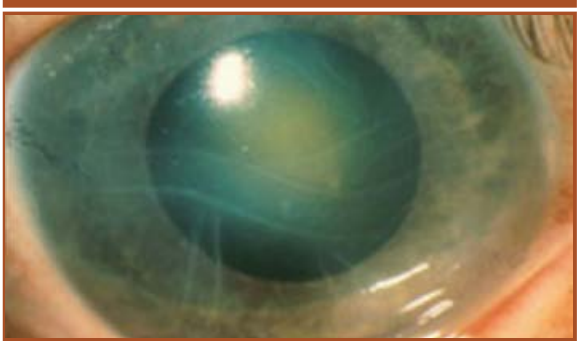

Fig. 3: Examination of the cornea showing Haabs striae and mild corneal oedema. (Source: American Association for Pediatric Ophthamology and Strabismus)

Fig. 4: Gonioscopic view during goniotomy showing the needle incising the anterior chamber angle structure and opening up the drainage angle. (Courtesy of Dr Sharon Freedman, Duke Eye Center, USA)

Fig. 5: Examination of the anterior chamber angle using a gonioscopy showing a tube of the glaucoma drainage implant.

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Radiology Quiz

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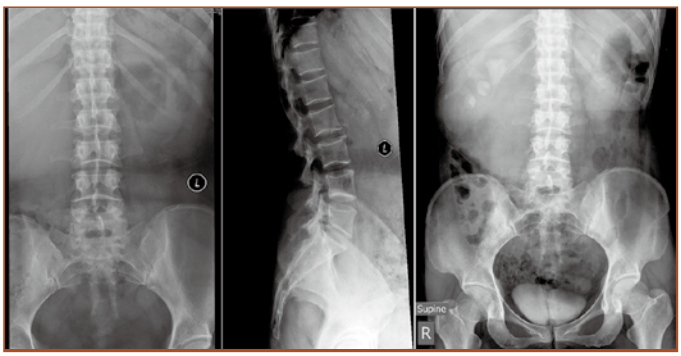

A 50 year old lady repeatedly attended the family medicine clinic for worsening low back pain.

Questions

1: What are the findings on the AP and lateral radiographs of the lumbosacral spine?
2: What are the findings from the spot radiograph of the IVU study?
3: What further imaging study would be useful for further evaluation?

(See P.25 for answers)

Age Related Macular Degeneration: Advances in Treatments

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Introduction

Age-related macular degeneration(AMD) is one of the commonest blinding disease worldwide, in particular in the developed countries.[1-4] In the United States it has been reported that up to 11.5% of the population is affected.[5] Studies in China have shown a prevalence of 3.0%-10.3% for early AMD and 0.1-1.1% for late AMD.[6,7] In Hong Kong although no exact figure has been reported, the problem is expected to be similar in magnitude or even more as Hong Kong has a predominant ethnic Chinese population with an ageing population structure.

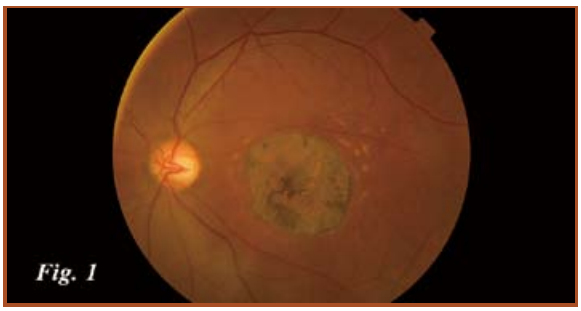

Fig. 1

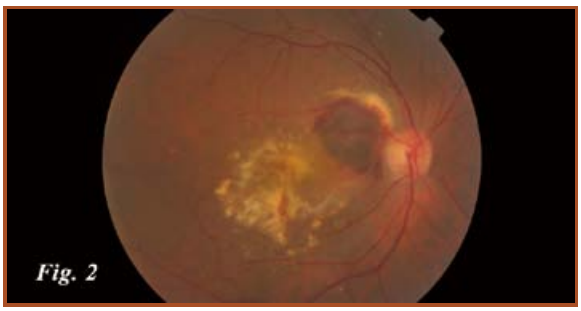

Fig. 2

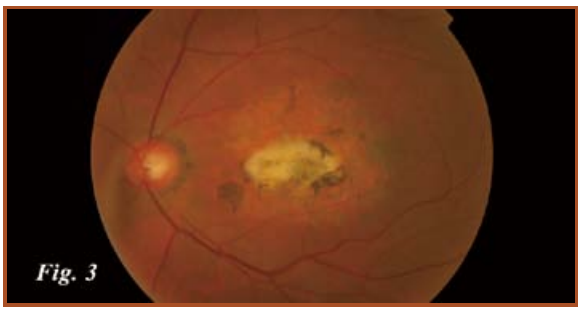

Fig. 3

AMD is well known to have two forms, the "dry" type and the "wet" type. The "Dry" type consists of a spectrum of degenerative changes with the most severe form known as geographic atrophy with accelerated atrophy but without exudation or haemorrhage. Fig.1 shows the macula of a patient suffering from the dry type with geographical atrophy in the centre. The "Wet" type, or better called neovascular age related macular degeneration, is characterised by membrane formation in the choroid, subretinal exudation, haemorrhage and eventual disciform scar formation. Fig.2 illustrates the appearance of the macula in the presence of haemorrhage and exudates due to wet AMD. Fig.3 shows an extensive scar in the centre in another patient due to late stage wet AMD.

AMD is classified based on clinical features and severity. A summary of the classification system has been charted in Table 1.[8,9] In addition, for wet AMD, when laser therapy is the mainstay treatment they are classified according to the location of the lesions from the centre of the macula.

Table 1. Categorisation of age-related macular degeneration (AMD) according to the Age-Related Eye Disease Study (AREDS).

	Brief description	Clinical features	Visual acuity
Category 1	Free of AMD in both eyes	<5 small drusen in one or both eyes	20/32 or better in both eyes
Category 2	Mild to borderline AMD	Multiple small or intermediate drusen in one or both eyes Pigment abnormalities in one or both eyes	20/32 or better in both eyes
Category 3	Absence of advanced AMD in both eyes	Intermediate or large drusen Geographical atrophy Features not involving central macular	20/32 or better in both eyes
Category 4	Advanced AMD in one eye	Advanced MAD or geographical atrophy in worse eye No such features in better eye	20/32 or better in both eyes

Clinical findings

Clinical features of AMD include reduction and distortion of central vision. In advanced cases, patients

may develop central visual field defects. Typically wet AMD patients may have more drastic changes in vision compared with dry AMD patients. Physical examination may show macula drusen, pigmentary changes, atrophic changes of the retinal pigmentary epithelium(RPE) with easy visualisation of underlying choroidal plexus in patients suffering from the dry type and subretinal fluid, pigmentary epithelial detachments, subretinal haemorrhage, lipid deposits and the characteristic choroidal neovascularisation may be seen in patients with the wet type.

Apart from clinical findings, the diagnosis of AMD depends on ocular imaging, namely, fundus fluorescein angiogram(FFA) and optical coherence tomography(OCT). FFA findings of hyperfluorescence due to dye leakage from CNV is diagnostic of wet AMD. Other features may include hypofluorescence due to blockage by subretinal haemorrhage, RPE hypertrophy, or hyperfluorescence due to window defect from RPE atrophy or staining of drusen. Optical coherent tomography is a non-invasive cross-sectional imaging modality which helps visualisation of drusen, RPE detachments, subretinal and intraretinal fluid, macula oedema and CNV. Because of its noninvasive and fast nature, it has now superseded FFA as monitoring tool for treatment response.

Pathophysiology

The exact pathophysiology of AMD is still largely unknown. However recent studies have been able to reveal part of the problems lies in the RPE/photoreceptor/Bruch's membrane complex which is responsible for the metabolism of the photoreceptors. With increase in age, the RPE function declines and there would be accumulation of deposition of material between the RPE layer and the Bruch's membrane and results in formation of drusen. Further degeneration may lead to dysfunction of the Brush's membrane, which separates the choriocapillaries from RPE. The malfunction of the Bruch's membrane together with increased levels of vascular endothelial growth factors can result in abnormal choroidal vessels growing beneath the RPE and subsequently the retina. These abnormal new vessels may result in leakage and bleeding and eventual scar formation.

Multiple signalling pathways have been thought to take part in the pathophysiology. Inflammation may be initiate by drusen formation. Drusen contains inflammatory components from the complement cascade pathway.[10] The complement factors, in particular complement factor H has been under great attention. Complement factor H is an inhibitor of the complement pathways and abnormal complement factor H will trigger complement cascade activation and inflammatory response to subretinal tissue.[11] Evidence has been found from patients with complement factor H polymorphism[11-14]. On the other hand complement factor B and complement component 2 have been found to be protective.[15] Oxidative stress is another factor implied in the pathophysiology, possibly through increases in vascular endothelial growth factor and neovascularisation. [16,17]

Treatments

There is no confirmed effective treatment for dry AMD.[18,19] On the other hand, there are various treatments developed for wet AMD in recent years focusing on the use of different anti-vascular endothelial growth factors[20,21], conclusions have yet to be drawn regarding which one is the most effective one.[22-26] However the high drug cost still poses serious implications on the treatment, as the drugs are still self-financed in various places including Hong Kong.

Due to the "non-treatable" nature of dry AMD and the high cost of treatment of wet AMD, effort has been made for disease prevention, with the aim to first improve the final visual outcome as some pathological changes may be irreversible and second, to reduce the economic burden of the health care system in the long run. Various preventive and therapeutic measures will be discussed below.

Preventive measures

Balanced diet

There have been reports that a balanced diet will be important in the prevention of AMD development. According to the Rotterdam Study, the effect of vitamin and mineral enriched diet on prevention of AMD progression has been reviewed. It has been found that for those with above-median intake of vitamin C, E, beta-carotene and zinc, there is a marked reduction of 35% risk of development of AMD.[27] This has implications in the importance of anti-oxidants in fighting against AMD. This has additional benefits as no further effort is required apart from taking a balanced diet, and would be easy to achieve.

Nutritional supplements

The Age-Related Eye Disease Study(AREDS) was a large-scale prospective study completed in 2001 with the aim to explore the effect of an active nutrient supplement to the development of advanced AMD.[28] The supplement formula contained above-normal doses of vitamins A, C, E and zinc. Results showed that there is a 25% reduction in risk of progression to advanced AMD if recommended doses of the ingredients were taken on a daily basis by high risk individuals (i.e. categories 3&4). The results were not significant for low risk groups compared with placebos. However, in later reports, there have been shown that for smokers, the risk of lung cancer would be increased due to the presence of beta carotene.[29] This leads to a subsequent revision of the formula which was tested in another trial, the Age-Related Eye Disease Study-2(AREDS2).[9] The newer AREDS formula removed beta-carotene and added in lutein, zeaxanthin, omega-3 fatty acids, hoping to enhance the protective effect against AMD progression, at the same time reducing the risk of lung cancer development among smokers taking the supplements.[8]

However, with the publication of results of AREDS2 in 2013, the new formula failed to demonstrate the additional benefit in risk reduction with lutein, zeaxanthin and omega-3 fatty acids.[8] The removal of beta-carotene probably made a safer formula for smokers or ex-smokers, nevertheless the number of lung

cancer cases in the AREDS2 trial was too small to draw such a conclusion.

It has to be noted that the benefit of 25% risk reduction was only applicable to the high risk group if the formula was taken on a daily basis. Its applicability to the general population is not certain. In addition whether the intermittent intake of such formula would have a similar effect is uncertain.[4] In addition this formula does carry side effects apart from causing lung cancers, including yellow discoloration of skin, genitourinary symptoms and self-reported anaemia, though most of them are minor and reversible.[28] These have to be discussed with patients if the supplements are to be used on a regular basis.

Apart from these quantified formulas, there have been heated debates whether some natural supplements, namely the berry extracts could have the protective effects via the anti-oxidants they contain. The most well known ones include blueberry and wolfberry. Ocular benefits mainly focus on the antioxidant anthrocyanin they contain. It is able to absorb blue-green light and protect cells from light stress.[30] In addition it has also been found to reduce AMD risks.[31-33] There have also been discussions claiming its angio-genic and anti-cancer capabilities.[34-36] However most of the findings are still preliminary and in vitro, and the potential side effects of these extracts are largely unknown. Further evidence is needed to justify their use in treatment.

Smoking cessation

Smoking is the most well established causative factor for AMD. It has been associated with increased oxidative stress, platelet aggregation, higher fibrinogen level and reduced plasma high-density lipoprotein and antioxidant levels.[37] Current evidence has shown that for smokers of 10 pack-years or more, the odds ratio for development of advanced AMD was 1.55.[38] In another study on twins, current smokers have 1.9-fold increased risk of having AMD. The risk still maintains at 1.7 fold for ex-smokers.[39] In addition if the AREDS formula has to be used, as mentioned smokers are more susceptible to develop lung cancer. Therefore smoking should be stopped for this additional reason.

Weight reduction

Dry AMD has been reported to be linked with higher body-mass-index(BMI).[2,40] An odds ratio of 1.93 was reported in obese people with BMI ≥30 kg/m^2.[2] Another study found that for those with BMI ≥30 kg/m^2, the relative risk was 2.35, and 2.32 for those with BMI of 25-29 kg/m^2.[40] The evidence has added reasons for ocular benefits for weight reduction apart from general health benefits.

Therapeutic modalities

There is no current effective treatment for dry AMD. On the other hand rapid progress has been made regarding the treatment of wet AMD in recent years, namely laser and anti-VEGF. However note has to be taken as wet AMD in general progresses faster than dry AMD and eventual scarring and fibrosis may make the central vision significant worse and sometimes unsalvageable. Prompt treatment is required to prevent these end stage changes from occurring in order to preserve vision.

Direct focal laser

The earliest trials of treating wet AMD in the 80s and 90s have been focusing on using direct focal photocoagulation via laser to damage the abnormal choroidal neovascularisation membrane(CNVM) in order to limit the formation of scar and subsequently preserve vision. The Macula Photocoagulation Study(MPS) was the first large randomised clinical trial in this aspect.[41] Nevertheless, apart from destroying the abnormal CNVM, laser also causes collateral damage of the normal retinal tissue. Therefore vision is unlikely to improve after treatment. However, this was the only treatment option available in the 80s and 90s. With better treatment modalities developed, there is now limited use of direct laser apart from pathologies outside the centre of macula. In the 90s, transpupillary thermal therapy(TTT) has also been advocated for the treatment of CNVM, but the outcomes were similar and so its use has been limited.[42]

Photodynamic therapy

Starting from the beginning of the century, a new type of treatment for wet AMD known as photodynamic therapy(PDT) has emerged. This utilises a photosensitive drug known as verteporfin. Patients would have intravenous infusion of the drug first before the eye would be treated with a special form of laser, which would only exert at special areas where the drug is photosensitised. The Treatment of Age-related Macula Degeneration with Photodynamic Therapy Study (TAP study) [43,44]and the Verteporfin in Photodynamic Therapy[45], the treatment efficacy has been confirmed. Comparing with direct focal laser, PDT was able to stop vision from deteriorating, and was able to stabilise vision if done in time. Although long-term results showed the vision would still decrease slightly before the stabilisation phase, with its reduction in collateral damage, it was accepted as a better option when compared with direct focal laser which created absolute scotoma. However, one has to be aware that restoration of lost vision is not possible with PDT alone. As a result, PDT is currently only used in some special types of AMD as a combined treatment.

Anti-vascular endothelial growth factors

In 2005, a kind of anti-vascular endothelial growth factor, bevacizumab, which was initially used for treatment of colorectal cancer, was found to be able to stabilise vision in wet AMD patients when the drug was injected intravitreally.[46] Another anti-VEGF, ranibizumab, has been studied extensively in two large-scale studies, namely MARINA and ANCHOR. The use of intravitreal anti-VEGF was also found to be not only stabilise, but also to improve vision in patients with wet AMD. The benefits in vision started after treatment initialisation and remained stable as long as repeated injections were given on a monthly basis.[47-49] It quickly emerged as the gold standard of treatment worldwide. It was first introduced in Hong Kong in 2007.

According to the original protocol of MARINA and ANCHOR trials, monthly injections of raibizumab for 24 months were required to maintain the visual outcomes. Although effective, there are always cumulative risks of intravitreal injection. The high cost of treatment may also cause problems to patients as there is no reimbursement from the government at the present moment. In order to

tackle these problems, multiple trails have been performed in search for alterative protocols with best efficacy and at the same time achieving best cost-effectiveness.
One of the most popular protocols used locally would be given an initial 3 monthly loading injections followed by monthly follow-ups and PRN re-injections, assisted with OCT imaging. This would reduce the cost of treatment and decrease the risks from repeated injections. Retreatment criteria used include 1) recent drop in vision, 2) new subretinal haemorrhage, 3) presence of new or persistence of subretinal fluid on OCT.

Long term results have been promising. 7 years after the initial MARINA and ANCHOR trials, almost half of the subjects achieved stable vision, and only about a third of the subjects had declined vision when compared with baseline.[50]

Another agent known as aflibercept has been approved by FDA for use in wet AMD patients since 2011. It is a dual agent which inhibit both VEGF-A and placental growth factor. Two related studies, VIEW 1 and VIEW 2 have shown non-inferiority of afilbercept to ranibizumab in vision maintenance at 52 weeks. It has also been suggested it may have potential benefit of 2-monthly injection during the maintenance phase while achieving similar efficacy of monthly injection of ranibizumab.[51] With similar cost per injection, this may have financial implications for the patient. Further studies are required to confirm the benefits.

Injections are done under topical or local anaesthesia, either in the operating theatre or in a sterile treatment room under strict aseptic technique. The absolute contraindications are active ocular or periocular infections, active ocular inflammation and hypersensitivity towards the drugs administered only. The injection is done through a 30-gauge needle at a distance around 3.5-4mm behind the limbus (the junction between the cornea and the sclera), and usually the temporal quadrants are chosen. 0.05mL of the drug is injected. After the injection, optic disc perfusion is checked and the patient is discharged with a course of topical antibiotics for 5-7 days at home.

Treatment is usually regarded as safe. Common side effects include conjunctival haemorrhage, transient reduction of visual acuity, eye pain and floaters. Significant ocular side effects including endophthalmitis, traumatic cataract, retinal detachment and transient increase in intraocular pressure are uncommon and occurred in less than 1 in 1000 intravitreal injections for different agents.[48,49,51] Regarding systemic side effects, the major effects would be arterial thromboembolic events, including strokes, myocardial infarction or vascular deaths. Overall rate of arterial thromboembolic events was 4.6% from MARINA and 5.0% from ANCHOR.[48,49] Data from VIEW 1 and VIEW 2 showed cardiovascular events to be 4.9% and cerebrovascular events to be 3.0% at 2 years, and the results were comparable to ranibizumab in VIEW1 and VIEW 2. [51]

Conclusion and Future Direction

Various trials are in progress for treatment of both dry AMD and wet AMD, including various neuroprotective agents, anti-inflammatory agents, immune modulating agents, radiotherapy and stem cell transplantation, aiming to retarding the pathological process or replenish neuroretinal tissue loss. Combination therapies are also in progress to achieve a synergistic effect in various parts of the pathologic pathway. Nevertheless these treatments are still under investigations and intravitreal anti-VEGF is still the most important treatment modality meanwhile.

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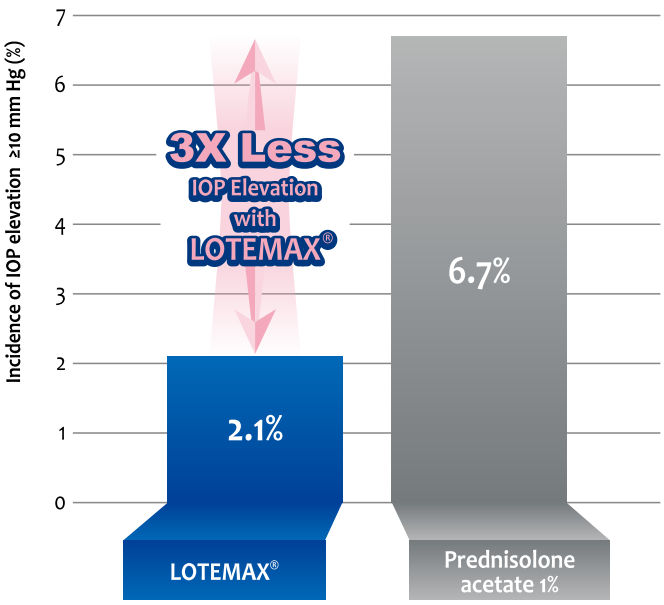

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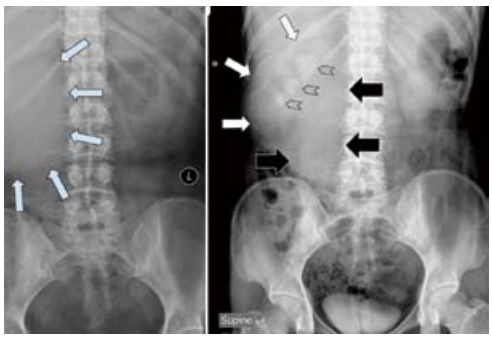
Answers to Radiology Quiz

Answer:

1. First set of X-rays revealed degenerative changes of the lumbrosacral spine. However, more importantly, asymmetric soft tissue densities were observed at the bilateral paraspinal regions, with increased soft tissue opacity at the right paraspinal region with an ill-defined right psoas shadow outline.



2. Spot radiograph from IVU revealed a large right paraspinal soft tissue mass. (Black arrows) Loss of fat plane with the right psoas shadow and displacement of the right kidney (white arrows) was suggestive of a retroperitoneal lesion. Right hydronephrosis was also seen (grey arrowheads), suggestive of right ureteric compression.

3. Further workup with contrast CT is warranted. Subsequent contrast CT revealed a large right retroperitoneal mass with renal and psoas invasion.

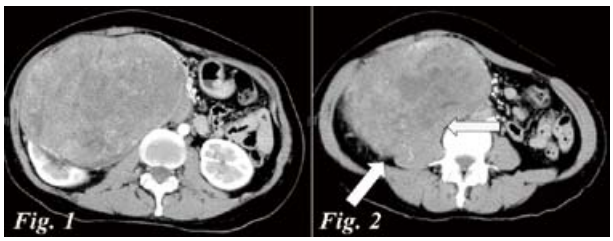


Fig.1 Contrast CT contained a large RT retroperitoneal mass with rental invasion.

Fig.2 Rt psoas shadow invasion with loss of fat plane (white arrows) is in line with plain radiograph findings.

The patient subsequently underwent radical nephrectomy. Pathology revealed undifferentiated leiomyosarcoma.

Discussion:

It is important to evalaate presence of extra-skeletal pathologies in all imaging studies, in particular the presence of asymmetrical or abnormal soft tissue densities. Never fall under the trap of satisfaction of search.

The psoas shadows are a useful aid in the diagnosis of various intra-abdominal and retroperitoneal lesions.

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Childhood myopia: update on effective prevention

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This article has been selected by the Editorial Board of the Hong Kong Medical Diary for participants in the CME programme of the Medical Council of Hong Kong (MCHK) to complete the following self-assessment questions in order to be awarded 1 CME credit under the programme upon returning the completed answer sheet to the Federation Secretariat on or before 31 August 2016.

Introduction

Myopia, (near-or short-sightedness), is one of the commonest human eye diseases and often has its onset during childhood. It is a spherical refractive error caused by excessive refractive power and/ or axial lengthening of the eye. As a result, the light rays entering the eye focus in front of, rather than, on the retina.[1] Nowadays, myopia has become a major health issue worldwide, especially in East Asia. The uncorrected refractive error is one of the major causes of visual impairment[2] and causing significant productivity loss with huge economic burden.[3]

Prevalence in children

Prevalence of myopia is increasing all over the world in many populations, with ethnic predominance in East Asians. In a study conducted in Australia, the prevalence of myopia were 42.7% and 59.1% in 12-year-old and 17-year-old school-aged children of East Asian ethnicity respectively, whereas the corresponding prevalence rates in European Caucasian children of the same age were 8.3% and 17.7% respectively.4 Prevalence of myopia in East Asian and Southeast Asian countries including Southern China and Hong Kong is now as high as 80%-90% in those completing high school, compared with 10% to 30% 50 to 60 years ago.[5] In Hong Kong, the prevalence of myopia in preschool children is also on the rise, increasing significantly from 2.3% to 6.3% over a ten-year period.[6]

High Myopia & Complications

High myopia is defined as ≤-6.0 dioptres (D). Unlike mild to moderate myopia, which is often benign, high myopia is associated with characteristic degenerative changes of the sclera, retinal pigment epithelium and choroid leading to potential blinding complications, including maculopathy,[7,8] choroidal neovascularisation, retinal detachment,[9] glaucoma,[10] and cataract.[11,12] The degenerative changes are principally thought to be related to mechanical stretching of the involved tissue as the eyeball lengthens. Table 1 summarises the possible complications associated with high myopia. With the increasing prevalence of high myopia and thus its associated complications, prevention of myopia progression at childhood becomes an important public health target.

Table 1: Complications associated with high myopia

Lens
Cataract
Dislocation of lens
Glaucoma
Chorioretinal abnormalities
Chorioretinal atrophy
Fuch's spot
Lacquer cracks
Staphyloma
Lattice degeneration
Retinal detachment
Choroidal Neovascularisation
Optic disc abnormalities
Tilted disc
Peripapillary atrophy

Table 2: Interventions for preventing myopia progression

1) Outdoor Activity
2) Pharmaceutical Agents
• High Dose Atropine (1%)
• Low Dose Atropine (0.01%-0.1%)
3) Contact Lenses
• Orthokeratology
• Rigid gas-permeable contact lenses
• Soft Contact Lenses
4) Spectacle Lenses
• Under corrected single visual spectacle lens
• Bifocal/Multifocal spectacle lenses
• Progressive addition spectacle lenses

Cause of Myopia

The exact cause of myopia remains to be elucidated. There are, however, many postulated theories, with genetics and environmental factors being two most important aspects. Results from both animal studies and clinical trials support the importance of environmental risk factors. Some studies reported that intensive near work, such as reading and writing, would lead to increased accommodation, which could result in myopia development.[13] On the other hand, increased outdoor time was found to have a protecting effect against the development of myopia.[14]

Another important factor for myopia is genetic factor. Children with myopic parents have a higher prevalence of myopia demonstrated in many studies.[15] It is now generally agreed that major genetic contributions to

high myopia exist. A number of chromosomal locations have been identified to be associated with high myopia, such as MYP1-MYP17. [16]

Prevention of Myopia Progression

A summary of different methods of intervention is shown in Table 2.

1) Outdoor activity

Recent studies demonstrated an association between increased time spent in outdoor activities and a reduced risk of myopia onset and progression. [17,18,19,20,21] One study in 2007[18] examined 514 children and found that comparing with children who remained non-myopic, those children who became myopic had significantly less time spent in outdoor activities. A similar result was shown in another subsequent study.[21] The strongest evidence of the protection effect from outdoor activities on myopia development came from a recent randomised control trial,[22] which is consisted of 6 intervention schools (952 students) and 6 control schools (951 students). One additional 40-minute class of outdoor activities was added in each school day to the interventional schools, comparing to those who continued their usual pattern of activity in the control schools. The 3-year cumulative incidence rate of myopia was reduced in the intervention group (30.4%) comparing to the control group (39.5%) ($p<0.001$).[22] However, the exact mechanism of outdoor activity on myopia prevention is still not fully understood. Many mechanisms have been postulated, which include 1) pupil constriction leading to less visual blur; 2) stimulation of the retinal release of dopamine, which is an eyeball growth inhibitor; and 3) high vitamin D serum levels which have been shown to be associated with less myopia.[23]

2) Pharmacological agent

Atropine is a nonselective muscarinic receptor antagonist and is now the most commonly used drug in slowing myopia progression. The postulated mechanism includes inhibition of accommodation, biochemical remodelling of sclera, and increase ultraviolet exposure secondary to pupil dilatation. Different concentrations of atropine eye drops have been investigated in randomised control trials. The Atropine in the Treatment Of Myopia (ATOM) study was a randomised, double-masked, placebo-controlled trial involving 400 Singaporean children. This study showed that the instillation of 1% atropine eye drops (Fig. 1) nightly in 1 eye over a 2-years period significantly reduced myopia progression by 77% (0.28D vs. 1.2D in control vs. atropine groups). Axial length elongation was also significantly reduced with this regimen (0.38+0.38mm vs. -0.02+0.35mm in control vs. atropine groups). However, discontinuation of treatment was shown to cause a partial rebound effect. Despite this, this regimen was still shown to reduce myopia progression significantly by 35% over a 3-years period (2 years atropine treatment, one year no treatment). The main side effects of atropine include photophobia due to mydriasis and decreased near vision due to cycloplegia. As a result, patients on treatment were required to wear photochromatic, progressive additional lenses. No systemic side effect related to atropine was reported but postulated side effects include dry eye, dry mouth, dry throat, flushed skin, constipation and difficulty in micturition. Other ocular side effects, e.g. allergic conjunctivitis, contact dermatitis of the eyelids were relatively uncommon. There was no significant retinal dysfunction 3 months after cessation of the eye drops. However, the ocular side effects mentioned above prohibited wide adoption of atropine use internationally.

Fig. 1: 1% Atropine Eye drop

Fig. 2: 0.01% Atropine Eye drop

In a subsequent ATOM2 study evaluating 0.5%, 0.1% and 0.01% in a total of 400 children,[24] the investigators found 0.01% atropine (Fig. 2) had much less side effects of cycloplegia and mydriasis than other doses, and still had significant effects on slowing down the increase of axial length and the progression of spherical equivalent refraction. The mean progression at 2 years was -0.49±0.63D in the 0.01% group, -0.30+0.6D in the 0.5% group and -0.38±0.6D in the 0.1% group. The mean increase in axial length was 0.27±0.25, 0.28±0.28, and 0.41±0.32mm in the 0.5%, 0.1% and 0.01% group. Furthermore, the rebound effect after cessation of drop was found to be greater in higher dose (0.5% and 0.1% group) than 0.01% group.[25] Therefore, compared with higher low dose atropine eye drops, nowadays, 0.01% atropine is the preferred option as the prevention of myopia.

3) Orthokeratology and other types of contact lenses

Orthokeratology (OK) is a type of contact lens that temporarily reshapes the cornea. It flattens the central cornea and steepens the mid-peripheral cornea to reduce relative peripheral hyperopia. Unlike other contact lenses, OK lenses are worn overnight by children, and are removed upon awakening. It is therefore particularly appealing to parents who want their children to be spectacle-free during the day. OK lenses have been reported to be able to slow down the axial length elongation,[27,28] with the following postulated mechanisms: (i) The change of the peripheral refraction towards a relatively peripheral myopic condition, which takes visual signals from the peripheral retina. These signals are essential for the regulation of vision, which influences the ocular growth. (ii) By moving the image forward at the peripheral retina, it leads to a multifocal image myopically defocused which may have an effect on slowing ocular growth. (iii) Changes in lens thickness leading to changes in the accommodative function of eyes.[29]

Unfortunately, complications, some of which are serious and blinding, e.g. recurrent corneal erosion, infective keratitis,[30,31,32] induced corneal astigmatism, corneal pigmentation, etc. are associated with the use of OK lenses. The risks associated with OK lenses must be carefully weighed against the possible benefits. Parents should be counselled adequately and made aware of these potentially blinding complications that may arise from its use.

Other types of contact lenses such as rigid gas-permeable contact lenses and conventional soft contact lenses have also been studied on myopia control. Currently the evidence on their efficacy on myopia control remained small.[33]

4) Spectacle lenses

Previous studies on under-correction with spectacles were found to be not effective on myopia control with contradicting results. Therefore, wearing spectacles with single vision lenses is not considered as an option for myopia control currently. On the other hand, spectacles with bifocal or multifocal lenses, progressive addition lenses and peripheral defocus modifying lenses are effective in reducing defective accommodative effort, which can produce hyperopic retinal blur and lead to the onset or the progression of myopia.[34,35] These methods were thought to prevent potential aberrant eye growth.

Conclusion

Myopia in children is a rising threat worldwide, especially in East Asia including Hong Kong. High or pathological myopia gives rise to potentially blinding complications that will be a burden to the health care system as well as the economy. Therefore, it is increasingly important to take interventions to prevent the onset and progression of myopia. To date, the most promising methods are increasing outdoor activities and the use of atropine eye drops. In particular, the use of low-dose atropine (<0.1%) should be the emerging trend of ophthalmological practices in myopia control. The use of orthokeratology, progressive addition spectacle lenses soft contact lenses may have some effects on myopia control but with potential complications.

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MCHK CME Programme Self-assessment Questions

Please read the article entitled "Childhood myopia: update on effective prevention" by Dr Jason C.S. YAM and Ms Yuning JIANG and complete the following self-assessment questions. Participants in the MCHK CME Programme will be awarded 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 August 2016. 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. **High myopia is defined as ≤-4.0 dioptres (D).**
2. **Both genetic and environmental factors are risk factors of myopia.**
3. **Refractive error is only caused by excessive refractive power of the lens.**
4. **Complications of high myopia are caused by the elongation of the eyeball.**
5. **High myopia may lead to potential blinding complications like glaucoma, cataract and retinal detachment.**
6. **Increasing time spent in outdoor activities has a protective effect on myopia development.**
7. **Atropine is a selective muscarinic receptor antagonist drug.**
8. **Low dose atropine (0.01%) is a preferred option for preventing myopia progression.**
9. **Orthokeratology is a type of contact lens that reshapes the cornea.**
10. **Use of OK lenses may lead to complications such as recurrent corneal erosion and infective keratitis.**

ANSWER SHEET FOR AUGUST 2016

Please return the completed answer sheet to the Federation Secretariat on or before 31 August 2016 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Childhood myopia: update on effective prevention

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

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

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

Contact Tel No.:_____ MCHK No.: _____ (for reference only)

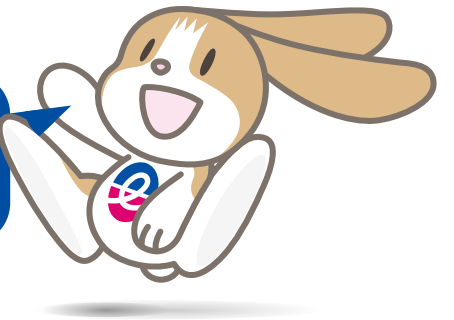
Answers to July 2016 Issue

Robotic Hair Transplant

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

Santen

Do you know the SCIENCE behind each signature Santen Dimple Bottle?



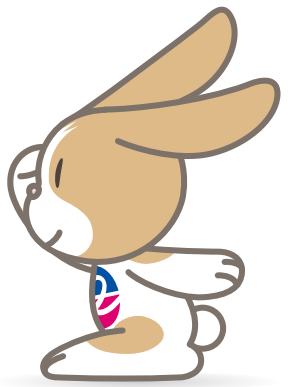
The shrink film label with a pull-tab is easy to remove.

The cap can be opened by being rotated just once. Also, the ten-cut cone-shaped cap will not roll over easily.

The product name is displayed in large fonts.

The dimples on both sides make the container easy to hold. The bottle is also soft and easy to press.

Transparent slots on both sides of the bottle enable users to check the amount of the remaining solution at a glance.



Developing Eye Drops Valued by Patients and Medical Professionals

Santen's researchers developed Dimple Bottles that won the Good Design Award in 2008. After launching the product, Santen received favorable responses from many users. They can instill the liquid without pressing the bottle hard, and there is no overflow from the eye. This bottle is extended to majority of our Santen products. Check out the full Saten portfolio with your local Santen sales representatives!

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For Healthcare Professionals Only

Shoes

Prof Jimmy LAI
HKU Professor

Prof Jimmy LAI

Photo 1: Oversized outsole shoes

I pick the topic on shoes because of the adage by John Wildsmith: you are either in your bed or in your shoes so it pays to invest in both". The earliest use of shoes was around 10,000 years ago which was evidenced by paintings found in caves. The invention of shoes designed to fit right and left feet came in the 19th century and that greatly improved comfort. I have done a small survey on the reasons of wearing shoes and 100% respondents claimed protection of feet from injury. I wear shoes for fashion in addition to protection. As an ophthalmologist, I look at the delicate eye structures. That may or may not explain why I pay attention to fine details of shoes. Like our eyes that occupy a small area of our head, our feet occupy a small area of the lowest part of our body. A pair of shoes with flamboyant colorations is not as exaggerating as a colourful shirt. This gives me the courage to try out eye catching shoes admittedly with properly matched garment. I am not a handsome man so I rather have others' attention on my shoes than on my face. Shoes also boost my confidence by giving me an extra 8 to 10cm height (Photo 1). Although I appreciate the work of shoe craftsman, I have no preference for hand-made shoes. Moreover, I like ready-to-wear shoes for immediate possession. I have no patience to wait a few months and I do not have peculiar aesthetic taste or unusually shaped feet for Bespoke shoes. I am a loyal follower of some of these brands: Alexander McQueen, Balenciaga, Belly Burton, Bottega Veneta, Burberry, Burluti, Corthay, Chanel, Christian Louboutin, Dior, Edward Green, Fendi, Giuseppe Zanotti, Gucci, Jimmy Choo, John Lobb, Louis Vuitton, Prada, Salvatore Ferragamo (Tramezza). I must declare no financial interest to all the above brands and my comments are based on personal feelings. Among these brands, I think Louis Vuitton produces the most diversified type of shoes including trainers, sneakers, boots and classical shoes that are elegant, classy and fashionable. This surely satisfies shopaholic like me. If you like to stray away from the conventional design of men's shoes, Christian Loboutin maybe the brand to go for. The company produces standout shoes with the designer's signature red sole and heavy embellishments that are good for stage show. Giuseppe Zanotti is another brand that produces glitter-encrusted leather shoes and sneakers with metallic foldover bar. If you prefer modest but on-trend shoes, Jimmy Choo and Axexander McQueen are good choices (Photo 2). Berluti shoes are in general pointy. Honestly they are not as comfortable as I once thought. Nevertheless they are stylish and come in a variety of colours with delicate shading and calligraphy etching. The colours are so distinctive that every pair of shoes is unique. Berluti provides after-sale patina service in case you are bored with the colour tone of your pair.

Photo 2: Heavy embellishments shoes

The most comfortable classic shoes from my experience are the Tramezza line of Salvatore Ferragamo. For trainers and sneakers, Bottega Venetta wins. These are just personal feelings and I have not explored into the shoemaker's knack of how this is achieved. John Lobb in my collection ranks second in terms of comfort. For Corthay and Edward Green I have to buy their Bespoke shoes because their ready-to-wear shoes rarely fit the size of my feet (Photo 3). I used to visit Edward Green shop in London to place my order. The shoes were tax exempted and were delivered to my home address in 3 months. Corthay shoes have similar style as Berluti's and they also have attractive patina but without being over the top. I have heard of other famous brands like Aubercy and Artioli but up till the time of writing I had not been able to visit their shops. Adidas and New Balance are my outfits for outdoor and indoor sports and Dr Martens, an extremely durable footwear, is my preference for hiking and snow walking.

7/8Days Tasmania

Roundtrip from Sydney

Emerald Princess | Dawn Princess

Sydney, Australia | Melbourne, Australia | Wineglass Bay & Oyster Bay, Australia [Scenic Cruising] | Hobart (Tasmania), Australia | Port Arthur (Tasmania), Australia | **Sydney, Australia**

12Dec2016
2*, 29^ 🧧 Jan2017

HKD9,079up

*This is an 8 eight days sailings, will stay overnight in Melbourne

^Port order may vary. Burnie replaces Wineglass Bay & Oyster Bay and stay overnight in Hobart.

13Days New Zealand

Roundtrip from Sydney

Sun Princess | Emerald Princess | Dawn Princess

Sydney, Australia | Fjordland National Park [Scenic Cruising] | Dunedin (Port Chalmers), New Zealand | Akaroa, New Zealand | Wellington, New Zealand | Napier, New Zealand | Tauranga, New Zealand | Auckland, New Zealand | Bay of Islands, New Zealand | **Sydney, Australia**

4Oct | 15, 26Nov | 9Dec2016
13, 16 🧧 Jan | 5, 18Feb |
3, 18Mar2017

~~**HKD15,201**~~up

HKD13,329up

10Days Panama Canal

Roundtrip from Ft. Lauderdale

Coral Princess | Island Princess

Ft. Lauderdale, Florida | Aruba | Cartagena, Colombia | Panama Canal [Scenic Cruising] | Colon, Panama | Limon, Costa Rica | Ocho Rios, Jamaica | **Ft. Lauderdale, Florida**

Oct2016-Apr2017

HKD11,160up

14Days Cape Horn

Sail between Santiago (Valparaiso) to Buenos Aires

Crown Princess

Santiago (Valparaiso), Chile | Puerto Montt, Chile | Amalia Glacier, Chile [Scenic Cruising] | Punta Arenas, Chile | Ushuaia (Tierra del Fuego), Argentina | Cape Horn [Scenic Cruising] | Falkland Islands (Stanley) | Puerto Madryn, Argentina | Montevideo, Uruguay | **Buenos Aires, Argentina**

18Jan 🧧 | 1*, 15Feb2017

HKD15,943up

*This is a reverse sailing, Punta Del Este replaces Montevideo

15 days Sailing between Ft. Lauderdale and Los Angeles itineraries are also avaliable:

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Photo 3: Various brands

I only fancy lace-up shoes, trainers, sneakers, ankle boots and selected buckle classic shoes. I never buy loafers. Besides the sense of security after lacing up, I consider shoe laces and socks adornments. Heedful selection of shoe laces and socks will add charisma to one's semblance. I never wear sandals because I regard toes the ugliest parts of the body. I never expose them in the public except when swimming or bathing in Japanese onsen.

All have been said with men's footwear but manufacturers for ladies shoes also have lines that are neutral. Women's flat shoes, sneakers and trainers are in general more ornate and exquisite than men's footwear (Photo 4). Because they are narrower in design, they have to be oversized to fit my feet. Oversized women's shoes also make them look more masculine.

Photo 4: Ladies footwear

Footwear can be made from leather of calf, lamb, suede, python, alligator, ostrich, lizard, stingray. I have tried shoes made of eel skin but they are not durable. Alligator shoes are peculiar because the rounded or irregularly squared scales make each pair of alligator shoes look unique. In Europe, when you purchase footwear made of exotic leather from alligator and python, the shop will issue a CITES certificate for re-export of the leather. (Convention on International Trade in Endangered Species of Wild Fauna and Flora). The certificate contains the name of the species and country of origin. It certifies that specimen was legally imported. Customs in certain countries may require inspection of the certificate for re-export of the footwear. The Convention was signed in 1973 and entered into force in 1975. All European Union Member States are parties to the Convention. The purpose of CITES is to ensure that no species of wild fauna is subject to unsustainable exploitation because of international trade.

Photo 5: Cleaning midsole with brush and detergent

Lastly I always remember caring for my shoes after bringing them home. Proper care of shoes will prevent leather on them to become dry, hardened, scuffed and cracked. Different leathers require different cleaning and polishing processes. It would be impossible for one to memorise all the shoe care guides. Sending footwear to expert shoe care service is a wise decision. However, a basic cleaning procedure after use is important. Simply take a soft cloth to wipe away any dust or debris on the shoe. Apply shoe conditioner to moisturise and shoe wax to protect the leather at times. Suede is particularly hard to clean. Fortunately suede brush and eraser are available for such purpose. For sneakers and trainers that have white midsoles, brushing with detergent and rinsing with tap water should be done to remove stains (Photo 5). Always remember to wash the white shoe laces of sneakers and trainers as well. Let the shoes air dry for 24 hours before putting into shoe bags and shoe boxes. Shoetrees are crucial in shoe care. They deodorise the shoes, draw moisture out of the leather and allow recently worn shoes to dry out and contract to their natural architecture (Photo 6). Humidity is a threat to the leather of shoes. It can lead to mould growing on the leather that will leave permanent stain and foul smell. Turning on dehumidifier and air-conditioner is the solution when the weather is warm and humid. Shoe deodoriser aerosol spray is essential for smelly shoes.

Photo 6: Shoetrees

Shoes are intimate friends of the feet. Good shoes are made with great leather. A good pair of shoes fit like gloves. Together with matching attire, an eye catching pair of shoes will bring out your personal silhouette and elegance without reserve. Once you have invested in a quality pair, put a little time and effort into looking after them and they will last a long time.

Co-organizers

The Federation of Medical Societies of Hong Kong

THE HKFMS FOUNDATION LIMITED
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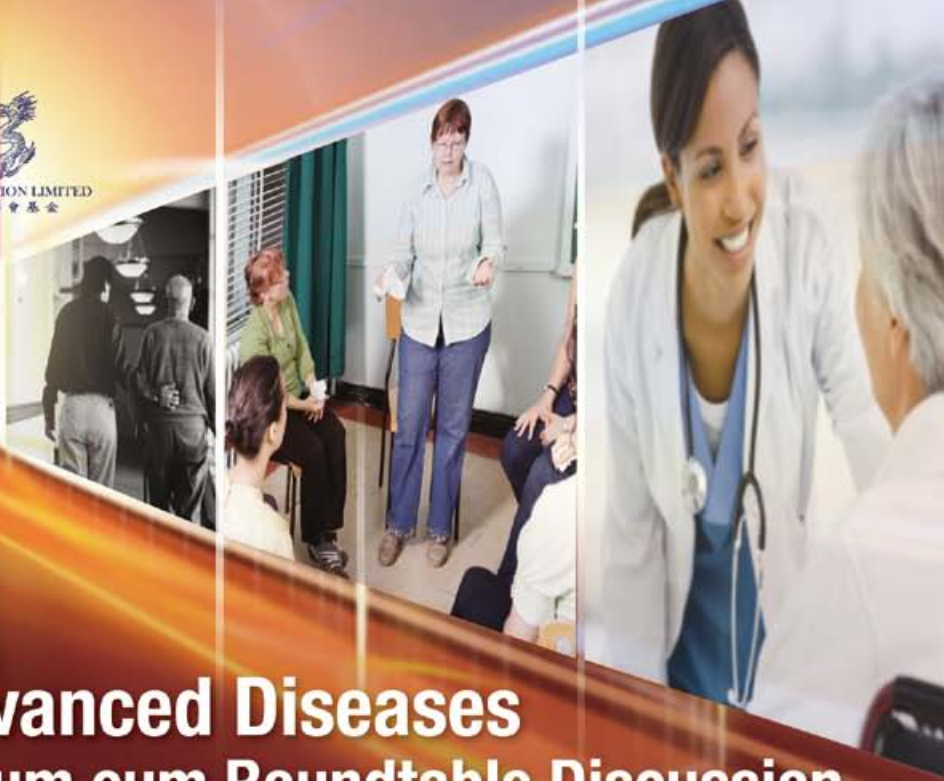

Care for Advanced Diseases
CME Symposium cum Roundtable Discussion

Dates : 27 Aug 2016 (Sat)
Time : 3:00pm – 6:15pm
Venue : Lecture Hall, 4/F, Duke of Windsor Social Service Bldg., 15 Hennessy Road., Wanchai, Hong Kong
Language Medium: Cantonese (Supplemented with English)

Rundown

2:45pm	**Registration**
3:00pm	**Chairperson: Dr Mario CHAK,** ***President of The Federation of Medical Societies of Hong Kong***
	Application of Principles and Philosophy of Palliative Care Dr Raymond LO, ***Immediate Past President of The Federation of Medical Societies of Hong Kong***
	Pain Control in Advanced Cancer Dr YUEN Kwok-keung, ***Chairman of Hong Kong Society of Palliative Medicine***
	Psychological Support for Patients and Families at the End of Life Dr Theresa LAI Tze-kwan, ***Chairperson of Hong Kong Palliative Nursing Association***
	Advance Care Planning and Advance Directives Dr TSE Chun-yan, ***Honorary Advisor of Hong Kong Society of Palliative Medicine***
	Q&A
4:45pm	**Registration & Tea Reception**
5:15pm	**Roundtable Discussion**
6:15pm	**End of Programme**

Remarks: FREE registration. All are welcome. RSVP by fax to 2865 0345 or email to him@fmshk.org on or before 19 August 2016 (Fri) as seats are limited. Reply form can be downloaded from website: http://www.fmshk.org

Enquiry: Mr Him Fung at 2527 8898 or him@fmshk.org

Accreditations: CME/CNE has been applied and pending confirmation

Survey result on「香港寧養紓緩服務」would be announced during the event

Medical Diary of August

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
	1	★ HKMA Council Meeting 2	3	4	5	6
★ HKMA Dragon Boat Fun Day 7	8	★ HKMA Kowloon West Community Network - New Challenges of Erectile Dysfunction Management 9	★ HKMA Central, Western & Southern Community Network - Current Management of Meniere's Disease 10	★ HKMA Kowloon East Community Network - Update Management on Psoriasiform Dermatosis in Clinical Practice ★ Certificate Course on Diabetes Mellitus (Session 3) - Management of DM Complications ★ HKMA New Territories West Community Network - Latest Update in GERD Management ★ HKMA Structured CME Programme with HKS&H Session VII: Biologic Therapies in the Management of Psoriasis 11	12	13
14	15	16	17	★ HKMA Hong Kong East Community Network - The Latest Updates on the Management of Atrial Fibrillation – Real Life Evidence on NOACs ★ FMSHK Officers' Meeting 18	★ HKMA Central, Western & Southern Community Network - Topic 1: Clinical Wisdom in Managing Patient Presenting with Acute Chest Pain Topic 2: Management Update on Acute Coronary Syndrome 19	★ HKMA KECN, HKCFP & UCH – CME Course for Health Personnel 2016 (Session 3) - Management of Blurred Vision 20
21	22	23	24	★ HKMA Kowloon East Community Network - Better LUTS Management, Better Days for Your Patients ★ FMSHK Executive Committee Meeting ★ FMSHK Council Meeting 25	26	27
28	29	30	31			

Date / Time	Function	Enquiry / Remarks
2 TUE 9:00 PM	**HKMA Council Meeting** Organiser: The Hong Kong Medical Association; Venue: HKMA Wanchai Premises, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong	Ms. Christine WONG Tel: 2527 8285
7 SUN 2:00 PM	**KMA Dragon Boat Fun Day** Organiser: The Hong Kong Medical Association; Chairman: Dr. YAM Chun Yin; Venue: Sai Kung	Miss Denise KWOK Tel: 2527 8285
9 TUE 1:00 PM	**HKMA Kowloon West Community Network - New Challenges of Erectile Dysfunction Management** Organiser: HKMA Kowloon West Community Network; Chairman: Dr. CHAN Siu Man, Bernard; Speaker: Dr. YIP Wai Chun, Andrew; Venue: Crystal Room IV-V, 3/F, Panda Hotel, 3 Tsuen Wah Street, Tsuen Wan, NT	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
10 WED 1:00 PM	**HKMA Central, Western & Southern Community Network - Current Management of Meniere's Disease** Organiser: HKMA Central, Western & Southern Community Network; Chairman: Dr. TSANG Chun Au; Speaker: Dr. CHOW Chun Kuen; Venue: HKMA Central Premises, Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
11 THU 1:00 PM	**HKMA Kowloon East Community Network - Update Management on Psoriasiform Dermatosis in Clinical Practice** Organiser: HKMA Kowloon East Community Network; Chairman: Dr. AU Ka Kui, Gary; Speaker: Dr. HO Ka Keung; Venue: Lei Garden Restaurant, Shop no. L5-8, APM, Kwun Tong, No. 418 Kwun Tong Road, Kowloon	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
1:00 PM	**Certificate Course on Diabetes Mellitus (Session 3) - Management of DM Complications** Organiser: HKMA-HK East Community Network; Chairman: Dr. LEUNG Kwan Kui, Terence; Speaker: Dr. TING Zhao Wei, Rose; Venue: Empire Grand Room, 1/F, Empire Hotel Hong Kong, Wan Chai, 33 Hennessy Road, Wanchai	Ms. Candice TONG Tel: 2527 8285 1 CME Point
1:00 PM	**HKMA New Territories West Community Network - Latest Update in GERD Management** Organiser: HKMA New Territories West Community Network; Chairman: Dr. TSUI Fung; Speaker: Dr. LI, Ernest Han Fai; Venue: Plentiful Delight Banquet, 1/F, Ho Shun Tai Building, 10 Sai Ching Street, Yuen Long	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
2:00 PM	**HKMA Structured CME Programme with HKS&H Session VII: Biologic Therapies in the Management of Psoriasis** Organiser: The Hong Kong Medical Association & Hong Kong Sanatorium & Hospital; Speaker: Dr. CHAN Chun Yin, Johnny; Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	HKMA CME Dept. Tel: 2527 8452 1 CME Point
18 THU 1:00 PM	**HKMA Hong Kong East Community Network - The Latest Updates on the Management of Atrial Fibrillation – Real Life Evidence on NOACs** Organiser: HKMA Hong Kong East Community Network; Chairman: Dr. WONG Chun Por; Speaker: Dr. CHAU Mo Chee, Elaine; Venue: Empire Grand Room, 1/F, Empire Hotel Hong Kong - Wan Chai, 33 Hennessy Road, Wanchai	Ms. Candice TONG Tel: 2527 8285 1 CME Point
8:00PM	**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. Nancy CHAN Tel: 2527 8898

Date / Time	Function	Enquiry / Remarks
19 FRI 1:00 PM	**HKMA Central, Western & Southern Community Network - Topic 1: Clinical Wisdom in Managing Patient Presenting with Acute Chest Pain Topic 2: Management Update on Acute Coronary Syndrome** Organiser: HKMA Central, Western & Southern Community Network; Chairman: Dr. YIK Ping Yin; Speaker: 1. Dr. TSANG Tat Chi 2. Dr. CHAN Hon Wah, Raymond; Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
20 SAT 1:00 PM	**HKMA KECN, HKCFP & UCH – CME Course for Health Personnel 2016 (Session 3) - Management of Blurred Vision** Organiser: HKMA Kowloon East Community Network & Hong Kong College of Family Physicians & United Christian Hospital; Chairman: Dr. AU Ka Kui, Gary; Speaker: Dr. CHUNG Chung Yee, Derek; Venue: 1. Lecture Theatre, G/F, Block K, United Christian Hospital (UCH), 130 Hip Wo Street, Kwun Tong, Kowloon 2. Conference Room, G/F, Block K, UCH (video conference)	Miss Hana YEUNG Tel: 2527 8285 1.5 CME Point
25 THU 1:00 PM	**HKMA Kowloon East Community Network - Better LUTS Management, Better Days for Your Patients** Organiser: HKMA Kowloon East Community Network; Chairman: Dr. MA Ping Kwan, Danny; Speaker: Dr. CHUNG Yeung, Vera; Venue: V Cuisine, 6/F., Holiday Inn Express Hong Kong Kowloon East, 3 Tong Tak Street, Tseung Kwan O	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
7:00 PM	**FMSHK Executive Committee Meeting** Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
8:00 PM	**FMSHK Council Meeting** Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898

Upcoming Meeting

31/8/2016 – 2/9/2016	**Medical Fair Asia 2016** Organiser: Messe Duesseldorf Asia; Venue: Marina Bay Sands, Singapore	Ms Cathy Ng Tel: 2143 2281
4/9/2016 8:50AM-5:00PM	**Li Shu Pui Symposium 2016 – Ambulatory Medical Practice** Organiser: Hong Kong Sanatorium & Hospital Venue: Ballroom, JW Marriott Hotel Hong Kong, Pacific Place, 88 Queensway, Hong Kong	2835 8800 Website: www.hksh.com/lsp-registration
8-9/10/2016	**The 9th Hong Kong Allergy Convention - Novel Strategies for Prevention and Treatment of Allergic Disorders** Organiser: Hong Kong Institute of Allergy; Venue: Hong Kong Convention and Exhibition Centre	HKAC 2016 Secretariat Tel: 2559 9973

THE FEDERATION OF MEDICAL SOCIETIES OF HONG KONG
香 港 醫 學 組 織 聯 會

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For enquiry and booking, please contact the Secretariat at 2527 8898.
http://www.fmshk.org/fmshk.html?id=35