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New Edition of Medical and Dental Directory
Submit your data NOW! (Refer to Page 9)

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The Cover Shot

Water reflection is always amazing. Early morning is the best time for this kind of shot when the water is calm. The autumn leaves have transformed the scenery into a masterpiece of colour. This picture was taken on 25th Oct., 2005 at Jiu Zhau Gau 九寨溝 at about 8am.

Nikon D100, Focal length 28mm, F5.6, 1/125sec.

Dr. Raymond HS LAM
MBBS, DO, FCOphth.HK, FCSHK, FHKAM(Ophthalmology)
Specialist in Ophthalmology

New Year Message from the President

Dr. Raymond SK LO

President
The Federation of Medical Societies of Hong Kong

Dr. Raymond SK LO

Time flies and we are stepping into another bright new year. On behalf of the Federation of Medical Societies of Hong Kong, may I wish you a happy, healthy and prosperous year of 2013.

The year 2012 has been another fruitful and productive year for the Federation. I am delighted to report that with your support and the concerted efforts of our Exco and Council, the Federation has continued to flourish and serve you better. To date, our membership has steadily increased to 132 professional societies, with the newest associate member being the Hong Kong Council of Social Service. This would serve as a good opportunity for a wider collaboration between the medical and the social sector, especially for the support of our affiliated Foundation projects.

As the umbrella organisation of medical, dental, nursing and allied health societies of Hong Kong, the Federation endeavours to promote the networking and fraternity of our members. We would like to invite our existing member societies, new and old, to update us on their activities and development in our Medical Diary. Further, our programme with RTHK radio one will be resumed again due to popular demand, and please tune in to hear the latest progress from our member societies on every Thursday lunch time at 1:30 pm. Our ninth edition of the Medical and Dental Directory will be in press early this year, serving as a useful reference for referrals.

Meanwhile, the Federation will continue to strengthen her support on member services. We have recruited an extra staff to assist member societies with secretariat needs and conference/seminar preparation. For the education role, we shall continue to collaborate with more partners for a wider perspective. Last year, we have co-organised a joint scientific meeting with Macau colleagues, which was attended by over 800 delegates. More certificate courses, public health talks and tailored-made seminars had also been held. We look forward to even more joint educational events with members and professionals this year.

As for the advocacy role, the Federation has been active, in expressing our members' viewpoints on recent professional issues and public concerns, through responding to consultative documents, nominating representatives to government committees and Legislative Council meetings, as well as addressing the public media. While the Federation maintains to be apolitical and non-partisan, we shall continue to strive as a leader with neutral platform, upholding our professionalism and promoting the health of the public.

Continuing growth of the Federation will rely on your advice and participation. The efforts you kindly contribute towards the Federation, are also sure efforts that contribute towards our health professions and society at large. Thank you in anticipation of your support, and we look forward to working alongside with you in the near future.

FMSHK Executive Committee (2012-2013)

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Editorial

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Editor

Dr. Nancy SY YUEN

General practitioners come across many different eye diseases in their daily practices. Hence it is very important to be well equipped with the knowledge to deal with eye diseases encountered and refer cases that may signify urgent or important eye diseases.

One of the top most prevalent blinding eye diseases is glaucoma, which has been the leading cause of irreversible blindness in Hong Kong since 1999. Development in diagnosis and management of glaucoma had been tremendous in the past decade and many systemic diseases are also noted to be associated with this important disease. Hence a detailed account of glaucoma is covered in this issue.

Diabetes mellitus, on the other hand, as one of the most common chronic medical illnesses, is also encountered in clinic settings in either the private or public sector. The long term control of the disease has also drawn much attention of the public through public education conducted in the media by both the government and other health care providers. It is also a systemic disease that has the most involvement of the eye in terms of direct effects of diabetic retinopathy, earlier development of cataracts, refractive changes in the early phases or as risk factors for many eye diseases including glaucoma. In view of the prevalence and relevance to clinical practice, Dr. Angie Fong and Dr. Timothy Lai will discuss on the management of diabetic retinopathy. With the conjoint effort of various disciplines, we are confident that blindness caused by diabetes can be controlled.

Optic neuritis, another important blinding though less prevalent eye disease, will be reviewed and discussed by Dr. Andy Cheng and Dr. Carmen Chan. Association with systemic diseases and investigative approaches will be covered.

Another very common but less vision-threatening eye disease that had a prevalence of up to 30% in reported studies will be the dry eye syndrome. Many of these patients are managed well in primary care settings. However, in view of the recent diverse types of dry eye treatment options, Dr. Douglas Lam will review on various types of eye drops and treatment in the section on drugs.

Refractive surgeries had been around in developed countries like USA and Hong Kong for over 30 years. According to a report by the American Academy of Ophthalmology, there were up to 12 million people worldwide with refractive surgeries performed and each year up to 700,000 cases are performed in USA. Myopia is common in Hong Kong and advances in refractive surgeries applicable to different age spectra will be discussed by Dr. Victor Woo.

Last but not least to add to the contents of the issue, Dr Raymond Lam is so kind to share with us some of his techniques in taking inspiring photographs and some of his shots taken in various corners around the world. We are eager to work with all disciplines in the health care sector to ensure the best ophthalmic health in our population and share the same inspiration we have with the delightful photos and scenery.

I shall like to take this opportunity to wish you a most enjoyable reading with this issue on Ophthalmology and a happy new year.

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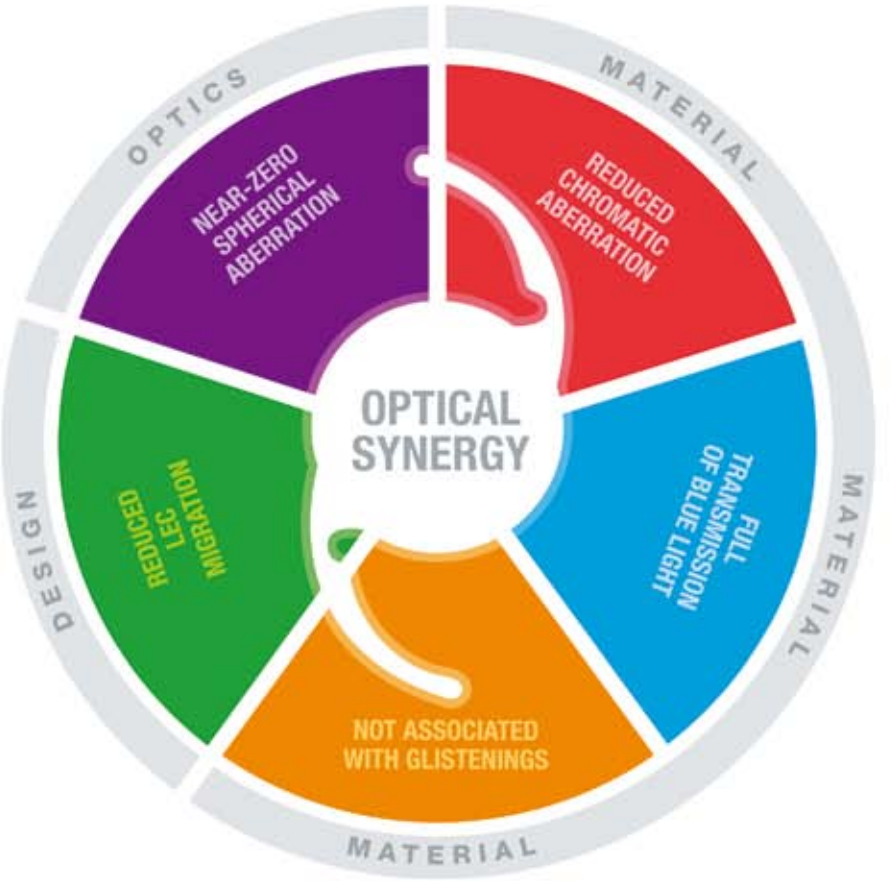

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Glaucoma- Advances in Diagnosis and Management

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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 January 2013.

Introduction

Glaucoma is a highly prevalent disease. It ranked second in the causes of blindness worldwide whereas it ranked first in causing permanent blindness in Hong Kong since 1999.

It was reported to affect around 1% of the population in most of the published population studies. It is a disease that showed increased incidence and prevalence with advancing age. It affected up to 2 to 3% of adults aged over 40 in most reported studies and could rise to up to 10% in populations aged 80 and over. [1,2,3]

Vision loss caused by glaucoma before diagnosis and treatment is irreversible, and the loss can be very disabling, some of the cases can have vision deteriorated to no light perception. Other common eye diseases e.g. Age-related macular degeneration, affect mainly central vision and in most of these patients, peripheral vision is spared and can be used for navigating around the environment.

Hence early and timely detection and treatment of glaucoma is very important for prevention of blindness from this disease.

What is glaucoma?

Glaucoma is an optic neuropathy with characteristic appearances of the optic disc (e.g. optic disc cupping, notching and peri-papillary atrophy) and specific patterns of visual field defects that are associated frequently but not invariably with raised intra-ocular pressure (IOP). Besides, there are also theories on impairment of the vascular supply to the optic nerve that contributes to the progression of the disease.

There are previous reports quoting normal IOP ranging from 10-21 mmHg with a mean of 16 mmHg. However, it was known that IOP fluctuates physiologically and will increase with age. Besides, there are increasing reports of glaucoma patients without raised IOP. Hence there is no definite cut-off value for IOP to define a glaucoma patient nowadays. Of all diagnosed cases of glaucoma, 50% have screening IOP < 21mmHg.[4]

Besides, there are increasing reports of a high proportion of patients of up to one-third of all glaucoma patients in their series are not having increased IOP which is classified as normal tension glaucoma. With advancing knowledge on normal tension glaucoma, more and more systemic associated diseases are identified.[5]

Figure 1: The diagnosis of glaucoma requires a full assessment which includes IOP measurement by applanation, visual field assessment and optic disc examination.

Glaucoma is not a single disease but includes a whole spectrum of diseases which can be further classified according to presentation, specific eyeball structure and underlying causes of the glaucoma.

Classification of glaucoma[6]

There are various classifications of glaucoma. It can be classified according to:

1. Presentation
   - Acute or Chronic
   - Chronic can be high pressure or normal tension glaucoma
2. Aetiology - Primary or Secondary
3. Structural - Open angle or Closed angle

How do glaucoma presents?

Symptoms and signs depend on the rate and degree of rise in pressure. In patients who suffer from a rapid rise in eye pressure to 50-60mmHg in acute angle closure glaucoma, they will present with sudden onset of painful red eye with dramatic drop in vision and may see halos around lights. There can be associated headache and vomiting. On examination, the eyes will appear red and when examined with a bright torch can reveal cloudy cornea with fixed and dilated pupils showing poor reaction to light. On palpation, the eyeballs may feel hard and tender. In view of the marked symptoms, most patients who suffer from acute glaucoma will present themselves early. This is an ocular emergency that requires urgent medical attention to reduce the eye pressure.

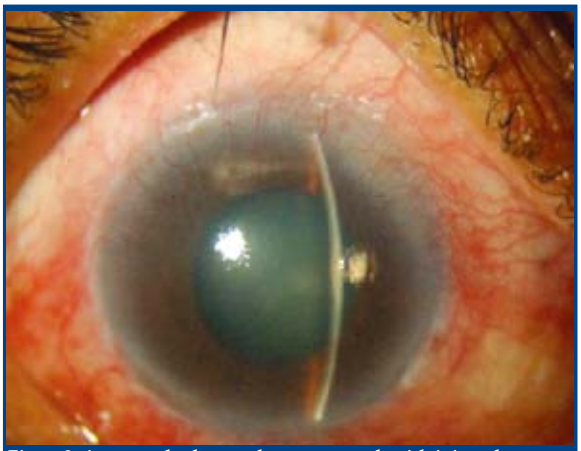

Figure 2: Acute angle closure glaucoma attack with injected eye, hazy cornea and mid dilated pupil

On the other hand, chronic glaucoma, which constitutes the main bulk of patients, and accounts for most cases of blindness caused by glaucoma, usually presents late. This is because most chronic glaucoma patients do not suffer from any symptom e.g. pain or any noticeable vision disturbance. They enjoy good vision especially in early and moderate disease when their central visual acuity is very good. Chronic glaucoma usually leads to slowly developing visual field defects without affecting the central vision and most patients realise their vision loss only in advanced stage of glaucoma with advanced constricted visual fields. Patients suffering from early or intermediate stages of glaucoma can have perfect vision in visual acuity examinations in optical shops. Eye pressure measurement may or may not review a high IOP and hence glaucoma cannot be excluded or diagnosed simply by a visual acuity check or simple IOP measurement by commonly used non-contact methods.

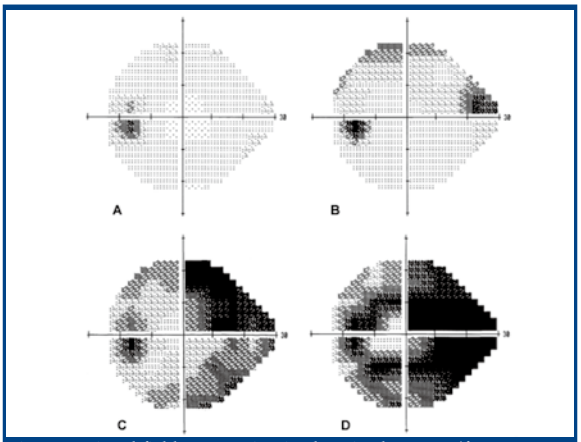

Figure 3: Visual field progression in chronic glaucoma (from A to D). The black area indicates areas of visual field that were lost with more and more advanced glaucoma.

Nowadays, more and more patients are being picked up by examinations of the optic disc and assessments by family physicians, in fundus photos for diabetic retinopathy screening, and by ophthalmologists. The vision loss caused by glaucoma is irreversible and hence early and timely detection and treatment is very important.

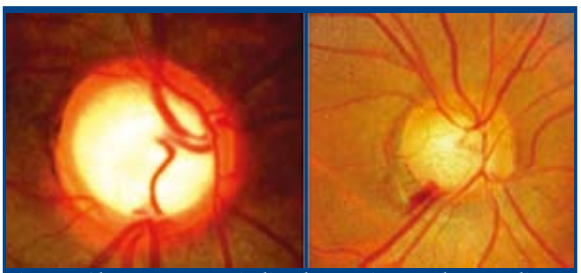

Figure 4: Glaucomatous optic disc changes, increased cup-to-disc ratio, sphincter haemorrhage and peri-papillary atrophy.

Treatment of glaucoma

Treatment of acute glaucoma

Urgent treatment is required in acute glaucoma. For example, acute angle closure glaucoma, which is known to be more prevalent in the Chinese population, needs urgent treatment to lower the IOP. If the patient presents early, systemic treatment with diamox (acetazolamide) or mannitol may abort the attack. Other treatments include Gutt 4% pilocarpine, which acts by constricting the pupil and hence reverses the pupil block. The fellow eye shall be treated with Gutt 1% Pilocarpine also to protect it from attack. Patients should be urgently referred to an ophthalmologist for full assessment; laser therapy or operation may be urgently needed in some cases because the high pressure, if sustained, may lead to blindness in hours.[7]

All patients who have acute angle closure attacks need laser iridotomy treatment to prevent future attacks. Some patients may volunteer a history of blurred vision with halos which aborted after a few hours. These may be cases of intermittent attack which need detailed assessments and prophylactic laser iridotomies too.

Besides, because of the high risk of development of chronic glaucoma in patients who have an acute attack history, all patients who have previous acute angle closure glaucoma must be monitored lifelong.[8]

Treatment of chronic glaucoma

Early diagnosis and treatment is most important in preventing blindness from chronic glaucoma. There was previously a diagnostic triad for the diagnosis of glaucoma, namely, increased IOP, characteristic glaucomatous visual field defect, and increased cup-to-disc ratio.

However, we now know that this triad no longer holds true. There are a group of glaucoma patients with normal eye pressure, referred to as normal tension glaucoma. Hence, in dealing with chronic glaucoma, we must identify both the high pressure and normal tension types of glaucoma.

With the advancement in technology, more and more cases of normal tension glaucoma are diagnosed and treated. Recent reports on normal tension glaucoma enlighten us on systemic diseases association of normal tension glaucoma patients and the contribution of many of these systemic diseases to the progression of visual field loss in this group of patients.

According to the Collaborative Normal Tension Glaucoma (CNTG) study reports, 20% of NTG subjects showed VF progression despite a lowering of IOP of more than 30% from baseline.[9] Thus, non-IOP-related elements have been sought as possible risk factors for NTG progression. There is growing evidence from clinical studies that circulatory abnormalities, including low blood pressure (BP), nocturnal hypotension, and unstable mean ocular perfusion pressure (MOPP), may be involved in the pathogenesis and progression of glaucomatous optic nerve disease.[10]

The diagnosis and treatment of normal tension glaucoma have now become multi-disciplinary, as circulation abnormalities of the optic nerve are noted to be associated.[11,12]

Besides a full examination by ophthalmologists on IOP, optic disc and visual field, there are new advances on ocular scanning. With the advent of more accurate scanning technology of optical coherence tomography (OCT), we can now also measure the thickness of the nerve fibre layer which further aids to diagnose and monitor glaucoma.

For both types of high pressure glaucoma or normal tension glaucoma, complete evaluation is very important and early diagnosis and treatment is most crucial in preventing blindness from glaucoma.

The main assessment now includes:

1. Risk factors assessment
   - Age
   - Ethnicity
   - IOP
   - Family history
   - High myopia
   - High hyperopia
   - Systemic vascular diseases e.g. hypertension and diabetes
   - Use of long term steroids
   - Sleep apnoea, Raynaud's disease
   - Previous eye trauma
2. IOP – can be high or normal
3. Gonioscopy – examination of the angle
4. Optic disc examination
5. Visual field examination with computerised automated visual field machine
6. Corneal thickness measurement in some cases
7. Other ocular imaging e.g. optical coherence tomography
8. Other systemic investigations e.g. neural imaging or cardiovascular assessment in selected cases of normal tension glaucoma

The mainstay of treatment for glaucoma for both high pressure and normal tension type is to lower the IOP, and this can be effected in most cases with topical medications. Systemic diseases associated with normal tension glaucoma need to be controlled too. There are various groups of eye drops that can control the IOP.

Table 1: Classes of drugs used to treat glaucoma

Class of drug	Drug names	Mechanism of action	Side effects
β-adrenergic antagonists (topical)	Timolol, levobunolol, betaxolol, carteolol	Decrease of aqueous humor production	Bronchospasm, bradycardia, congestive heart failure, depression, confusion, impotence
Adrenergic agonists (topical)	Brimonidine (α-2 selective), dipivefrin	Decrease of aqueous humor production and decrease resistance to outflow	Conjunctival hyperemia, allergic reactions, malaise, headache, SNC depression
Cholinergic agonists (topical)	Pilocarpine	Increase of aqueous outflow	Eye or brow pain, increased myopia, decreased vision
Prostaglandin-like analogues (topical)	Latanoprost, unoprostone, travoprost, bimatoprost	Increase of aqueous humor outflow	Conjunctival hyperemia, increased iris pigmentation, hypertrichosis
Carbonic anhydrase inhibitors (topical and systemic)	Dorzolamide and brinzolamide (topical), acetazolamide (oral)	Decrease of aqueous humor production	Paresthesias, depression, malaise, anorexia, allergic reactions, renal calculi

Treatment must be individualised. The success of treatment of glaucoma depends very much on the compliance and understanding and rapport with patients. Patients must know their disease condition. Glaucoma can be controlled with resulting good vision throughout life but cannot be cured. Patients have to receive treatment and be monitored lifelong.

Besides, treatment of many associated systemic diseases e.g. sleep apnoea, nocturnal hypotension are also very important in the long term control of normal tension glaucoma.[13,14]

In cases with narrow angle morphology, laser iridotomies or iridoplasty may be needed. In cases with open angle, there are also options of laser trabeculoplasty which may help in the IOP control. The indication for different laser treatments of particular patients depends on the results of their detailed assessments.

Surgeries for glaucoma are mostly reserved for patients who have their eye pressures not controlled with topical medications or lasers or both. The most commonly performed surgery is trabeculectomy which is a surgery

that creates a drainage bleb for the aqueous humour under the cover of the upper lid. New advances in surgical treatment include the development of safer and more predictable surgery of non-penetrating deep sclerectomy/non-penetrating trabecular surgery which can achieve similar IOP control to conventional trabeculectomy but with a smooth post-operative course and markedly less complications. This new type of non-penetrating surgery is applicable in many different types of glaucoma.[15,16]

For patients who have failed conventional surgeries or for cases of recalcitrant glaucoma, other surgical options, e.g. implantation of a glaucoma drainage device or destruction of the ciliary body by means of laser, may be employed in some cases.

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MCHK CME Programme Self-assessment Questions

Please read the article entitled "Glaucoma- Advances in Diagnosis and Management" by Dr. Nancy SY YUEN 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 January 2013. 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. Glaucoma is the top blinding eye disease in Hong Kong.
2. Glaucoma vision loss is reversible.
3. Chronic glaucoma patients can be totally asymptomatic.
4. Acute glaucoma must be managed urgently because vision can be lost within hours if untreated.
5. Glaucoma needs lifelong follow up and management.
6. IOP lowering is the mainstay of treatment for both high pressure and normal tension glaucoma.
7. Patients who suffered acute angle closure glaucoma needs no further follow up after the acute attack is aborted.
8. There is growing evidence from clinical studies that circulatory abnormalities, including low blood pressure (BP), nocturnal hypotension, and unstable mean ocular perfusion pressure (MOPP), may be involved in the pathogenesis and progression of glaucomatous optic nerve disease.
9. High myopia, high hyperopia are risk factors for glaucoma.
10. Diagnosis of glaucoma need detail assessment of eye pressure measurement by applanation, optic disc examination and visual field examination.

ANSWER SHEET FOR JANUARY 2013

Please return the completed answer sheet to the Federation Secretariat on or before 31 January 2013 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Glaucoma- Advances in Diagnosis and Management

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Answers to December 2012 Issue

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Advances in the Management of Diabetic Retinopathy

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Dr. Angie HC FONG Dr. Timothy YY LAI

Introduction

Diabetic retinopathy (DR) is one of the most common causes of preventable blindness in the developed world. In the United States, it has been estimated that 40% of people with Type II diabetes and 86% of people with Type I diabetes have diabetic retinopathy.[1] The META-EYE study, a meta-analysis involving pooled analysis of over 22,000 individual data from 35 population-based studies from 1980 to 2008 around the world, showed that the overall prevalence of any DR was 34.6%. In addition, the prevalence of proliferative diabetic retinopathy (PDR) was 6.96%, diabetic macular oedema (DMO) was found in 6.81%, and 10.2% had vision threatening DR, which is defined as PDR or DMO.[2]

The presence of DR has important systemic implications on the patients' general health. DR has been found to be strongly associated with all-cause mortality and cardiovascular complications in both type 1 and 2 diabetic patients. A meta-analysis of 20 observational studies of 19,234 patients showed that any degree of DR increased the chance for all-cause mortality and/or cardiovascular events (including myocardial infarction, angina pectoris, coronary artery bypass graft surgery rate, transient Ischaemic attacks, non-fatal stroke, and lower leg amputation) by 2.3 times.3 DR is also a predictor of all-cause mortality, with an odds ratio of 2.41 for type 2 diabetic patients and 3.65 for type 1 diabetic patients.[3]

Systemic management of diabetic retinopathy

Multiple systemic risk factors have been implicated in the progression of diabetic retinopathy, including hyperglycaemia, hypertension, dyslipidaemia, long duration of diabetes, ethnicity (especially Hispanic or South Asian), pregnancy and puberty. Some of these risk factors are modifiable and optimising the control of these systemic risk factors will be beneficial to prevent the development and progression of DR.

Control of hyperglyaemia

The role of intensive glycaemic control has been investigated in multiple studies. The definition of intensive glycaemic control differ among various studies, ranging from HbA1c < 6.0%[4] to HbA1c < 6.5%.[5] The DCCT (Diabetic Control and Complications Trial) found that intensive glycaemic control to levels close to an individual without diabetes resulted in a 73% risk reduction in the progression of diabetic retinopathy relative to control group with no target glycaemic levels.[6] Another randomised control trial, the ACCORD (Action to Control Cardiovascular Risk in Diabetes) study aimed to investigate the effects of tight glycaemic control (target HbA1c <6.0%), dyslipidaemia controlled by oral fenofibrate and blood pressure control in type 2 diabetic patients with cardiovascular disease or risk factors. The trial was terminated prematurely due to increase in all-cause mortality in the treatment arm. However subsequent subgroup analysis has shown a 12% reduced risk of DR progression with tight glycaemic control.[4] Another meta-analysis involving 14 clinical trials with 28,614 patients with type 2 diabetes found that intensive glyceamic control lowered the risk for composite microvascular complications and retinopathy by 12% and 20% respectively.[7] Regarding safety, the study found that intensive glycaemic control did not significantly influence the risks of all-cause mortality or cardiovascular mortality, but the risk of severe hypoglycaemia was significantly increased when intensive glycaemic control was targeted.

Control of systemic hypertension

Hypertension is another risk factor targeted by investigators in the treatment of DR. In the United Kingdom Prospective Diabetes Study (UKPDS), a landmark trial in which 5,102 patients with newly diagnosed type 2 diabetes were followed for 10 years, tight blood pressure control (<150/85mmHg) with captopril or atenolol was found to reduce the progression of DR by 37% in 9 years, compared with less tight blood pressure control (<180/105).[8] Another study, the Renin Angiotensin System Study,[9] found that rennin angiotensin system blockage with enalapril or losartan reduced DR progression by 65% and 70% respectively in patients with type 1 diabetes. Harindhanavudhi et al additionally found that the benefit of angiotensin system blockage on DR seemed to vary with glycaemic control and was only beneficial when baseline HbA1C was >7.5% in patients with type 1 diabetes.[10]

Control of dyslipidaemia

Control of dyslipidaemia has also been identified to be beneficial in the control of DR. The Fenofibrate Intervention and Event Lowering in Diabetes (FIELD) Study investigated the use of fenofibrate in 9,795 patients with type 2 diabetes with total cholesterol of 3.0-6.5mmol/L and total cholesterol to high density lipoprotein-cholesterol ratio of >4.0 or triglyceride of 1.0-5.0mmol/L. Patients were randomised into receiving fenofibrate 200mg daily versus placebo.[11] In one sub-study with 1,012 patients where 512 patients received fenofibrate and the rest received placebo, it was found

that patients in the treatment arm had a lower risk of DMO, overall diabetic retinopathy and PDR (hazard ratios of 0.69, 0.69 and 0.87 respectively). The need for laser treatment was also lowered by 34% in the treatment group compared with placebo.

Summarising the above evidence, we can conclude that tight glycaemic control, blood pressure control and treatment of dyslipidaemia are the mainstay of systemic treatment for DR. Intensive glycaemic control and tight blood pressure control with Angiotensin Converting Enzyme Inhibitor (ACEI) or Angiotensin Receptor Blocker (ARB) agents have been shown to reduce the risk of progression of DR, and the use of fenofibrate in addition to statins in hyperlipidaemia can reduce the progression of DR and need for laser treatment.

Ocular management of diabetic retinopathy

Diabetic Macular Oedema

Diabetic macular oedema (DMO) is one of the main causes of visual loss in diabetes (Fig. 1). If left untreated, a considerable proportion of patients will suffer from gradual visual loss. The Early Treatment Diabetic Retinopathy Study (ETDRS) showed that 33% of untreated eyes lost 3 or more lines of vision at 3 years.[12] The current treatment options for clinically significant macular oedema include focal or grid laser photocoagulation, intravitreal steroid and anti-VEGF therapy.

Figure 1. Fundus photograph of a patient with diabetic macular oedema showing macular thickening associated with hard exudate and retinal microaneurysms and haemorrhage.

Laser photocoagulation

Laser photocoagulation has been standard treatment for DMO until the recent availability of ocular pharmacotherapy. The ETDRS has shown that grid laser photocoagulation can cause a 50% risk reduction of moderate visual loss (a drop of three or more Snellen acuity lines) in treated eyes with DMO compared with no treatment.[12] However, laser photocoagulation for DMO is not ideal because it can only prevent visual loss and cannot improve vision, and the destruction of retinal tissues often leaves laser scars which can create visual field loss close to the central vision.

Intravitreal steroid

Intravitreal administrations of corticosteroids, such as triamcinolone acetonide (TA), fluocinolone and dexamethasone, have been investigated in the treatment of DMO. Steroid works by inhibiting the arachidonic acid pathway, thereby lowering the production of vascular endothelial growth factor (VEGF), which causes macular oedema. TA also reduces the breakdown of the blood retinal barrier and can reduce leakage of fluid from capillaries. Different methods of delivery have been developed, including direct intravitreal injection of TA, or intravitreal corticosteroid implants.

A systemic review of 7 randomised controlled trials involving 632 DMO eyes has investigated the use of intravitreal TA and steroid implants for chronic or refractory DMO.[14] All studies were found to be in favour of ocular steroid in terms of visual improvement and decrease in central macular thickness on optical coherence tomography (OCT) examination. This beneficial effect was evident from the third month to up to 2 years after treatment. However, intravitreal TA was associated with side effects such as increased intraocular pressure in up to 40% of patients and increase in cataract formation, which require regular follow-ups and management.

Ozurdex (Allergan Inc, Irvine, CA, USA) is a dexamethasone drug delivery implant which is recently marketed for the treatment of uveitis or macular oedema secondary to retinal vein occlusions. Once the implant is injected into the eye, it slowly dissolves in the vitreous cavity and the drug effect can last for around 4 months. It has also been used for the treatment of DMO. Zucchiatti et al performed a retrospective review of 9 patients receiving Ozurdex 0.7mg for persistent DMO.[13] At 3 months, the mean visual acuity improved by 1.5 lines with significant reduction in central retinal thickness measured by OCT. However, at 6 months, there was rebound of oedema due to absorption of the drug pellet and the visual acuity returned to baseline level. Further studies are now being conducted to evaluate the longer-term efficacy of Ozurdex for DMO.

Anti-vascular endothelial growth factor (Anti-VEGF) therapy

The availability of anti-VEGF agents such as bevacizumab (Avastin, Roche, Switzerland) and ranibizumab (Lucentis, Novartis, Basel, Switzerland) has revolutionised the treatment of various retinal diseases including DMO and PDR. VEGF plays a major role in the development of both DMO and PDR. Retinal hypoxia associated with diabetes triggers VEGF production by the retinal pigment epithelial cells, pericytes and endothelial cells of the retina, which in turn causes the breakdown of the blood-retinal barrier and stimulates angiogenesis, leading to DMO and PDR.

Treatment using anti-VEGF agents involves injection of the drug directly into the vitreous cavity at the pars plana using a 30-gauge needle under aseptic environment. By blocking the action of VEGF, anti-VEGF agents can decrease the amount of exudation and neovascularisation in DMO and PDR respectively. However, since anti-VEGF agents will usually be absorbed in around 4 weeks, repeated monthly injections are frequently required for the treatment

of DMO. The treatment responses following anti-VEGF therapy are monitored regularly by visual acuity assessment and evaluation of macular thickness by OCT and repeat treatment is performed when there is worsening of visual acuity or increase in macular thickness (Fig. 2). Currently, there are two anti-VEGF agents which are commonly in use by ophthalmologists for treatment of macular diseases and these include bevacizumab (Avastin, Roche, Switzerland) and ranibizumab (Lucentis, Novartis, Switzerland).

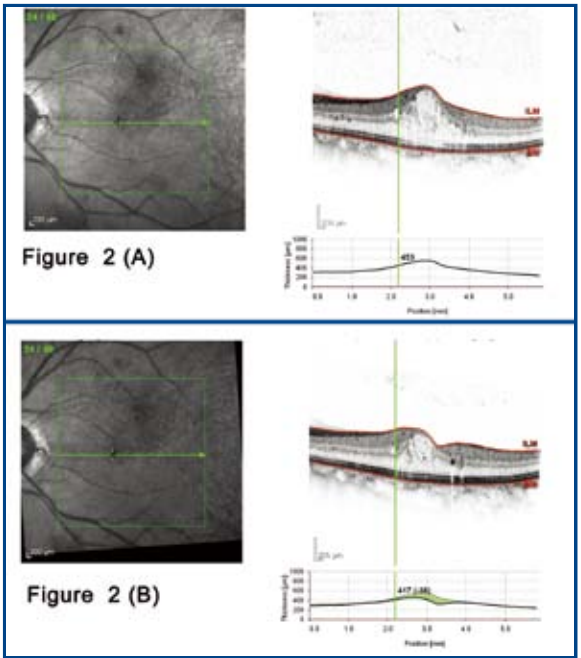

Figure 2. (A) Spectral domain optical coherence tomography scan of a patient with diabetic macular oedema showing macular thickening with cystic spaces. (B) Six weeks after intravitreal ranibizumab injection, repeat spectral domain optical coherence tomography scan demonstrating reduction of macular oedema with restoration of foveal depression.

Since the first publication on the use of intravitreal bevacizumab (Avastin, Roche, Switzerland) for the treatment of DMO in 2006,[15] more than 80 reports have been published regarding its use in DMO. Intravitreal bevacizumab appeared to be effective in improving vision and reducing macula oedema in DMO. However, there were very few well-designed randomised control trials available to evaluate its efficacy and safety, making the results difficult to generate conclusive evidence on the use of intravitreal bevacizumab injection for DMO.[16] Moreover, bevacizumab was originally designed for intravenous use in cancer patients and therefore there might be some theoretical increased risks of systemic adverse events in using intravitreal bevacizumab for DMO due to its longer plasma half-life in the systemic circulation.

At present, the only anti-VEGF agent approved by regulatory authorities for the treatment of DMO is ranibizumab. The efficacy of intravitreal ranibizumab for DMO has been demonstrated by two parallel, 24-month phase 3 double-masked randomised control trials (RISE and RIDE studies).[17] In these studies, 377 patients (RISE) & 382 patients (RIDE) were randomised into receiving monthly 0.3mg ranibizumab, 0.5mg ranibizumab or sham injections, followed by potential rescue macular laser at 3 months. The results showed that a significantly greater proportion of ranibizumab-treated eyes gained 15 or more letters in vision compared with sham controls, with approximately 34-45% of ranibizumab-treated eyes gaining 15 letters or more of vision compared with 12-18% of sham groups. The ranibizumab treated groups also required significantly fewer macular laser procedures compared with the sham group. There were significant improvements in macular oedema with reduction in central foveal thickness in the ranibizumab-treated groups compared with sham injection. The treatment was found to be safe with low incidence of adverse events and no significant increase in deaths or nonfatal myocardial infarction or cerebrovascular accidents in the ranibizumab groups compared with the sham group.

Another study, the RESTORE trial, is a 12-month phase 3 double-masked randomised control trial which investigated the effects of combining ranibizumab and macular photocoagulation laser for DMO.[18] Over 300 patients were randomised to receiving ranibizumab monotherapy, laser monotherapy, or a combination of ranibizumab and laser. At 1 year, patients treated with ranibizumab either as a monotherapy or in combination with laser had significantly better vision than patients in the laser group alone (6.1, 5.9 and 0.8 letters gain respectively). There were no significant differences in visual gain and the number of injections between the ranibizumab or ranibizumab plus laser treatment arms. Therefore, treatment of DMO using intravitreal ranibizumab monotherapy appeared to be justified without the addition of laser.

Another newer anti-angiogenesis agent, aflibercept (Eylea, Bayer, Berlin, Germany), is a fusion protein which has recently been approved for the treatment of neovascular age-related macular degeneration (AMD). It is a highly potent anti-angiogenic agent and binds to all forms of VEGF-A as well as placental growth factor (PIGF). The efficacy of aflibercept in the treatment of DMO was investigated in a phase 2, multi-centre randomised control trial (DA VINCI).[19] Two different dosing, 2mg aflibercept every 4 weeks for 6 months, or 2mg aflibercept every 4 weeks for 3 months followed by monthly pro ra nata treatment until 6 months, resulted in significant visual improvements compared with the laser-treated group (11.4, 10.3 and 2.5 letters gain respectively). Further phase 3 clinical trials are now being conducted to evaluate the efficacy of aflibercept in the treatment of DMO.

In summary, anti-VEGF therapy appears to be an effective treatment for DMO that can result in substantial visual gain. There are less ocular side effects compared with intraocular steroids, but its major drawback is the need for repeated monthly injections. Currently, the efficacies between different anti-VEGF agents for DMO are unclear and large-scale comparative studies like the CATT trial (which compared ranibizumab and bevacizumab for the treatment of neovascular AMD) is needed to make a definitive conclusion.[20]

Algorithm in treating patients with DMO

In dealing with patients with DMO, one should first

determine whether there is central macular involvement. If the leakage points are extrafoveal, focal laser may be considered. If there is central involvement without visual loss one may observe and treat according to the ETDRS guidelines, but if there is central involvement with visual loss then treatment with ranibizumab monotherapy may be considered.[21] Ancillary investigations like OCT and fluorescein angiography (FA) are also useful in the management of patients with DMO. OCT can reveal structural abnormalities such as thickened posterior hyaloid, epiretinal membrane and vitreomacular traction which may be amenable to vitreoretinal surgery, whereas FA will be useful to exclude macular ischaemia and will provide prognostic information about the treatment outcome.

Proliferative Diabetic Retinopathy (PDR)

The hallmark of PDR is the development of abnormal new blood vessels on the surface of the retina and these abnormal vessels can bleed easily causing vitreous haemorrhage and may form fibrovascular membranes leading to retinal detachment. Moreover, neovascularisation can develop in the drainage angle with iris neovascularisation (NVI), resulting in neovascular glaucoma (NVG). At present, pan-retinal photocoagulation (PRP) laser therapy is the definitive treatment of PDR, Nonetheless, recent evidence suggest that anti-VEGF can cause rapid regression of retinal neovascularisation with effects as soon as 1-2 days after anti-VEGF injection. Anti-VEGF may also be used in combination with PRP, as an adjunctive therapy before vitrectomy, or used in the treatment for NVI and NVG.

Anti-VEGF as primary therapy for retinal neovascularisation

Arevalo et al reported a retrospective study of 43 eyes of 39 patients with PDR treated with intravitreal bevacizumab.[22] The results showed that 39.5% of patients had total regression of the neovascularisation without leakage on FA, while 34.9% had partial regression and 25.6% had no regression. Three treatment-naive eyes with vitreous haemorrhage resolved after treatment and subsequently avoided vitrectomy. The study concluded that bevacizumab is safe and effective either alone or in combination with PRP in causing regression of retinal neovascularisation in patients with PDR.

Anti-VEGF as adjunctive therapy before vitrectomy

Ahmadieh et al performed a randomised controlled trial which showed eyes receiving intravitreal bevacizumab 1 week prior to vitrectomy had significantly less incidence of postoperative vitreous haemorrhage at 1 week and one month, compared with sham injection.[23] The main concern with anti-VEGF adjunctive therapy, however, is the development of tractional retinal detachment after bevacizumab injection. Risk factors include long duration of diabetes of >15 years, >13 days from injection to vitrectomy and higher dose (2.5mg) of bevacizumab used. Therefore careful monitoring following anti-VEGF therapy for PDR is mandatory.

Anti-VEGF in the treatment of iris neovascularisation and neovascular glaucoma

In a retrospective study of 28 eyes with NVI treated with intravitreal bevacizumab, 71.4% showed significant regression, 21.4% had partial regression and 7.2% had no change. Visual acuity improved in 17.9% and 91% had IOP reduction after injection.[24]

Based on the above evidence, anti-VEGF therapy seems to be a promising treatment modality in the treatment of PDR. However, one must be cautious in patient selection as indiscriminate use may cause more harm than good. Patients with eyes that have dense preretinal fibrotic neovascular tissue or vitreous haemorrhage without a definitive plan for vitrectomy are not suitable candidates for anti-VEGF therapy.

Conclusions

Epidemiological studies have demonstrated increasing prevalence of diabetes and diabetes-related eye disease worldwide and DR is an important disease marker for cardiovascular morbidity and mortality. Therefore, physicians should be aware of the systemic medical implications of DR when treating patients, especially when anti-VEGF agents are now available to treat eye diseases as systemic use might be associated with thrombo-embolic complications. In managing patients with DR, one must be holistic and optimally manage systemic diseases such as hyperglycaemia, hypertension and hyperlipidaemia. Major advances in ocular pharmacotherapy have allowed for not only visual preservation but visual gain in eyes with DMO. Careful monitoring and timely referral to ophthalmologists with expertise in treating retinal diseases is essential to ensure optimal visual outcomes for patients with DR.

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Dermatological Quiz

Dermatological Quiz

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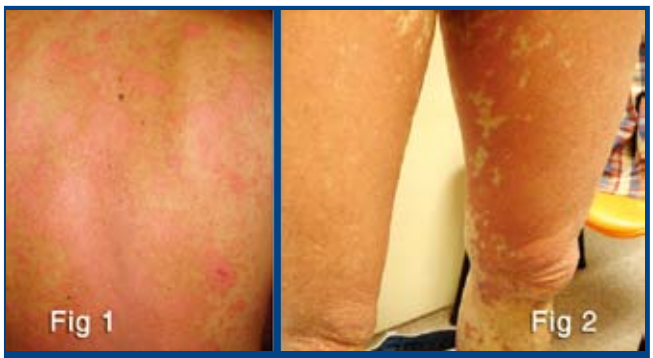

Generalized confluent erythema & scaling at back (Fig. 1) & thighs (Fig. 2)

This 40-year-old man had acute onset of mildly pruritic scaly skin rash at the face and scalp, which spread extensively to his trunk and limbs within a month (Fig. 1 & 2). There were no associated arthralgia or nail lesions. His general condition remained well without any systemic upset like fever and chill. He had no previous or family history of chronic skin diseases and no preceding drug history. The skin condition did not respond to topical steroid and oral antihistamines.

Questions:

1. What is your preliminary diagnosis?
2. What are the key features of this disease?
3. What are the main differential diagnoses of this disease?
4. How do you manage this patient?

(See P. 37 for answers)

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What Eye Drops Should I Prescribe? An Overview of Drug Treatment for Dry Eye Syndrome

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Introduction

Dry Eye Syndrome (DES) is a common disorder, which can present with various eye symptoms (grittiness, burning sensation, tearing, ocular discharge, pain and even blurring of vision). DES was recently redefined as "a mulitfactorial disease of the tears and ocular surface that results in symptoms of discomfort, visual disturbance, and tear film instability with potential damage to the ocular surface. It is accompanied by increased osmolarity of the tear film and inflammation of the ocular surface."[1] It has been estimated that up to 25% of patients report symptoms of DES in general ophthalmology clinics.[2] It is also been reported that DES is more commonly encountered in women and elderly.[3,4] According to a local epidemiology study done recently, the prevalence of DES is estimated to be 7.7% in Hong Kong.[5] Though DES is not a vision-threatening disease, it can significantly affect the patient's quality of life. This article will provide an overview of DES and its drug treatments.

Tear film and DES

The tear film is important to the eye in numerous ways, such as: lubricating the ocular surface; maintaining nutrients and oxygen to the cornea; acting as part of the refractive surface; providing enzymes to prevent infection; and removing debris and foreign bodies from the ocular surface. The tear film is composed of the anterior lipid layer, the middle aqueous layer and the posterior mucin layer. Any disturbance in the composition of the tear film may lead to DES. DES can be divided into tear deficiency type and evaporative type. Tear deficiency type DES can be classified into non-Sjogren syndrome and Sjogren syndrome (with lacrimal gland involvement). Evaporative type DES can be due to meibomian gland disease, exposure-related (lagophthalmos), eyelid abnormalities (entropion or ectropion), and ocular surface diseases secondary to Steven-Johnson syndrome and ocular cicatricial pemphigoid.

Clinical Diagnosis

A comprehensive clinical history is essential to identify underlying causes of DES. Systemic diseases such as rheumatoid arthritis, systemic lupus erythematosus, thyroid diseases and diabetes may be associated with DES. Also, medications including anti-histamines and antidepressants can cause dry eyes. History of ocular surgeries, in particular refractive surgeries, may predispose to DES. Environmental factors can also lead to DES. Preliminary study shows that more than 3 hours of daily computer use is associated with DES.[5] Clinical tools commonly used in diagnosing DES are based on subjective and objective measures. The Ocular Surface Disease Index (OSDI®) is a 12-item scoring survey available on the internet, in which the patient rates his or her own ocular symptoms on a scale of 0-4. Then, the survey gives a total score ranging 0-100, the higher score being more symptomatic. Objective approaches to DES include: tear film breakup time, fluorescein or lissamine green staining of the cornea and conjunctiva, Schirmer's test, tear osmolarity and ocular inflammation (MMP-9) detection tests. Since no single test can definitively diagnose DES, a multi-factorial approach is required to manage DES.

Drug treatment

Treatment of DES primarily aims to restore and stabilise the tear film, and topical eye drops, gels and ointments are the mainstay of drug delivery to the ocular surface. Artificial tears of different viscosities can be of the preserved, e.g. benzalkonium chloride (BAK), or the non-preserved form. Preservative-free artificial tears are favoured as BAK can cause eye irritation or allergies. Those patients who need frequent applications are advised to use preservative-free tear substitutes. Buffers of different forms are used to normalise the altered osmolarity in dry eyes. Other preparations contain vasoconstrictors in order to reduce conjunctival injections. MIMS Hong Kong lists more than 30 listed ophthalmic drugs under the classification "Ocular Lubricants" with indications to treat dry eyes. Hypromellose, or hydroxypropyl methylcellulose, is the active ingredient in most artificial tear preparations. Over-the-counter (OTC) artificial tears such as Eye Glo®, Eye Mo®, GenTeal®, Isopto Tears® and Lac-Oph® contain hypromellose, and they can be used for general eye discomfort. 0.9% sodium chloride (Lamabak®, Normal Saline Xepa-Soul®) can be used as well in treatment of tear insufficiencies. However, these artificial tears serve to replace tear volume only and require frequent application. More advanced preparations (Bion Tears®, Blueye®, Computer Eye Drops®, Moisture Eyes®, Tears Naturale®) have glycerin or dextran incorporated so as to increase the stability of the tear film and prolong tear break-up time. Carboxymethylcellulose sodium is another active ingredient which significantly enhances tear stability, and it is available in Optive® and Refresh Tears®. Systane® contains PEG 400 and propylene glycol to increase its viscosity, thereby lengthening the retention time of the drug in the eye.

As for eye gels, sodium hyaluronate preparations (Hialid 0.1® and Vismed®) or carbomers (GenTeal gel®, Lacryvisc® and Liposic eye gel®) is added in order to provide more protection to the ocular surface. However, eye gels may compromise vision transiently after drug application, and therefore gels are recommended for night-time use. Ointments are of the highest viscosity, e.g. Duratears® containing white petroleum, anhydrous liquid lanolin and mineral oil, and they are used in the most severe cases of DES.

Aside from artificial tears, steroid eye drops (fluorometholone, dexamethasone, and prednisolone acetate) are used by ophthalmologists in cases where ocular surface inflammation is evident. Topical steroids are used with caution, as vision-threatening side-effects of topical steroids include elevated intra-ocular pressure, cataract formation, higher risk of infective keratitis, corneal and scleral melting.

With the recent introduction of topical 0.05% cyclosporine emulsion (Restasis®), DES can be treated with higher success. A 2nd line immunomodulator, topical cyclosporine targets ocular surface inflammation and stimulates tear production without the side effects of steroid. At present, Restasis® is prescribed by ophthalmologists exclusively, and patients are required to attend regular follow-ups for monitoring their response.

Apart from topical preparations, Doxycycline taken orally can be used to reduce ocular surface inflammation and promote tear production. However the potential side effects of Doxycycline must be discussed with the patient, and close monitoring of its use is recommended.

Alternatively, diet supplements containing omega-3 and omega-6 fatty acids and flaxseeds have shown improvements in tear production and tear volume, as well as reduction of ocular inflammation in DES.[6,7]

Conclusion

DES is a multi-factorial disease and should be assessed both subjectively and objectively. Comprehensive systemic workup and ocular examination are often required. Judicious and accurate use of drugs to treat DES can significantly improve patients' symptoms and quality of life.

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Optic Neuritis Management 2013- a Hong Kong Perspective

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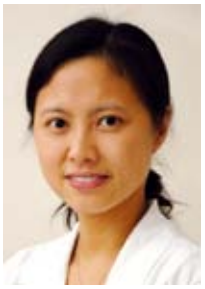


Dr. Carmen KM CHAN Dr. Andy CO CHENG

Introduction

Optic neuritis is one of the most commonly encountered neuro-ophthalmologic conditions. It is usually managed by general ophthalmologists, neurologists, as well as neuro-ophthalmologists. Whilst it is typically idiopathic or associated with multiple sclerosis (MS), there are other causes of optic neuritis (e.g. autoimmune, infectious) and conditions which mimic optic neuritis, which require a different management approach. In this article, we shall provide a revision of the standard management of 'typical' optic neuritis, as well as review some other conditions which may have similar initial presentations to optic neuritis, in particular neuromyelitis optic (NMO) and optic perineuritis (OPN).

Management of typical optic neuritis

Our current management of typical optic neuritis is very much guided by the Optic Neuritis Treatment Trial (ONTT).[1] In order to know how to translate the study findings to clinical practice, we need to know what disease entity they intended to study. In the opening paragraph of the original ONTT paper[1], acute optic neuritis was described as an acute demyelinating disease of the optic nerve, isolated or associated with MS; presents with sudden visual loss, followed by **spontaneous** improvement over several months. This constitutes our current understanding of 'typical' optic neuritis. The classic clinical features of typical optic neuritis include acute/ sub-acute, usually unilateral, visual loss; with dyschromatopsia, and pain on eye movement. Any type of visual field defect is possible, although diffuse and central visual loss predominated at presentation in the ONTT.[2] A relative afferent pupillary defect is an important objective finding, if the disease is unilateral. Two thirds of the patients do not have disc swelling (i.e. retrobulbar optic neuritis). Typical optic neuritis can recur in the same eye or in the fellow eye; the 10-year risk for recurrence in the ONTT was 35%.[3] According to the ONTT, the diagnosis of typical optic neuritis is a clinical one, and does not require any laboratory studies or lumbar puncture. However it is nowadays usual to obtain magnetic resonance imaging (MRI) of the brain and orbit, with and without gadolinium, to exclude other anatomical causes of optic neuropathy; to confirm the presence of optic neuritis; and to look for demyelinating white matter lesions for prognostic purposes.[4] The best sequence to look for optic neuritis is T1-weighted images of the orbit, with fat suppression; and the presence of optic neuritis is confirmed by contrast enhancement (present in up to 94% of acute optic neuritis[3]). Whilst acute optic neuritis can also be demonstrated by increased signal intensity on non-contrast T2-weighted, fat suppressed images, this finding is also found in optic atrophy (due to increased cerebrospinal fluid around the optic nerve) and therefore this is not diagnostic of optic neuritis in patients with chronic optic neuropathies. Computer tomography of the orbit, even with contrast, is not useful for confirming/ excluding optic neuritis. Pattern reversal visual evoked potential (VEP) P100 peak latency is typically increased in demyelinating optic neuropathies, however an abnormal VEP is not diagnostic of optic neuropathy. The results of pattern reversal VEP can be affected by any component of the visual pathway, including uncorrected refractive error, cataract and maculopathy. VEP is not necessary for the diagnosis of optic neuritis.[4]

In the ONTT, 457 patients with acute unilateral optic neuritis within 8 days of presentation, were randomised to receive oral prednisone (1mg/ kg/day) for 14 days; or intravenous methylprednisolone (1g/day) for 3 days, followed by oral prednisone (1mg/ kg/day) for 11 days; or oral placebo. It was found that the use of intravenous methylprednisolone hastened the visual recovery compared with placebo but did not affect the visual outcome from 6 months onwards. Therefore expectant management for acute typical optic neuritis is an option. Treatment with oral prednisone alone increased the recurrence rate of optic neuritis and **the use of oral corticosteroids alone at ONTT dosage is contraindicated in typical optic neuritis**. Treatment with intravenous methylprednisolone had an additional advantage of lowering the rate of MS development in the first 2 years, but the beneficial effect is not sustained at 3 years and beyond.[5] Treatment with intravenous immunoglobulin or plasma exchange is not usually considered helpful for typical optic neuritis.[6,7]

The brain MRI is the single most important predictor for the development of MS after a single episode of optic neuritis. In the ONTT, 25% of patients with no lesions on baseline brain MRI developed MS during the 15-year follow-up, compared with 72% of patients with 1 or more lesions.[8]

If a patient has an episode of acute optic neuritis and at least 2 white matter lesions on brain MRI, this is termed clinically isolated syndrome (CIS). Three randomised, double-masked, placebo controlled studies (CHAMPS[9], ETOMS[10] and BENEFIT[11]) demonstrated that treatment with interferon beta can delay the onset of clinical definite MS after a CIS. However it is worth noting

that MS is much less common in Southern Chinese than in Caucasian countries. In addition, interferon beta treatment is not without side effects and its use in CIS, without evidence of progression on MRI, is not currently funded by the Hong Kong Hospital Authority.

Atypical optic neuritis

Acute demyelinating and idiopathic optic neuritis can recover spontaneously without treatment. However there are other forms of optic neuritis which can lead to blindness if not treated promptly. These include NMO; inflammatory/ autoimmune (e.g. associated with sarcoidosis, systemic lupus erythematosis; or it can be isolated, like chronic relapsing inflammatory optic neuropathy), infective optic neuritis (e.g. paranasal sinusitis; syphilis and tuberculosis, especially with human immunodeficiency virus co-infection) and OPN. There are also other forms of optic neuritis which may or may not benefit from treatment, including paraneoplastic optic neuritis and neuroretinitis.

If typical optic neuritis requires no investigations, how can a clinician distinguish typical optic neuritis from the more 'sinister' atypical ones? This relies on the accurate clinical assessment and observation of the disease course. Optic neuritis can be considered atypical, if it occurs in an atypical patient, presents atypically or has an atypical disease course. The clinical features which should prompt further investigations are summarised in Table 1. It is important to note that in the ONTT, the mean age of the patients was 32 years, 77% of them were female and 85% white.[1] Therefore any patient who does not fit this standard description may be considered atypical, and that includes all Chinese patients!

Table 1: Features which suggest atypical optic neuritis

Atypical patient
- Children; older patients
- Non-Caucasian
- Male
- History of acute myelitis (may suggest neuromyelitis optica)
- History of autoimmune diseases
- History of vaccination (may suggest acute demyelinating encephalomyelitis)
- Current systemic infection or exposure to infective agents

Atypical presentation
- Very sudden onset (may suggest ischaemic optic neuropathy)
- Bilateral, severe disc swelling, or presence of macular star
- Painless, or very painful (may suggest inflammatory optic neuropathy)
- Additional ocular/ orbital/ systemic features or signs

Atypical clinical course
- Progressive worsening for more than 2 weeks and lack of recovery after 8 weeks[12]
- Rapid response to iv methylprednisolone or relapse when steroid tailed off (may suggest inflammatory cause)

There are also conditions which can mimic the clinical presentation of acute optic neuritis. These include other optic neuropathies (anterior and posterior ischaemic optic neuropathies; Leber hereditary optic neuropathy and compressive optic neuropathy) and other ocular conditions (e.g. acute zonal occult outer retinopathy). Making the correct diagnosis will prevent unnecessary and possibly harmful interventions in these patients.

Neuromyelitis Optica (NMO)

NMO is an autoimmune, inflammatory, demyelinating disease of the central nervous system (CNS) characterised by preferential involvement of the optic nerves and spinal cord. NMO could follow a monophasic or polyphasic course, of which the latter is more commonly seen.[13] The relapsing nature of NMO in the polyphasic form can lead to confusion with MS. There was previously debate of whether NMO is a variant of MS or other neuro-inflammatory disorders such as acute disseminated encephalomyelitis, or as a distinct disease entity. The more guarded clinical course, the neuropathological findings and the presence of the autoimmune antibody against aquaporin 4 (Anti-AQP4 or NMO-IgG, a type of water channel that is most abundant in the CNS), suggest that NMO is, in fact, a distinct clinical entity.

Diagnosis of NMO is currently based on the revised Wingerchuk criteria published in 2006 (Table 2).[14] This diagnostic combination was estimated to be 99% sensitive and 90% specific for NMO.[14]

Table 2. Revised Wingerchuk criteria for diagnosis of Neuromyelitis Optica (NMO)[14]

1. Optic neuritis
2. Acute myelitis
3. At least 2 of 3 supportive criteria
   - Contiguous spinal cord MRI lesion extending ≥ 3 vertebral segments
   - Brain MRI not meeting diagnostic criteria of MS
   - NMO-IgG seropositive status

Although clinically NMO mainly affects the optic nerves and spinal cord, cerebral MRI abnormalities were found in 60% of patients at their first relapse.[13] Most of them are non-specific and clinically silent. The distribution of brain lesions were found to be related to sites of high AQP4 expression, mainly localised in the hypothalamus, periaqueductal brainstem surrounding the ventricular system and sometimes extending into the cerebral white matter and cerebellum.[15]

NMO-IgG is a serum autoantibody that targets aquaporin 4, a type of water channel in the CNS responsible for brain water homeostasis and the maintenance of blood brain barrier (BBB). It is found in the astrocytic end feet abutting the capillaries in the CNS. NMO-IgG was found to be 76% sensitive and 94% specific for NMO. Recent evidence suggested that NMO-IgG may play an important pathogenic role in NMO by inducing an increase in permeability of BBB, complement cascade activation and astrocytic cytotoxicity.[13]

Differentiating between NMO and MS is very important, as they differ in optimum treatment and prognosis. From a neuro-ophthalmologist point of view, we shall focus our discussion here on the management of NMO-related optic neuritis.

Most authors agree that treatment of acute attacks of NMO-related optic neuritis is by high dose intravenous corticosteroids (methylprednisolone 1g/d for 3 to 5 days), initiated as soon as possible, followed by high dose oral steroid.[13] In patients with aggressive or

refractory disease unresponsive to steroid therapy, plasmaphoresis (5-7 exchanges over 2 weeks) may be considered.[13] With these treatment, about 60% of the patients recover moderately to markedly, depending on the time interval between attack onset and therapy initiation. Rituximab, an anti-CD20 monoclonal antibody, has also been shown to be effective in some patients with aggressive and otherwise therapy-resistance disease. Maintenance immunosuppression (e.g. azathioprine 2.5-3 mg/kg/day) should also be considered to reduce relapses.[13]

NMO carries a guarded prognosis and poor recovery rate in terms of visual outcome, Extended Disability Status Scale and with a higher mortality, especially if not treated promptly and appropriately. It was estimated that in patients with relapsing NMO, 50% of the patients have permanent severe visual loss (<20/200) or para / quadriplegia within 5 years, and mortality after 5 years was 17-32%, mainly due to respiratory failure.[13;16]

Optic perineuritis (OPN)

OPN is an uncommon inflammatory disorder affecting the optic nerve sheath and perineurium, characterised by visual disturbances with signs of optic nerve dysfunction.[17] It has been described to be secondary or related to neurosyphilis, Wegener's granulomatosis, sarcoidosis or Crohn's disease.[18-21] On the other hand, it is most commonly idiopathic and is considered to be a form of non-specific orbital inflammatory disease predominantly affecting the optic nerve sheath and perineurium.

The initial clinical presentation of OPN may mimic that of demyelinating optic neuritis and can cause diagnostic confusion.[22] In OPN, however, apart from pain and signs of optic nerve dysfunction, there may also be symptoms and subtle signs suggestive of orbital inflammation.[17] These include diplopia or signs like mild proptosis, ptosis or extraocular motility deficits. Presence of orbital signs in patients presenting with presumed optic neuritis suggests the possibility of OPN.

Neuroimaging of the brain and orbit is essential for the definitive diagnosis of OPN. MRI of the brain and orbit with contrast is the gold standard for diagnosis of OPN, which may show perineural enhancement around the optic nerve with 'dirty fat' sign (Figure 1). There may also be subtle enhancement of surrounding orbital structures including the extraocular muscles.[17;22] If MRI is not readily available, computed tomography (CT) with contrast may sometimes be able to show thickened optic nerve with surrounding streaky fat enhancement. (Figure 2).[17]

Management of non-infectious and idiopathic OPN requires prompt initiation of high dose oral (prednisolone 80 mg /day) or intravenous steroid (methylprednisolone 1 g/d for 3 days) with subsequent slow oral tapering.[17;22] Most patients experience a dramatic improvement in terms of pain reduction and visual improvement within 24 hours after initiation of treatment.[17;22] Poor treatment response and visual outcome are associated with delayed initiation of treatment.[17] Steroid should be tapered slowly to reduce subsequent relapse of the disease.[17;22] These clinical characteristics were different from that of typical demyelinating optic neuritis.

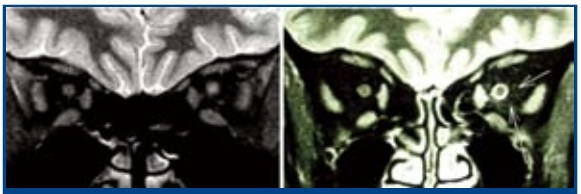

Figure 1. Magnetic resonance imaging (coronal short TI inversion recovery images) of the orbit in patients with (left) left optic neuritis showing hyperintensity of the optic nerve, and (right) left optic perineuritis showing perineural hyperintensity around the left optic nerve with dirty fat sign depicted with arrows. Note also the hyperintensity of the left medial rectus muscle.

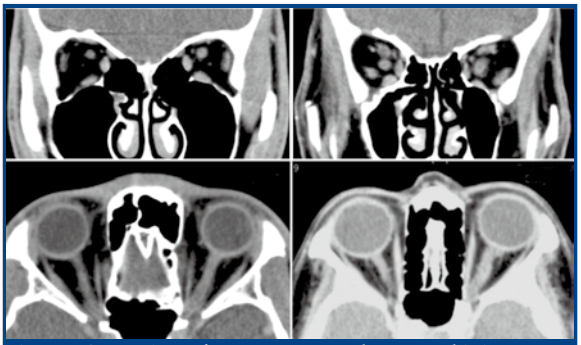

Figure 2. Comparison of contrast computed tomographic images of patients with (left panel) optic neuritis and (right panel) optic perineuritis. Coronal and axial scans of the patient with left optic neuritis show an enlarged and contrast-enhancing left optic nerve. In the patient with left optic perineuritis, there is streaky enhancement of perineural tissue (dirty fat sign) which is not evident in optic neuritis.

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What is the situation in Hong Kong?

Most of the older published studies on optic neuritis were based on non-Chinese populations (mainly Caucasian population). It is well known that, however, the clinical characteristics of optic neuritis in Chinese may be quite different from the Caucasian population. In view of this knowledge gap, our group has performed a retrospective case series on patients presenting with acute or subacute optic neuropathy in the Chinese population.[17]

In our series of Chinese patients presenting with acute / subacute optic neuropathy, 14% were found to be suffering from OPN and the others were optic neuritis.[17] Among the patients with optic neuritis, 68% were idiopathic optic neuritis, 28% were MS-related and 4% were NMO-related.[17] The rate of NMO-related optic neuritis is similar to another study in Chinese patients (4%) published by Zhang et al. in 2007.[23] These rates for OPN and NMO-related optic neuritis were higher than that in the Caucasian population.[17] Our findings suggested that in Chinese patients presenting with features suggestive of optic neuritis, it is important to consider the possibility of OPN and NMO-related optic neuritis as these optic neuropathies require prompt and prolonged course of treatment. Delayed or early cessation of treatment carries a high risk of severe permanent visual loss. This is in contrast to "typical" demyelinating optic neuritis, in which high dose intravenous steroid

treatment only hastens the initial visual recovery but does not improve the final visual outcome.[24]

Summary: management algorithm

Our suggested management algorithm is summarised in Figure 3. If a patient with suspected optic neuritis is first seen by a family physician or neurologist, referral to an ophthalmologist/ neuro-ophthalmologist is recommended to exclude other causes of visual loss. Thorough ophthalmic history taking and examination (specifically looking for signs of ocular inflammation and orbital signs) are required to elicit features which may suggest atypical optic neuritis. Chinese ethnicity may already be an atypical feature for optic neuritis which warrants further workup as suggested.[17] If no atypical features are present, theoretically no further investigations are necessary, although an MRI brain and orbit with contrast is desirable to rule out atypical causes and look for brain lesions (in case of typical or MS-related optic neuritis). Neuroimaging is necessary if atypical features are present. In situations where MRI may not be promptly available, a CT brain and orbit with contrast, though suboptimal for the investigation of optic neuritis, may be performed as a second choice. CT cannot reliably diagnose/ exclude optic neuritis, but it can detect large space occupying lesions/ sinusitis, and sometimes OPN can have characteristic features on CT as mentioned above. If a patient presents with clinical signs and symptoms very suggestive of optic neuritis and has severe visual loss, MRI is not available, but other investigations including contrast CT and blood tests excludes infection and other diagnoses; intravenous pulsed steroid therapy (ONTT regime) can be considered if there are no systemic contraindications. This will cover for typical optic neuritis, NMO, OPN and inflammatory optic neuropathies. Once the patient has been started on corticosteroids, very rapid response or a relapse of symptoms on tapering treatment may suggest an inflammatory aetiology (atypical optic neuritis) and this should prompt slower tapering of corticosteroids and work up for other causes of optic neuritis.

Conclusion

Whilst observation is an option for typical demyelination optic neuritis, delaying treatment may result in irreversible visual loss in infective/ inflammatory/ NMO-related optic neuritis and OPN. Chinese ethnicity itself may already be an atypical feature in a patient with optic neuritis, which prompts further investigations and a lower threshold for treatment.

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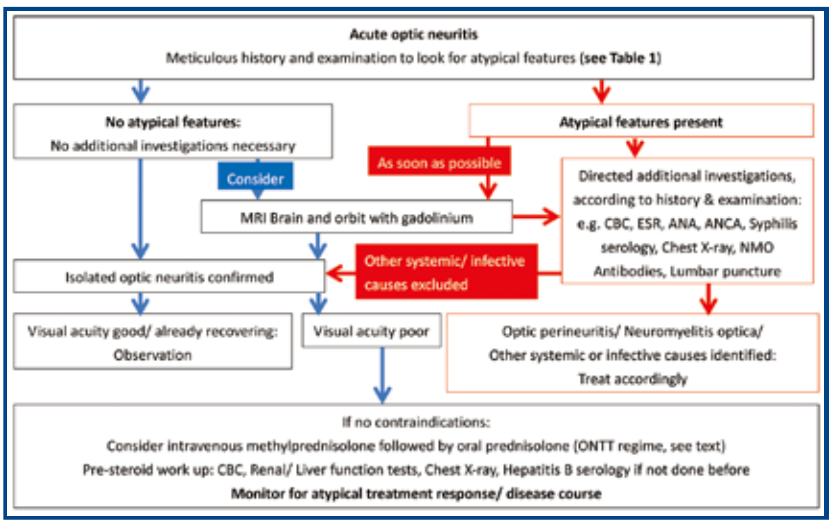

Figure 3: Suggested management flowchart of a patient who presents with symptoms of acute optic neuritis
Abbreviations: ANA= antinuclear antibodies, ANCA= anti-neutrophil cytoplasmic antibodies, CBC= complete blood count, ESR= Erythrocyte Sedimentation Rate, MRI=Magnetic Resonance Imaging, NMO=Neuromyelitis Optica, ONTT= Optic Neuritis Treatment Trial

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Advances in Refractive Surgery

Dr. Victor CP WOO

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Honorary Professor, Eye Institute, The University of Hong Kong

Dr. Victor CP WOO

Surgical options to correct refractive errors had been available for more than 30 years. Over the years, with further understanding of refraction changes of the human eye and many of the optical principles for perfect vision, together with the fast development in technology, we have been witnessing ever improving and evolving techniques now applicable in refractive surgeries for myopia, hyperopia, astigmatism and presbyopia.

Some of the techniques become obsolete after more advanced techniques are introduced. Hence we will focus on techniques that are most adopted with well supported literature or data and also touch on some new techniques in this article.

Most people would have heard of LASIK, this is the abbreviation for (Laser Assisted in-situ Keratomileusis). This is the most popular procedure nowadays. In USA, over 12 million patients had undergone the LASIK procedure since it was first approved. According to the report -Eye Health Statistics at a Glance released by the American Academy of Ophthalmology in 2009, more than 700,000 LASIK procedures were performed each year.[1] With the introduction of Femtosecond Lasers, the current standard of modern refractive surgery is FemtoLASIK which involves the creation of corneal flaps by Femtosecond laser followed by corneal stromal abrasion to eliminate the refractive error by excimer laser.[2] Advancements in FemtoLASIK in the last 10 years mainly involve higher speed machine and lower energy, which then offers more comfort for the patients and a smooth surgical interface which gives better result and vision quality.[3] The ablation profile of the excimer laser has also improved with standard aberration-free correction thus eliminating most of the night glare that was the most commonly reported adverse effects in earlier reports regarding LASIK.[4] Moreover, improvement in tracking system of the laser ablation enables more accurate correction of astigmatism. Figure 1.

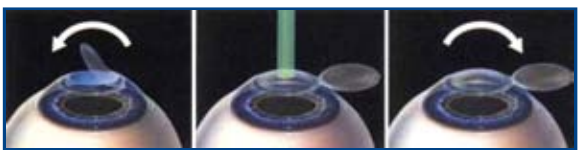

Figure 1: LASIK procedure (from right to left), lifting of superficial corneal flap, ablation of cornea according to refraction to be corrected, repositioning of the superficial corneal flap.

Recent advances also enable those who have entered the presbyopic age to benefit from LASIK by modifying either the Q value with an increased depth of focus or creating a multifocal cornea. This procedure has been named PresbyLASIK.[5] Figure 2.

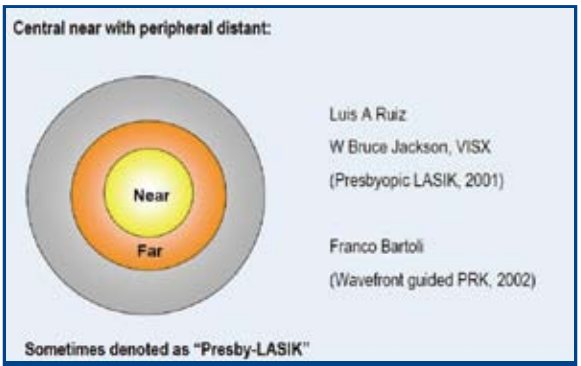

Figure 2: Presby-LASIK

Another means of correction of presbyopia is the introduction of corneal inlay with the assistance of Femtosecond LASIK creating a pocket and placing the inlay accurately in the pupil centre creating a pinhole effect and enabling reading without using accommodation.[6] Figure 3.

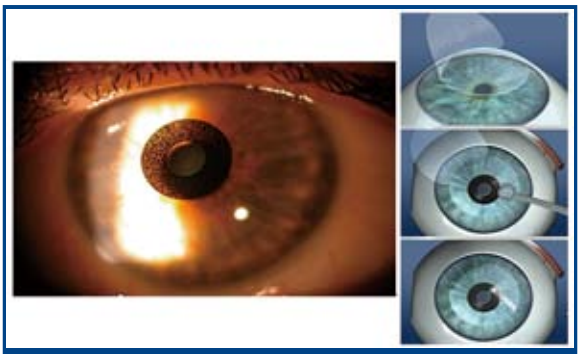

Figure 3: Corneal inlay

A new technology basing on the same principle of FemtoLASIK is the much improved technique of Femto laser keratomileusis. By passing accurately twice through the cornea at a calculated depth, a small lenticels of cornea can be extracted through a small opening and change the refractive power of the cornea. This procedure (named SMILE), has the advantage of elimination the need to manipulate the superficial

corneal flap as in LASIK and may avoid many flap-related complications. The refractive results await more data to be collected.[7]

The old technique of Photo-refractive Keratoplasty (PRK) has also made a step forward in the form of transPRK or no touch laser correction. Instead of making a flap the Excimer laser will blow off the epithelium and go straight to stromal ablation. Recovery will be slower than LASIK but is useful in patients with thin cornea and loose epithelium.[8]

For patients who are older and start to develop cataract, cataract extraction by phacoemulsification and small wound incision, followed by insertion of premium Intra-ocular lens (IOL) can now correct myopia, hyperopia, regular astigmatism and presbyopia. With the recent application of Femto laser in Laser cataract surgery, clear lens exchanges have been made a lot safer and accurate and can be used in patients not suitable for LASIK who are near the cataract age.[9] Figure 4.

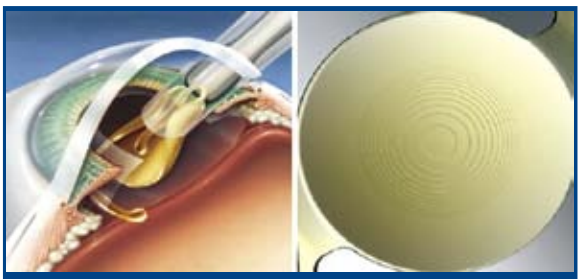

Figure 4: Left:Phacoemulsification of cataract and insertion of IOL, right: multifocal IOL

Phakic lens implant (i.e. inserting an extra IOL without taking out the patient's own lens) has been used in patients with high degree of myopia and hyperopia not suitable for LASIK for many years. The type of lens used has evolved from the anterior angle fixing lens to the iris clip lens to the now popular posterior chamber ICL (intraocular contact lenses).[10]

With advances in modern refractive surgery, we can now correct a much wider range of refractive errors, and these techniques can be applied in more complex cases and extended age range of patients to help them to remain spectacles free with good quality vision. Detailed assessment is needed in each individual case in consideration for various means of refractive surgeries. Pros and Cons of surgeries and expected vision quality will be discussed in consultations.

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Laser assisted *in-situ* keratomileusis (LASIK) can only be performed by a trained ophthalmologist and for specified reduction or elimination of myopia, hyperopia, and astigmatism as indicated within the product labeling. Laser refractive surgery is contraindicated for patients: a) with collagen vascular, autoimmune, or immunodeficiency diseases; b) who are pregnant or nursing women; c) with signs of keratoconus or abnormal corneal topography; d) who are taking one or both of the following medications: Isotretinoin (Accutane) and Amiodarone hydrochloride (Cordarone). Potential side effects to laser refractive surgery may include glare, dry eye, as well as other visual anomalies. LASIK requires the use of a microkeratome that cuts a flap on the surface of the cornea, potential side effects may include flap related complications. Patients are requested to consult with their eye care professional and *Patient Information Booklet* regarding the potential risks and benefits for laser refractive surgery, results may vary for each individual patient.

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Travel Photography

Dr. Raymond HS LAM

MBBS, DO, FCOphth.HK, FCSHK, FHKAM(Ophthalmology)
Specialist in Ophthalmology

Dr. Raymond HS LAM

Taking a good picture has never been easier. It's no longer necessary to twiddle with focus rings, nor to adjust the aperture or the shutter speed. No more light meters, no more selecting the most suitable type of films; and no need to drive yourself half demented calculating the aperture from the guide number for the flash you are using. Now everything is taken care of by your camera's automatic mode. A well- exposed, sharply- focused picture appears within a micro-second of your pressing the shutter release. But still some pictures look better than others. Everyone wants to know the essence behind taking that most elusive of delight: 'the perfect picture'.

Strong colours and patterns

Sometimes I find it difficult to imagine the number of places I have visited over the years. At the last count I have been to over 60 different countries. If I had lived a hundred years ago, I would have been counted the greatest traveller or explorer on Earth. With the development of fast modern transport, and communication, travelling has never been easier. The two combine to make today's travel photography a far more relaxed pursuit than it was a generation ago.

Light reflection

Travelling without a camera or anything capable of taking a picture, and this includes the ubiquitous mobile phone, is, I feel, a grave mistake. How else is one to review and relive the wonderful moments of a trip? How else to experience the joy of gathering with family and friends to pore over photo albums? This is also the time to showcase your photographic talent and skill and indeed to gently revel in the delight and surprise that your photographs afford. So, do you think you would like to learn a little more about travel photography?

Animals

There are many more doctors who are better photographer than I, but, after over 40 years of photographic experience (I started as a toddler) I know a few tricks that are useful in making a picture look better.

All things start with good preparation. Plan your trip well and make sure you are going at the right time of the year, so that you will not miss what you are looking for nor be hampered by adverse weather conditions. You cannot bring along everything you have, and so you should carefully select those pieces of equipment that you handle best. Having said this, I personally would never miss the chance of buying a new lens or camera that might give me the edge on taking even better photographs. It is always worthwhile to read up about the places you intend to visit, making a note of locations which you feel might be more picturesque .Books with lots of pictures are most useful for this purpose. Studying postcards can also be very useful both for how and where to take a photograph as well as how not. When you study a picture, pay special attention to the season it was taken, the time of the day, the direction of light and the angle of view. All these can make a great deal of differences to your own eventual picture.


A different viewpoint

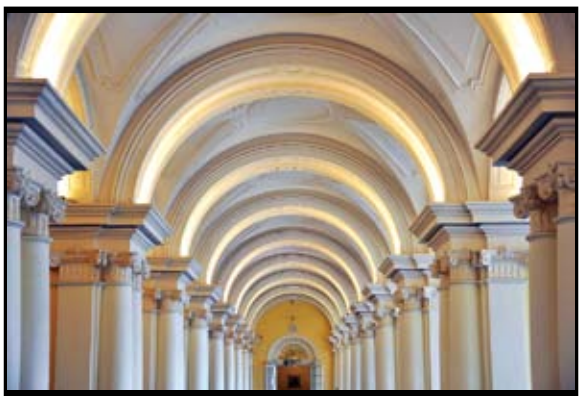

Landmarks

Arriving at a new and strange place, the temptation to shoot almost everything is well-nigh irresistible. But try to make the subject in your picture stands out. Eyes get tired easily wandering around the picture, wondering what one should be looking for. Our old teaching was to crop tightly, but now you can always do this on your computer later. So I say, ask you wife and children to wear red and this always works. To defocus the background, you need a lens with a large aperture and that would be very expensive and heavy to carry.


Children are willing to pose

People in local costume or people at work or leisure can be very interesting subjects. Start a dialogue and it will make things go much more smoothly, because cameras, especially big ones, can be rather intimidating. Children are usually more than willing to pose for you. When taking pictures of children try to set your camera at their eye level. With the high ISO sensor of today's cameras, flash is now rarely my choice of light source. It destroys the atmosphere and flattens the features.

Architectures tell us about the history of a place and are usually the best representation of the places you have visited. Use your imagination and try to shoot from different angles and if possible return at different time of the day if you think that the location is special, or a landmark that you just cannot miss. Save some time for the interior of beautiful architecture or you will regret that afterwards. Make use of light reflection from rivers, the lake or any nearby pool of water. When shooting landscape try to place something of interest in the foreground if at all possible. This increases the depth perception and helps to guide the eye into the main subject. Inclusion of a local inhabitant in the landscape usually tells a better story. Framing the picture with arches or whatever you find about you is also a good idea.

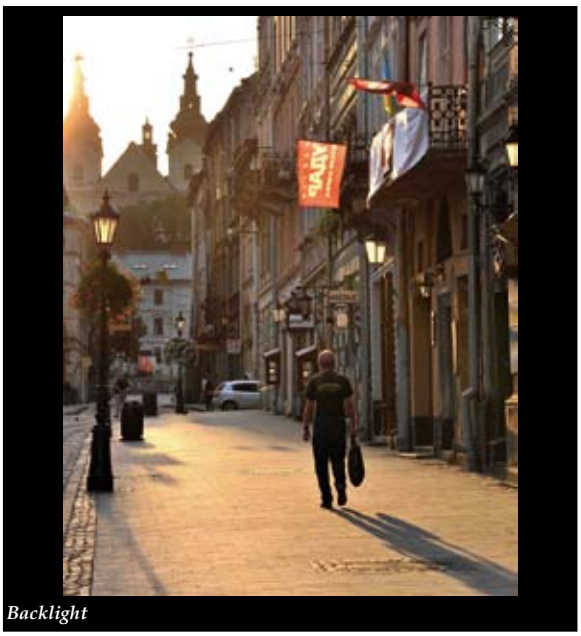

Symmetry is pleasing


Frame your picture

Sunrise and sunset give the most picturesque shots, and so be prepared to sacrifice your sleep to get up early. With the new digital technology, night scenes are no longer a problem, even without a tripod, but I would certainly carry one. The best night scenes are taken just before the sky gets really dark, at twilight when the colours and shadows are richly crepuscular. But don't leave after the sun has set, for this is when the sky is going to present her most interesting colours. Dawn and dusk light have a colour quality that can be unpredictable and consequently exhilarating.

Objects displayed in shops or markets are attractive subjects too. They also serve to identify a place. Look for details and a series of these items make the album much prettier. Animals, food, still-life can all be interesting subjects. Pay special attention to strong colours and patterns: symmetrical patterns are usually pleasing. Bad weather or back lighting is not always a problem. Sometimes you may find that an overcast sky, or a downpour provides shots with the best mood. Indeed, overcast weather is often best for portraitures and plant photography since harsh light is eliminated. Light reflection from the water-saturated ground or a surface after rainfall can give amazing effects in night scenes.

Sunrise and sunset



Backlight

Still life

Rainy days

There are no set rules for a good picture. With imagination, creativity and practice everyone can be a good travel photographer and one soon realises how much more fun is added to your trips when you have a camera in hand.

Foreground interest

Start a dialogue before shooting

Before the sky is dark

The right season

Federation News

New Member

The Hong Kong Council of Social Service

Official Representative :
Ms Ada IP, Director (Human Resources and Administration)
13/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, HK
Tel : 2864 2911 Email : ada.ip@hkcss.org.hk

Introducing HKCSS

The Hong Kong Council of Social Service is an umbrella organisation representing the non- governmental social service sector, with around 400 Agency Members, that provide over 90% of the social welfare services for those in need through their 3,000 service units all over Hong Kong.

Our Position

The Council represents, with clear vision and professional expertise, non-governmental social service agencies committed to sustain and develop social services in Hong Kong.

Our Values

The Council and its member agencies believe in social justice and equality, and the intrinsic rights of every individual. While society is obliged to provide individuals with the basic social and economic resources to develop their potential, individuals in turn should carry out their responsibilities towards their families and society, to be self-reliant and to achieve self-actualisation.

Our Vision

To build a social service sector that is highly accountable, efficient, effective and responsive to social needs, upholding the long-term sustainable development of society and the well-being of our citizens.

Our Mission

To promote the development of social welfare together with its member agencies, through:

- Enhancing accountability of social welfare service agencies;
- Promoting improvement of social welfare services;
- Facilitating agencies to better serve the community;
- Advocating equality, justice, social integration and a caring society;
- Setting the local welfare sector as a model of excellence in the international community

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
		1	2	3	4 *Joint Surgical Symposium- Periodic PR Bleeding	5
6	7 *A Young Lady with Bladder Tumor	8 *FMSHK Officers' Meeting *HKMA Council Meeting	9 *Hong Kong Neurosurgical Society Monthly Academic Meeting –Low Grade Glioma	10 *HKMA Kowloon East Community Network - Allergic Rhinitis & Asthma: An Update *HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2013 – Latest Development in the Treatment of OA Knee	11	12 *Refresher Course for Health Care Providers 2012/2013
13	14	15 *HKMA Kowloon City Community Network- Advance in Rheumatological Practice	16 *HKMA Yau Tsim Mong Community Network- Latest Update in Gastro-esophageal Reflux Disease Treatment	17	18	19
20	21	22	23	24 *International Colorectal Disease Symposium *HKMA Kowloon East Community Network- New Directions in Managing Type 2 Diabetic Patients *Clinical Meeting (1) A Constant Guest is Never Welcome (2) A Patient with Blood-stained Sputum *FMSHK Executive Committee Meeting	25 *International Colorectal Disease Symposium *HKMA Yau Tsim Mong Community Network - Allergic Rhinitis & Asthma: An Update	26 *HKMA 8th Sports Night
27	28	29	30 *HKMA Central, Western & Southern Community Network - Update on Gout Management	31 *HKFMS Foundation Committee Meeting		

Date / Time	Function	Enquiry / Remarks
4 FRI 8:00 am	**Joint Surgical Symposium- Periodic PR Bleeding** Organiser: Department of Surgery, the University of Hong Kong & Hong Kong Sanatorium & Hospital, Chairman: Prof. Law Wai-Lun, Speakers: Dr. Angus CHAN & Dr. Jenson POON, Venue: Hong Kong Sanatorium & Hospital	Department of Surgery, Hong Kong Sanatorium & Hospital Tel: 2835 8698 1 CME point
7 MON 7:30 pm	**A Young Lady with Bladder Tumor** Organiser: Hong Kong Urological Association, Chairman: Dr. Ning-hong CHAN, Speaker: Dr. Jeremy TEOH , Venue: Multi-disciplinary Simulation and Skills Centre, 4/F, Block F, QEH	Dr. Hing-hoi HUNG Tel: 2958 6006 1 CME point
8 TUE 8:00 pm	**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
8:00 pm	**HKMA Council Meeting** Organiser: The Hong Kong Medical Association, Chairman: Dr. TSE Hung Hing, Venue: HKMA Head Office (5/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong)	Ms. Christine WONG Tel: 2527 8285
9 WED 7:30 am	**Hong Kong Neurosurgical Society Monthly Academic Meeting –Low Grade Glioma** Organiser: Hong Kong Neurosurgical Society, Chairman: Dr. PO Yin Chung, Speaker: Dr. CHAN Ngo Lun, Allan, Venue: Seminar Room, Ground Floor, Block A, Queen Elizabeth Hospital	Dr. Gilberto LEUNG Tel: 2255 3368 1.5 CME points
10 THU 1:00 pm	**HKMA Kowloon East Community Network - Allergic Rhinitis & Asthma: An Update** Organiser: HKMA-Kowloon East Community Network, Chairman: Dr. AU Ka Kui, Gary, Speaker: Dr. CHAN Hing Sang, Venue: Lei Garden Restaurant, Shop No. L5-8, APM Millennium City 5, 418 Kwun Tong Road, Kwun Tong	Ms. Candice TONG Tel: 2527 8285 1 CME point
2:00 pm	**HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2013 – Latest Development in the Treatment of OA Knee** Organiser: The Hong Kong Medical Association, Speaker: Dr. WU Wing Cheung, Stephen, 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
12 SAT 2:30 pm	**Refresher Course for Health Care Providers 2012/2013** Organiser: The Hong Kong Medical Association, Speaker: Dr. Chiu Ying Wah, Venue: Training Room II, 1/F, OPD Block, Our Lady of Maryknoll Hospital, 118 Shatin Pass Road, Wong Tai Sin, Kowloon	Ms. Clara Tsang Tel: 2354 2440 2 CME points
15 TUE 1:00 pm	**HKMA Kowloon City Community Network- Advance in Rheumatological Practice** HKMA Kowloon City Community Network- Advance in Rheumatological Practice Organiser: HKMA Kowloon City Community Network, Chairman: Dr. CHIN Chu Wah, Speaker: Dr. TSUI Hing Sum, Kenneth, Venue: Sportful Garden Restaurant, 2/F, Site 6, Whampoa Garden, Wonderful Worlds of Whampoa, 8 Shung King Street, Hung Hom	Ms. Candice TONG Tel: 2527 8285
16 WED 1:00 pm	**HKMA Yau Tsim Mong Community Network- Latest Update in Gastro-esophageal Reflux Disease Treatment** Organiser: HKMA Yau Tsim Mong Community Network, Speaker: Dr. LAI Kam Chuen, Venue: Pearl Ballroom, Level 2, Eaton Smart, Hong Kong380 Nathan Road, Kowloon	Ms. Candice TONG Tel: 2527 8285
24 THU (25)	**International Colorectal Disease Symposium** Organisers: Minimal Access Surgery Training Centre, Hong Kong Society for Coloproctology & Dept of Surgery, PYNEH, Chairman: Dr. Hester CHEUNG, Venue: Pamela Youde Nethersole Eastern Hospital	Symposium Secretariat Tel: 2595 6362
1:00 pm	**HKMA Kowloon East Community Network- New Directions in Managing Type 2 Diabetic Patients** Organiser: HKMA Kowloon East Community Network, Chairman: Dr. MA Ping Kwan, Danny, Speaker: Dr. YEUNG Chi Kin, Venue: East Ocean Seafood Restaurant, Shop 137, 1/F, Metro City Plaza 3, 8 Mau Yip Road, Tseung Kwan O, Kowloon	Ms. Candice TONG Tel: 2527 8285 1 CME point
6:30 pm	**Clinical Meeting (1) A Constant Guest is Never Welcome (2) A Patient with Blood-stained Sputum** Organiser: Hong Kong Thoracic Society & American College of Chest Physician (HK & Macau Chapter), Chairmen: Dr. Wing-ching WONG & Dr. King-ying WONG, Speakers: Dr. Lai-yun NG & Dr. Ching-ho SZETO, Venue: LG1 Lecture Room, Ruttonjee Hospital	Dr. Fanny KO Tel: 2632 2785 1.5 CME points (HKCP) 2 CME points (HKCFP) 1.5 CME points (CSHK)
8:00 pm	**FMSHK Executive Committee Meeting** Organiser: The Federation of Medical Societies of Hong Kong, Venue: Council Chamber, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
25 FRI 1:00 pm	**HKMA Yau Tsim Mong Community Network - Allergic Rhinitis & Asthma: An Update** Organiser: HKMA Yau Tsim Mong Community Network, Speaker: Dr. YIP Kim Kwong, Gary, Venue: Jade Ballroom, Level 2, Eaton Smart, Hong Kong, 380 Nathan Road, Kowloon	Ms. Candice TONG Tel: 2527 8285 1 CME point
26 SAT 7:00 pm	**HKMA 8th Sports Night** Organiser: The Hong Kong Medical Association, Venue: Wanchai Ho Choi Banquet and Seafood Restaurant	Ms. Dorothy KWOK Tel: 2527 8285
30 WED 1:00 pm	**HKMA Central, Western & Southern Community Network - Update on Gout Management** Organiser: HKMA Central, Western & Southern Community Network, Chairman: Dr. LAW Yim Kwai, Speaker: Dr. LEE Ka Wing, Gavin, Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	Ms. Candice TONG Tel: 2527 8285
31 THU 8:00 pm	**HKFMS Foundation Committee Meeting** Organiser: The Federation of Medical Societies of Hong Kong, Venue: Council Chamber, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898

Upcoming Meeting

30-31/3/2013	**9th CT Coronary Angiography Teaching Course 2013 – Intermediate & Advanced Level** Organiser: Hong Kong College of Radiologists, Speakers: Dr. Szilard Voros, Dr. Stephen Cheung, Dr. Jack Shu, Dr. Sonny Chiu & Dr. John Hoe, Venue: Pamela Youde Nethersole Eastern Hospital, Chai Wan, Enquiry: Ms. Lai Wai Yee, Ada Tel: 2871 8788
10-13/7/2013	**9th Asian Dermatological Congress 2013** Organisers: Asian Dermatological Association, Hong Kong College of Dermatologists & the Hong Kong Society of Dermatology and Venereology, Chairman: Prof. Henry HL CHAN, Venue: Hong Kong Convention & Exhibition Centre, Enquiry: ADC 2013 Secretariat Tel: 3151 8900

Answer to Dermatological Quiz

1. Pityriasis rubra pilaris (PRP), classic adult type, with erythroderma

2. PRP is a chronic papulosquamous dermatosis of elusive aetiology, characterizsed by reddish scaly plaques with orange hue (Fig. 1 & 2), palmoplantar keratoderma, and keratotic follicular papules. This patient had progressed to the erythrodermic stage with distinct well demarcated areas of uninvolved skin ("islands of sparing") (Fig. 2). The onset of classic PRP can be acute and typically spreads in a craniocaudal direction. Pruritus is only present in about 20% of cases. The diagnosis is usually based on a correlation between the clinical features and histopathological pattern.

3. The main differential diagnosis is psoriasis, and in practice it is often misdiagnosed & treated as such. The distinct red-orange hue (especially at palmoplantar keratoderma), the classical "islands of sparing", the keratotic follicular papules with a nutmeg grater appearance, and a histopathological finding of alternating vertical and horizontal orthokeratosis and parakeratosis are useful features to distinguish it from psoriasis. Other differential diagnoses include erythrokeratoderma variabilis, and other causes of erythroderma such as drug reaction, atopic dermatitis and cutaneous T-cell lymphoma.

4. In general, the treatment options of PRP are similar to those of psoriasis, but often more recalcitrant and difficult to manage. In contrast to psoriasis, topical corticosteroids usually have little therapeutic effects in PRP and phototherapy is also less effective. In severe case, the treatments of choice are oral retinoid (oral isotretinoin or acitretin) or low dose methotrexate. Even if the responses to treatments are unsatisfactory, the acquired classic adult type may resolve spontaneously within 1-3 years. However, the familial form of the disease may possibly persist for life.

Dr. Lai-yin CHONG
MBBS(HK), FRCP(Lond, Edin, Glasg), FHKCP, FHKAM(Med)
Private Dermatologist

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NEW **DEXILANT** dexlansoprazole

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- Effective across the spectrum of GERD[3,4]
- Maintain long-term healing and therefore quality of life[5,6]
- Lifestyle-friendly PPI: once daily, taken with or without food[1,7,8]

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Presentation: Dexilant 30mg and 60mg capsules. Indication: Healing of all grades of erosive esophagistis (EE) for up to 8 weeks, maintenance of healed erosive esophagistis for up to 6 months, treatment of heartburn associated with symptomatic non-erosive gastroesophageal reflux disease (GERD) for 4 weeks. Dosage and administration: 60mg once daily for up to 8 weeks for healing of EE, 30mg once daily for maintenance of healed EE, 30mg once daily for 4 weeks for symptomatic non-erosive GERD. Dexilant can be taken without regard to food and should be swallowed in whole. Contraindication: contraindicated in patients with known hypersensitivity to any component of the formulation. Interaction: atazanavir, ampicillin esters, digoxin, iron salts, ketoconazole, warfarin, Tacrolimus. Precaution: gastric malignancy. Adverse reaction: diarrhea, abdominal pain, nausea, upper respiratory tract infection, vomiting, flatulence.

Reference:1. Dexilant prescribing information (DEX051 HK1 TT, HK Branch). 2. Wittbrodt ET et al., Clin Exp Gastroenterol 2009;2:117-28. 3. Fass R et al., Aliment Pharmacol Ther 2009;29:1261-72. 4. Sharma P et al., Aliment Pharmacol Ther 2009;29:731-41. 5. Metz DC et al., Aliment Pharmacol Ther 2009;29:742-54. 6. Howden CW et al., Aliment Pharmacol Ther 2009;30:895-07. 7. Lee RD et al., Aliment Pharmacol Ther 2009;29:824-33. 8. Lee RD et al., Aliment Pharmacol Ther 2010;31:1001-11.

For further information, consult full prescribing information.

* 96% of patient on Dexlansoprazole 60mg achieved 24-h heartburn-free days[6]

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