Terrace Eye Centre

newsletter

MARCH **2017**

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Terrace Eye Centre welcomes Dr Sing-Pey Chow

Dr Sing-Pey Chow (MBBS (Hons) BMedSc FRANZCO) **is an Ophthalmologist with sub-specialty fellowship training in the management of corneal and anterior segment diseases, including corneal transplantation, cataract surgery, pterygium surgery and refractive surgery.**

She graduated from medical school with Honours at the University of Melbourne in 2006, and completed her Ophthalmology training at the Royal Victorian Eye and Ear Hospital in Melbourne. She then completed another three years of sub-specialty training in corneal and anterior segment diseases in the United Kingdom at Bristol Eye Hospital and Moorfields Eye Hospital before returning home to Australia via an observership at the Edward S. Harkness Eye Institute, Columbia University in New York.

She has been an invited speaker at international conferences including World Cornea Congress and European Society of Cornea and Ocular Surface Disease Specialists (EuCornea), and her work has been published in prestigious peer-reviewed journals including *Ophthalmology* and *Cornea*. She has also recently been awarded the Association for Research in Vision and Ophthalmology (ARVO) International Travel Grant in 2016 for her work on outcomes following corneal transplantation.

She is a Fellow of the Royal Australian and New Zealand College of Ophthalmologists (RANZCO) and a member of the Cornea Society. She is also a visiting Consultant Ophthalmologist at the Royal Brisbane and Women's Hospital.

Availability:

Dr Chow realises that vision and eye health are an important priority and will endeavour to see patients within 1–2 weeks of a new referral. She operates regularly at Queensland Eye Hospital.

We have moved

Terrace Eye Centre has moved into our brand new rooms just a few doors down at Level 2, 87 Wickham Terrace.

The move has increased our floor space by fifty percent, providing more space for our two new doctors, new equipment such as high resolution Heidelberg Spectralis OCT angiography and new improved waiting facilities for patients and their families. All of the staff and doctor partners would like to sincerely thank our referrers who have supported Terrace Eye Centre over the past forty years. We are very excited with these improvements that will enable us to continue providing the highest possible service to your patients into the future.

IN THIS ISSUE

Dr Michael C Hogden BSc MBBS (Hons) FRANZCO | Vitreoretinal Surgeon and Medical Retinal Specialist

What is a retinal detachment?

The retina is the delicate neural tissue layer that lines the inner wall at the back of the eye, and is responsible for the detection and basic processing of visual stimuli.

The retinal pigment epithelium (RPE) lies beneath the retina. The RPE and retina are closely held together by three cohesive forces: (i) An active suction force derived from fluid being pumped from the potential subretinal space; (ii) direct interdigitation between the RPE cells and photoreceptors, and (iii) 'glue-like' extracellular matrix elements within the subretinal space.

A retinal detachment occurs when these forces are overcome and fluid begins to accumulate under the retina, separating it from the RPE.

Most retinal detachments are caused by a break in the retina, which is termed a *rhegmatogenous retinal detachment*. Typically, breaks occur in the peripheral retina when the vitreous gel begins to degenerate with age and separates from the retina, forming a *posterior vitreous detachment* (PVD). Alternatively, the retina can also be detached by traction from scarring on the surface of the retina, such as that in proliferative diabetic retinopathy or following previous retinal detachment repair, called proliferative vitreoretinopathy (PVR). Finally, retinal detachment may also occur through the exudation of fluid into the subretinal space due to choroidal inflammation or tumours.

'Floaters and flashes'

The development of a PVD, retinal tear or retinal detachment is often heralded by symptoms of flashes of light (photopsias) and increased floaters. The photopsias accompanying a PVD or retinal detachment are characterized by a brief series of flashes, or white arc-shaped 'lightning', commonly in the temporal visual field. These unusual visual sensations are produced through mechanical traction on the retina by the separating vitreous gel.

Floaters are commonly described as dots, strands or cobwebs which move freely across the vision and are most noticeable when looking at a plain background, such as a white wall or blue sky. They are caused by vitreous opacities casting shadows on the retina. Hence the appearance of photopsias, or a sudden increase in floaters may indicate the development of a posterior vitreous detachment and necessitates a careful dilated retinal examination to check for retinal tears or detachment. Other causes of floaters include vitreous haemorrhage and uveitis (intraocular inflammation).

Who is at risk?

In the absence of ocular trauma or a high myopic refractive error (short sightedness), a PVD is rare before the age of 40. Its incidence increases with age, and by age 70, over 60% of people will develop the condition (Foos and Wheeler, 1982). The risk of a retinal detachment therefore also rises with age, with an overall incidence of around 10/100,000 (Saidkasimova et al., 2009).

Generally, people with a PVD do not develop problems. However, when a PVD is accompanied by significant symptoms such as floaters and flashes, the likelihood of developing a retinal tear ranges between 10–26% (van Overdam et al., 2001; Sharma et al 1999; Hikichi and Trempe, 1994; Coffee et al., 2007). Also, for those with a PVD with a symptomatic flap tear, up to 50% may later develop a retinal detachment (Neumann and Hyams, 1972).

Apart from the development of a PVD, other important risk factors for retinal detachment include cataract surgery, myopia, trauma, previous retinal detachment and genetic predisposition, such as those with Sticklers syndrome.

Diagnostic features of retinal detachment

A progressive visual field defect, commonly described as a 'curtain' or 'dark shadow' that develops in the periphery and extends towards the central vision, is the telltale symptom of a retinal detachment. Visual acuity often becomes affected when subretinal fluid accumulates and reaches the macula. The central vision may also be reduced by haemorrhage and pigment liberated within the vitreous cavity.

When the retina is detached, it becomes oedematous and looks wrinkled and more opaque. Careful inspection of the retina is necessary to reveal the cause of the detachment, which will often be a retinal flap tear, atrophic retinal hole or an area of retinal traction.

Who and when to refer?

Patients with a retinal detachment should be immediately referred to a facility that can offer surgical repair by a vitreoretinal specialist. Patients who are reporting flashes and floaters should be referred to their nearest Ophthalmologist or Optometrist for a comprehensive ophthalmic examination. If the vision is blurred or a field defect has developed, patients should ideally be seen on the same day.

How is a retinal detachment treated?

1. Retinal detachment

The most common procedure for surgical repair of retinal detachment is a vitrectomy with gas tamponade. This is an intraocular procedure, using specialized equipment to remove the vitreous gel and relieve traction on the detached retina, allowing the retina to re-attach to the RPE. It is particularly suitable for those who have a posterior vitreous detachment, had previous cataract surgery, and those with significant blood or debris in the vitreous.

Scleral buckling is an alternative technique for patients with retinal dialysis following trauma, patients without a PVD, and in young myopic patients with round atrophic retinal holes. Unlike vitrectomy, scleral

buckling does not promote the development of cataract. It involves sewing a prosthesis made of silicone rubber or sponge onto the sclera to alter the shape of the eye.

Both vitrectomy and scleral buckling procedures work by apposing the retinal breaks to the underlying RPE and applying either cryotherapy or laser to produce permanent adhesion. In cases with poor prognosis or repeat operations, silicone oil can be used instead of gas, and scleral buckling can be combined with vitrectomy.

2. Retinal breaks

If a retinal break (tear, hole or dialysis) is surrounded by little or no subretinal fluid, it should promptly be surrounded by laser photocoagulation to prevent the spread of subretinal fluid and development of symptomatic retinal detachment.

Post-operative care for retinal detachment

Following retinal detachment surgery, patients are routinely prescribed topical steroids and antibiotics. The patient is usually advised to restrict movement in the early post-operative period and will have been provided with specific instructions to posture.

The gas tamponade inside the eye causes poor vision until it clears. The patient will often be able to see a fluid level in their vision that gradually moves lower in the visual field as the gas subsides.

Patients are not allowed to travel by air or to high altitudes whilst their eye contains gas, as the increased altitude causes the gas to expand and will raise intraocular pressure. This can result in severe ocular pain and cause a central retinal artery occlusion and permanent visual loss.

The most common complication of retinal detachment surgery is re-detachment. Deterioration in vision after an initial improvement, or the development of an enlarging visual field defect, requires immediate examination by the vitreoretinal surgeon. Vitrectomy is also not generally painful. Symptoms of pain, particularly if associated with reduced vision, should also be promptly reported.

Scleral buckling surgery can be uncomfortable for the first few weeks following surgery and may also be associated with early post-operative double vision. Implant exposure or infection is a potential long-term complication, so unilateral conjunctivitis is an eye with a scleral buckle necessitates an urgent referral.

When to refer to an Ophthalmologist: Red flags and the 'Golden Eye Rules'

Dr Sing-Pey Chow MBBS (Hons) BMedSc FRANZCO | Cornea, Cataract and Refractive Surgeon

A patient comes to you with a painful red eye for the past three days. What associated factors should make you concerned? When should you refer them to see an Ophthalmologist?

Australian Ophthalmologist Dr John Colvin AM (1929–2005) first developed thirty-five 'Golden Eye Rules' to teach medical students, and his lectures were legendary for the use of trumpets, megaphones, a gong and bongo drums to herald each teaching point! These principles have since been taught worldwide, and I have summarised them into five practical points below:

1. **Beware of the unilateral red eye.**
   This can sometimes be due to a serious underlying ocular condition such as a corneal ulcer or infection, intraocular inflammation (uveitis) or acute angle closure glaucoma. This is particularly important if the patient is a contact lens wearer, or has had recent trauma.
2. **Any blurred vision always requires prompt investigation.**
   Flashes, floaters and visual field defects may be signs of a retinal detachment, and prompt treatment can lead to better outcomes. There are three vitreo-retinal surgeons at Terrace Eye Centre, ensuring that there is always someone available for retinal emergencies throughout the year.
3. **Refer any squint (strabismus) or new onset diplopia.**
   This could signal an underlying cranial nerve palsy, or an intraocular or orbital pathology, and warrants prompt investigation. Any child with a squint or 'lazy eye' should be assessed by an Ophthalmologist, as early treatment of amblyopia is important in preventing visual loss.
4. **Beware of herpes zoster ophthalmicus (HZO) if the nose is involved.**
   This is because the nasociliary branch of the trigeminal nerve innervates the cornea as well as the tip and side of the nose. Any patient with HZO and ocular symptoms should have a comprehensive eye examination.

5. **Topical steroids can be dangerous.**
   It should never be initiated in a patient with suspected herpetic eye disease without first being seen by an Ophthalmologist, as this can lead to corneal perforation. Other potential side effects include cataract formation, raised intraocular pressure or glaucoma, and worsening of fungal corneal infections.

We are always happy to see any patient with a visual or ocular concern, and it is always better to seek a specialist assessment rather than wait and worry about potentially missing a serious diagnosis. We have appointments available for patients to be seen at short notice.

Oncogenomics UM Synopsis

Dr Antonia Pritchard | Senior Research Officer | Oncogenomics Group

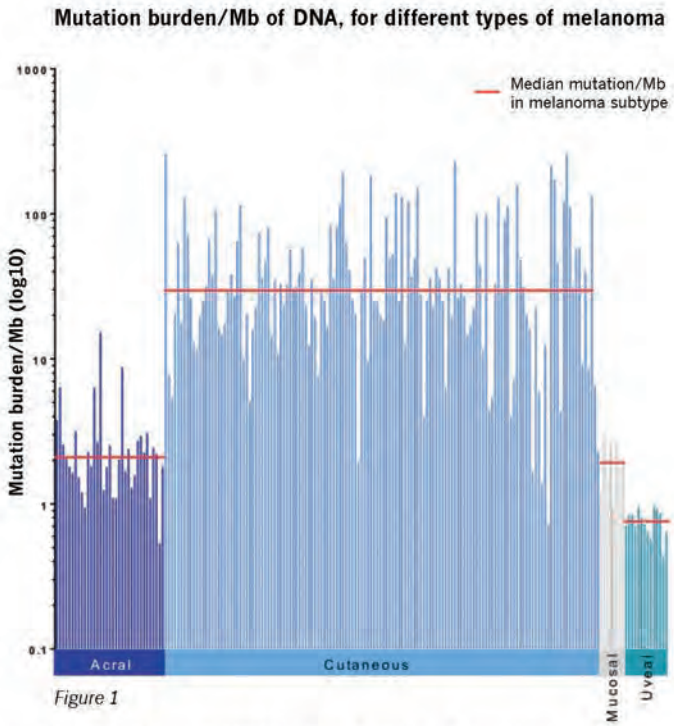

Figure 1

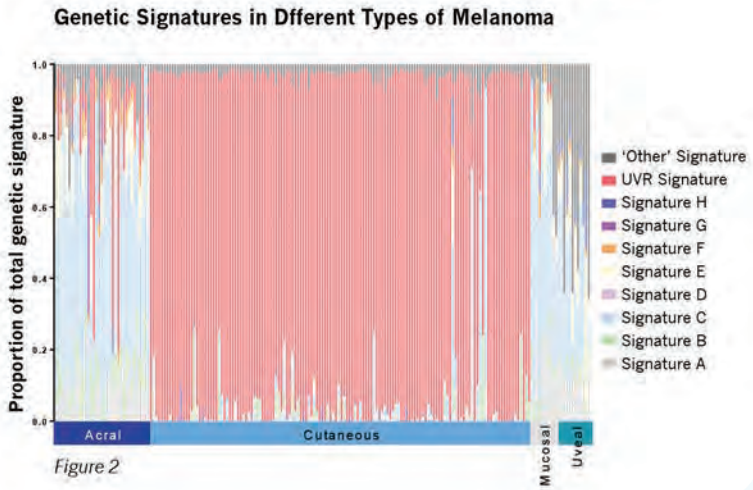

Figure 2

Since 2012, a multi-centre team consisting of Prof. Nicholas Hayward's Oncogenomics group at the QIMR Berghofer Medical Research Institute and Queensland Ocular Oncology Service clinicians, Drs William Glasson, Sunil Warrier and Sidney Finnigan, have undertaken research into the genetics of uveal melanoma (UM).

The previously described driver mutations controlling tumourigenic growth are in genes *GNAQ, GNA11, EIF1AX, SF3B1*; additionally, loss of the *BAP1* gene is known to be a strong driver of metastasis. Recently, we identified a new driver mutation, in a gene called *PCLB4*. Together these findings mean that only a very small fraction of UM have no identifiable tumourigenic driver gene.

In a world first, we have performed comprehensive analysis of the number and types of mutations compared between the four subtypes of melanoma; acral, cutaneous, mucosal and uveal. These molecular analyses have revealed that UM has the lowest number of acquired mutations, while cutaneous melanoma has the highest (Figure 1). Further in-depth characterisation of the nature of these mutations involves examining the exact DNA basepair change and the sequence context they occur in, known as a mutation 'signature'. These can be used to identify causes of DNA changes in the tumours. Our analyses reveal that the majority of cutaneous melanoma mutations are driven by the ultra-violet radiation (UVR) signature (Figure 2), which is directly linked to the high number of acquired variants observed (Figure 1). Uveal, as well as mucosal and the majority of acral melanomas do not have a UVR mutation signature (Figure 2), confirming that these melanoma subtypes are not usually driven by UVR exposure.

The next major aim of our research is to identify UM specific molecular features that can be targeted therapeutically to treat this disease, which, particularly once metastasised, is highly resistant to current treatment options.

Ocular Oncology Unit

As part of the Terrace Eye Centre for over two decades, Queensland Ocular Oncology has looked after patients from all over Queensland, Northern New South Wales and the Northern Territory.

This service, under the care of Dr Bill Glasson, Dr Sunil Warrier and Dr Sid Finnigan, assesses all patients with intraocular tumours or surface tumours across a broad spectrum. Common intraocular tumour referrals include suspicious choroidal naevi through to large choroidal melanomas. On average, the service treats between 150-200 choroidal lesions per year with laser, brachytherapy or enucleation.

Dr Bill Glasson will be taking a sabbatical this year for seven weeks from June to July, with the aim of visiting a number of world-renowned oncology centres to gain insight into the most up to date treatments for ocular tumours. Dr Sunil Warrier, who joined our team in the last few years, has provided increased capacity for the service to handle referrals in a timely fashion. Dr Warrier trained in Liverpool and brings with him special expertise that has enhanced the ocular oncology service. Dr Sid Finnigan, who is based primarily at Redlands, continues to form a vital part of our Friday Oncology clinic which focuses on the comprehensive assessment of all new patients.