

CME

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Written by Dr Lee Shu Yen

"Academic medicine" is now the buzz word. We have been hearing snippets about how the Ministry of Health has played matchmaker and sealed the union between SingHealth with the Duke-NUS Graduate Medical School, to form an Academic Medical Campus (AMC) in the Outram campus. This has led to the oft asked question - "What is Academic Medicine?" and "How do we as clinicians here factor in the equation?"

If you google it, Wikipedia will read that "Academic Medicine describes the branch of medicine pursued by doctors who engage in a variety of scholarly activities. While the traditional role of clinical academics is to provide clinical care, do research, and teach, academics today may also spend some of their time in managerial and representative roles. Every academic has a different work description, which is one of the many attractions of an academic career."

So Academic Medicine strives to improve clinical knowledge and patient care in a more coordinated and structured manner, through teaching and research. It also means that we should each be able to find our own niche in the AMC. By the process of natural selection, we will each morph into clinicians, clinician educators and clinician researchers.

Hence the all importance of CME, subspecialty rounds and discussions. These help generate more questions for greater disease understanding, better our care for the patients and provide platforms for research ideas to take off. In the ratrace that we all face each day, doctors now appear to pay less emphasis on such important scholarly activities, with trainees making up the bulk of attendances. Perhaps with greater emphasis on education, new life will be injected into these scholarly rounds and there will be renewed banter heard.

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IS DMEK THE PREFERRED METHOD FOR ENDOTHELIAL KERATOPLASTY?

03

Presented by Dr Jessica Lie, Netherlands Institute for Innovative, Ocular Surgery on 7 July 2010 | **Written by Dr Gavin Tan**

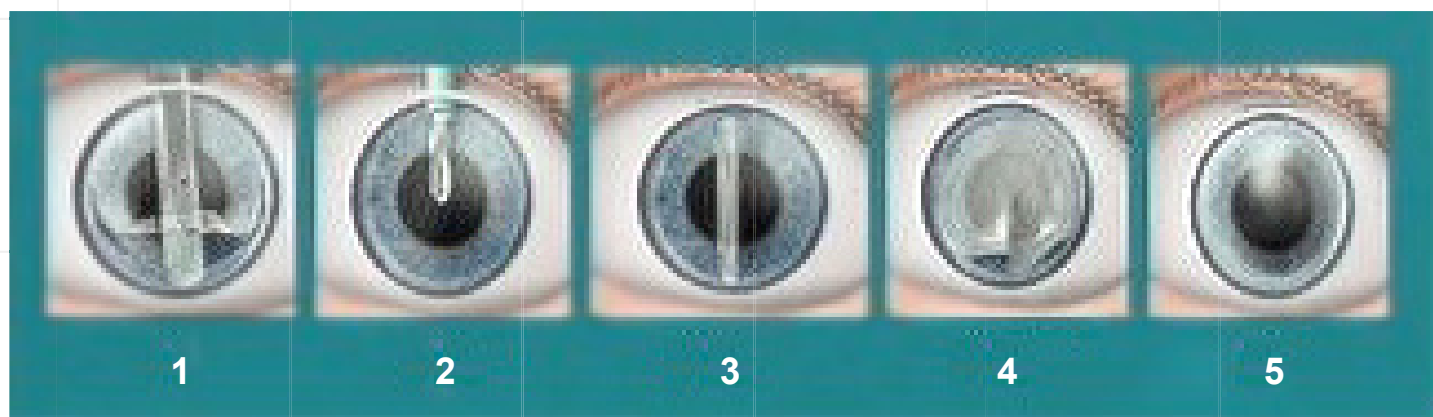
Posterior lamellar keratoplasty (PLK) is the preferred procedure today for management of cornea endothelial failure because of the smaller incision which induces less corneal astigmatism with a faster visual recovery. There are currently two main techniques used for PLK, the Descemet's stripping (automated) endothelial keratoplasty (DSEK/DSAEK) and the newer Descemet's membrane endothelial keratoplasty (DMEK). The differences are summarised in the following table:

| | DSAEK/DSEK | DMEK |
|-------------|-----------------------|---------------------|
| Donor Graft | DM+endothelium+stroma | DM+endothelial only |
| Thickness | 250um – 75um | 20um |

The main difference is the thinner donor graft in DMEK, which may allow for faster visual recovery. The DMEK technique also allows for more efficient use of donor corneas as the remnant tissue can be used for anterior lamellar cornea graft procedures.

Dr Lie described the results of the first 73 DMEK grafts preparations from donor corneas undertaken at the Netherlands Institute for Innovative Ocular Surgery (NIIOS). Graft preparation is done at the eye bank independent of the surgeries. There were 4 failures resulting in 69 DMEK grafts and 55 anterior remnants were available for Deep Anterior Lamellar Keratoplasty(DALK) (18 were ineligible due to media opacity).

The DMEK technique is described in **5 phases**.



Phase 1: Descemetorrhexis under air.

Phase 2: The graft is inserted with an injector using a no touch technique.

Phase 3: The graft is presented in the anterior chamber (AC) as a roll.

Phase 4: The graft is unrolled with air and fluid manipulation.

Phase 5: The graft is attached by inserting a large air bubble into the AC

Dr Lie reported the results for the first 135 DMEK cases at NIIOS. 86% were successful with 14% failure due to detachment or primary graft failure however, all had a successful second DSAEK procedure afterwards.

At 1 month 77% had best corrected visual acuity (BCVA) $\geq 6/12$ and at 6 month 96% have BCVA $\geq 6/12$. BCVA $\geq 6/7.5$ was achieved in 47% of eyes at 1 month and 74% of eyes at 6 months. 45% of eyes had BCVA $\geq 6/6$ at 6 months.

In contrast, following penetrating keratoplasty (PK), about 50% of eyes have BCVA $\geq 6/12$ at 1 year, and in DS(A)EK, about 60% of eyes have BCVA $\geq 6/12$ after 6 months. Whereas in DMEK, 77% achieve BCVA $\geq 6/12$ in 1 month suggesting that thinner grafts enable faster visual recovery.

With PLK, endothelial cell loss has been a concern due to the greater graft manipulation required. In PK, after 3 years the cell loss is expected to be about 50%. In this series, the observed cell loss following DMEK was 42% at 3 years. However, it should also be noted that in DMEK a larger graft (9.5-10.0mm) is used as compared with PK (7.5-8.0mm) or DS(A)EK (8.5-9.0mm) which results in a greater surface area of endothelial cells compared with PK. Given that endothelial cell density is measured, with greater surface area, the total number of endothelial cells loss would be greater for the same endothelial cell density.

With DMEK there is also a learning curve, with graft detachment being the main complication. In their series, graft detachment was noted in 20.0% of the first 45 cases. However the graft detachment rate fell to 13.3% of their next 45 cases, and in cases 91 to 132, a low graft detachment rate of 4.4% was reported.

In conclusion, it can be observed that there is a faster and often complete visual rehabilitation in DMEK as it is a minimally invasive procedure involving a closed system with no sutures and there is near perfect anatomical restoration of recipient cornea. There is also potential for more efficient use of donor tissue with the anterior remnant being available for anterior lamellar procedures.



FIGURE 1

Slit-lamp photographs of an eye before (left) and at 3 months (right) after Descemet membrane endothelial keratoplasty (DMEK) for Fuchs endothelial dystrophy. Note the near anatomical restoration of the transplanted cornea as well as its clarity after DMEK.

KEY LEARNING POINTS

1. DMEK has a steep learning curve but can provide faster and near complete visual rehabilitation in patients with cornea endothelial failure.
2. With DMEK there is a potential for more efficient use of donor tissue, since the remaining anterior lamellar can be used for an anterior lamellar keratoplasty

DESCEMET STRIPPING AUTOMATED ENDOTHELIAL KERATOPLASTY (DSAEK) **05** AND DE NOVO GLAUCOMA

Presented by Dr Tina Wong and Dr Jodhbir Mehta on 7 July 2010

Written by Dr Gavin Tan

The issues concerning cornea transplantation and glaucoma include the development of glaucoma after DSAEK, the need for cornea transplantation in glaucoma patients and the need for glaucoma procedures after DSAEK

Uncontrolled intraocular pressure (IOP) is a major risk factor for poor visual outcome, endothelial cell loss and graft failure following penetrating keratoplasty (PK). DSAEK is now the preferred surgical procedure for corneal endothelial dysfunction with superior visual outcomes and fewer complications. However, raised and uncontrolled IOP post DSAEK remains a principle concern for visual outcome and graft survival.

A retrospective comparative longitudinal study was done at SNEC to compare the development of elevated IOP with a need for sustained anti-glaucoma medication for at least 3 months (ocular hypertension, OHT) in 54 consecutive DSAEK grafts vs 108 diagnosis matched PK grafts.

At 18 months, the development of OHT was similar in PK (15%) and DSAEK (12%). No significant risk factor was found for the development of OHT post DSAEK. However it was noted that DSAEK eyes which developed OHT required more surgical procedures as compared with PK (34.8% vs 6.1% $p=0.005$), suggesting that the IOP rise post-DSAEK was more resistant to medical therapy than post-PK.

Another retrospective study was done to describe the effect of DSAEK on IOP control and corneal graft survival in 47 Asian eyes with pre-existing glaucoma or ocular hypertension. BCVA improved to 6/12 or better in 50% of patients compared with 87% of non-glaucoma patients. There were 8 graft failures (17%) with only one being related to a glaucoma procedure (post trabeculectomy). This suggests that pre-existing glaucoma or ocular hypertension was not a risk factor for DSAEK failure. 61.7% of eyes require further IOP lowering treatment post DSAEK. The majority of these patients (72.4%) only required additional anti-glaucoma medications to control IOP.

Risk factors for requiring more glaucoma interventions post DSAEK were pre-DSAEK glaucoma filtration surgery ($p=0.002$) and secondary intraoperative procedure post DSAEK (0.008).

In conclusion, following DSAEK, the incidence of IOP elevation was comparable to PK. Elevated IOP does not appear to have significant effect on VA and graft survival in the intermediate term. Eyes are more likely to require glaucoma management if trabeculectomy was present pre-DSAEK.

KEY LEARNING POINTS

1. Following DSAEK, incidence of IOP elevation was comparable to PK.
2. Elevated IOP In DSAEK patients does not appear to have significant effect on visual acuity and graft survival in the intermediate term.

Presented by Dr Chan Tat Keong on 21 July 2010

Written by Dr Leonard Yuen

Background

Cataract extraction with intraocular lens (IOL) implantation is a commonly performed surgery with a high success rate. However, occasionally this surgery may be complicated by anterior segment inflammation and/or infection.

If severe inflammation is observed in the early postoperative period, several conditions should be borne in mind, namely retained fragments, malpositioned IOL, endophthalmitis, and TASS.

TASS stands for "Toxic Anterior Segment Syndrome" and it is an acute, sterile postoperative anterior segment inflammation which can develop despite uneventful surgery. It can occur in isolation or as a cluster, and it can be confused with endophthalmitis, with similar signs including hypopyon and fibrin.

Presentation and Clinical signs

The most important differentiation point is the presentation. Post-operative acute endophthalmitis usually occurs between 4 to 7 days after surgery whilst TASS occurs within 12-24 hours. Patients typically present with extensive corneal edema, extending from "limbus to limbus", a fixed pupil and / or iris defects. Descemet's membrane folds may be seen and occasionally there are keratic precipitates as well. There is marked anterior segment inflammation together with fibrin and hypopyon. The IOP may be high and in severe cases, there may be associated trabecular meshwork damage and intractable secondary glaucoma. In protracted cases there may also be cystoid macular edema (CME). The inflammation is limited to the anterior segment and there is an absence of vitritis. Sight threatening complications of TASS include permanent corneal decompensation, intractable glaucoma and CME.

Causes and Differential Diagnosis

TASS is a toxic reaction and not an infection. There are numerous causes of TASS but the origin can be divided into three categories:

1. Extraocular substances that inadvertently entered the anterior chamber (AC) during or after surgery, for example, lint.
2. Products which have been introduced into the AC as part of the surgical procedure. This can be subdivided into:
 - a. Balanced salt solution (BSS) and other irrigating solutions, which may be of the wrong pH (<6.5 or >8.5), or wrong osmolality (<200mOsm or >400mOsm).
 - b. Retained ophthalmic viscoelastic devices (OVD) particularly denatured OVDs retained in re-usable instruments, for example from the previous day's case. De-ionized sterile water should be the last fluid used to flush out these instruments.
 - c. Preservatives including those from intracameral antibiotics and dilating agents.
3. Irritants on instruments that have accumulated due to inadequate or inappropriate instrument cleaning.

Management

Differentiating TASS from true infection is paramount as their management is different. Whilst endophthalmitis requires antibiotics, TASS requires intensive topical and frequently oral corticosteroids, with careful monitoring to ensure that it is not in fact an endophthalmitis. The role of immediate AC washout is controversial for TASS. The natural course is that it clears with treatment, rapidly in mild cases. However, clearing may occur over 3-6 weeks in moderate cases. In severe ones, there may be permanent corneal endothelial and trabecular damage requiring corneal graft and /or glaucoma surgery.

Landmarks

In the autumn of 2005 an outbreak of anterior segment inflammation occurred following uncomplicated cataract surgery. More than 300 patients had TASS and a portion had severe loss of vision. It was attributed to elevated levels of endotoxins in BSS, particularly in Endosol (Cytosol Laboratories, Braintree, Massachusetts, USA). Endosol was subsequently removed from the market and no other common factors were found in the outbreak.

There was a second TASS outbreak between January 17 and July 11, 2006. 140 centres in the US and Canada were affected, with 1 to 16 cases being identified in each centre. Multiple causes were found, with inadequate cleaning and sterilizing of instruments being the most important factor, particularly in inadequately sterilized re-usable cannulated instruments. Other sources include contaminated water baths (harboring heat stable endotoxins), enzymes and detergents for cleaning. In several cases of Intraoperative Floppy Iris Syndrome (IFIS) which required adrenaline in BSS, the adrenaline which was not preservative- or bisulphites- free was found to be the culprit. Intraocular anaesthetics and OVD were also found to be causative agents. A shorter surgery time with a faster turn over time resulted in flash sterilization which was inadequate and hence a buildup of toxic substances occurred which resulted in TASS. One patient also had an oil-based reaction when petroleum-based combination antibiotic-steroid treatment seeped into the eye from the compressive postoperative patching, which reacted with the implanted silicone 3-piece IOL.

Another outbreak occurred in Turkey that was associated with glutaraldehyde 2%. This chemical was accidentally used to clean re-usable instruments before autoclaving. 6 eyes of 6 patients were affected and none of the eyes recovered. The patients subsequently required further surgeries including penetrating keratoplasty, trabeculectomy or glaucoma tube implant.

More recently OVDs (Healon D, AMO, USA) with revealed elevated levels of endotoxins were also found to be a cause of TASS. Substances injected into the posterior segment may also enter into the anterior segment causing severe inflammation. Similarly, LASIK surgeons see Diffuse Lamellar Keratitis (DLK) which is a condition equivalent to TASS.

Finally there may be a disease entity termed "delayed onset acute anterior segment inflammation" which may be related to specific materials used in certain IOLs such as the Memory Lens (CibaVision) which contains residual polishing compound of aluminum oxide.

Due to these outbreaks, the American Society of Cataract Refractive Surgery (ASCRS) set up a TASS task force, which included ophthalmologists, epidemiologists, toxicologists, the Centre for Disease Control (CDC), the FDA, nursing professionals and industry representatives.

Outcomes

Following the collation and analyses of results of 909 cases of TASS identified from 50,114 (1.8%) cataract surgeries performed at reporting centers during the study period, poor instrument processing was found to be the most commonly associated factor with TASS. These included inadequate flushing of phacoemulsification and irrigation/aspiration (I/A) handpieces, use of enzymatic cleaners, detergents, ultrasonic baths (without changing the water after each case) which resulted in a buildup of heat stable endotoxins, and the re-use of single use products.

Other factors included antibiotics in BSS, stabilizers in adrenaline which may contain sulphites and bisulphites), intracameral antibiotics (for example, vancomycin is too acidic, gatifloxacin has Benzalkonium chloride, BAK), inappropriate agents for skin preparation (such as concentrated 10% Povidone iodine), and powdered gloves (Talc). Powder-less gloves can also be potentially toxic as their surface contains a releasing compound to facilitate release from the manufacturing mold.

Other issues relating to intracameral medications include “kitchen pharmacy”. As there is no standard unit dose of intracameral medications on the market, self preparation was performed in certain centers. For example, vancomycin 0.1% has been injected into the anterior chamber, however it was found to be too acidic. There are also dosage errors and osmolality issues with self-prepared medications.

Causes and Differential Diagnosis

In order to prevent TASS, a proactive prevention program and registry should be in place. This is highly dependent on ophthalmic surgeons to report all suspected cases of TASS, to limit epidemics specific to a particular product, and the need for detailed records on products and equipment used during surgery and product traceability.

TASS outbreaks should be reported to the healthcare cluster or local health departments and every outbreak must be investigated and changes made urgently to safeguard patient safety.

Prevention guidelines for TASS (“When sterile isn’t clean enough”):

1. Minimize the use of re-usable instruments
2. Thoroughly rinse phacoemulsification handpieces and I/A tips with sterile de-ionized water, the water in ultrasound baths should be replaced daily, all intraocular medications should be preservative free, and intracameral lidocaine, tryphan blue, miostat should not be mixed with sterile water but with BSS.
3. For the administrators, every new product introduced in surgery should be carefully evaluated preferably by an expert committee. New operating room staff should be supervised in their duties by experienced staff. Stringent instrument cleaning and sterilization protocols should be in place.

KEY LEARNING POINTS

1. **Accurate diagnosis of TASS is paramount. Presentation is usually during the first 24 hours and treatment of TASS consists of intense topical corticosteroids, and glaucoma medications as appropriate, with close follow up, especially in the first several hours and days.**
2. **The prevention of TASS requires a high index of vigilance for possible causes as well stringent instrument cleaning and sterilization protocols.**

Presented by Dr Winston Lim on 22 December 2010

Written by Dr Boey Pui Yi

Vascular orbital lesions can be classified by pathology according to Mulliken's classification, or by flow characteristics into low flow versus high flow lesions.

As computed tomography (CT) scans done today are all spiral volume acquisitions, reconstructions can be carried out in any plane, thus decreasing lens dose. The standard planes that are reconstructed are the axial and coronal planes, although parasagittal planes along the axis of the optic nerve may be useful in certain instances. However, as the scans are acquired very quickly, some within 1-2 seconds, some low flow lesions may be missed as they have yet to be opacified by contrast. Hence examinations should be tailored according to the clinical situation. For example, for low flow lesions, a delayed scan may be performed, or alternatively a Valsalva manoeuvre, to increase intra-thoracic pressure and thus filling of the lesion. Lying the patient prone will also achieve the same effect. For mid to high flow lesions, CT angiography to define the arterial input may be performed.

In CT angiography (CTA) an axial dataset is acquired, timed to the specific vascular phase that is required. The data set is then subjected to post processing, such as Maximum Intensity Projection where brighter (higher enhancing regions) vessels will be selected over the background and displayed. Magnetic resonance imaging (MRI) is useful for tissue characterization, for example, tissue that is inflamed or oedematous can be identified. It may also be useful in differentiating vascular tumours from vascular malformations. Low flow lesions tend to enhance as contrast flows through slowly, or in the case of tumour tissue, contrast is retained and thus demonstrate true mass effect. High flow lesions tend to demonstrate flow voids due to rapid flow.

Mulliken's classification divides vascular orbital lesions into 2 big groups, vascular tumours and vascular malformations/anomalies:

Vascular tumours

- Infantile haemangiomas
- Other vascular tumours

Vascular malformations/anomalies

- Capillary malformations (or telangiectasias)
- Cavernous malformations (angiomas/haemangiomas)
- Venous malformations (developmental venous anomalies)
- Arteriovenous shunting malformations
 - Classic cerebral (pial) arteriovenous malformations (AVMs)
 - Dural arteriovenous shunting malformations (AVMs) and arteriovenous fistulae (AVFs) including carotico-cavernous fistulae
 - Mixed pial-dural AVMs/AVFs
- Arterial malformations (no arteriovenous shunting)
 - Congenital angiodyplasia (fibromuscular dysplasia, Ehler's Danlos)
 - Intracranial aneurysms (berry/saccular, giant, serpentine)

- Mixed malformations
 - Venous cavernous type
 - AVM venous type
 - Cavernous AVM type
- Syndromatic CNS malformations

Orbital lesions can also be classified into high flow or low flow on MRI scans:

High flow lesions (AVMs / AVFs)

- Serpinginous signal voids
- Absence of a dominant mass
- Intraosseous extension with decreased marrow signal on T1-weighted images

Low flow lesions (haemangiomas, venous and lymphatic malformations)

- Intermediate signal on T1-weighted images
- Heterogenous high signal on T2-weighted images
- Prominent enhancement
- Involuting haemangiomas show focal areas of fatty replacement
- Venous lakes and phleboliths are features of venous malformations

High flow lesions include tumours such as proliferative infantile haemangiomas, or non-tumours such as vascular malformations such as AVMs, AVFs and dural AVFs. Low flow lesions include vascular tumours, capillary malformations, cavernous malformations, venous malformations/varix, lymphatic malformations, and mixed malformations.

Embolization may be an effective treatment for some of these lesions either alone or in conjunction with surgery. It can be performed through the ophthalmic artery, distal to the safety point, where the blood supply to the retina / choroid emerges. Nonetheless the patient still has to be counselled about risk of visual loss due to potential dislodgement of the glue or vasospasm of the ophthalmic artery during the procedure. Direct intra-operative injection into the lesion to control blood loss during surgery can also be offered. Agents used for embolization include sodium tetradecyl sulfate (STS), or alcohol, though the latter can cause more inflammation, which can be controlled with post-op steroids and NSAIDs.

The main imaging features for differentiating various orbital lesions are shown in following table (from Poon CS, Sze G, Johnson MH. Orbital lesions: differentiating vascular and nonvascular etiologic factors. AJR Am J Roentgenol. 2008 Apr;190(4):956-65.).

KEY LEARNING POINTS

1. Vascular lesions in the orbit have specific characteristics on CT and MRI scans.
2. They can be classified by pathology according to Mulliken's classification, or clinically by flow characteristics into low flow versus high flow lesions.

TABLE 1. KEY IMAGING FEATURES FOR DIFFERENTIATING ORBITAL LESIONS

| Lesion | Enlargement of Superior Ophthalmic Vein | Enlargement of Extraocular Muscles | Orbital Nerves Distinct from Lesion | Cavernous Sinus Engorgement | Prominent Arterial Supply | Phleboliths | Soft-Tissue Nodularity or Mass | Enhancement |
|----------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------|------------------------------------|----------------------------------------------------|-------------------------------------------------------------------------------------------|---------------------------------------------------------|-----------------------------------------|-------------------------------------------------------------------|---------------------------------------|
| Carotid cavernous fistula | Usually present | Present | Present | Present (usually asymmetric, can be symmetric if intercavernous communication is present) | Present | Absent | Absent | Marked (intense) |
| Superior ophthalmic vein or cavernous sinus thrombosis | Present | Present | Present | Present (usually asymmetric) | Absent | Absent | Absent | Marked (intense with filling defects) |
| Thyroid ophthalmopathy | Variable | Present (spares tendons) | Present | Absent | Absent | Absent | Absent | Variable |
| Soft-tissue orbital mass | Present (if lesion is at orbital apex, causing superior ophthalmic vein outflow obstruction) | Variable (involvement of muscles) | Present (except when nerves are directly involved) | Variable (if cavernous sinus is involved) | Absent | Absent | Variable (may not be nodular in cases of infiltrative metastasis) | Present (but variable) |
| Diffuse infiltrative soft-tissue process | Rarely (if mass affect at orbital apex causing superior ophthalmic vein outflow obstruction) | Variable | Present | Absent | Absent | Absent | Variable | Present (but variable) |
| Neurofibroma, schwannoma | Absent | Absent | Absent | Absent | Absent | Absent | Present | Variable |
| Normal variants of dilated superior ophthalmic vein or increased intracranial pressure | Present | Absent | Present | Absent | Absent | Absent | Absent | Intense |
| Venolymphatic malformation (lymphangioma) | Absent | Absent | Present | Absent | Absent | Variable (present in venous components) | Present | Moderate |
| Varix | Present (increased with Valsalva maneuver) | Absent | Present | Absent | Absent | Variable | Absent | Marked |
| Capillary hemangioma | Absent | Absent | Present | Absent | Present (may be less well visualized on MR angiography) | Absent | Present | Marked (intense) |
| Cavernous hemangioma | Absent | Absent | Present | Absent | Absent | Variable | Present | Present |

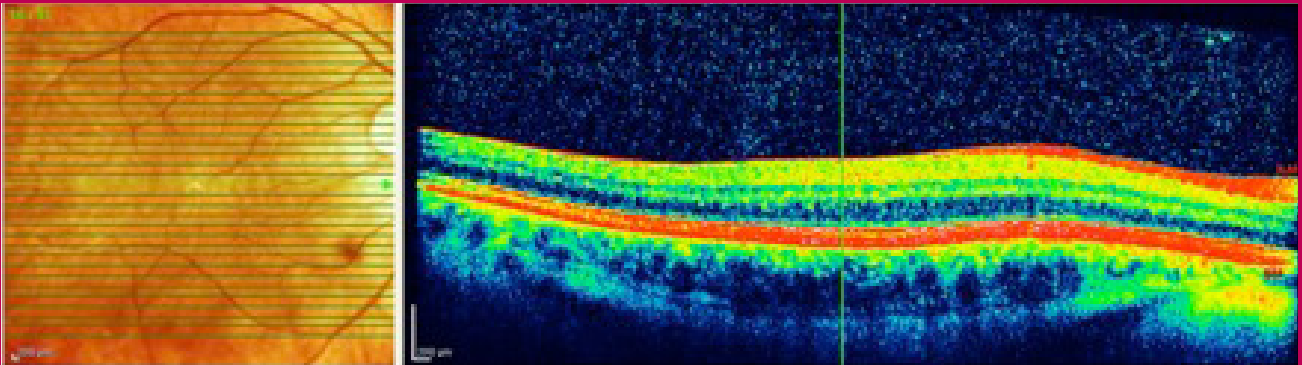


FIGURE 1A: Fundus photo and optical coherence tomography of the right eye

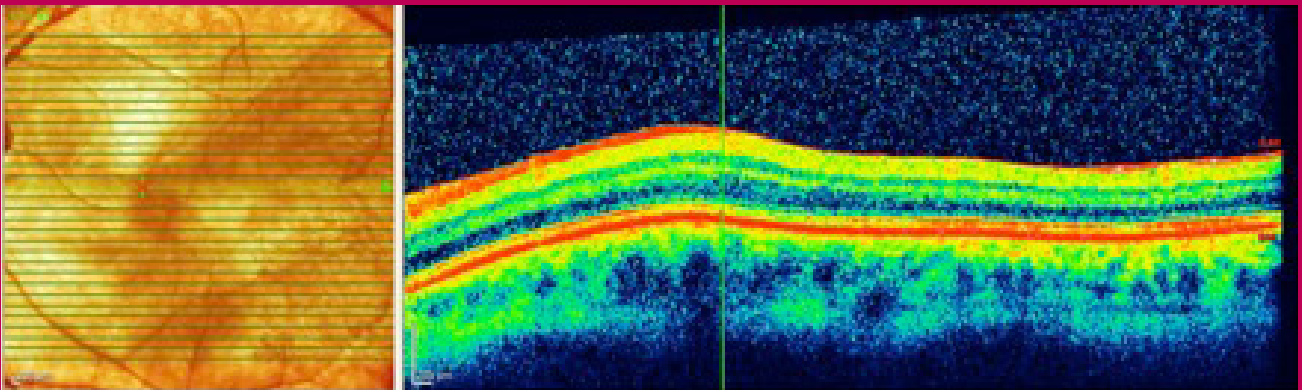


FIGURE 1B: Fundus photo and optical coherence tomography of the left eye (post operative)

THE QUESTION

A 60 year old healthy Chinese lady with a recent history of left uncomplicated cataract operation 3 months ago presents with a left pseudo-phakic retinal detachment. She underwent successful retinal detachment surgery with scleral buckle and vitrectomy.

Post-operatively, her visual acuity for the right and left eyes were 6/120 and VL 6/24 respectively.

Post-operative fundus photography and optical coherence tomography were performed.

What is the diagnosis for the primary ocular disease?

THE ANSWER**Foveal hypoplasia associated with ocular albinism**

Albinism is a heterogeneous condition which is characterised by genetic disorders of melanin synthesis. It can either affect the eyes, skin and hair (oculocutaneous albinism) or the eyes alone (ocular albinism).

Oculocutaneous albinism can be further divided into tyrosinase-positive or negative subtypes, which can be determined by the hair bulb incubation test. Tyrosinase-negative (complete) albinos are incapable of synthesizing melanin and hence have white hair and pale skin. There is a lack of melanin pigment in all ocular structures which manifest as pale iris and fundus with obvious large choroidal vessels. Other ocular associations include foveal hypoplasia, fewer uncrossed fibres in the optic chiasm, high refractive error, squints, nystagmus and absence of stereopsis. Tyrosinase-positive (incomplete) albinos are able to synthesise varying amounts of melanin and have differing severities of the oculocutaneous associations as mentioned above.

Ocular albinism with rhegmatogenous retinal detachment is rare and there is very limited literature on its management. The first published case report from Japan treated 1 case of rhegmatogenous retinal detachment successfully with a scleral buckle and photo-coagulation using a krypton laser.¹ Another case from Korea was treated by vitrectomy and endolaser photocoagulation but failed due to inadequate laser burns. A subsequent cryopexy with silicon oil was then performed with a successful outcome.²

Similarly in our case, laser photocoagulation may have been inadequate due to the lack of melanin pigment in the cells. Hence cryotherapy may be a better modality in terms of achieving chorioretinal adhesion in these eyes.

1. Hiroshi M, Hiroyuki M A case of ocular albinism with rhegmatogenous retinal detachment. Japanese Review of Clinical Ophthalmology 1999; Vol.93(6):861-864
2. Yang J, Lee S, Kang S, Park Y A Case of Retinal Detachment Surgery in Albinism Patient Journal of Korean Ophthalmology Society 2008; 49(5):840-844

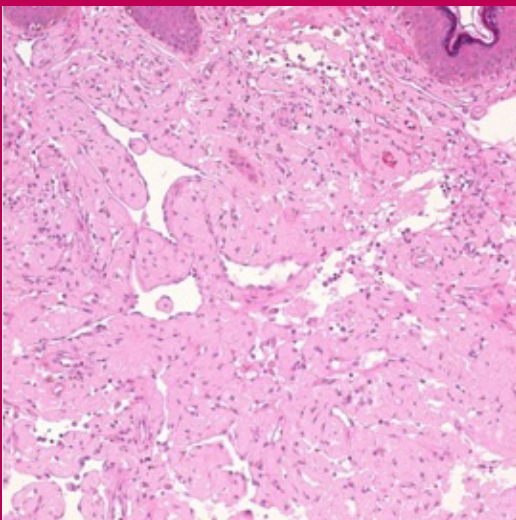
THE QUESTION

A 54 year old gentleman presented with bilateral chronic eyelid swelling for 3 years. There was no pain or limitation of movement and visual acuity was normal in both eyes.



Photo showing bilateral chronic periorbital swelling

He underwent an incisional biopsy of both eyelids. Intraoperatively, necrotic tissues were identified and sent for histopathological examination. The following are the histology findings.



H&E stain shows : amorphous eosinophilic deposition within the dermis

Question 1:

What are the possible differential diagnosis based on the history and intraoperative findings?

Question 2:

What is the diagnosis based on the H&E stain?

Question 3:

What further specific histopathologic stain and examination would help in the confirmation of the diagnosis?



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