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The official journal of the Ophthalmological Society of South Africa

AUTUMN 2023 | Vol 18 • No 2

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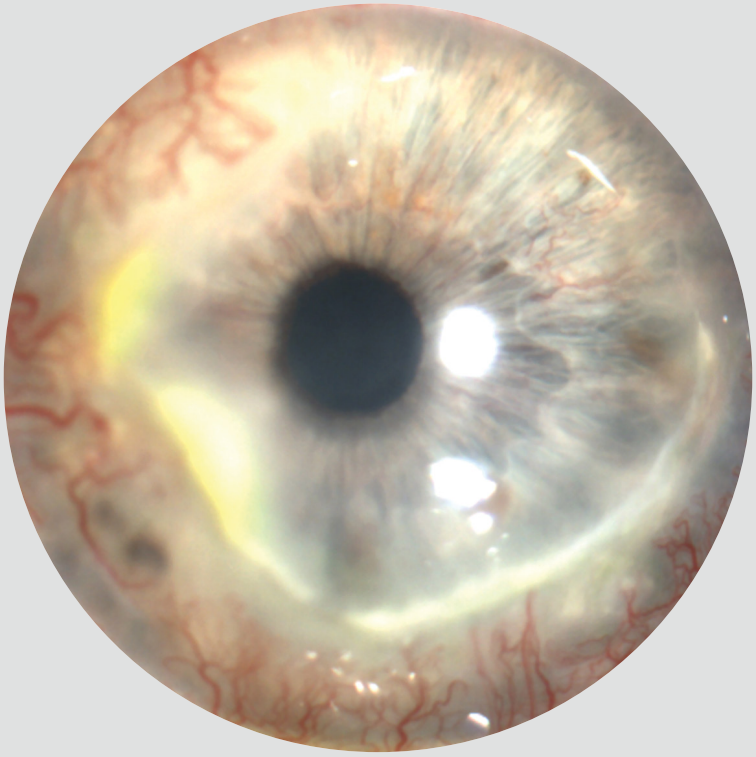

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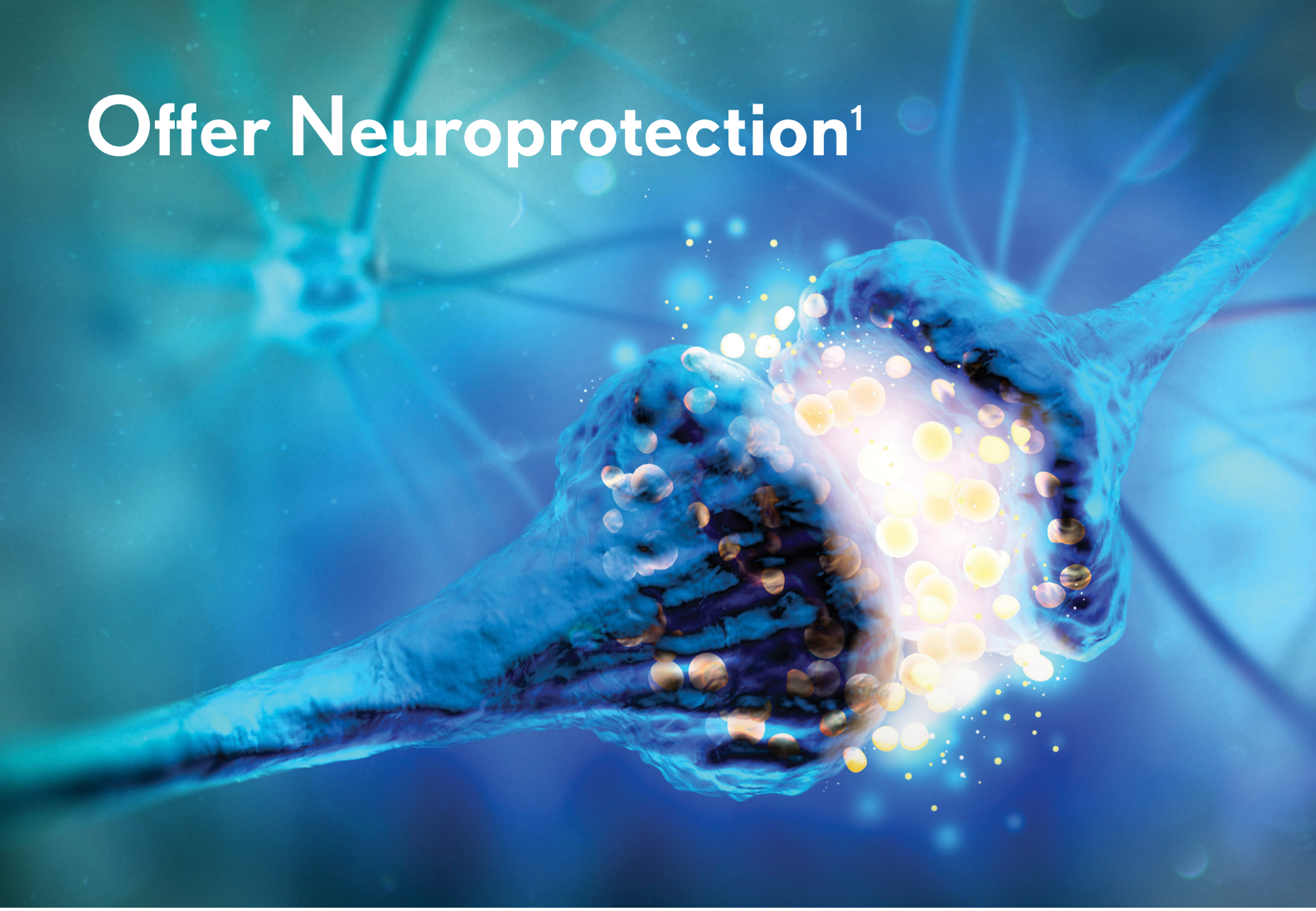

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SA Ophthalmology Journal

The official journal of the Ophthalmological Society of South Africa

ISSN: 2218-8304

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www.medicalacademic.co.za

Printing: Printed and bound by CTP Printers

Published by New Media,

a division of Media24 (Pty) Ltd

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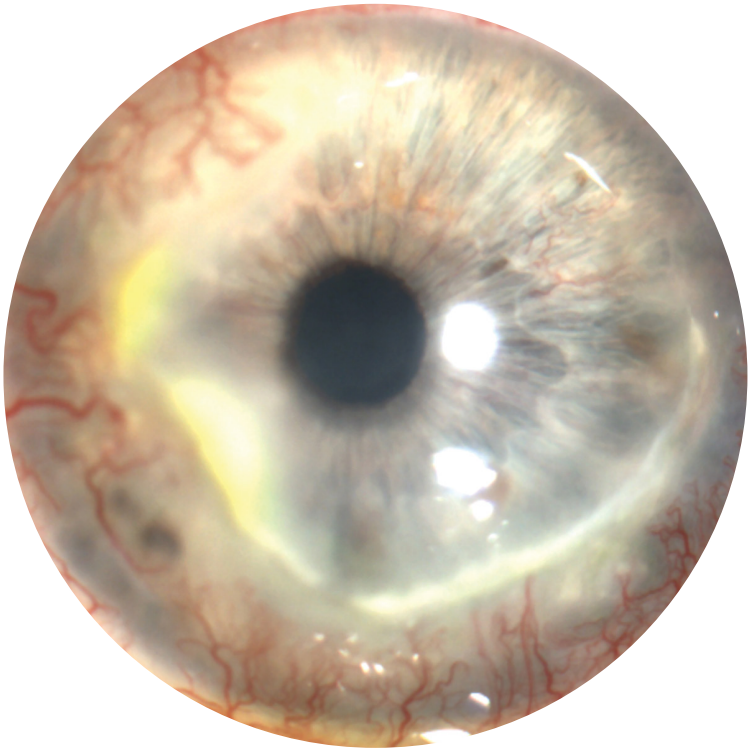

COVER PIC:
Moorens ulcer with perilimbal crescentic ulceration

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SA Ophthalmology Journal

CMSA specialist exams undergo changes

In this, our second issue of 2023, we have included a review article, two original studies and a case report. The review article resulted from collaborative work done between authors from the state and private sectors in Port Elizabeth. The original studies focus on familial dominant drusen and infective keratitis at academic hospitals, while the case report highlights an unusual pattern of geographic atrophy in a case of age-related macular degeneration.

As an aside, I think that it is useful for the ophthalmology community, especially those who are not based at academic centres, to be aware of the transformation that has occurred in the CMSA specialist exams (including ophthalmology) over the past two-three years. These changes were in the pipeline for a while already, but their implementation was fast-tracked because of the Covid-19 pandemic. Some aspects of these changes have been mentioned previously by Prof Tinley (Winter 2022, Vol. 17, no. 3).

In March 2020, the CMSA Senate decided to convert all exams from in-person to the online format, with a transition timeline of eight weeks. The categories below illustrate these major changes:

	Prior to COVID	During COVID
Theory exam:	Paper-based	Online (Speedwell platform)
Clinical/ Oral exam:	In-person, patient-based	Zoom-based, online
Clinical skills:	Centralised, in-person, patient-based	Decentralised, in-person, patient-based

The CMSA examination centres were increased from four to eight around the country. These changes required completely redesigned CMSA examinations, while attempting to adhere to international best practice, without compromising standards, as well as ensuring equity for candidates. The CMSA employed an executive director of education and assessment in the form of Prof Vanessa Burch, who has a PhD in medical education. Curricula needed to be carefully redefined, with blueprinting and prescribed texts (for those specialties that had not already done so). The underlying concept is to have online written exams which assess knowledge via single best answer (SBA) MCQs and/or short answer questions, using the concept of standard setting (Cohen/Angoff methods). Clinical (OSCE) and oral exams are intended to assess clinical reasoning and decision-making using multiple clinical scenarios, with structured answers and memoranda. Examiners/conveners/moderators hail from all the university training units and private specialists linked to training units in the country. Clinical competence is assessed via work-based assessments (WBAs) locally (and internally) at each university. The CMSA declared that the system of HODs signing off trainees prior to exams was unsatisfactory, clinical competencies were undefined, logbooks and time-in-training did not reflect competence, thus creating a platform for launching the WBA, under the direction of SACOMD and a national WBA task team, which was piloted in 2023 and is to be rolled out in 2024.

After a few rounds of exams in the above format, the Ophthalmology Council of the CMSA saw the need to re-introduce the clinical assessment of real patients in the OSCE, since it is much easier to recognise clinical signs from pictures that already show the signs to the candidate, compared to examining a patient to elicit the signs, before making a diagnosis and deciding on management. The CMSA Senate has, however, refused this request since it is not in keeping with the above plan, which if properly set-up, should test all the aspects of knowledge and skill required from any specialist. The CMSA president, Prof Fagan, has stated that the CMSA cannot afford the huge costs of running two different exam systems simultaneously, while the Deans of the SA universities prefer exam candidates not to have to bear the costs of travelling to exam venues all over the country. The new exam format is also in keeping with international trends. So, it seems that the new system is here to stay. The challenge will lie in creating a national standard at all training centres in South Africa, even those that are understaffed and lack proper equipment. Maintaining uniformity in the decentralised assessment of clinical skills will present a particular challenge in this regard.

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Prof Nagib du Toit
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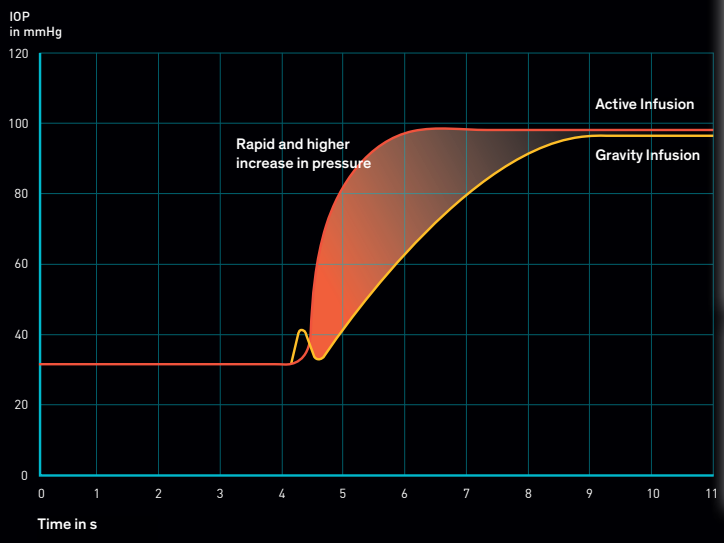

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1. The *South African Ophthalmology Journal* invites review articles, original studies and case reports for submission. Articles should be the original, unpublished work of the stated author. All materials submitted for publication must be submitted exclusively for publication in this journal. Written permission from the author or copyright holder must be submitted with previously published figures, tables or articles. Authors are solely responsible for the factual accuracy of their work.

2. A cover sheet is to be submitted with each manuscript. It should contain the title of the manuscript, the names of all authors in the correct sequence, their academic status and affiliations. The ORCID ID number for each author should be supplied (https://orcid.org/). The corresponding author should include his/her name, address, phone and email address.

3. Articles should be between 2 000 and 3 000 words in length. A 200-word abstract should state the main conclusions and clinical relevance of the article. Use the headings Background, Methods, Results and Conclusion. Five keywords are to be supplied at the end of the abstract.

4. Authors should declare any interests, financial or otherwise, regarding the publication of their article, under the headings of Funding and Conflict of interest. If none, this should be stated. An ethics statement regarding patient consent and/or Ethics Board approval should be included. Authors should also indicate whether the submission forms part of an 'MMed dissertation by publication' by stating so clearly on the title page.

5. All articles are to be in English and are to follow the Vancouver style of referencing. References should be numbered consecutively in the order that they are first mentioned in the text and listed at the end in numerical order of appearance. Identify references in the text by Arabic numerals in superscript after punctuation, e.g. ... trial.[13]

6. The following format should be used for references:
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   Chapter in a book: Young W. Neurophysiology of spinal cord injury. In: Errico TJ, Bauer RD, Waugh T (eds). *Spinal Trauma*. Philadelphia: JB Lippincott; 1991: 377-94.

7. Tables should carry Roman numerals, I, II etc., and illustrations Arabic numbers 1, 2 etc.

8. Abbreviations and acronyms should be defined on first use and kept to a minimum.

9. All figures, tables and photographs should also be submitted electronically. Each figure must have a separate self-explanatory legend. The illustrations, tables and graphs should not be embedded in the text file, but should be provided as separate individual graphic files, and clearly identified. Photographs should be saved as a 300 dpi JPEG file. Graphs and algorithms, which need to be editable, should be saved as MS Word documents or in PowerPoint. Tables should be saved either in MS Word or in a PowerPoint document. Photographs and X-rays need to be suitably anonymised. Permission should be obtained for the use of patient photographs.

10. Articles are to be submitted by email to the Editor-in-Chief, Prof Nagib du Toit at the following email address: journaleditor@ossa.co.za The text should be in MS Word. Pages should be numbered consecutively in the following order wherever possible: Title page, abstract, introduction, materials and methods, results, discussion, acknowledgements, tables and illustrations, references.

11. The Editor reserves the right to shorten and stylise any material accepted for publication.

12. For all accepted articles, authors will be requested to provide five (5) multiple choice CPD questions related to their paper.

13. Authors are to insert the following copyright notice on their article submissions:

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IOP = Intra Ocular Pressure

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SA Ophthalmology Journal

Corneal melt: a review of current strategies for resource-limited settings

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Abstract

Corneal melt is an ophthalmic emergency and a major cause of visual morbidity; corneal pathology is the fourth largest contributor to vision loss worldwide. Infective keratitis is the leading cause of corneal melting and blindness in both developing and developed nations, with a wide range of causative pathogens. It is substantially more common in low-income countries, where the disease burden is exacerbated by resource constraints; this review is aimed at practitioners working in such settings. Corneal melt is driven by proteinases, endogenous or exogenous, resulting in a progressive lysis of corneal stromal tissue and if untreated may be complicated by perforation or endophthalmitis. Management consists of identifying and treating the underlying aetiology, in conjunction with supportive treatment aimed at slowing the melting process and restoring corneal integrity. Anticollagenases are the backbone of medical therapy; anti-inflammatory drugs such as steroids, topical immunosuppressants or medroxyprogesterone acetate play an important role, and protecting the ocular surface with lubricants, autologous serum or contact lenses is essential. Surgical intervention is reserved for threatened or actual perforation, and includes patching with adhesives, grafting with amnion or autologous tenons capsule, and tectonic keratoplasty or epikeratoplasty. In addition, there are several novel and experimental interventions under development for the treatment of corneal melting.

Authors contributions: The authors fulfilled the criteria for authorship, and this paper represents honest work.

Disclosure statement: There are no conflicts of interest to declare.

Keywords: Cornea, melt, ophthalmology, update, medical, surgical, management.

What is known on the topic:

- Keratolysis is an ocular emergency requiring immediate intervention.
- Treatment is difficult, and outcomes are often poor.
- A multimodal treatment approach is usually employed.

What this review adds:

- Medical: The role of corneal cross-linking, scleral lenses and medroxyprogesterone acetate.
- Surgical: Autologous tenon capsule graft and the role of glycerine preserved corneal tissue.
- Novel and experimental therapies with promising results.

Introduction

Corneal melting, or keratolysis, is the loss of corneal epithelium with progressive dissolution of stromal tissue,[1] and can result from a variety of conditions including ocular infections and systemic autoimmune conditions. Keratolysis is an ophthalmic emergency and requires immediate intervention to reduce sight-threatening complications, including descemetocoele formation, perforation and endophthalmitis. The disease can be frustrating and difficult to treat, and without timeous intervention, visual outcomes are often poor.[2] There are some established management options, a few alternative applications of other interventions, and several emerging and experimental treatments.

Pathophysiology

The initiating event in keratolysis is an epithelial defect – the corneal epithelium plays a pivotal role in maintaining the health of the corneal surface, and corneal stroma without epithelial cover is less resistant to collagenolysis.[2]

Corneal melting is mediated by the activity of proteolytic tissue enzymes,

of which the matrix metalloproteinases (MMPs) are an important group.[1] MMPs are a family of calcium- and zinc-dependant endopeptidases with the ability to break down extracellular matrix tissue and tough collagenous fibrils; they have been implicated in both infectious and non-infectious corneal melting.[3] MMPs are balanced by counter-enzymes called tissue inhibitors of metalloproteinases (TIMPs), with a TIMP:MMP ratio of approx. 1:1 in healthy tissue.[4] MMPs are activated by three main pathways – 1) infiltrating inflammatory cells, e.g. macrophages, 2) by keratocytes in the cornea in response to injury, and 3) direct collagenase release from an invading pathogen.[2]

Injury and inflammation initiate a biochemical cascade which results in an imbalance of the normal MMP to TIMP ratio, with an increase in both absolute and relative amounts of MMPs in the affected tissue.[5] The end effect of these biochemical cascades is corneal stromal breakdown, known as a corneal melt.

Several other molecules are also involved in the melting process, including tumour necrosis factor and cytokines such as IL-6.[6] In peripheral ulcerative keratitis (PUK) and other inflammatory causes of corneal melt, several upstream events contribute to the melting process, including immune complex deposition in the peripheral cornea, perilimbal capillary occlusion, inflammatory cell recruitment, cytokine release and collagenase upregulation.[7]

Aetiology

Herpetic keratitis is the most common cause of corneal melting worldwide,[8] and is followed closely by other infectious diseases, collagen vascular diseases, and neurotrophic keratopathy.[2,9] Other common aetiologies include corneal trauma, limbal stem cell deficiency and exposure keratopathy;[1,10] any ocular or systemic condition resulting in a persistent epithelial defect can progress to corneal melting.[2] *Figure 1* is a flowchart detailing an approach to corneal melt.

A large number of collagen vascular diseases have been implicated in corneal melting; the most common being rheumatoid arthritis (RA) and granulomatosis with polyangiitis (formerly Wegener's granulomatosis). Others include systemic lupus erythematosus (SLE), Crohn's disease, polyarteritis nodosa and relapsing polychondritis.[10] Stevens Johnson syndrome is a well-known immune-mediated cause, and a Moorens ulcer is an inflammatory corneal melt in which no underlying systemic collagen vascular disease can be identified.

Both topical and systemic drugs can cause corneal melting. Keratitis medicamentosa is most commonly seen after topical non-steroidal anti-inflammatory (NSAID) use,[11] but can occur with almost any chronic topical treatment. Systemic medications of concern include biological agents, such as immunoglobulins and antibodies, used to treat autoimmune conditions and malignancies. Cetuximab, erlotinib, nivolumab, afatinib and several other agents have been implicated; as these are becoming more widely available it is important for eye care practitioners to be aware of their ocular side effects.[12-14]

Neurotrophic keratitis is another common cause of corneal melt. There are several congenital causes of neurotrophic keratitis, including Riley Day syndrome, corneal dystrophies (e.g. Reis Buckler and Lattice dystrophy), and congenital corneal hypoesthesia. More common acquired causes include Herpes Simplex Virus keratitis, diabetes mellitus, leprosy, ocular infections, or any lesion of the trigeminal nerve. Iatrogenic causes of corneal hypoesthesia include corneal surgery, contact lens wear, and chronic topical medication use.[15] Other iatrogenic causes of corneal melt to consider include corneal collagen cross-linking, the use of mitomycin C, and any procedure in which corneal epithelial integrity is compromised.

Xerophthalmia should be considered when treating severely malnourished children with corneal melting – keratomalacia is an indicator of severe vitamin A deficiency and should be managed as a medical emergency due to the high associated mortality rate.[9]

All the above causes induce an injured state in the cornea, and the resulting signalling cascade is biochemically similar regardless of the underlying aetiology.

Clinical features

Clinical features differ depending on the cause. In general, the diagnosis of a corneal melt must include an epithelial defect and stromal thinning; other associated features vary depending on the underlying disease process.

The aetiology of corneal melting can be divided into inflammatory and non-inflammatory causes. Inflammatory causes result from immune activation and

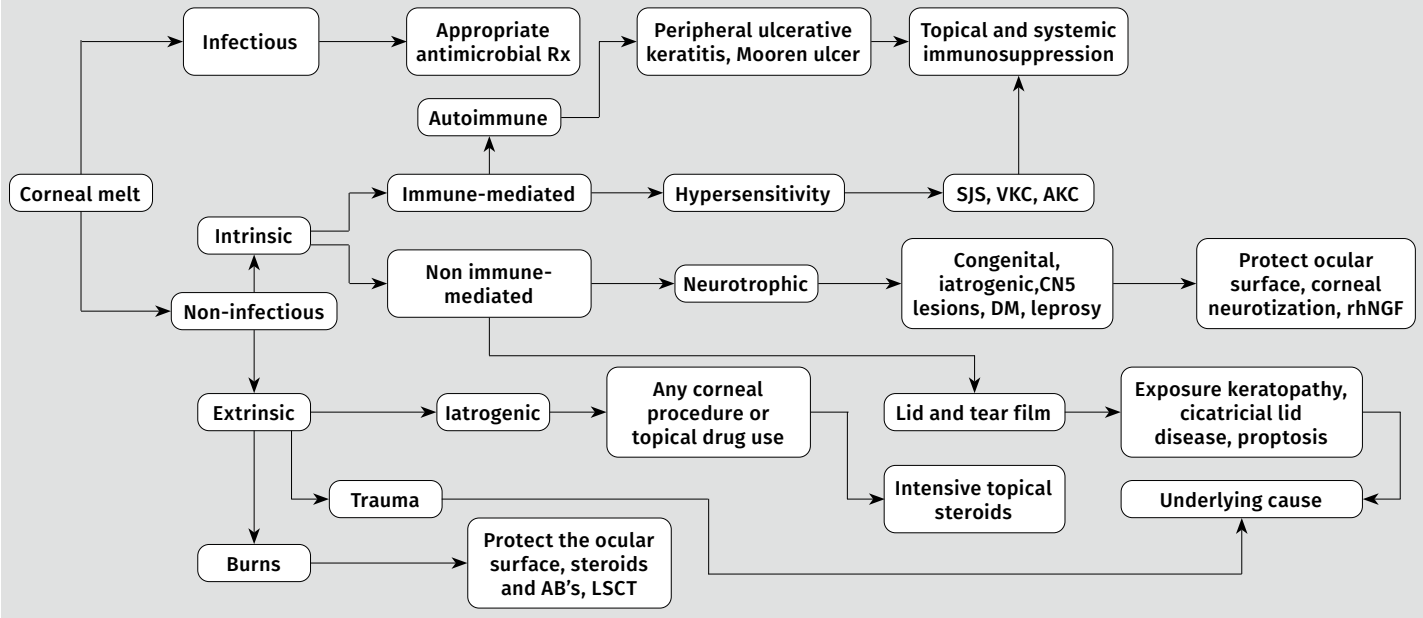

Figure 1: Approach to Corneal Melt

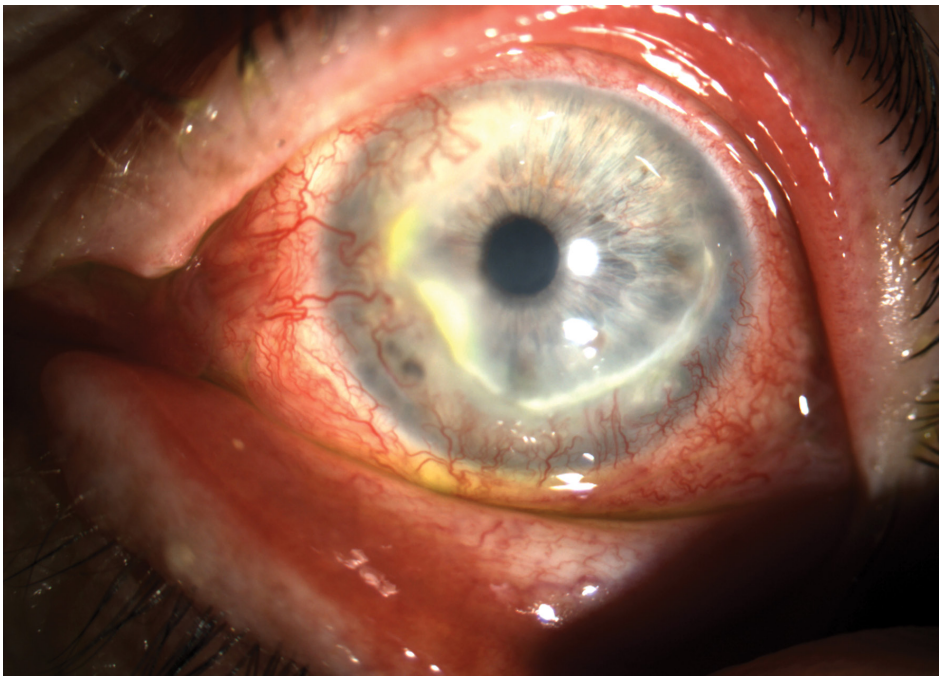

Figure 2: Moorens ulcer with perilimbal crescentic ulceration

include PUK and Moorens ulcer. Features are variable, typically with perilimbal crescentic ulceration and circumferential spread *(Figure 2)*; scleritis or episcleritis may occur with extension into the sclera.[16]

The clinical features of non-inflammatory causes vary widely due to the large number of causative conditions. Infective aetiologies may be associated with stromal infiltrates, epithelial dendrites, a corneal abscess, descemetocele, or hypopyon.[17]

Investigation

Investigation should be individualised based on the medical and ocular history, signs and symptoms, and the characteristics of the ulcer and surrounding tissues.[17]

Investigations in inflammatory melts aim at identifying the underlying systemic condition, and should include: FBC, U&E, LFT's, Hepatitis studies, CMP, s-ACE, CRP, RPR/FTA, ANA, ENA, anti-CCP, Rh Factor, C-ANCA, P-ANCA, urinalysis and chest X-Ray.

When suspecting an infectious cause, investigations aim at identifying the causative organism and may include corneal scraping, corneal biopsy, and conjunctival/corneal swabs for herpes virus or acanthamoeba PCR.

For other causes such as trauma, corneal burns, keratitis medicamentosa, nutritional deficiencies or iatrogenic causes, a good history will lead to the correct diagnosis.

Management

General medical management

The first priority is to address the underlying cause. In addition, preservative-free lubricants, oral tetracyclines (e.g. doxycycline 100mg BD), and oral ascorbic acid (1g BD) should be used in all cases, unless specifically contraindicated. Doxycycline is a proteinase inhibitor which has anticollagenase activity; it facilitates wound healing and is anti-inflammatory, but is contraindicated in children <12 years, pregnant or breastfeeding women, and in patients with renal or hepatic impairment. Topical acetylcysteine 10% also has anticollagenase activity and is used as an adjunct.[18] Topical ascorbic acid 10% scavenges free radicals but should not be used in acid burns; topical citric acid 10% is a powerful inhibitor of neutrophil chemotaxis used in corneal burns, but instillation is painful.

Cycloplegia may be used for comfort. Autologous serum is frequently used in refractory cases; while caution has been advised in its use for PUK and Moorens ulcer due to the theoretical risk of instilling systemic antibodies onto the corneal surface, there are several reports describing its successful use in these conditions.[19]

Infectious causes are managed with antimicrobial agents and adjunctive treatments. Topical aminoglycosides, including gentamycin and tobramycin, are epitheliotoxic and inhibit wound healing and re-epithelialisation[20] – these drugs are commonly required in the treatment of severe bacterial keratitis, but should be stopped as soon as it is safe to do so in the setting of persistent epithelial defects with melt. Similarly, a number of drug preservatives are toxic to the corneal epithelium, and preservative-free medication should be used where possible.

The mainstay of treatment in inflammatory corneal melting is systemic immunosuppression, and co-management with a rheumatologist is important.[17] Due to their rapid onset of action systemic steroids are commonly used to control acute disease; disease modifying anti-rheumatic drugs (DMARDS) are frequently used for long term control, and biological agents may be required in refractory cases.[21] Methotrexate, azathioprine, infliximab and other immunomodulatory agents may be used to treat PUK and Mooren's ulcer, as well as other causes of sterile corneal melt according to local guidelines.

Neurotrophic keratopathy and exposure keratopathy are treated initially by protecting the ocular surface. Lid taping, especially at night, is non-invasive and provides modest protection; silicone bandage contact lenses may be used, provided the eye is monitored closely for infection.[17] Botulinum toxin induced ptosis or tarsorrhaphy can be used, taking into consideration the underlying pathology and visual potential. These are usually reserved for cases where long-term management is expected.

Iatrogenic causes, e.g. post corneal cross-linking (CXL) or surgery, are usually treated with intensive topical steroids. Caution is advised with the use of topical steroids in other causes of corneal melting; although they may reduce scarring, steroids inhibit fibroblast migration and collagen synthesis, resulting in worsening of the thinning and delayed re-epithelialisation.[22,23]

Autologous serum:

Many ocular conditions are treatable with autologous serum tears, derived from clotted whole blood. Serum has a pH similar to that of tears, and contains epidermal growth factor, vitamin A, lysozyme, fibronectin, and occasional white blood cells.[24] Although there is a paucity of robust clinical trials examining the use of serum tears, a recent review by Shtein and colleagues found encouraging evidence for its use in persistent epithelial defects (PED).[25] The high levels of transforming growth factor β-1 (TGF β-1) in serum have raised concern, and for this reason several different concentrations have been used. Cho *et al.* reported faster healing of PED when using 100% serum,[26] and to date there is no evidence to support using diluted serum. There are

no globally accepted standards for the preparation of serum tears, and the need for immediate freezing after preparation complicates their use, but despite these challenges blood-derived tears are still widely used.[25] A commercially available preparation of umbilical cord serum (Optiserum®, Next Biosciences) is an alternative to autologous serum, and has been reported to be non-inferior to autologous serum in the management of several ocular conditions.[27]

Topical immunosuppressants

Topical immunomodulators such as cyclosporin and tacrolimus have been used for several decades to treat inflammatory corneal melting,[28] but there are difficulties to their use. Two per cent (2%) cyclosporin has been the treatment of choice, but this is only available from compounding pharmacies, which makes access to the drug difficult. The commercially available preparation (0.05%, Restasis®, Allergan) is generally considered too weak to treat high grade inflammation but may be considered in certain circumstances – Westland *et al.* reported successful treatment of corneal melting secondary to vernal plaque ulcers with this concentration applied 8x/day.[29]

Tacrolimus is much more potent than cyclosporine and has better tissue penetration due to its low molecular weight. The preferred concentration is 0.02%, again, only available from compounding pharmacies. Tacrolimus is available as a commercial skin ointment of 0.03% (Protopic®, Leo Laboratories), which has been described for ocular use in several studies and appears to be safe and well tolerated.[30]

Medroxyprogesterone acetate

Medroxyprogesterone acetate (MPA) (Depo-Provera®, Pfizer) is best known as a long-acting injectable contraceptive. In addition to its activity of binding progesterone receptors, it also has an affinity for glucocorticoid receptors, exerting an anti-inflammatory effect.[31] The cellular cascade in corneal ulceration includes both polymorphonuclear cell (PMN) dependant and PMN-independent collagenase release; MPA inhibits PMN-dependant collagenase, promoting collagen synthesis for repair.[32] It is easily compounded to a 1% solution by mixing one vial in 15ml of normal saline. MPA has less concomitant suppression of wound repair than steroids and is useful as a substitute in corneal burns after 10-14 days of steroids. It has been successfully used in corneal melting[33] and in post-infectious corneal thinning.[32] It has the major advantage of being cheap and widely available and appears to be safe for ocular use.

Corneal cross-linking

Corneal collagen cross-linking (CXL) is a well-established treatment used for slowing the progression of keratoconus. It involves soaking the cornea with vitamin B2 (riboflavin) and irradiating it with ultraviolet light. The resulting free radicals form covalent bonds in the collagen fibres, known as cross-links, stiffening the cornea. It has been used as an adjunctive, salvage and even solitary therapy for infectious keratitis, and has well established antimicrobial properties.[34] The stiffening effect also slows down collagen breakdown by making the cornea more resistant to proteolytic enzymes. A review of 104 eyes with corneal melting from infectious keratitis found a beneficial effect to CXL in 85% of cases.[34] There is no consensus on the ideal regimen, and its efficacy in fungal and acanthamoeba keratitis is unclear, but it should nevertheless be strongly considered in cases of infectious corneal melt unresponsive to antimicrobial treatment.

Silicone contact lenses

Therapeutic contact lenses are useful for protecting a damaged ocular surface and for splinting a wound in microperforations. A high-water content silicone hydrogel lens is satisfactory, except in eyes with aqueous tear film deficiency, in which a contact lens with a low water content requiring less hydration is preferred. The lens acts as a mechanical scaffold, protecting the epithelium from lid-induced mechanical trauma and enabling the epithelium to slide over the posterior lens surface prior to adhering to the stroma; new collagen is laid down under the intact epithelium forming a scar. Concurrent topical aminoglycosides should be avoided because of their epitheliotoxicity.[17]

Scleral contact lenses

Scleral contact lenses may be used in corneal melting, as they provide a protective environment for the cornea to heal. The lens reservoir vaults over the cornea and can be filled with artificial tears, antibiotics, amniotic membrane or blood derived products, providing a constant supply of nutrients, drugs or lubrication to the corneal surface for extended periods.[35] Scleral lenses have been used for decades for other indications, and it is important to remember their role in non-healing epithelial defects.

Surgical management

Glue

Cyanoacrylate glues have been used for decades in corneal melt complicated by micro-perforation.[36] Cyanoacrylate glues may be used for perforations less than 2mm in diameter, and are considered as first line surgical management for small corneal perforations.[17] Cyanoacrylate glues polymerise on contact with free anions on the ocular surface, and are usually used together with a 2-4mm sterile drape patch prepared using a skin biopsy punch to create a circular overlay patch *(Figure 3)*; a bandage contact lens

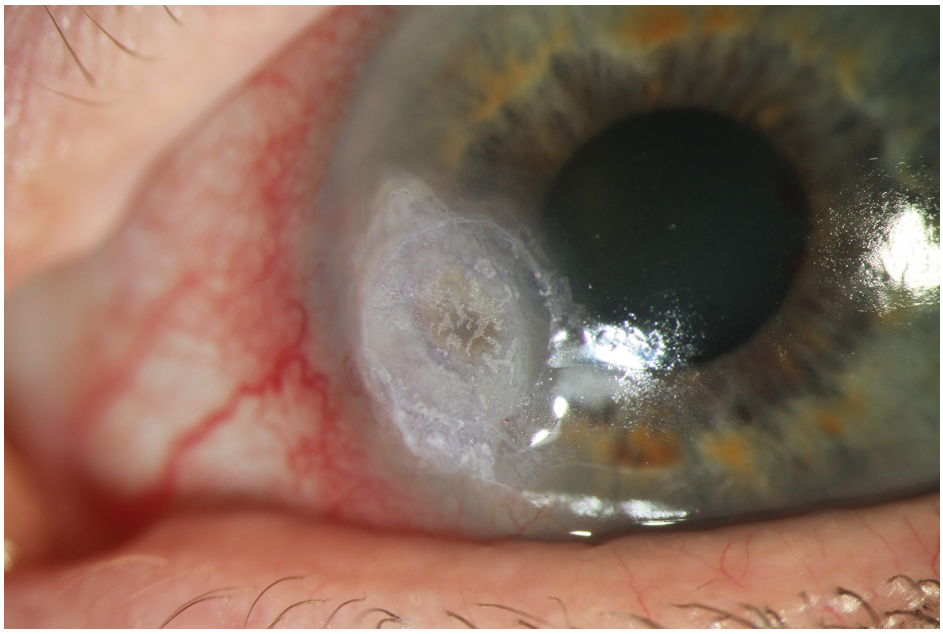

Figure 3: A small peripheral perforation sealed with cyanoacrylate glue

is then placed over the patch to reduce discomfort. In general, cyanoacrylate glues are non-biodegradable; they enable healing beneath the seal, frequently with vascularised scar formation. Repeated applications are toxic and should be avoided if possible.[37]

Amniotic membrane

Amniotic membrane (AM), derived from embryonic mesoderm, is the innermost layer of the placenta. It may be purchased commercially or prepared in-house and is available in fresh-frozen and freeze-dried forms. Fresh-frozen AM provides a better substrate for limbal epithelial cell growth, releases intact wound-healing modulatory factors, and its basement membrane is better preserved than in the dried form.[38] The fresh-frozen form is quick to harvest and easy to prepare, and once frozen can be stored for several months. Placental tissue is readily available wherever elective caesarean sections are performed and is usually otherwise discarded.

AM was first described for use in PEDs in 1997; since then several studies have established its use in corneal melting and perforation.[10] It secretes anti-inflammatory and anti-fibrotic molecules, and contains mesenchymal stem cells, invaluable for their ability to regenerate extracellular matrix and prevent scar formation. Amnion is well suited as a transplant tissue because of the absence of MHC class 2 antigens, making it biocompatible with non-HLA matching recipients.[39] In addition to its anti-inflammatory and anti-scarring properties, AM also provides a physical scaffold for extracellular matrix remodelling, allowing bridging of a perforation. It is used in a wide variety of ocular conditions,[40] including corneal melting, ocular surface disease, persistent epithelial defects, descemetocele, bullous keratopathy, pterygium surgery, conjunctival reconstruction, limbal stem cell deficiency, and even glaucoma and vitreoretinal surgery.

Several application techniques are in use for the placement of AM, including patching, grafting, and others. Patching generally refers to the placement of a large piece of membrane over the whole cornea and suturing to the limbus or episclera; grafting is the placement of a piece of membrane cut to size and sutured to the edges of the defect.

Amniotic membrane is frequently used with human fibrin sealant[41] – a biodegradable, non-toxic adhesive comprised of human fibrinogen and human thrombin. Placement as a clinic procedure is gaining popularity due to the availability of fresh-frozen AM mounted on a ring conformer (Prokera®, Bio-Tissue) and dehydrated discs that can be applied directly to an ulcer and covered with a contact lens.

Tenon's capsule graft

Tenon's capsule is a fascial sheath arising two millimetres posterior to the limbus; it contains fibroblasts which have the ability to produce connective tissue, and it incorporates into corneal tissue when transplanted.[42] Using autologous Tenon's capsule as a transplant tissue has been described in corneal melting and perforation, traumatic scleral injuries and leaking trabeculectomy blebs.[10] The graft is usually taken inferiorly and glued or sutured onto the defect; it may be combined with AM or tissue adhesives for tectonic support.[43] There are a number of advantages to using autologous tissue for transplantation – because no immune response is evoked, tissue rejection does not occur, and as there is no antigenic sensitisation there is a higher likelihood for success in corneal grafting at a later stage. Unlike with corneal tissue and AM there is no reliance on donor tissue and the associated infrastructure; important advantages in resource constrained healthcare settings where access to transplant tissue is limited.[42]

Tectonic keratoplasty

Surgical reconstruction with a tectonic graft of donor corneal tissue is the treatment of choice in corneal melts that have progressed to large or complex perforations. The aim of this procedure is to restore the structural integrity of the cornea and seal large perforations to save the eye from sight threatening complications such as expulsion of the intraocular contents or endophthalmitis.[44] Tectonic grafts do not require optical grade tissue, as these grafts will often fail in time and require a secondary optical keratoplasty to restore vision as a delayed secondary procedure. Glycerol preservation of non-optical grade corneal tissue extends its shelf life above a year without refrigeration, rendering it available in an emergency for the restoration of globe integrity; it is also significantly cheaper to procure and store than fresh tissue, and is commercially available at a fraction of the price.[45] Glycerol-preserved cornea lacks antigen presenting cells and does not stimulate recipient t-cells, leading to lower rejection rates and higher chances of success with future transplants than if fresh tissue was used.[45] In addition to corneal tissue, tectonic patch grafts may be performed with a variety of other materials, including donor sclera or biological collagen preparations (e.g. Lyoplant® pericardial tissue, Braun).

Tectonic epikeratoplasty has been used in corneal melting as an alternative to keratoplasty, and involves the placement of a corneal button over the melted or perforated area with suturing to the sclera; this large graft is left in place to allow the underlying defect to heal.[44] An advantage of this procedure is that it may be performed with corneal tissue that is unsuitable for optical keratoplasty due to stromal scarring or a poor endothelial cell count, making it an attractive option in low-resource settings.[10]

Once corneal integrity has been restored, the melting process has been arrested and the inflammatory or infectious aetiology has been fully addressed, a secondary optical keratoplasty can be performed to restore vision *(Figure 4)*.

Novel and experimental therapies

Zinc-scavenging hydrogel contact lenses are made by incorporating dipicolylamine (DPA) into the structure of silicone hydrogel lenses. DPA has a high selectivity for binding zinc ions, deactivating MMP-1, MMP-2, MMP-9 and collagenase-A; these contact lenses have been shown to prevent porcine corneal breakdown *in vitro*. Advantages of DPA-containing contact lenses are numerous – a localised application with no systemic drug absorption, excellent MMP deactivation, and the ability to mass-produce and sterilise the product cheaply, all make this an exciting prospect for resource-limited settings.[3]

Ilomastat (Galardin, Selleck) is a synthetic broad spectrum MMP inhibitor which has been used to prevent scar formation in glaucoma surgery;[46] topical application has been shown to significantly reduce corneal melting from alkaline injuries in rabbit eyes.[47]

Platelet Activating Factor (PAF) is a strong inflammatory mediator, chemokine and protease inducer; topical LAU0901, a novel PAF inhibitor, has been shown to inhibit corneal melting and perforation in animal models.[48]

Decellularised porcine corneal stroma is a promising intervention, with

several human trials showing good results.[49,50] Porcine corneal xenotransplant material can be sourced as a by-product of the food manufacturing industry and has the potential to be manufactured and distributed cheaply.

Another novel source of biological collagen-based stromal scaffold material is decellularised and decalcified scales of the Tilapia fish, which are mechanically robust and can be sutured onto host cornea – Initial trials in rabbit[51] and porcine[52] hosts have shown promising results, and a human trial is underway.

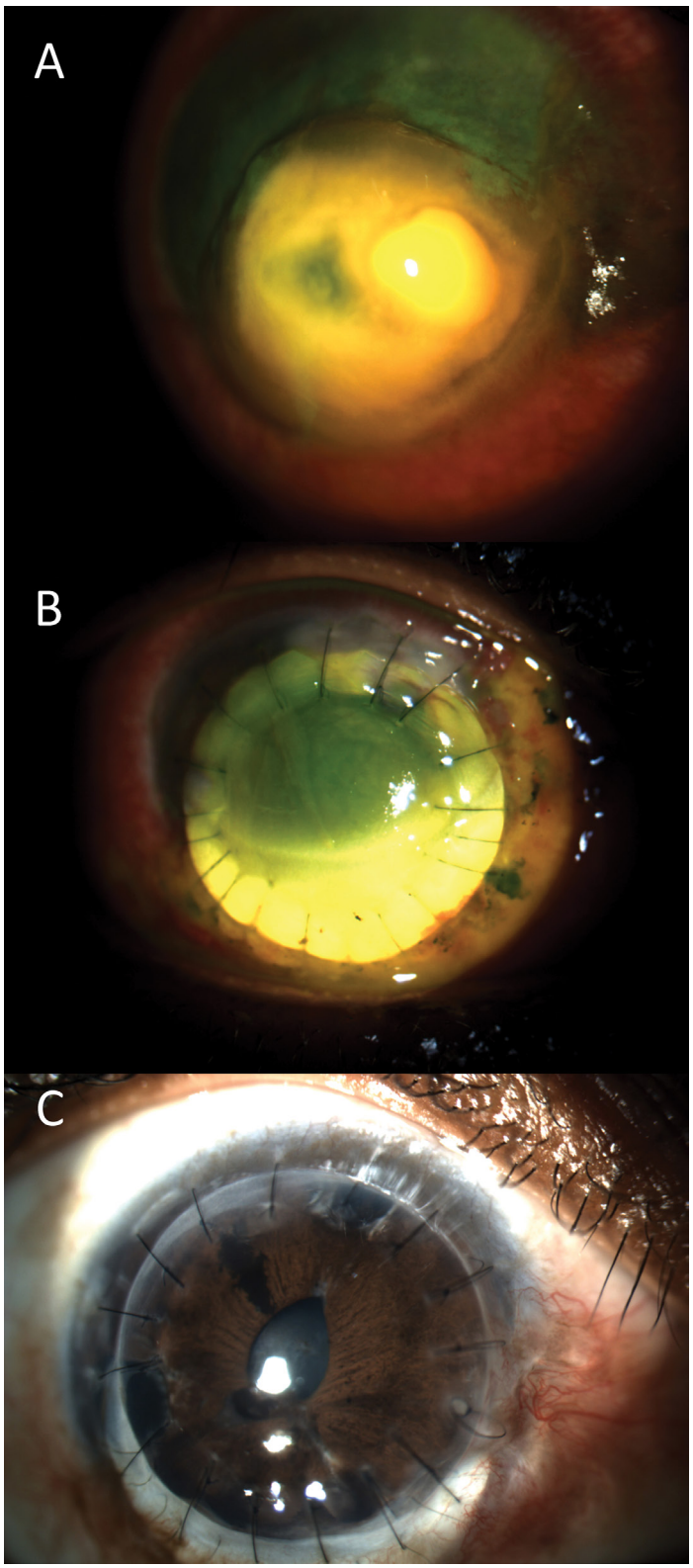

Figure 4: A) Corneal melt secondary to Aspergillus keratitis B) Tectonic corneoscleral patch graft C) Secondary optical keratoplasty

3D bioprinted corneal stroma, tissue adhesive gel (GelCORE), acellular bioengineered collagen, and cell-populated scaffolds are all on the horizon and have the potential to address the global shortage of suitable corneal transplant tissue, which is particularly acute in Asia and Africa.[50]

Conclusion

No perfect algorithm exists for the treatment of corneal melting – in general, a stepwise approach is usually employed, with inexpensive, non-invasive modalities tried first, expensive or invasive treatment options follow.

Corneal melting can be caused by a number of underlying diseases, and treatment thereof is often difficult and frustrating. The primary aim of the ophthalmic practitioner is to identify and address the underlying pathology as soon as possible; where a systemic disease is the cause of the melting, it is important to involve an appropriate specialist early on in the management process.

There are several effective therapeutic options, familiar from other indications, but frequently overlooked in corneal melts; remember the place of corneal collagen cross-linking in recalcitrant infectious causes, scleral contact lenses and the use of amnion, especially in refractory cases. Surgical reconstruction is often required in more severe cases with significant thinning and perforation if the melting process is not arrested timeously.

No single treatment exists for any particular disease, and significant variation in response between patients can be expected. Several therapeutic interventions are frequently employed simultaneously, and treatment should always be individualised based on underlying aetiology and the patient's response to implemented interventions.

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A descriptive study of patients seen with familial dominant drusen at an academic hospital in Gauteng, South Africa

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Abstract

Background: Familial Dominant Drusen (FDD) is a rare disease with an uncertain incidence worldwide. No information has been documented on the magnitude and clinical features of FDD locally. This study aims to describe the clinical features of patients diagnosed with FDD at the ophthalmic clinic of Dr George Mukhari Academic Hospital and calculate the magnitude of the disease.

Methods: We conducted a retrospective descriptive review of electronic ophthalmic medical records of patients clinically diagnosed with FDD between 2013 and 2021 at an academic hospital in Gauteng, a province in South Africa.

Results: A total of 39 patients (0.08% of all patients seen) were diagnosed with FDD, The median age at presentation was 61 years (interquartile range: 56-70 years); all participants were African, and 72% were females. All FDD were bilateral, with the same pattern seen in each of the two eyes, and 74% were intermediate to large. The median visual acuity was 20/50 in the left and right eye, respectively. Drusen were distributed along the arcades (51%) and both at the macula and along the arcades (44%), while in 5% of cases, there were only macular drusen.

Conclusion: The proportion of patients seen with FDD did not differ significantly over time. Most patients were of advanced age and may have had AMD rather than FDD. Molecular diagnosis may assist in definitive diagnosis, and it is necessary to follow up and monitor these patients to pick up the early visual changes of developing choroidal neovascular membranes. In addition, further study is needed to assess if these patients developed choroidal neovascular membranes.

Keywords: Familial dominant drusen; Doyne honeycomb Retinal Dystrophy; Malattia Leventinese

Introduction

Familial dominant drusen (FDD), also known as Doyne honeycomb retinal dystrophy or malattia leventinese,[1,2] is a rare genetic macular dystrophy characterised by the presence of small yellow-white accumulations of extracellular material under the retinal pigment epithelium in the posterior pole of the eye of patients younger than 60 years.[3] The condition is caused by a single autosomal dominant mutation in the EFEMP1 gene on the short arm of chromosome 2p16, which encodes fibulin-3, an extracellular matrix glycoprotein.[4] The diagnosis of FDD is made clinically and confirmed by genetic testing to prove an EFEMP1 mutation.[5] At an early stage of the disease, the patient may have blurry vision, but there is severe visual impairment at advanced stages.[4] The prevalence of FDD is unknown worldwide; however, the condition has been commonly described in isolated case reports in different populations such as Chinese,[6,7] Japanese,[8] Indian,[3,9] American[1] and Scandinavian populations.[10]

As a result of the progressive loss of vision, an early diagnosis of FDD is important. Even though there is no known effective treatment for the condition, studies have shown that the use of anti-vascular endothelial growth factor (anti-VEGF) therapy for choroidal neovascular membrane (CNVM), a complication of the condition, improves the vision and resolves subretinal fluid,[9] while others have found that treatment options such as the use of lasers helped to clear drusen deposits, however, the treatment did not result in a reduction in the risk of developing CNVM and was not shown to limit the occurrence of geographic atrophy or visual acuity loss.[11]

Patients over the age of 60 years may develop age-related macular degeneration (AMD),[12] which is the leading cause of irreversible blindness in the elderly population worldwide.[13,14] FDD has a similar presentation to AMD but must be distinguished from AMD. The main differentiating features include age of onset before 60 years (most often 30 – 40 years), the radially elongated or honeycomb-shaped distribution of the drusen, the presence of drusen nasal to the disc and a positive family history. It is worth noting that there is no treatment for FDD but, patients are monitored for signs of choroidal neovascularisation. FDD is a rare disease worldwide and most studies published are case report.[1,6,7,8,9,10] The prevalence of FDD is therefore unknown. This study aims to describe the clinical features of patients diagnosed with FDD at the ophthalmic clinic of the Dr George Mukhari Academic Hospital between 2013 and 2021 and calculate the magnitude of the disease.

Materials and methods

Study design: This retrospective study of clinically diagnosed patients with FDD in the ophthalmic clinic at the Dr George Mukhari Academic Hospital (DGMAH) between January 2013 and December 2021. The clinical and demographic data and images for patients diagnosed with FDD were extracted from electronic medical records.

Study Setting: The DGMAH is a 1 650-bed hospital that serves as a referral hospital for the greater part of Pretoria North in Gauteng, the North West province and Polokwane in Limpopo. It is an academic

facility for the Sefako Makgatho Health Sciences University, formerly known as the Medical University of Southern Africa and the University of Limpopo Medunsa Campus. It is situated near the township of Ga-Rankuwa. The ophthalmology clinic is fully equipped with a colour fundus camera that captures the retinal images of patients with retinal disease. Each patient underwent a fundoscopic examination, and a fundus photograph was taken for those clinically diagnosed with FDD. Molecular diagnosis and optical coherence tomography (OCT) were unavailable during study period.

Study population: All patients diagnosed with FDD in the ophthalmic clinic at this academic hospital between 2013 and 2021, where an average of 5 000 new patients with different eye conditions are seen annually.

Inclusion and exclusion criteria: All medical files of patients presenting to the ophthalmic clinic and diagnosed with FDD within the study period were included in the study. The patients with incomplete information in their medical records were excluded from the study.

Sampling technique and sample size: As FDD is a rare condition, all cases of FDD seen during the study period (n = 39) were included.

Data collection: The ophthalmic clinic register books were used as the starting point to search for and identify patients diagnosed with FDD. Electronically captured images on the fundus camera were used to identify patients with FDD. The hospital files of all patients diagnosed with FDD were retrieved from the hospital records department and reviewed by the researchers.

All images captured during the period of the study were reviewed by a senior experienced ophthalmologist. Demographics, such as age at presentation, gender, race and place of residence, were documented. Clinical data, including visual acuity and the site and the size of the drusen temporal to the disc, were extracted from the ophthalmic clinic register books and patients' medical files. The size of the drusen was categorised into three groups, namely small (< 63 µm in diameter); intermediate (63 to 125 µm) and large (> 125 µm).

Statistical analysis: All statistical analyses were performed using Statistical Package for Social Sciences software, version 26.0 (Released 2011; IBM Corp., Armonk, NY, USA). Descriptive statistics such as median and interquartile ranges were used to present continuous variables, while frequencies and percentages displayed categorical variables.

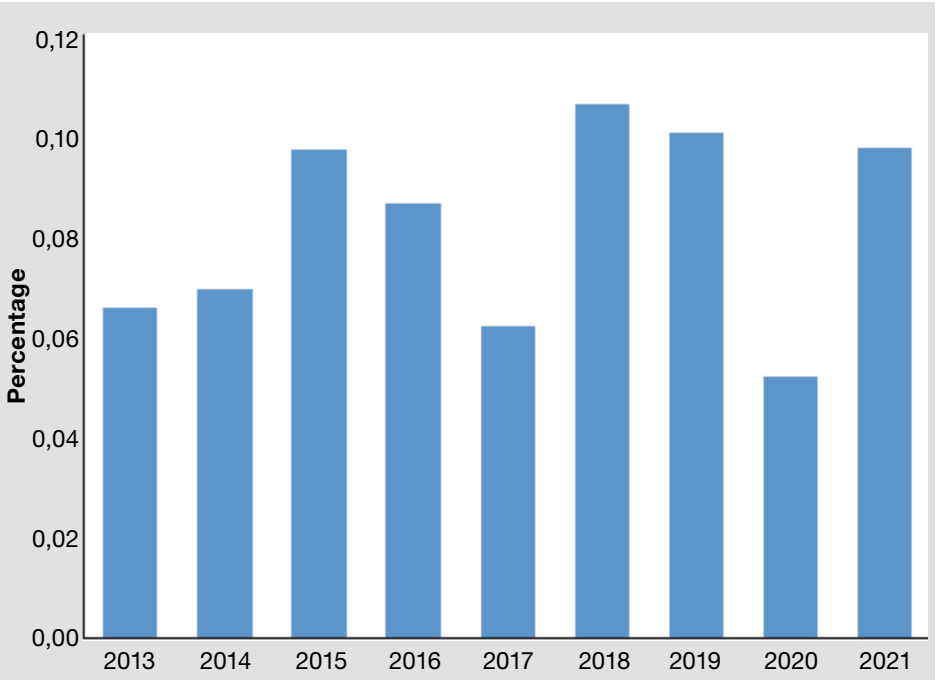


Figure 1: FDD per year as a proportion of new ophthalmic patients presenting to our clinic

Table I: Demographic information

	No	%
Age (years)		
<60	17	43
60-69	12	31
70+	10	26
Gender		
Male	11	28
Female	28	72
Eye affected		
Both	38	97
Size of Drusen		
Small	10	26
Intermediate	18	46
Large	11	28
Distribution of Drusen		
Macula	2	5
Arcades	20	51
Combined	17	44

Table II: Distribution of Visual Acuity

	No	%
Visual Acuity left eye		
6/7.5	1	3
6/9	6	16
6/12	4	11
6/15	12	32
6/20	3	8
6/30	8	22
Hand movement	3	8
Visual Acuity right eye		
6/9	8	22
6/12	4	11
6/15	8	22
6/20	3	8
6/30	12	32
Hand movement	1	3
NLP no light perception	1	3

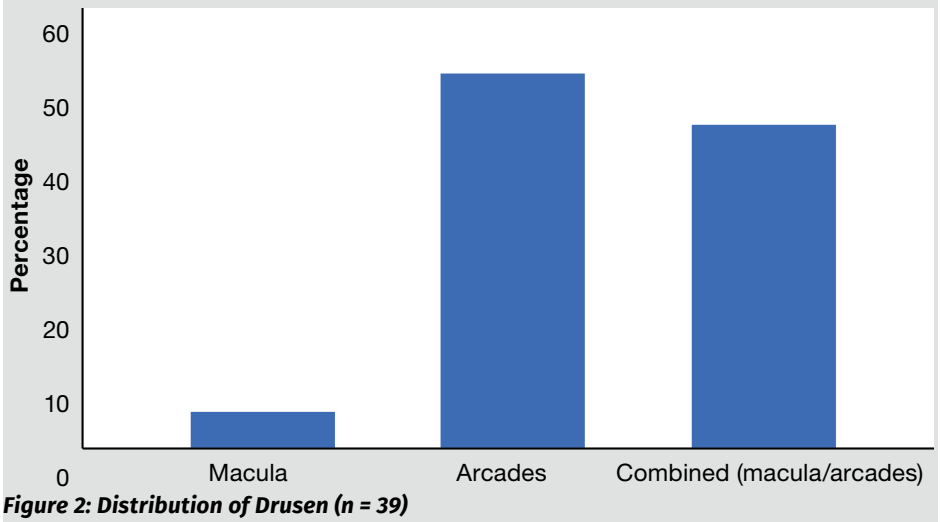
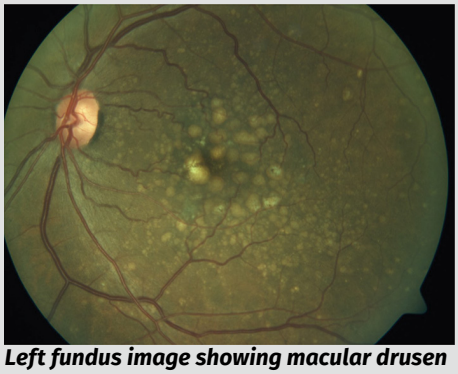
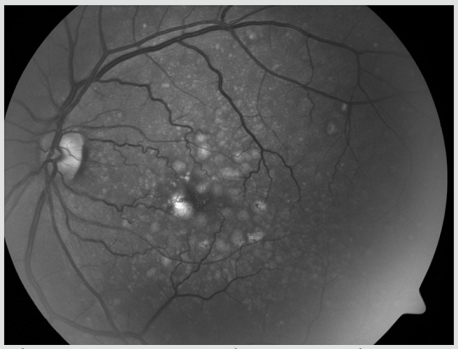


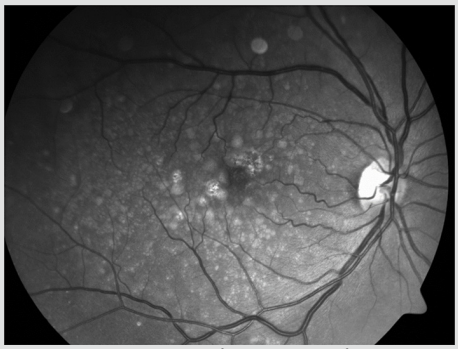
Figure 2: Distribution of Drusen (n = 39)

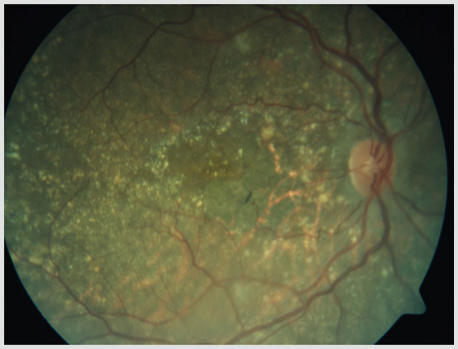
Figure 3: Different images of distribution of drusen

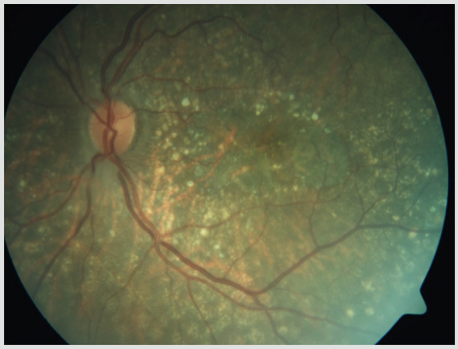

Right fundus image showing macular Drusen

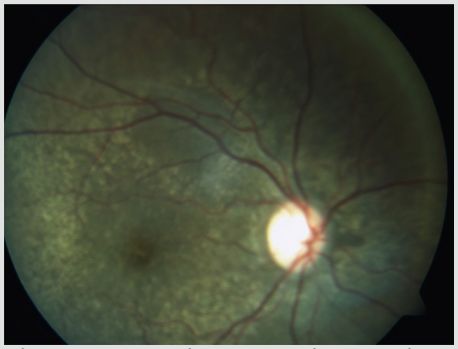

Left fundus image showing macular drusen

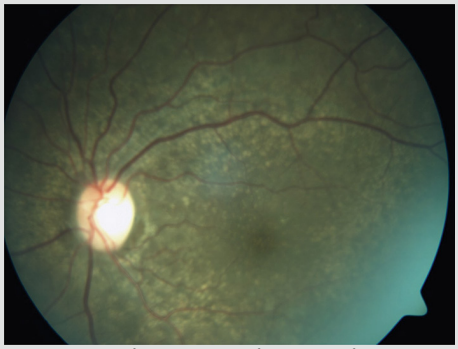

Right fundus red free image showing macular drusen

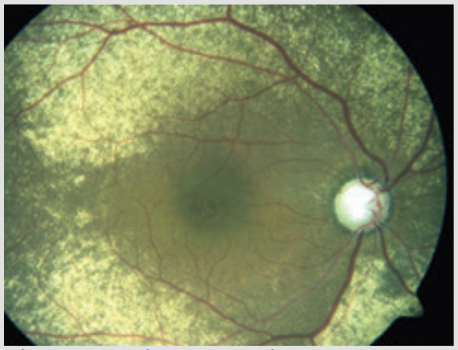

Left fundus red free image showing macular drusen

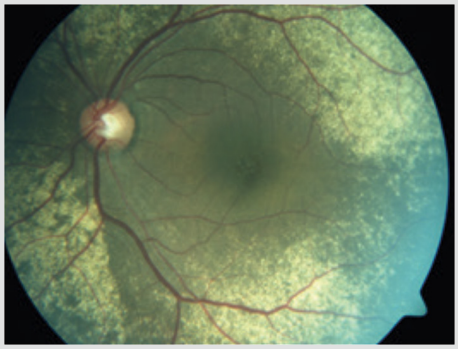

Right fundus image showing combined drusen (involving arcades and macular)


Left fundus image showing combined drusen (involving arcades and macular)


Right eye fundus image showing combined drusen with an island on superior arcade


Left fundus image showing combined drusen


Right fundus image showing drusen along the arcades


Left fundus image showing drusen along the arcades

A chi-square test for trends was used to assess the number of patients diagnosed with FDD as a proportion of the total number of patients seen throughout the study period and a *p*-value of less than 0.05 was considered statistically significant.

Ethical considerations: Ethical clearance to conduct the study was obtained from Sefako Makgatho Health Sciences University Research Ethics Committee (REF: SMUREC/M/163/2022: PG). Permission for the study was sought from the management of the DGMAH, where data were collected. The confidentiality of the patients was maintained throughout the study by allocating a numbering system to the images and clinical records to retain anonymity.

Results

A total of 47 142 new ophthalmic patients were seen during the study period, of which 39 (0.08%) had been diagnosed with FDD. *Figure 1* illustrates the proportion of FDD cases per year. The chi-square test for trends did not show statistically significant differences in FDD proportions over time (p = 0.9905); however, the proportion of FDD cases increased slightly from 0.07% in 2013 to 0.1% in 2021.

Table I presents the demographic information and the clinical findings of patients with FDD. The median age at presentation was 61 years, with an interquartile range of 56 to 70 years. All the patients were 40 years or older and were African. There were more females (28; 72%) than males (11; 28%). Most patients (38; 97%) were from local areas, specifically Soshanguve and Ga-Rankuwa. The size of the drusen was as follows: 26% (n = 10) small, 46% (n = 18) intermediate, and 28% (n = 11) large.

Macular drusen only were seen in 5% (2/39) of the patients *(Figure 2)*. Drusen distributed along the arcades were observed in 51% (20/39) of the patients, and 44% (n = 17) had drusen both at the macula and along the arcades. The disease was bilateral in all cases, with the same pattern in each eye. The median visual acuities are 20/50 in the left and right eyes, respectively. The different images of the FDD are shown in *Figure 3*.

Discussion

To the best of our knowledge, this is the first study to evaluate the magnitude of FDD in developing countries, particularly in South Africa. The majority of the studies in the literature were case study reports

carried out in developed countries.[1,6,8,9,10] We retrospectively analysed data from 2013 to 2021 and found that the proportion of patients diagnosed with FDD was 0.08%, which did not differ significantly over the study period.

In most case reports, FDD presents between the ages of 30 and 50 years and is more commonly seen in females [3,7,8,9,10,19] In their study, Käsmann and Völcker (1990) stated that, apart from genetics and family history, this entity (FDD) was characterised by the earlier appearance of drusen in patients aged 20 to 30 years.[20] In our study, the majority (72%) of the cases were also females, but all were of advanced age at presentation, which likely means that some of the patients had AMD and were misclassified as FDD. A molecular diagnosis would have helped reach a definitive diagnosis and facilitate genetic counselling.[21]

Case study reports have shown that drusen are distributed along vascular arcades, in the central macula and in the peripapillary region.[3,9] In our study, distribution along the arcades was most common (51%), followed by a combination of drusen distributed along the arcades and in the macula (44%), while macular distribution was seen in 5% of the patients. Fundus examination revealed that all cases were bilateral; in most cases (74%), the drusen were intermediate to large. Even though there is no treatment to prevent the progression of FDD, home monitoring using an Amsler grid is advised to pick up the early visual changes of developing CNVM.[21]

This study has several limitations. It was conducted in one hospital setting and the findings from a population-based study would be more appropriate for estimating the prevalence of FDD. Secondly, OCT imaging, fundus autofluorescence and genetic testing were not performed, which would have helped reach a definitive diagnosis. Moreover, to the best of our knowledge, no study is available on the prevalence of FDD worldwide, making it difficult to compare findings. Lastly, this study retrospectively reviewed previously acquired images and clinical notes with no access to additional information such as family history and no longitudinal data.

Conclusion

In conclusion, this study demonstrated that the number of patients presenting with FDD, as a proportion of the total number of patients who attended the ophthalmic clinic at this academic hospital, was small and did not differ significantly throughout the study period. Most of our patients were female and 40 years or older, which might indicate that some had been misdiagnosed as FDD when they had AMD. Drusen distributed along the arcades, and drusen both at the macula and along the arcades were commonly present in both eyes, and drusen of intermediate to large size were the most prevalent. It is necessary to follow up and monitor these patients for the possible development of CNVMs. In addition, further study is needed to assess if these patients developed CNVMs.

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Retrospective review of infective keratitis management at Groote Schuur Hospital in Cape Town, South Africa

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Abstract

Aim: To describe the demographics of patients presenting with infective keratitis (IK) at Groote Schuur Hospital (GSH) and review their presentation, management and complications.

Method: Retrospective review of 40 folders for patients who presented with IK from 1 January 2022 to 30 August 2022. Patient information from each folder was entered on SPSS 26 folder and descriptive statistical analysis was done.

Results: Forty folders of 26 males and 14 females were reviewed. The most common infective keratitis risk factors were human immunodeficiency virus infection, previous eye trauma and contact lens use, in 37.5%, 30% and 22.5% respectively. The mean duration of symptoms was nine days. Presenting visual acuity of equal to or worse than 6/60 was found in 87.5% of the study subjects. Corneal ulcer diameter greater than 4mm was seen in 50% of patients. Positive corneal scraping culture were found in 65% (26/40). The commonest cultured microorganisms was staphylococcus epidermidis and pseudomonas aeruginosa, which accounted accounted for 26.9% (7/26) and 19.2% (5/26) of positive culture cases. Topical ofloxacin and cephazolin antibiotics were first antibiotics of choice in 77.5% of patients on day one of hospital admission. Surgical interventions performed in this study group were; glue patches (12.5%), evisceration (7.5%), scleral patch graft (5%) glycerol corneal graft (2.5%).

Conclusion: Patients presented to GSH with sight threatening IK. The commonest comorbidity in these patients was HIV infection. Corneal scrapings had higher yield on culture than gram stain. Staphylococcus epidermidis and pseudomonas accounted for the highest number of positive cultures. Topical ofloxacin and cephazolin were the commonest drugs used for treatment. Surgical intervention was required in 27.5% of patients. Severe IK portends a poor visual outcome if not aggressively managed.

Funding: The authors have no financial interest in any of the material covered in this paper nor are there any conflicts of interests.

Introduction

The national burden of infective keratitis (IK) in South Africa remains unknown. Infective keratitis as a cause of unilateral blindness is estimated at 2-3 million per year[1]. The epidemiology of IK differs according to studies and geographical locations. The visual outcome can depend on virulence of the infecting organism, susceptibility to treating medications and compliance with treatment guidelines.

Complications of IK include corneal perforations, endophthalmitis and corneal opacities. These complications can necessitate expensive and not readily accessible surgical interventions.[2,3] The burden of consequent blindness causes economic hardships to the affected and is also an economic burden to the country. Definition of blindness uses visual acuity (VA) in the better eye thus unilateral corneal blindness from IK may be underreported when blindness

is discussed. A study on cost of patient care could shed light on local costs in South Africa.

Objectives

To describe the demographics of patients presenting with IK, associated factors, visual function before and after treatment.

To describe corneal scraping culture and sensitivity patterns.

To describe medical and surgical management of IK.

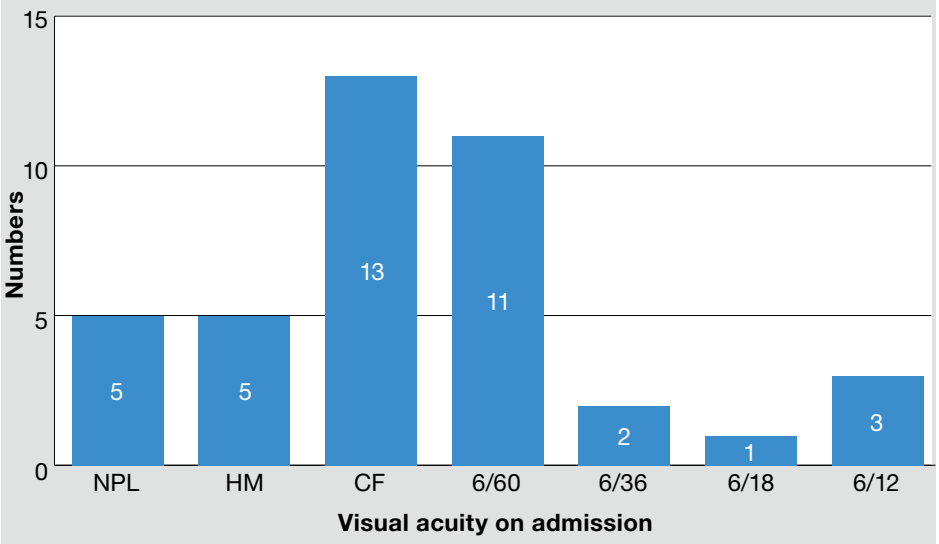

Figure 1: Snellen visual acuity on admission

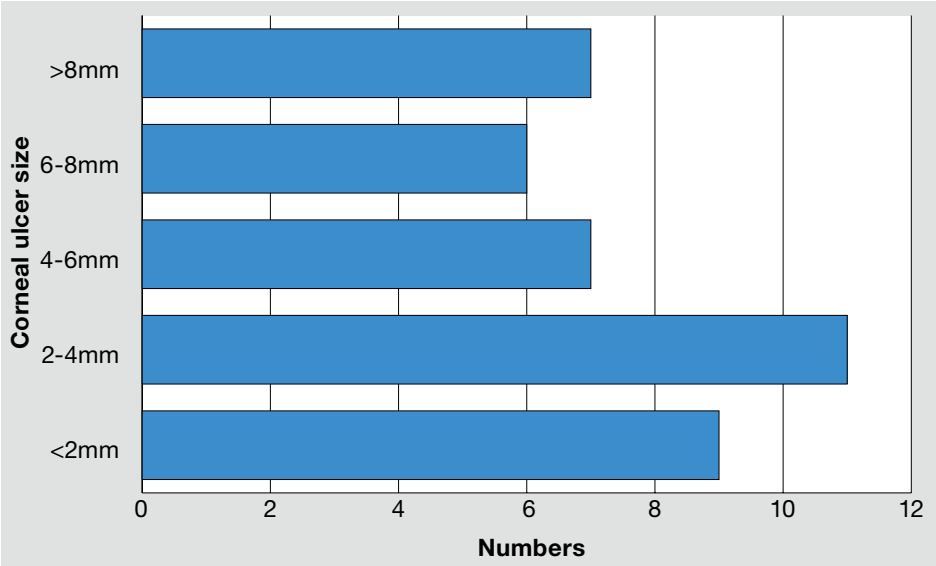

Figure 2: Corneal ulcer size on hospital admission

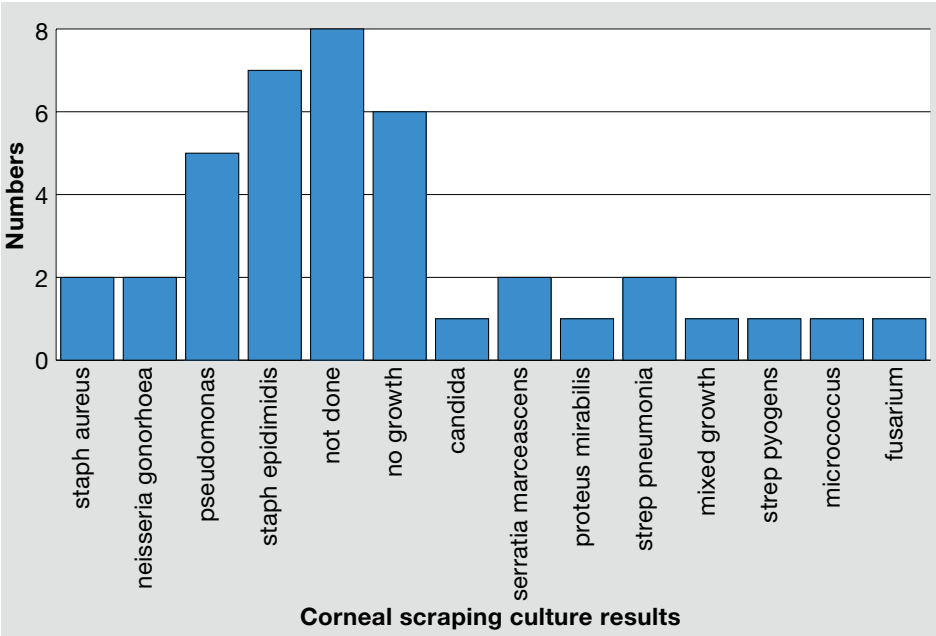

Figure 3: Corneal scraping culture results

Methods

A retrospective audit of all patient folders with ICD H16 (coding for infective keratitis) from 1 January 2022 to 30 June 2022 was done. Folders were obtained from Hospital Records Department. Information on each patient; age, gender, duration of symptoms, risk factors, clinical corneal examination findings, medications used and progress notes up to discharge were transcribed into an IBM SPSS 26 version folder with no patient identification details. Laboratory results of corneal scrapings were checked from National Health Laboratory Services (NHLS) website. Patient drug charts were reviewed and drug application confirmed against nurse signatures. Frequency and tapering of topical medications was checked. No folder was discarded even if there was some missing information. Approval for the study was received from the UCT Department of Surgery Research Committee. Analysis was done on available data. Statistical analysis, graphs and tables was done using SPSS 26 software.

Results

Patients' information was reviewed from 40 folders. It showed that 65% (26/40) of the patients were male and 35% (14/40) were females. The mean age was 45 years (SD 15) with a median of 46 years. The corneal ulcer was restricted to the right eye in 60% (24/40) and 2.5% (1/40) had bilateral ulcers. The main reported risk factors were noted to be:

- 37.5% (15/40) were Human Immunodeficiency Virus (HIV) positive, with (10/15) of these not on antiretroviral treatment.
- 30% (12/40) reported previous trauma to the eye. 22.5% (9/40 had previous eye surgery with five out of these nine having had previous eyelid surgery.
- 22.5% (9/40) had contact lens related infective keratitis
- 20% (8/40) had recurrent corneal ulcer disease
- 10% (4/40) had been using topical steroids prior to corneal ulcer.

The mean duration of corneal symptoms was nine days (SD 4.7). Corneal ulcer with hyopyon was present in 40% (16/40) of subjects on presentation. Visual acuity (VA) at presentation is as shown on

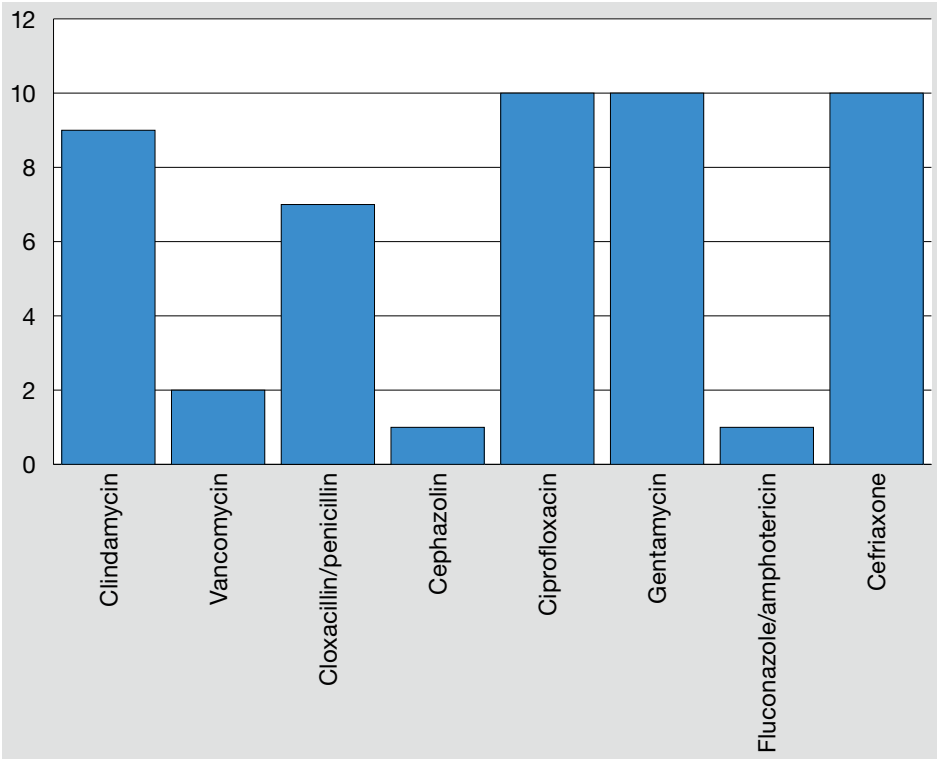

Figure 4: Corneal culture sensitivity results

Figure 1. It shows that 87.5% (35/40) had initial VA less than or equal to 6/60.

Fifty per cent had corneal ulcer size of less than 4mm while the rest had above 4mm as shown on *Figure 2.* Central corneal ulcers were found in 47.5% (19/40) of patients while the rest had paracentral and peripheral ulcers. Corneal scars were noted in 50% of patients on discharge from the ward.

Corneal scraping for microbiological examination was done in 80% (32/40) of patients of which 87.5% (28/32) was done on day of admission. Reasons for late scrapping were: patients already on antibiotics, thin descemetocoele and perforated corneal ulcers. Gram stain demonstrated that 12.5% (4/32) of specimens were gram positive, 9.4% (3/32) showed gram negative and 25% (8/32) showed neutrophils only. The overall yield on gram stain was thus 46.9% (15/32). The mean number of days it took for culture results to be available on the laboratory website was 4.5 days with a maximum of 16 days for fusarium.

Corneal scraping culture results are as shown on *Figure 3.* Positive culture results were noted in 81.3% (26/32) of corneal scrapings of which six specimens grew two microorganisms. There was no growth on 18.8% (6/32) of specimens. The commonest cultured microorganisms were staphylococcus epidermidis and pseudomonas, which accounted for 26.9% (7/26) and 19.2% (5/26) of cultured microorganisms respectively. Only two patients had positive fungal cultures, namely candida and fusarium.

The commonest drug sensitivity was to gentamycin, ciprofloxacin and ceftriaxone, with least sensitivity to cephazolin as shown on *Figure 4.* Resistance to penicillin was registered in 27% (7/26) of positive culture specimens.

Topical antibiotics were started on day of admission in 97.5% (39/40) of patients with 77.5% (31/40) put on hourly topical antibiotics, while 20% (8/40) were put on two hourly and 2.5% (1/40) on four hourly. The majority (57.5%) of patients had tapering of antibiotic frequency on day four and five. Topical antibiotics that were adminstered on admission are shown on *Figure 5.* It shows that 77.5% were started on ofloxacin and cephazolin. Topical steroids were adminstered in 27.5% (11/40) patients during the course of admission. Subconjuctival antibiotics were adminstered to two patients.

The commonest steroid used was dexamethasone which was used in 15% (6/40) of patients. This was started on average on day seven of hospital admission in patients who used topical steroids. Oral doxycyline was given to 65% (26/40) of the patients, while 60% (24/40) received vitamin C. Acyclovir was adminstered to 15% (6/40) of patients for clinical suspicion of coinfection with herpes simplex virus.

The mean length of hospital admission was 10 days (SD 7.4), with a mode of five days. The longest hospital stay was 61 days in a patient who was diagnosed with tuberculosis and tertiary syphilis during hospital stay. The patient who had IK secondary to fusarium stayed in hospital for 26 days.

The study showed that 27.5% (11/40) lost one snellen VA line and 20% (8/40) had no change in VA. Corneal perforation occured in 22.5% (9/40) of patients and three patients had complete corneal melting. The study revealed that 7.5% (3/40) of the patients required eviscerations. The three eviscerations presented with VA of NPL with completely melted corneas. There

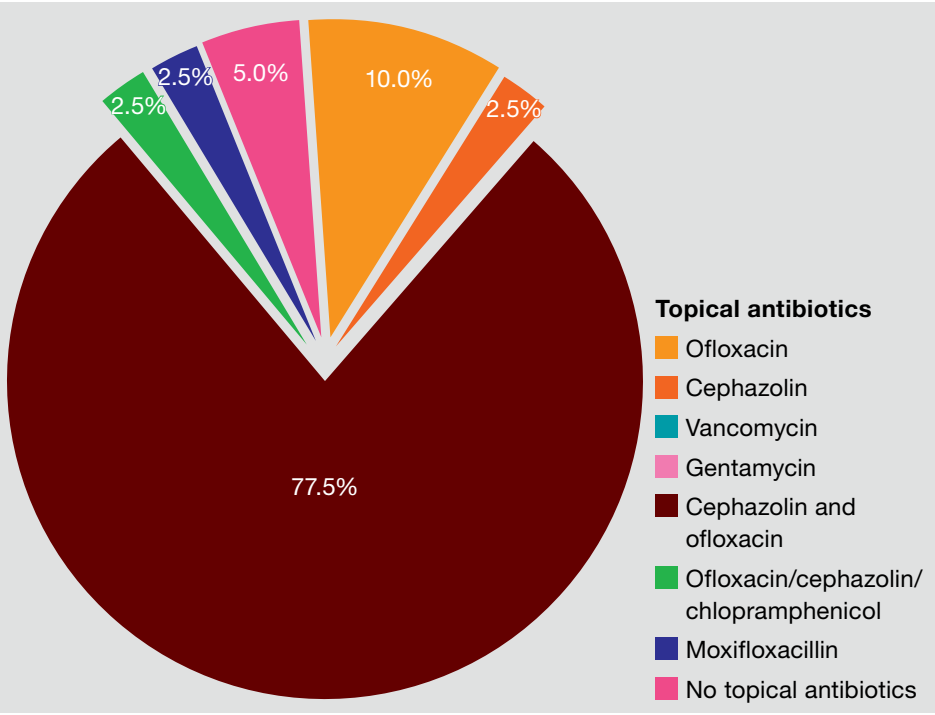

Figure 5. Topical antibiotics used on day 1 of hospital admission

was one glycerol preserved corneal graft surgery, two scleral patch grafts and five glue patches done.

Chi square tests did not show any significant association between change in final VA with corneal ulcer size (p = 0.281), no association between change in final visual acuity with gender (p = 0.634), nor with any risk factor for infective keratitis.

Discussion

The study describes IK presentation and management at GSH. This serves as an assessment of all relevant team players namely doctors, nurses and the laboratory scientists that receive the corneal scraping specimens for culture and sensitivity to guide doctors in choice of antibiotics to prescribe. The ultimate aim is to improve care and reduce avoidable blindness secondary to IK.

The mean age of 45 years is within the commonly reported mean age for IK of 35 to 55 years.[4,5,6] The average duration of symptoms of nine days and median of seven days is less than the median of 19 days mentioned in a study by Schafyenaar *et al.* which was done in a rural setting in South Africa.[7] This is, however, not unexpected since GSH is a tertiary hospital in an urban setting. The hospital however acts as a secondary level care due to absence of full time ophthalmologists or ophthalmic medical officers at some secondary level hospitals in Cape Town. This can be improved by equiping primary and secondary level care centres with expertise and general community congentisation to improve early health seeking behaviours. Duration of symptoms before admission was almost similar to the mean of 8.9 days from a study from New Zealand by T Wong *et al.*, showing comparative behaviour with our study.[8]

This study confirmed the known risk of IK among HIV infected patients though there could also be other confounding risk factors. Jeng *et al.* showed that there was approximately a tenfold increased risk of IK in HIV infected individuals compared to non-infected persons.[9] These HIV positive patients were all referred to clinics on discharge to be investigated for antiretroviral management according to South African guidelines. Contact lens related IK of 22.5% in this study is lower than quoted rates of 35-65% reported in major hospital centres in the western world.[2,3,2,10,11] This could be explained by possible low contact lens use in patients who present to state hospitals in South Africa. A study on uptake of contact lenses in South Africa could not be found by the authors.

Previous eye trauma was found in 30% of patients, which is higher than quoted rate of 15% in a study from France by T Bourcier *et al.*[11] This could be explained by high general and/or ocular trauma in South Africa.[12] A study from India of 1 644 patients demonstarted that ocular trauma (predominantly vegetative) accounted for 60.3% of corneal ulcers.[13] The urban setting of GSH could account for the lower risk of vegetative trauma. Previous eye surgery (iatrogenic trauma) was however reported in 22.5% (9/40) of the patients.

Visual acuity on presentation was 6/60 or worse in 87.5% (35/40) of patients which is worse than a study on severe IK by T Wong *et al.* in which 40% of study patients had VA worse than 6/60.[8] Study from East Africa by Burton *et al.* had initial VA worse than 6/60 in 81% of patients.[14] This could be explained by differences in aetiology of infective keratitis, virulence of causative microorganisms, size and centrality of corneal ulcers and other associated risk factors like HIV.

High percentage (47.5%) of central corneal ulcers could account for poor initial and final VA due to optic axis involvement and induced astigmatism from the ulcers and corneal opacities. A study by Ibrahim *et al.* showed that initial VA was significantly associated with ulcer size and location.[15] This study showed that 22.5% of patients with previous history of eye surgery had VA which was worse than 6/60 while a study by Butlet *et al.* on IK in above 60-year-old patients showed that patients with previous eyes surgery also had poor initial VA.[16] Compromised corneas from previous eye surgery can be a high risk for poor visual outcomes because of corneal scars.

This study showed that 53.1% (17/32) of patients lost at least one line on Snellen VA, while only 18.8% (6/32) gained at least one line on discharge. This could have changed the ratios if eight patients had final VA recorded in the notes. It has to be recognised that this was only a change in VA within a mean of nine days of hospital admission. The reviewed patient folders had VA measured using Snellen Charts not Logmar Charts, thus improvement in vision could not have been adequately captured. Prospective studies tend to have more robust measurement of VA than retrospective studies due to their study designs. The Steroids for Corneal Ulcer Trial subsequent follow up study showed that VA in bacterial keratitis continues to improve up to 12 months after initial presentation.[17] This audit did not seek to find VA changes on subsequent follow up visits due to time constraints.

Culture result positivity was higher in current study than results from two South African studies, a retrospective and a prospective study, which yielded positive cultures in 51.3% and 42.1% of corneal scrapings respectively.[18,19] These studies, however, showed higher culture yield than a study on severe sight threatening IK by Otri *et al.* which had positive cultures in 41.7% of the specimens.[20] The overall positive yield from gram stain of 40%, is much lower than the microbiological culture yield of 65% in this study. It could either mean poor specimen sampling from the doctors on the glass slides, or general limitations of gram staining as a diagnostic tool. A study by Burton *et al.* at Kilimanjaro Centre for Community Ophthalmology showed that 65% of specimens did not show any organism on gram stain, this compares favourably with this study.[14] A reported gram stain diagnostic accuracy 65%-75% for bacterial keratitis and 35%-95% for fungal keratitis was reported by Badiee *et al.* and Gopinanthan *et al.* in studies that confirmed diagnosis with polymerase chain reaction (PCR).[21,22] Gram staining thus still remains a good diagnostic tool. Standardisation of corneal scraping collection methods need to be explored in our unit. The study did not explore numbers of patients who were already on antibiotics on referral to GSH; this could have led to negative culture results.

The culture results appear to be similar to a previous South African study which confirmed staphylococcus epidermidis as the commonest organism causing IK. It accounted for 26.9% in this study compared to 36% from the study by Schaftenaar *et al.*[7] Our results differed from Proxenos *et al.* study which showed that streptococcus pneumoniae comprised 57% of the cultured bacteria while this study showed streptococcal pneumoniae accounting for 8.3% (2/24) culture results.[18] The Koetsie *et al.* study at a tertiary hospital in South Africa showed predominance of staphylococcus species as causative to IK.[23] Cultures from this study could have been commensals but it is difficult to be conclusive. Commensals have been known to cause multiple systemic diseases, thus could account for the infective keratitis.[24] An eight year study from a tertiary hospital in India showed that staphylococcus species accounted for 64.5% of all

bacterial isolates with predominance of staphylococcus epidermidis.[25] These different studies show that culture results differ according to location but confirm staphylococcus epidermidis as causative.

There were no polymerase chain reaction (PCR) viral tests for any of the collected specimens, thus polymicrobial infection involving bacteria and viruses could not be confirmed. Six patients in this audit were on acyclovir, suggesting viral coinfection was clinically suspected. A prospective study in Cape Town by Smit *et al.* concluded that polymicrobial infections were common in IK.[19]

Fungal growth was found in 7.7% (2/26) of the positive cultures results, which is higher than a larger retrospective study at McCords Eye Hospital in KwaZulu-Natal in South Africa by Proxenos *et al.* which showed that fungal growth accounted for 2.5% of cultured microorganisms, and also marginally higher than the study in Cape Town by Smit *et al.* which had 7.4% of cultures being fungi.[18,19] The prospective study by Smit *et al.* used calcium alginate swab as is used at GSH to collect the sample, while the Proxenos *et al.* study used disposable blades. A study by Jacob *et al*. showed that there was no significnt difference between using clacium alginate swabs and Bard Parker blades in quality of specimen collected.[26]

Topical ofloxacin and cephazolin was the initial empiric antibiotic choice in 77.5% of patients though sensitivity to cephazolin was only reported on one culture result. Sensitivity to cephazolin is seldomly tested by our laboratory due to lack and cost of cephazolin antibiotic plates. Different antibiotic protocols are mentioned in different studies. Topical antibiotic choice in the department follows the Food and Drug Adminstration (FDA) recommendation of fortified ofloxacin and cephazolin.[5,6] Subconjuctival adminstration of antibiotics was administered on two patients who had poor initial response to topical antibiotics. This route was chosen because of the scleral extension of the corneal ulcer.

Otri *et al.* studied severe corneal ulcers and used cefuroxime and gentamycin in 61.2% of patients.[20] The American Academy of Ophthalmology recommends cephazolin and gentamycin or fluoroquinolone as empiric therapy for unknown aetiology of bacterial keratitis.[4] Only 20% of patients were on one antibiotic on admission. Monotherapy is considered an acceptable management in many studies for mild infective keratitis (grading scale by Acharya *et al.*) or infection outside the visual axis.[27,28,29] Only 30% of patients on monotherapy in this audit complied with this definition. This violated department protocol on dual therapy for severe infective keratitis.

Frequency of application of topical antibiotics was hourly in 77.5% of patients on admission. No patients were put on a loading dose of quarter or half hourly as is recommended for severe keratitis.[4] Frequency higher than hourly might require a higher nurse patient ratio. No patients received subconjunctival antibiotics on day of admission but later during the course of treatment. Subconjunctival injection is often recommended when there is impending scleral extension and when there is limitation of nursing staff to apply hourly medications.[30] Drug charts reviewed confirmed hourly application of topical antibiotics by nursing staff. There could be adequate staffing of nurses at GSH to apply hourly medications. Topical steroids were used in 30% (12/40) of the patients possibly due to fear of complications from topical steroids and the severity of the corneal ulcers under this study. Steroids could have helped reduce size of corneal opacities in the long run.

The three eviscerations were inevitable due to corneal melt with VA of no light perception. These patients all presented more than 14 days after onset of symptoms. Delay in presentation could have accounted for the poor outcome. Unfortunately, there were no corneal scrapings nor bacterial culture on the two patients who had eviscerated globes. Eviscerated globes should have been submitted for microbiological cultures. The culture result from one patient who had eyeball evisceration was streptococcus pyogenes. This organism has been known to cause severe ocular morbidity.[31] Wills eye Hospital study on microbial keratitis by Cruz *et al.* showed that 1.8% (17 of 965) of study patients had eviscerations, these were all severe IK from mostly pseudomonas and streptococcal infections.[32] Another study showed that eviscerations were done in IK mostly caused by staphylococcus, propionibacterium and pseudomonas, unlike in this study.[33] Severe IK seems to be a high risk for complications leading to eviscerations.

Study limitation and strengths

This was a retrospective study with inherent reliance on patient notes, which were not always legible. There was reliance on nurses' signatures to confirm drug administration. The study missed IK treated as outpatients as it was difficult to locate the patient folders due to poor ICD 10 coding by the doctors in the out patients department. There was an assumption that the technique of corneal scraping was adequate, though different doctors did the scrapings. No patient folder was discarded to represent actual practice and reality in our department.

Conclusion

This retrospective study showed that many patients present to Groote Schuur Hospital with sight threatening IK. The commonest comorbidity in these patients was HIV infection. Patients were timeously started on empiric topical antibiotics. Corneal scrapings had higher yield on culture than gram stain. Staphylococcus epidermidis accounted for the highest number of positive cultures. There was little improvement in VA during hospital admission. Surgical intervention was done in 27.5% of study subjects. Severe IK portends a poor visual outcome if not aggressively managed.

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AD/COM/1.0 04/2023

Vol 18 | No 2 • Autumn 2023
SA Ophthalmology Journal

Unusual pattern of geographic atrophy in a case of age related macular degeneration

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Abstract

Purpose: To report a case of atypical geographic atrophy associated with age related macular degeneration.

Methods: Case report and review of literature.

Summary: In this case report and literature review we present a patient with age related macular degeneration. Over the course of 17 years of follow up, our patient developed geographic atrophy which started in the peripapillary area and enlarged in an arcuate pattern along the vascular arcades, initially as discrete, round lesions which later coalesced.

Results: A 69-year-old white female presented with age related macular degeneration. She was sequentially treated for exudative disease in both eyes. The left eye progressed to end stage disease with a large area of geographic atrophy in the macular region, the right macula was spared. Concurrently, a very atypical progression of atrophy of the retina developed bilaterally, originating in the peripapillary area, and extending along the arcades in an arcuate pattern. While this atrophy initially presented as well defined, roughly circular areas, it went on to enlarge and coalesce over the course of the 17 year follow up.

Keywords: Age related macular degeneration, choroidal neovascular membrane, drusen, perivascular atrophy, peripapillary atrophy, geographic atrophy

Conclusions: While abundant information about GA associated with AMD exists, to our knowledge, this is the first reported case of this pattern of atrophy.

Conflict of interest: None of the authors have any proprietary interest. No funding was received for this paper.

Introduction

Age related macular degeneration (AMD) is a disease of the macula which encompasses progressive loss of vision due to late-onset neurodegenerative changes of the photoreceptor- retinal pigment epithelium complex.[1]

AMD is the most common cause of severe loss of vision in developed countries, affecting 10% of the population over 65 years and over 25% of people older than 75 years.[1,2] With an increasingly aging population, these numbers are expected to rise.[3]

While severe loss of vision is most commonly due to choroidal neovascularisation (CNVM), geographic atrophy (GA) accounts for 20% of legal blindness attributed to AMD, affecting over eight million people worldwide.[2,4,5] It may occur in patients with both exudative and non-exudative AMD.[6,7,8]

GA manifests as areas of outer retinal atrophy which progressively enlarge and result in absolute scotoma of vision. [9,10] The mechanisms and natural progression of GA remain poorly understood.[11] Clinical presentation is with lesions of variable topography, in the extrafoveal area which enlarge over time, affecting the fovea in the late stages of disease.[12]

We report on a case of unusual retinal atrophy associated with AMD.

Case report

A 69-year-old white patient presented, in 2004, with bilateral AMD. She was a non-smoker and reported no significant medical history. Family history included paternal loss of vision of unknown aetiology.

On presentation, best corrected visual acuity (BCVA) in the right eye was 1.0, BCVA in the left eye was 0.25 with no improvement on pin hole testing. Anterior segment examination was normal bilaterally. Both eyes had undergone phacoemulsification with implantation of monofocal intraocular lenses. Intraocular pressures were 12 mmHg and 14 mmHg in the right and left eye respectively. The vitreous bodies were syneretic and synchytic bilaterally. On fundoscopy of the right eye the C:D ratio was 0.3, the vasculature appeared normal. Dense medium and large drusen were present in the centre of the macula as well as reticular pseudodrusen in the superotemporal quadrant of the macula. No pigmentary changes were present. *(Figure 1A)* Fundoscopy of the left eye revealed a C:D ratio of 0.3. The

vasculature was normal. An area of GA was present at the macula, measuring 1 disc diameter. Intermediate and large drusen were also present in the macular region, which was clinically swollen. *(Figure 1B)* Exudative AMD was confirmed in the left eye by fluorescein angiography, with diffuse leakage at the macula signifying the presence of an occult choroidal neovascular membrane. *(Figure 2)* This swelling resolved with intravitreal triamcinolone injection and full fluence photodynamic therapy.

Three years later, the right eye developed a CNVM, by which time intravitreal bevacizumab was available and administered in a treat and extend fashion. After two years of treatment, swelling of the right macula had resolved and treatment was stopped.

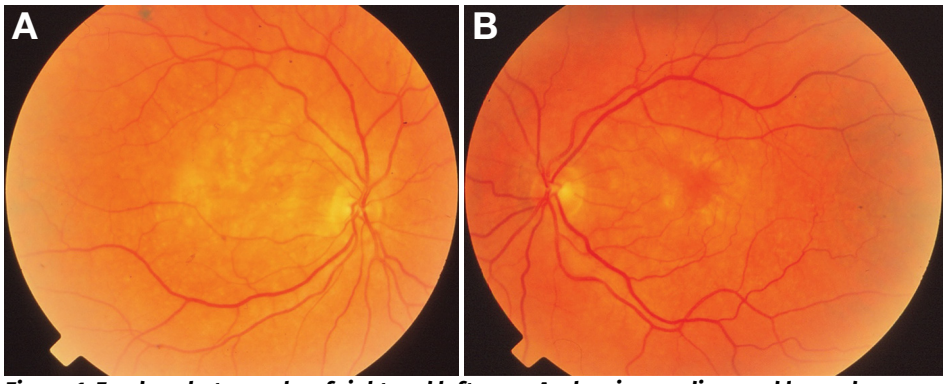

Figure 1: Fundus photographs of right and left eyes. A, showing medium and large drusen at the macula and reticular pseudodrusen in the superotemporal quadrant of the macula. B, showing intermediate and large drusen at the macula with early atrophic changes.

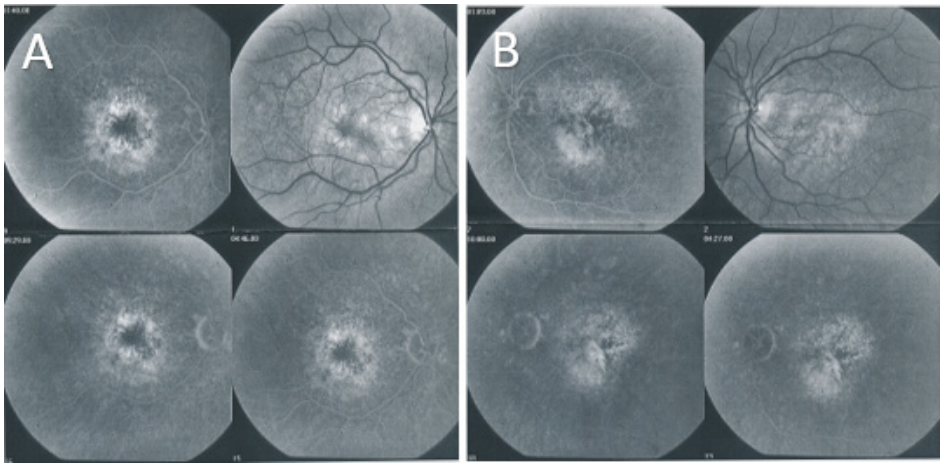

Figure 2. Fundus fluorescein angiogram right and left eyes. A, showing confluent drusen in the perifoveal area as well as perivenous pseudoreticular drusen. B, showing perivenous pseudoreticular drusen, multiple drusen in the perifoveal area and diffuse leakage at the macula.

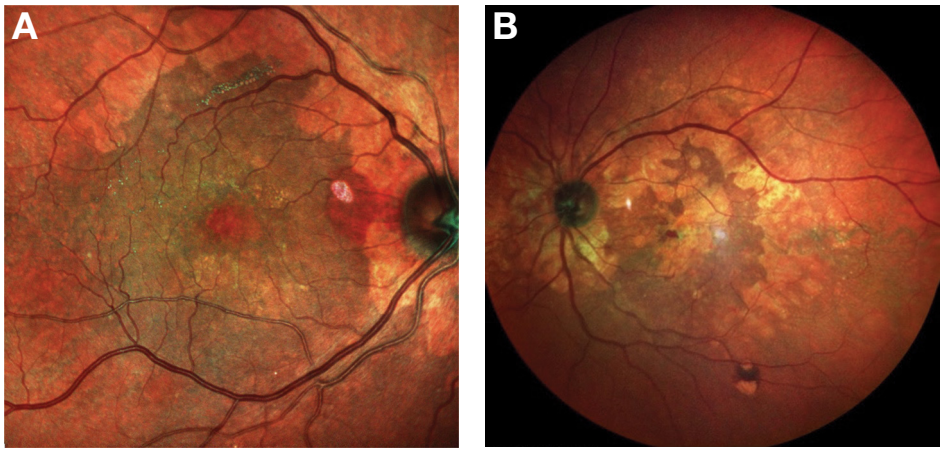

Figure 3. Multicolour images right eye and left eyes. A, showing temporal peripapillary haemorrhage of 0.75 disc diameter. B, showing peripapillary atrophy extending in an arcuate pattern along the superior vascular arcade. Geographic atrophy and pigmentary changes at the macula with drusen temporally.

Twelve years later, leakage recurred, with central subretinal fluid on ocular coherence tomography (OCT) and an area of haemorrhage, temporal to the optic disc. *(Figure 3A)* Intravitreal bevacizumab was initiated with three loading doses, a month apart and continued according to the treat and extend protocol. By this time, central geographic atrophy had worsened in the left eye with a resultant BCVA of count fingers. *(Figure 3B)* The right central macula was spared from atrophic changes, with a BCVA of 1.0 in that eye.

During 17 years of follow up, the area of the left macular atrophy enlarged to a size of 2 disc diameters. In addition to this, an unusual, progressive pattern of geographic atrophy formed bilaterally. This peripapillary atrophy extended in an arcuate fashion along the vascular arcades, to a greater extent in the temporal, than the nasal region and along the superior arcade more than the inferior arcade. There were no associated pigmentary changes. Although initially occurring as focal, discrete patchy areas, the atrophy later became confluent and more extensive, involving the entire length of the superotemporal arcades, bilaterally. *(Figure 4).*

Discussion

We report a patient with AMD with associated atypical GA. The pathophysiological course of the development of GA, involving retinal pigment epithelium (RPE) atrophy, loss of adjacent photoreceptors and atrophy of the corresponding choriocapillaris and outer neurosensory retina, is poorly understood.[12,13] Recent evidence indicates that lipofuscin accumulation in the lysosomal compartment of RPE cells is central to the mechanism of disease.[14,15,16] Postmitotic RPE phagocytosis of photoreceptor outer segments is believed to contribute to lipofuscin accumulation. Excessive lipofuscin accumulation is a common pathophysiological mechanism in various degenerative and genetically inherited retinal diseases as lipofuscin contains toxic compounds which interfere with normal cell function.[17,18,19,20]

The clinical presentation of GA is characterised by one or more clearly defined, roughly circular, patches of 175um or more in diameter, with partial or total loss of pigment of the RPE, revealing the underlying choroidal vasculature.[10,21] These atrophic patches typically occur parafoveally and progressively coalesce as new atrophic areas occur. This characteristically results in a horseshoe pattern of atrophy which can later advance to a ring of atrophy that encircles the fovea, termed foveal

sparing.[22,23] End stage disease occurs if the fovea is eventually involved, which results in severe loss of vision.[24]

The Classification of Atrophy Meeting (CAM) has recently devised an alternative nomenclature for the different stages of AMD.[25] Incomplete retinal pigment epithelial and outer retinal atrophy (iRORA) in patients with AMD with conventional drusen and complete retinal pigment epithelial and outer retinal atrophy (cRORA), which describes an endpoint of atrophy that occurred in the presence of drusen.

Geographic atrophy has progressed to end stage disease, as described above, during follow up of our patient's left eye. However, unique to this case, are the bilateral large areas of peripapillary, arcuate areas of atrophy, that have formed in a pattern associated with the retinal vessels.

A significantly higher incidence and more severe presentation of peripapillary retinal pigment epithelium changes (PPRCs) has been reported to occur in patients with AMD than in cases of other eye conditions known to be associated with RPE changes.[26]

A grading scale of PPRCS of 0-4 seen of fundus autofluorescence was: 0, absent; 1, uneven background; 2, focal hyperautoflourescent dot and spots; 3, light reticular pattern; 4, dense reticular pattern of hyperautoflourescent and hypoautoflourescent changes.[26] According Cohen *et al*., 2016, only 3% of AMD eyes in which PPRCs were found, had grade 4 disease. The areas of atrophy were far more localised to the peripapillary region than those seen in our patient. OCT of those cases graded 2 and 3 exhibited deposits both above and below the optic disc, in the areas if PPRCs, as is seen in our case. However, this study was not longitudinal and was therefore not able to form relationship between PPRCs and a final atrophic stage, peripapillary atrophy (PPA). Other studies have suggested that an association may exist between PPA and advanced AMD.[9,12,27,28] PPA which is not confluent with the macular area of atrophy is highly prevalent.[29] Once again, the PPA described in these studies, is far more localised to the immediate peripapillary area than that seen in our case.

Several differential diagnoses can be considered in this case:

Pigmented paravenous retinochoroidal atrophy (PPRCA) is a rare disease characterised by perivenous accumulation of pigment clumps which are associated with peripapillary and radial areas of retinochoroidal atrophy, located along the retinal veins. It has been postulated

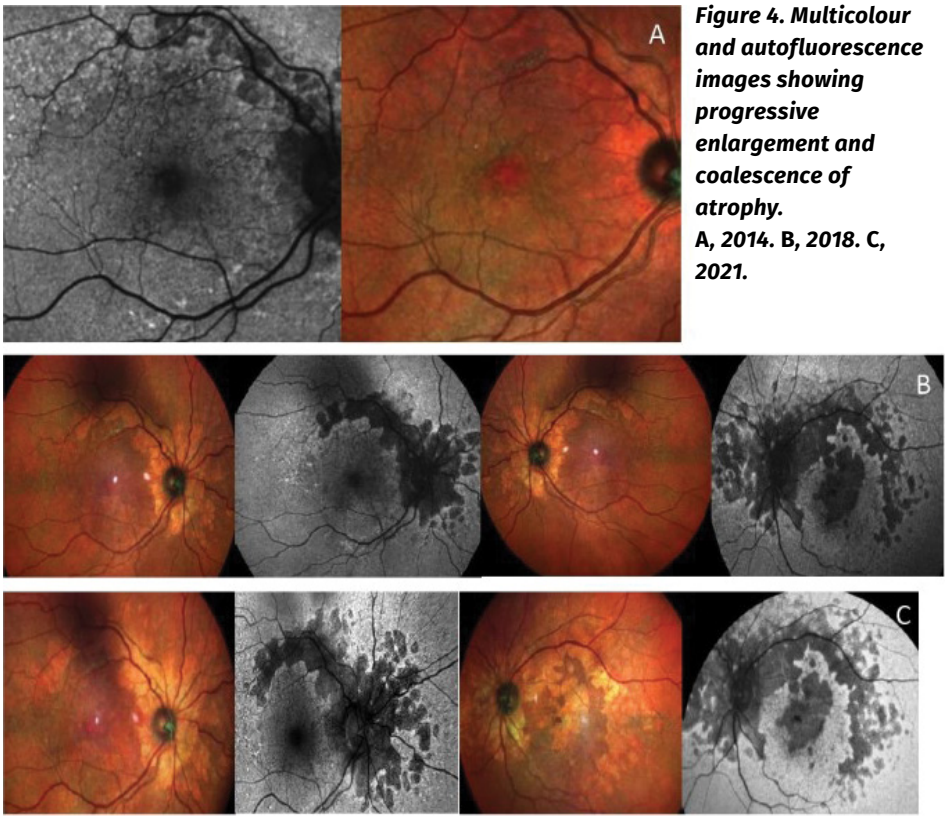

Figure 4. Multicolour and autofluorescence images showing progressive enlargement and coalescence of atrophy. A, 2014. B, 2018. C, 2021.

to be a developmental disorder of the retinal blood vessels during the embryonic stage of ocular development.[30] Patients may be asymptomatic or have mild loss of vision. Over and above the lack of pigment aggregation which typifies this disease, the changes seen in PPRCA are invariably located at a distance from the optic nerve head and not in the immediate peripapillary zone as seen in our patient. The macula has been reported to be affected in a minority of cases, none of which report an association with choroidal neovascularisation.

Helicoid peripapillary chorioretinal degeneration, initially described by Sveinsson, is a rare pathology, characterised by slowly progressive, bilateral disease. It encompasses well defined, wing shaped areas of atrophy of the choriocapillaris and RPE, radiating from the optic nerve to the macula and peripheral fundus.[31] Autosomal dominant (AD) inheritance is via a mutation in the *TEAD1* gene, located on chromosome 11p15.[32] Good response to intravitreal ranibizumab has been reported when complicated by CNVM.[33] While the areas of atrophy extend from the optic nerve, they characteristically occur as tongue shaped strips, unlike the morphology of atrophy in our patient.[32]

Pericentral retinitis pigmentosa or pericentral pigmentary retinopathy is a rare disease, characterised by bilateral, symmetrical transparency of the RPE that follows the retinal vasculature. The areas involved occur in association with bone spicule pigment clumping, in an arcuate pattern, extending from the disc. Inheritance may be autosomal recessive or AD. No pigment clumping was seen in our patient.

Serpiginous choroidopathy results from active inflammation, known as serpiginous choroiditis.[34,35] As the typically bilateral inflammation resolves, the RPE, choriocapillaris and choroid atrophy in the juxtapapillary area, extending radially to involve the macula and peripheral retina.[36] This diagnosis can be excluded as our patient had no clinical evidence of previous inflammation.

Angioid streaks are crack- like dehiscences of Bruch membrane which result in corresponding changes of the RPE and choriocapillaris.[37] While CNVM is a major complication, the cracks appear as dark, reddish brown bands, which surround and radiate out from the optic disc toward the peripheral retina, with no relationship to the vessels, differentiating this disease from our patient's.[38]

Myopic eyes may present changes in the peripapillary area such as peripapillary detachment, peripapillary retinoschisis, peripapillary microfolds, and paravascular cysts.[39] The diagnoses were easily excluded on OCT imaging of our patient. In addition, her axial lengths were 23.66 mm and 24.21 mm in the right and left eyes respectively.

Conclusion

Geographic atrophy involves atrophy of the retinal pigment epithelium (RPE)

atrophy, loss of adjacent photoreceptors and corresponding choriocapillaris. This represents the end stage of AMD. While abundant information about GA associated with AMD exists, to our knowledge, this is the first report of this pattern of atrophy.

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On a lighter note

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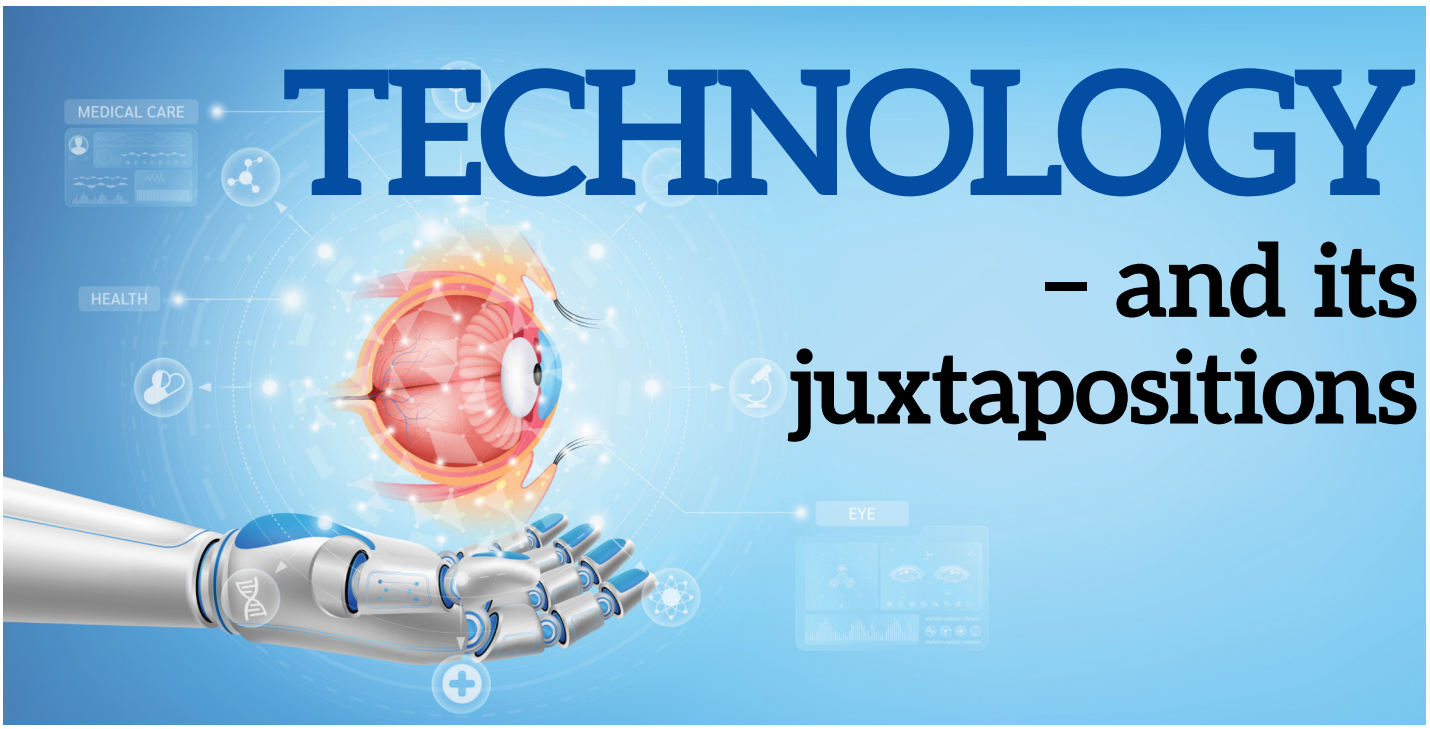

TECHNOLOGY – and its juxtapositions

William Gibson, the acclaimed futurist, said "the future is already here, it's just not evenly distributed". As I start off my time as president of the society, this is the topic foremost in my mind. Today my thoughts are on its implications in two areas. Firstly, thinking about how we help our profession and vocation remain economically sustainable amidst the ever-increasing cost of new equipment. Secondly – what will be the impact of the buzziest new technology around, Artificial Intelligence (AI).

Equipment costs are spiraling and when coupled with increased pressure from funders – it is starting to make many practices uneconomical. With machines that are increasingly complex, that require specialised skills to maintain – is the individual practice model still viable? This is perpetuated by a lot of the trade and industry that are reaching a celling in terms of their company growth – skills and incentives are lacking to repair and/or maintain equipment. So will ophthalmologists be forced, like radiologists into group practices to spread equipment costs over a wider set of doctors and patients. This has potentially dire impacts on what has historically been a specialty characterised by independence, autonomy, and innovation. Since our public sector colleagues already lack funding, the impacts of the lack of resources are far more devastating

At the same time, we have the dramatic explosion of advancements in AI. A side-effect of the Covid pandemic was the arrival of AI a decade earlier than predicted. Much like the Covid pandemic, when data needed to be analysed in real time – not a five-year randomised double-blind controlled cross-sectional study – agile growth in AI is happening incredibly fast with little time to reflect or analyse; time lost in viewing the bigger picture or pausing to figure out the question means getting left behind.

Medicine is very much a conservative profession. We will not be swallowed up by AI yet, and that gives us time to use it to our advantage. In ophthalmology – there are a series of opportunities for us to enhance our practices by leveraging AI ranging from more affordable and adaptable electronic medical records to improved diagnostics using AI engines to improve speed and accuracy of OCT reporting.

Retinal screening for diabetic retinopathy is taking off very slowly; but the broader cardiovascular risk profiling from retinal screening is still in its infancy – eye screening will become useful to many other specialist fields in medicine as well, especially cardiology. In addition, it's exciting that there are already mechanisms to take fundus photos with mobile phones that are high enough quality to be analysed using artificial intelligence. The practical implications of this in a primary healthcare setting is overwhelming.

We need the time to ensure we are not replaced by AI or become purely reliant on it. There was a time, when part of a registrars training involved taking apart a slitlamp and reconfiguring it themselves. There are times now when I wish I had that skill to do my own maintenance and repair – though I would struggle to find the time. Keeping that in mind, is why we cannot overcommit and lose our skills in pure reliance on the machine.

As we do this, we need to also be thoughtful about the risks for both patients and as professionals. We are only just starting to think through the implications around data privacy and the legal and ethical issues surrounding liability – who gets sued when the AI is wrong? For radiologists – the answer is Google gets sued. Up to know, that is a huge component of why AI reporting has not been forced into implementation.

To manage these risks, we will need to ensure that AI is not just applied because it is there – it must have a clearly defined use and purpose; and be used in tandem with trained, experienced professionals.

Alan Kay, the computer scientist, said "If you want to predict the future, invent it". I see AI as part of the way in which we could bridge the ever-expanding gap. Running costs are escalating but remuneration is being heavily clamped down upon at every turn for practitioners. Putting cheaper tools in the hands of ophthalmologists will help improve our productivity and allow us to focus our time and skills in the moments that matter.

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POAG, primary open-angle glaucoma.

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Potpourri

The strange case of the lady who could not rub her eye

I did right phaco on Mrs X about a year ago. There were no complications, and she was very happy with the surgery. She now presents back to me with the complaint that, for the last three weeks she is unable to rub her right eye. She said that there was nothing wrong with her hand or arm or with her eye. She could rub it freely before the phaco three weeks ago. However, Mrs X could rub her left eye.

For some reason she has developed a phobia of rubbing her right eye a year after cataract surgery. I suggested that to overcome this, she forces herself to do it. I made her rub it in the consulting room and helped guide her hand to rub her right eye (gently of course).

I therefore suggest a new subspeciality: psycho-neuro-ophthalmology! I think these doctors will be very busy.

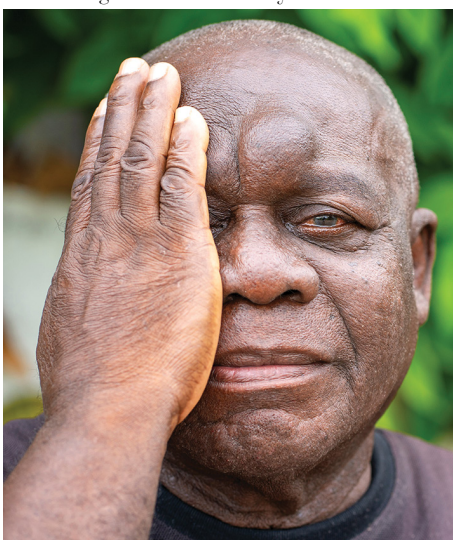
EMDR therapy

Talking about psychology, have you heard of Eye Movement Desensitisation Therapy?

Apparently, this is a fairly well-known technique amongst psychotherapists. The therapist asks the patient to look at their (the therapist's) finger. The finger is then moved horizontally from side to side and the patient asked to follow it. This is just like testing horizontal pursuit movements except that the finger is moved at a greater speed. While this is going on conventional psychotherapy takes place. It seems the therapy now is much more effective. Maybe it's some form of hypnosis? I should have used this technique on Mrs X.

Double trouble: a lesson in cultural diversity

A Zulu-speaking patient presented to me with bilateral cataracts. The right one was a mature white cataract. The left eye was a moderate cataract with 6/36 vision. I did phaco on right eye with no complications. At the one-week check-up I covered the right eye and asked him what he could see. He could see 'okay' and could read the 6/35 letters as expected. I then covered the left eye and asked him how his vision was with the right eye. 'Double' he answered. I tried again and he kept telling me that he saw double with the newly operated eye. I thought perhaps it was monocular diplopia. I looked at the IOL and it was perfectly positioned. I was totally confused. But not for long. My trusted Zulu-speaking ophthalmic assistant heard what was going on and exchanged a few words in Zulu with him. She then explained to me that he was using the word 'double' to explain that his vision was double as good as the other eye!



Weaning off steroid drops after phaco

I saw another ophthalmologist's post phaco drop regimen. It was very complicated with three different bottles each to be used at different time intervals. To make it even more complicated the steroid drops had to be tapered after two weeks. Compare this to my standard for 90% of my cases: One drop

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FUC AD 21_01

Tobradex four times per day. Finish the bottle. But why taper the steroid? I have never done this. Approximately 5% of my patients get the 'rebound reaction' but I don't believe this is what it is. The rebound reaction is increased inflammation after stopping the steroid without tapering. I think that this is not a rebound but simply a need for more steroids. To back-up this belief, I have always simply prescribed another bottle of Tobradex to these 5% of patients. This has always worked and there has never been another rebound after finishing second bottle thus supporting my theory.

Another thing I noticed on this doctor's instructions: No dental work for four weeks post-op. I have never instructed my patients to avoid dental work and my rate of endophthalmitis over the last 30 years has been extremely low. Do any readers also give this instruction?

A physiotherapist treats eye pain

A recent article in the *Indian Journal of Ophthalmology* (January 2023) describes myofascial pain syndrome of the occipitofrontalis muscle and its ophthalmological implications.

As I've mentioned in a previous article (Clive's Corner SAOJ Autumn 2022), one of the first things I ask a patient with retrobulbar eye pain is if they have neck trouble. This case report describes a 65-year-old male patient with headache and pain in the eyes. The ophthalmologist thought that the pain was arising from the neck via the occipitofrontalis muscle and so he sent the patient to a physiotherapist.

The physiotherapist used a 'hair pull technique' where the hair is lifted and pulled in the direction of the hair at the back of the patient's head and neck. The physio also used a 'cranial base release technique' where the therapist puts their fingers under the supine patient's head and gently separates the atlas from the occiput in a stretching motion. Immediately after this therapy, the patient reported significant pain relief.

So, why not teach your ophthalmic assistants how to do these techniques and bring the patient back for regular treatments?

Multiple YAG treatments

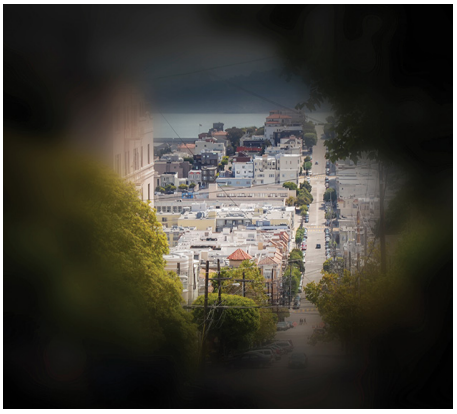
An optometrist referred a patient to me for YAG for PCO. The optometrist told the patient that this was a one-off procedure that never had to be repeated similar to phaco surgery. I advised the patient that the optometrist was correct for most patients most of the time but that there are the following exceptions when more than one YAG treatment is needed:

- If the anterior capsule starts to contract threatening phimosis requiring relaxing radial YAG cuts
- If the previous YAG capsulotomy was too small resulting in dysphotopsia and necessitating enlargement of the capsulotomy
- If another fibrinous membrane or more Elschnig pearls develop behind the IOL
- If pigment clumps deposit on the IOL.

I have a collection of cases of multiple YAG treatments that have been published on line if anyone needs this to show to a sceptical medical aid enquiry.

Phaco on retinitis pigmentosa patients

Just a reminder about this issue because it is quite rare that we do phaco on RP or Usher syndrome patients. The following precautions need to be taken:



Have a CTR (Morcher ring) ready because zonular laxity is common in these patients.

Consider placing a CTR even if the zonules hold up well during the surgery. This is because the risk of post-op capsular phimosis is greater in these patients.

Reduce the microscopes light intensity during phaco to avoid phototoxic retinopathy.

Consider making a slightly larger capsulorrhexis in anticipation of later anterior capsule contraction. But remember you can always enlarge a small rhexis, but you cannot reduce the size of a rhexis that is too large. I discovered this some time ago when the IOL kept popping up out of the bag as I was finishing a case. I had made the rhexis too large and now I had no way of reducing its size.

Add a NSAI drop to the post-op regimen because CME is more common in these patients. Consider starting the drops three days pre-op and continuing post-op for longer than usual.

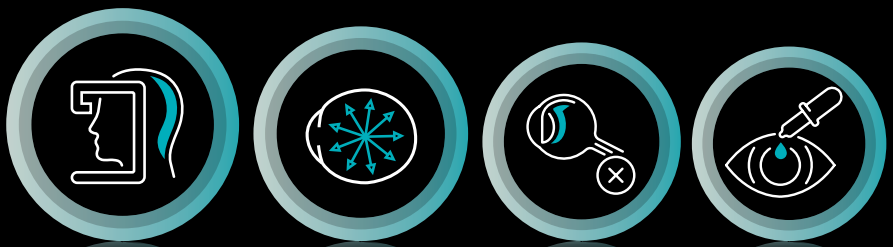
Post op PCO is very common in these patients. Consider early YAG and monitor carefully for anterior capsule contraction when radial YAG cuts should be done early to avoid phimosis.

If you have any other tips for these patients, please e-mail me at clivenovis@mweb.co.za.

Dr Clive Novis Dip Optom, MBBCh(Wits), MMed(Wits), FCS(Ophth)
clivenovis@mweb.co.za

When GLAUCOMA works on the nerves

causing tunnel vision, **it's time to reduce IOP** [1,2]



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IOP = Intraocular pressure

References: 1. Weinreb RN, Aung T, Medeiros FA. The pathophysiology and treatment of glaucoma: a review. *JAMA.* 2014; **311**(18):1901-1911. **2.** South African Glaucoma Society. Glaucoma algorithm and guidelines for glaucoma, 2016. Available at https://www.sags.co.za/doctors-information. Accessed 12 December 2019. S4 TRAVATAN® Eye Drops, solution (0,004 %), 40 µg of travoprost per ml in a sterile ophthalmic solution, 36/15.4/0333, Novartis SA (Pty) Ltd. S3 SIMBRINZA® 10 mg/ml + 2 mg/ml eye drops, suspension. Reg. No.: 50/15.4/0358. Each ml contains 10 mg of brinzolamide and 2 mg of brimonidine tartrate. S4 DUOTRAV® eye drops, 1 ml of solution contains 40 µg travoprost and 6,83 mg timolol maleate equivalent to 5 mg timolol, A40/15.4/0511, Novartis SA (Pty) Ltd. S3 AZOPTIC® Eye Drops (Suspension), 10 mg brinzolamide per ml, 34/15.4/0382, Novartis SA (Pty) Ltd. S4 AZARGA® eye drops, suspension, 1 ml of suspension contains 10 mg brinzolamide and 5 mg timolol (as timolol maleate), 44 /15.4/0046, Novartis SA (Pty) Ltd. **Note:** Before prescribing, consult full prescribing information.

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NOVARTIS

The 51st National Congress of the Ophthalmological Society of South Africa

8-11 March 2023

Durban Convention Centre, South Africa

Due to the Covid-19 Pandemic the presentation of the Humanitarian Award was delayed in 2021 and 2022. These were presented at the 51st National Congress of the Ophthalmological Society of South Africa.

Prof Nagib du Toit – 2021 Humanitarian Award (absent).

Dr Shabbir Hussain – 2022 Humanitarian Award.

Dr Agnus Risko – 2023 Humanitarian Award.

Dr Bayanda Mbambisa and Prof Ismail Mayet – recipient of the DJ Wood Award.

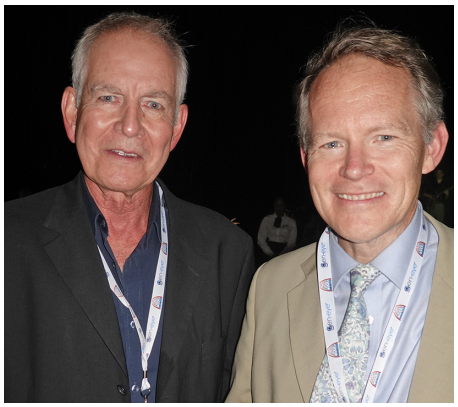
Sister Fundiswa Magaqa – this year's Citizen's Award.

Dr Leandi Linde – Registrar Presentation Award winner.

Dr Bayanda Mbambisa – president of OSSA and Dr Farah Moti – next president of OSSA.

Dr Pierre Wassermann (Congress chairman) and Prof Harminder Dua.

The organisers: Rhyno Kriek and Heyns du Preez.



Prof Trevor Carmichael and Mr David Verity.

Dr Agnes Risko, Dr Jan Talma, Prof Wayne Marais.

Dr Kerry Alberto and Dr Genevieve Ephraim.

Dr Karsten Visser and Prof Ferenc Kuhn.

Dr Frik Potgieter and Prof Harminder Dua.

Prof Wayne Marais and Prof Ferenc Kuhn.

Keeping rural communities safe from BLINDNESS

At the 2023 Ophthalmological Society of South Africa (OSSA) conference, which ran from 8 to 11 March, medical volunteering organisation Tshemba Foundation shared their plans to address prevent blindness in rural areas.

The Tshemba Foundation shared their plans to address this issue – by kickstarting a blindness prevention programme at Tintswalo Hospital in rural Acornhoek, Mpumalanga.

More than 80% of people in South Africa affected by vision impairment live in rural areas, where access to specialist diagnostic and treatment services is limited.

Until now, the non-profit's ophthalmic efforts have focused on cataract surgery, running 10 to 15 cataract camps per year. Since 2014, they have successfully facilitated over 700 cataract surgeries. However, screenings in the community and clinics have indicated an urgent need for non-surgical interventions in diagnostics and treatment.

"Diabetic retinopathy, glaucoma and allergic keratoconjunctivitis are some of the main contributors to vision impairment. These can all be treated with short-term or chronic medication, or laser eye treatment, provided they are detected early," says Professor John Gear, physician and medical director at Tshemba Foundation.

As part of the blindness prevention programme, Tshemba Foundation is calling on ophthalmologists to volunteer their time and expertise for specialist diagnostic and treatment services at Tintswalo Hospital's eye clinic. The non-profit is also recruiting volunteer optometrists to support their drive to prevent blindness in rural South Africa – with a particular focus on vision outreach in local schools.

As a Tshemba volunteer, ophthalmologists and optometrists will receive complimentary, self-catering accommodation at the organisation's unique Volunteer Centre in Moditlo Private Game Reserve. In addition to clinical interventions, volunteers can expect to manage acute referrals from Tintswalo Hospital and the surrounding clinics, as well as teaching and writing of foundational management protocols for common outpatient eye pathology.

Professor John Gear, physician and medical director at Tshemba Foundation.

"For volunteers, this presents an opportunity to gain invaluable eye pathology experience, and provide training to permanent medical staff at the hospital. In just two weeks, volunteers can positively impact an entire community of people for years to come," says Gear.

To date, Tshemba Foundation has hosted 260 volunteers from around the world, to the benefit of over 28 000 patients. There are both long-term, and short-term opportunities available that can accommodate busy schedules, while still maximising the impact of volunteering at Tintswalo and the local clinics in the area. To find out more, visit www.tshembafoundation.org/volunteer-programme.

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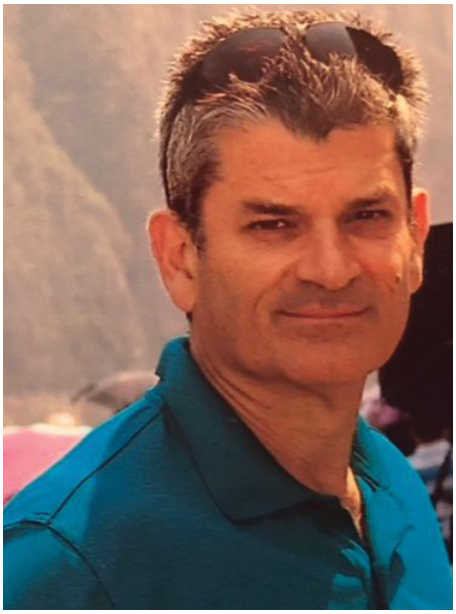
SAD LOSS for the ophthalmology community



Dr Savvas Christodoulou died unexpectedly on 18 March 2023 at the Unitas Hospital in Centurion. Savvas, aged 64, passed away surrounded by his loved ones after suffering a cerebral haemorrhage.

He was a keen golfer and went to gym regularly. Shortly after he qualified as an ophthalmologist, Savvas and other colleagues opened practices on the East Rand.

Around that time, the group started a Journal Club, a semi-formal semi-academic regular meeting of peers. Once a month, on a Monday, evening for two hours the ophthalmologists of the East Rand would get together to discuss journal articles, interesting cases, and other matters of interest to ophthalmologists.

Savvas was a regular attendee at these meetings. Over the last 27 years he seldom missed a meeting. Savvas did not only attend and sit quietly at the back. He almost always presented one or two difficult cases and was always humble enough to ask the opinion of his peers. He was wise enough to accept that he did not know everything.

"Our colleagues passing is a great loss to all of us but we can take comfort in knowing that he lived a full and meaningful life and that he touched the lives of so many people in profound ways. His legacy will live on in the countless patients whose lives he transformed and stories that we all share, said Dr Clive Novis," who delivered the eulogy.

Savvas is survived by his wife, three sons, and a step-daughter.

Savvas and his wife Linda and their four children. From left to right: Mark, Carmen-Daisy, Cameron, Daniel

2023 South African congresses and meetings

MAY

American Society of Cataract and Refractive Surgery Annual Meeting (ASCRS)

Date: 5-8 May 2023
Venue: San Diego Convention Center, San Diego, CA, USA
Website: https://annualmeeting.ascrs.org

JUNE

The 35th APACRS Annual Meeting

Date: 8-10 June 2023
Venue: Suntec Singapore Convention & Exhibition Centre, Singapore
Website: https://apacrs2023.org

The annual ESO Congress

Date:15-17 June 2023
Venue: Prague Congress Centre, Prague, Czech Republic
www.SOE2023.org

10th World Glaucoma Congress

Date: 28 June-01 July 2023
Venue: La Nuvola Convention Center, Rome, Italy
Website: www. worldglaucomacongress.org

JULY

4th World Congress on Ophthalmology and Optometry

Date: 17-18 July 2023
Venue: Best Western London Heathrow Ariel Hotel, Harlington, UK.
Website: www.ophthalmology.ophthalmologyconferences.com

37th European Ophthalmology Congress

Date: 31 July- 01 August 2023
Venue: Vancouver, Canada (Venue to be announced)
Website: www.clocate.com/european-ophthalmology-congress/67538/

SEPTEMBER

40th Congress of the European Society of Cataract and Refractive Surgeons (ESCRS)

Date: 16-20 September 2023
Venue: Milano Convention Centre Milan, Italy
Website: https://www.escrs.org

OCTOBER

The 23rd Euretina Congress

Date: 5-8 October 2023
Venue: RAI Amsterdam, The Netherlands
Website: www.euretina.org

The World Ophthalmology Congress (WOC)

Date: 09-11 October 2023
Venue: New York (Venue to be announced)
Website: www.suntextmeetings.com/ophthalmology

NOVEMBER

American Academy of Ophthalmology Congress (AAO)

Date: 3-6 November 2023
Venue: Moscone Convention Center, San Francisco, USA
Website: https://www.aao.org

7th World Congress on Eye and Vision

Date: 06-07 November 2023
Venue: Stockholm, Sweden (Venue to be announced.)
Website: www.vision.ophthalmologyconferences.com

2023 International congresses and meetings

June

SOE- European Society of Ophthalmology

Date: 15-17 June 2023
Place: Prague, Czech Republic
Website: https://soevision.org

10th World Glaucoma Congress- World Glaucoma Association

Date: 28 June-01 July 2023
Place: Rome, Italy
Website: https://worldglaucomacongress.org

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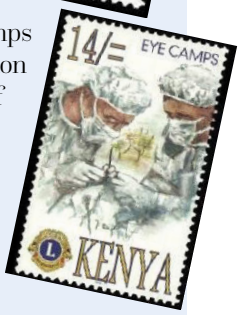
Forefathers of ophthalmology:
TRAILBLAZERS IN THE FIELD'S DEVELOPMENT



Juzer Surka is an ophthalmologist and enthusiastic philatelist, who first started collecting stamps and commemorative envelopes at a young age. In subsequent years he has focused his collection and research on medically themed stamps, and more specifically, on those relating to the field of Ophthalmology, where he has collected to date in the region of around 60 original stamps.

In 2005, as a member of the National Committee for the Prevention of Blindness, he was instrumental in getting the South African Postal Service to release a stamp to raise awareness around the prevention of blindness – an elegant, plain white stamp with the word 'Hello' written in braille. It was a first of its kind in the history of South African philately.

Philately is the study and collection of postage stamps. As stamp collecting gained in popularity over the years and around the world, collectors became more specialised in their areas of interests and collections; medical philately being no exception. Many countries have produced stamps on a wide range of medical topics, from health promotion and disease prevention to medical advances and notable clinicians. Ophthalmology has its fair share of stamps, and discovering them can be a fun and interesting way to learn about some of the history and heritage of the specialty.

In this series, we intend to look at various ophthalmology-related stamps, starting with famous clinicians and scientists. We hope the reader finds this to be informative and fun.

The eye of Horus

In ancient mythology, the god of darkness, Seth, who was also Horus' uncle, removed Horus' eye during their rivalry for the Egyptian throne. After killing and dismembering Osiris, Horus' father, Seth murdered and dismembered his own brother. To restore the eye, Thot, the Moon God and patron of sciences, intervened and reassembled it. Each component of the eye represents a prevalent symbol in Egyptian hieroglyphics.

1937 - Egypt. The Eye of Horus

Above: 1972 - Egypt, Social Work Day

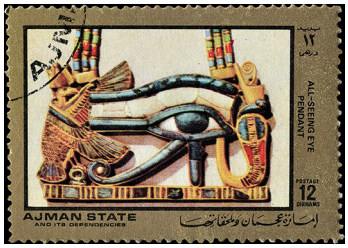
Above: 1986 - Honouring the Eye of Horus

Left: 1987 - Egypt

1981 - Egypt Air Post. Seeing Eye medallion

1975 - Egypt coact with WHO in producing this stamp

Together, the fractions of the eye should have equalled a whole, but they only amounted to 63/64. It was believed that Thot withheld the missing 1/64 by utilising magic. This emblem was highly esteemed by the Egyptians and is commonly found in stamp designs.



Ajman State - The All-Seeing Eye pendant

Editorial credit: ilapinto / Shutterstock.com

Hesy-Ra (2700 BC)

Hesy-Ra was an ancient Egyptian official who served during the Third Dynasty, around 2700 BCE. He was a physician and held the title of 'Chief of Dentists and Physicians'.

Hesy-Ra's papyrus, also known as the *Hesy-Ra's Medical Papyrus* is a collection of medical texts that describe various medical treatments, surgeries, and herbal remedies used by ancient Egyptians. The papyrus is believed to be the oldest known medical document, predating the more famous *Ebers Papyrus* by a few centuries.

1971 - UAR, (United Arab Republic)

1980 - Egypt

The papyrus is divided into two parts. The first part contains instructions for diagnosing and treating various ailments, including dental problems, stomach disorders, and parasitic infections. The second part of the papyrus provides instructions for preparing and using herbal remedies, including the use of plants such as castor oil and juniper berries.

One of the most interesting aspects of the papyrus is its emphasis on preventive medicine. Hesy-Ra recommends a healthy lifestyle, including a balanced diet, regular exercise, and proper hygiene, as a means of preventing disease.

Overall, the Hesy-Ra's papyrus provides valuable insights into the medical practices of ancient Egypt and demonstrates the sophistication of their medical knowledge and practices.

Iry- 2550 BC first eye doctor (Chief of Physicians)

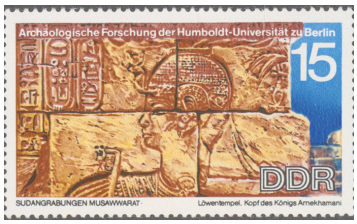
Iry was an ancient Egyptian physician who lived around 2500 BCE, during the 4th Dynasty of Egypt (2575 BC - 2465 BC). He is believed to be the first recorded ophthalmologist (eye doctor) in history. Iry's name found inscribed on the wall of his tomb in the necropolis of Saqqara, near Memphis.

Iry's title was Chief of the Physicians, indicating that he held a high position in the medical profession of ancient Egypt. His expertise in treating eye diseases was recognised and respected by the pharaoh and his court.

Iry's medical knowledge and skills were documented in the *Edwin Smith Papyrus*, (ca. 1650-1550BC) one of the oldest known medical texts in the world. This papyrus contains detailed descriptions of various medical cases, including eye diseases, and the treatments used by Iry and other physicians of his time.

Iry's treatments for eye diseases included the use of various herbal remedies, salves, and ointments. He also performed surgeries, such as the removal of cataracts, which involved using a sharp instrument to puncture the eye and then using a small hook to pull out the cloudy lens.

Iry's legacy as the first recorded eye doctor in history is a testament to the ancient Egyptians' advanced knowledge and understanding of medicine and the human body.



Printed in 1970 shows head of the King Arne-khamani

The Eber's Papyrus, (1500 BC)

One of the oldest medical texts in history, The *Eber's Papyrus* (1500 BC) contains a variety of medical remedies and treatments, including those related to ophthalmology or eye care.

The text describes a range of eye conditions and their treatments, including trachoma (a bacterial infection that causes inflammation of the eyelids), cataracts, and glaucoma. For example, for trachoma, the papyrus recommends a poultice made from figs, dates, and oil, which was applied to the eyes to reduce inflammation.

The *Ebers Papyrus* also contains remedies for other eye conditions, such as conjunctivitis, which was treated with a mixture of honey, milk, and animal fat applied to the affected eye. For cataracts, the papyrus suggests the use of a mixture of honey and sour milk, while for glaucoma, it recommends an ointment made from roasted pig eyes.

Although the remedies in the *Ebers Papyrus* were based on a combination of superstition, magic, and observation, some of them have been found to have some scientific merit. For example, honey has been shown to have antibacterial properties and may help in the treatment of certain eye infections.

Overall, the *Ebers Papyrus* provides a fascinating glimpse into the medical practices of ancient Egypt, including those related to ophthalmology. While some of its remedies may seem strange or even dangerous by modern standards, they demonstrate the ingenuity and resourcefulness of the ancient Egyptians in treating various medical conditions.

Editorial credit: rook76 / Shutterstock.com

1981 - Germany: shows Ebers Papyrus

Susutra (The birth of cataract surgery: Uncovering its origins)

Cataract surgery has come a long way since its inception. In the past 70 years, countless pioneers have contributed to the field, including Sir Harold Ridley, who paved the way for intraocular lenses. Yet, the true origin of cataract surgery remains a mystery.

While it's impossible to give credit to every individual who has helped shape cataract surgery, one name stands out in the history of medicine – Sushruta. Many ophthalmologists credit this ancient Indian physician as the first to perform any type of cataract operation.

Born in 600 BC in the city of Benares, Sushruta wrote the *Sushruta Samhita*, a treatise on surgery that contained details of cataract surgery. Unfortunately, only one copy of this text exists today, written in Sanskrit and largely unknown to medical historians of the 19th and early 20th centuries.

Despite this lack of knowledge, Sushruta's contributions to cataract surgery cannot be denied. His legacy lives on, and his innovative techniques have paved the way for modern-day surgeons to continue improving the field.

Below left: 1979: Sri Lanka, Susruta.

Below right: 1981: Germany

Editorial credit: Zoltan Katona / Shutterstock.com

1987: Hungary showing Hippocrates.

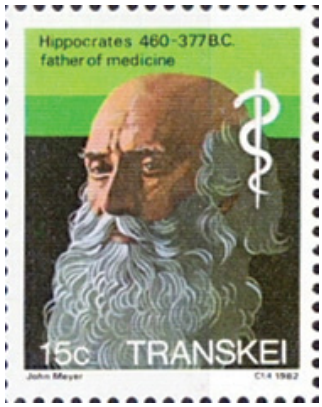
Editorial credit: rook76 / Shutterstock.com

1996 - Greece, honouring Hippocrates.

Hippocrates

Hippocrates, a Greek physician who lived in the 5th century BC, is often regarded as the father of modern medicine. While he is famous for his contributions to the fields of internal medicine and surgery, his involvement in eye surgery is not well known.

However, Hippocrates did write about eye surgery in his medical treatises, including one called *On the Eye*. In this text, he described procedures for removing cataracts using a needle, which was inserted through the side of the eye and used to push the cataract to the bottom of the eye where it would be absorbed.



1982 - Transkei (South Africa) Hippocrates.

It is also believed that Hippocrates performed surgeries to treat strabismus and even performed a procedure to repair a patient's ruptured cornea. While Hippocrates' techniques may seem crude by modern standards, they were ground-breaking at the time and helped to pave the way for modern-day eye surgery. His contributions to the field of medicine continue to be studied and celebrated today.

Prof J Surka MBBS, DOMS, MS(Ophth), FCS(Ophth)SA
Email: Jsurka@akrus.co.za

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References: **1.** Clareon® IOL Directions for Use. **2.** Lane S, Collins S, Das KK, Maass S, Thatthamla I, Schatz H, Van Noy S, Jain R. Evaluation of intraocular lens mechanical stability. J Cataract Refract Surg. 2019 Apr;45(4):501-506. **3.** Alcon Data on File, 2017. **4.** Lehmann R, Maxwell A, Lubeck DM, Fong R, Walters TR, Fakadej A. Effectiveness and Safety of the Clareon Monofocal Intraocular Lens: Outcomes from a 12-Month Single-Arm Clinical Study in a Large Sample. Clin Ophthalmol. 2021;15:1647-1657. **5.** Oshika T, Fujita Y, Inamura M, Miyata K. Mid-term and long-term clinical assessments of a new 1-piece hydrophobic acrylic IOL with hydroxyethyl methacrylate. J Cataract Refract Surg. 2020 May;46(5):682-687. **6.** AcrySof® IQ Vivity® Extended Vision IOL Directions for Use. **7.** Bala, Chandra, et al. Multi-country clinical outcomes of a new nondiffractive presbyopia-correcting intraocular lens. Journal of Cataract and Refractive Surgery Publish Ahead of Print DOI:10.1097/j.jcrs.0000000000000712. **8.** Varma, Devesh, et al. Clinical Outcomes of a New Non-Diffractive Presbyopia-Correcting Intraocular Lens From Two Large Confirmatory Studies. American Academy of Ophthalmology. Abstract: PA005. **9.** Alcon Data on File, 2022. [A01970-REP-211731] - Optical Equivalency of Clareon Monofocal and AcrySof IQ IOL Version 1.2 REF-15172. Based on Studies with AcrySof®; Clareon® and AcrySof® are optically equivalent with the same -0.2µm aspheric design resulting in improved depth of focus.
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References: 1. ARGOS User Manual

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Alcon

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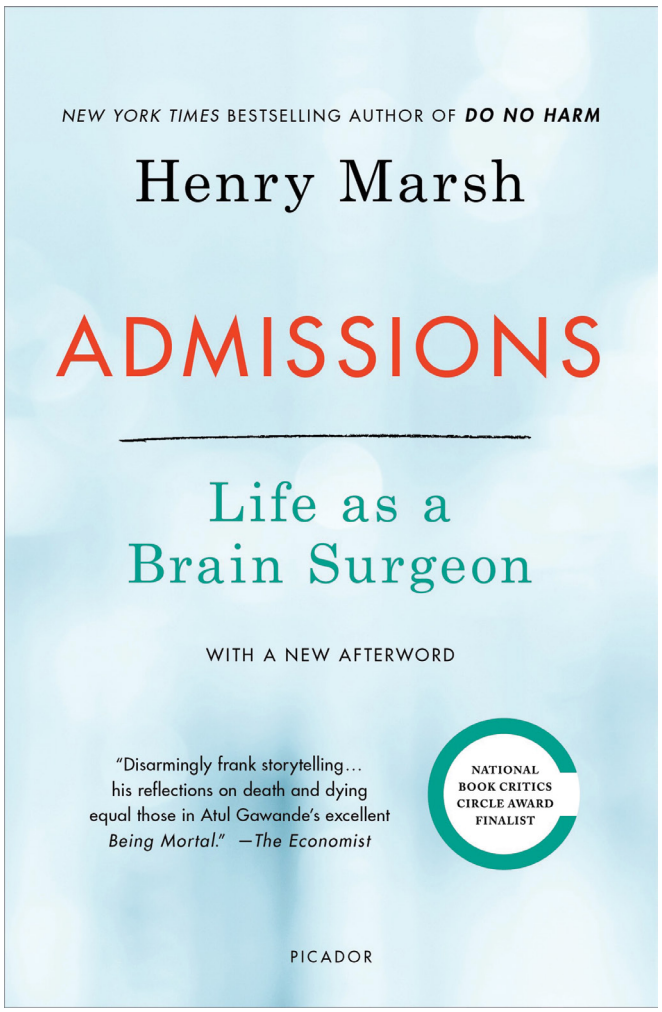
ADMISSIONS
Life as a Brain Surgeon



Author: Henry Marsh

Year of Publication: 2017

Number of pages: 271

Reviewer: Clive Novis

About a year ago I reviewed Mr Marsh's first book called *Do No Harm: Stories of Life, Death, and Brain Surgery* (published in this journal Autumn 2022). I so enjoyed that book that I immediately bought his second book: *Admissions*.

Admissions did not disappoint. Often, an author's second book does not live up to the expectations created by the first book. But this book is just as good.

Henry Marsh is a prominent neurosurgeon, now retired and living in the UK.

One of the things I liked about his first book was his openness. In this book he dives straight in on the very first page by admitting that he has a 'suicide kit'. He has collected several drugs, vials, poisons, drip sets, needles, tourniquets that he intends to use on himself when he feels that his time has come to die. He watched his father succumb slowly to Alzheimers disease and does not want to suffer the same fate. But he is not altogether sure that he will use these lethal drugs on himself when faced with the actual decision.

He talks of his cognitive dissonance: knowing and accepting that we are going to die but still clinging on to hope. He compares this to the cognitive dissonance of believing in a soul and afterlife as the hope that if the dying have that they will yet live. Another example that he discusses is his own yearning to retire and be free of all the responsibility and human misery that he must deal with daily versus his fears about what he will do and become after retirement.

Henry Marsh was born in 1950, so he was 67 years old at the time of writing this book. Interestingly he says that he seems to have more anxiety in theatre now than in his younger days. He says that he feels that the defensive psychological armour that he had worn for so many years is starting to fall away, leaving him 'as naked as his patients'. Earlier in his career he found that the difficult and dangerous operations were the most attractive and exciting ones, but as he grew older his enthusiasm for these cases was diminishing.

Working in a large NHS hospital was frustrating most of the time for him with the usual bureaucratic inefficiencies, paper work, etc. He tells several stories of meetings with administrators and other public officials that were not always cordial. But he also enjoyed teaching and mentoring younger surgeons even though he says that watching and guiding a trainee surgeon do an operation is much harder than doing it yourself. And he loved the close relationship that he usually built up with his trainees saying that this is one of the great pleasures of a surgeon's life.

In his first book, he admits to many of his weaknesses and confesses many of his mistakes. "We all have guilty secrets, and silence them with self-deception and exaggerated self-belief." Some of the cases that went wrong are truly tragic, but he describes them in detail together with insights into his own feelings of failure. He tells how at one stage he was admitted into a psychiatric institution. He talks of other failures in his life including his marriage and of his recurring nightmares (usually about surgical complications). To readers, he exposes his heart and soul.

As I write this review, a terrible war is still raging in Europe. Chapter 12 of this book is titled 'Ukraine'. He visited Ukraine frequently throughout his neurosurgical career to operate and to teach Ukranian doctors. He loved Ukraine and described not only the geography but the wonderful people (patients, colleagues, etc) that he met and worked with. He explains that Ukraine was one of the "great historical watersheds, where Europe met Asia, where democracy met despotism". I can only imagine his anguish now as the Russian invasion enters its second year.

All doctors will enjoy reading this book and will learn a lot about neurosurgery. There are also several YouTube videos featuring Henry Marsh which I also enjoyed.

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