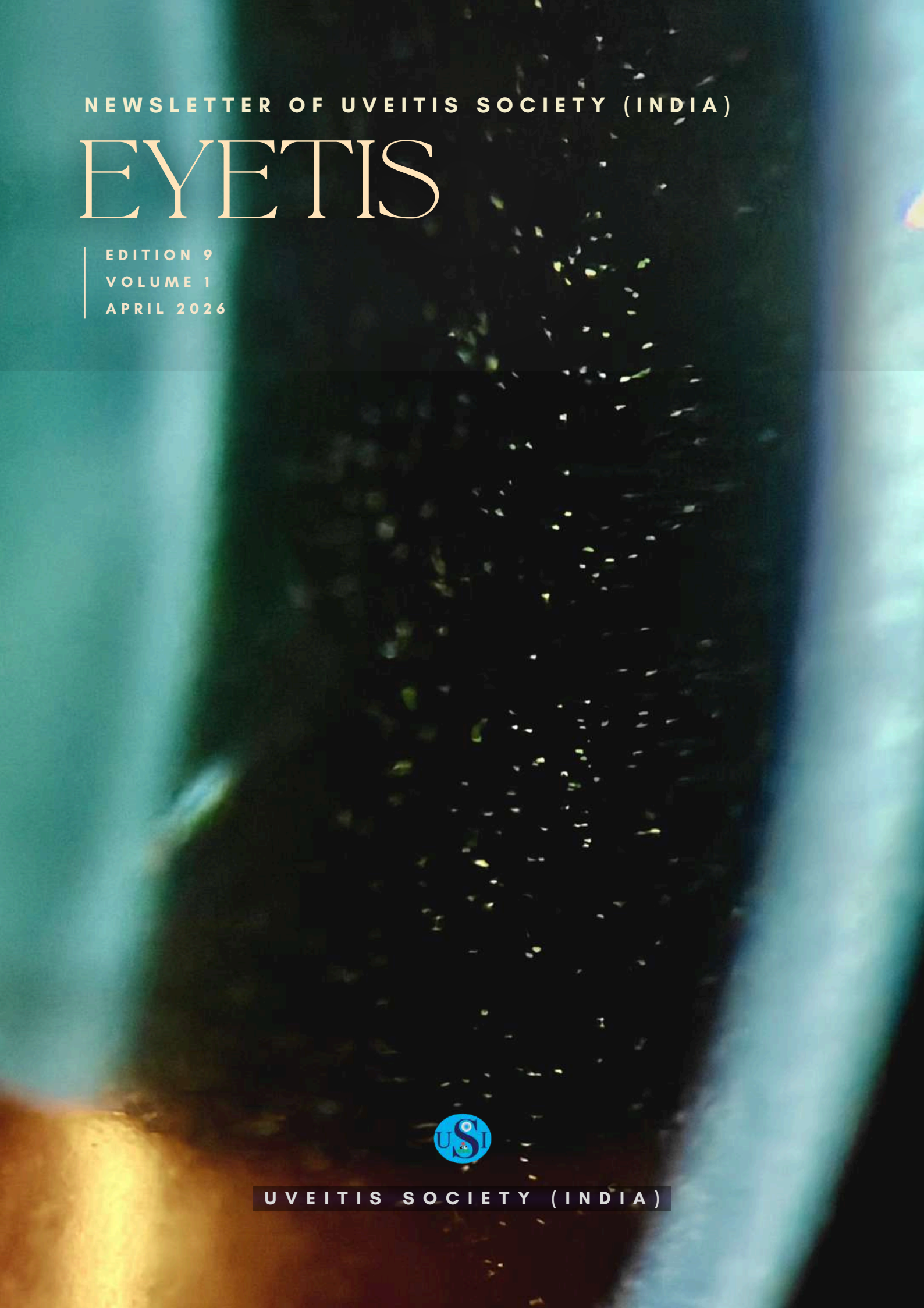
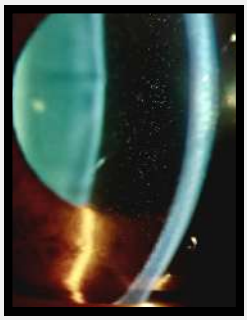


NEWSLETTER OF UVEITIS SOCIETY (INDIA)

EYETIS

EDITION 9
VOLUME 1
APRIL 2026

UVEITIS SOCIETY (INDIA)



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Eyetis, Official Newsletter of Uveitis Society (India)
Edition 9 | Volume 1 | April 2026
Official Publication of Uveitis Society (India)

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FROM THE EDITOR'S DESK

EDITOR-IN-CHIEF

Dr. Vandana Pradeep
Consultant Uvea and
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Kerala

Every publication carries a voice. For this issue, we wanted it to reflect the discipline we practice: thoughtful, curious, collaborative, and constantly evolving!

Uveitis challenges us to look deeper, to connect patterns, to balance science with judgement. This newsletter aspires to do the same.

You will find here foundational learning, expert insights, emerging evidence, and conversations that span generations. Each section has been curated with care — not only to inform, but to engage, provoke thought, and strengthen our collective understanding.

This edition is the result of many quiet hours of reflection and planning, along with unstinting and generous contributions with a shared belief that our community deserves a platform that grows with it.

> "We wanted this newsletter to feel as thoughtful as the way we practice."

If these pages spark reflection or curiosity, we have achieved what we set out to do.

FROM THE EDITOR'S DESK

ASSOCIATE EDITOR

Dr. Richa Pyare
Consultant Medical
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Uveitis specialists are time travellers. Each day, we peer through a device invented more than a century ago by the Nobel laureate Allvar Gullstrand, wielding Victorian optics along with cutting edge immunology and pharmacotherapy to defeat an ancient foe. Each patient encounter becomes an act of temporal synthesis: one eye on the beam illuminating the anterior chamber, the other on the immune pathways we are learning to redirect. This newsletter travels the same temporal terrain.

While Professor Andrew Dick, Duke Elder Chair of Ophthalmology, anchors us in how immunological research forms the core of clinical practice, the foundational past that orients our present;

Dr. Panayiotis Maghsoudlou's article on Iluvien points us toward tomorrow's pharmacotherapy. Between these temporal poles, we feature an interview with Professor Amod Gupta, the doyen of Indian uveitis, exciting clinical cases from members nationwide, and prize-winning articles from NextGen USI 2025 winners Dr. Tanya Jain and Dr. Anup Kelgaonkar.

I hope you enjoy traveling the time-verse with this issue as much as we found assembling it!

THE PRESIDENT'S MESSAGE

Dr. Padmamalini Mahendradas
Head of Uveitis and Ocular Immunology
Narayana Nethralaya
Bengaluru

It gives me immense pleasure, on behalf of Uveitis Society (India), to present the first issue of the USI Newsletter for the year 2026. This publication is the result of the collective efforts of our members and the dedicated work of our Editor-in-Chief, *Dr. Vandana Pradeep*, and Associate Editor, *Dr. Richa Pyare*, whose commitment has been instrumental in bringing this issue to fruition.

For the first time, USI has introduced a young editorial team, and their enthusiasm is clearly reflected in the quality and breadth of the content presented. I sincerely acknowledge and thank the national and international faculty who have generously contributed their clinical expertise, significantly enriching this edition.

The theme of this issue focuses on topics commonly encountered in our clinical practice, with the aim of enhancing knowledge and supporting improved patient care. This edition includes the life history of Padma Shri Dr. B K Jain, reflections by Prof. Amod Gupta, a revisit of the fundamentals of ocular syphilis, in-depth discussions on VKH, genetics and uveitis, the MUV project, immunology, recent updates, clinical pearls, quizzes, case reports, snapshots, and sections on arts and culture.

I extend my sincere gratitude to all contributors, office bearers, and the editorial team for their invaluable contributions. As we move forward, we hope the USI Newsletter continues to inform, inspire, and connect our fraternity. We look forward to continued contributions from all our members.

With best wishes for a successful and fulfilling 2026.

Dr. Padmamalini

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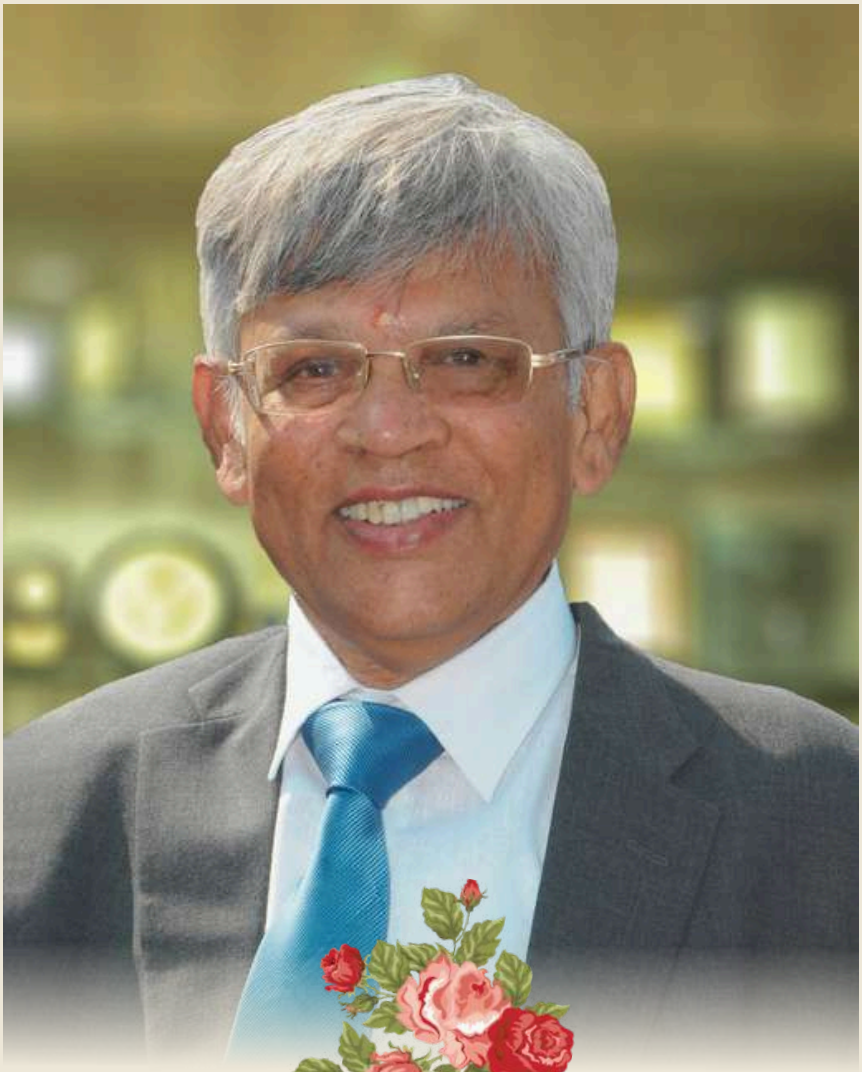
Celebrating

THE LIFE OF



PADMA SHRI
DR. BUDHENDRA KUMAR JAIN
(5 December 1948 - 27 February 2026)

The Bhagavad Gita outlines three primary paths toward spiritual realization: the path of devotion (bhakti yoga), the path of knowledge (jnanayoga), and the path of action (karma yoga). Dr. Jain was a living embodiment of the Karma Yogi, consistently practicing Nishkama karma—the performance of duty without attachment to personal gain. His life reflected the essence of Bhagavad Gita verse 2.47:

कर्मण्येवाधिकारस्ते
मा फलेषु कदाचन

"Karmanye vadhikaraste ma phaleshu kadachana"
You have a right to perform your prescribed duties, but you are not entitled to the fruits of your actions.

Dr. Jain's journey began when he left behind the comforts of a wealthy family in Satna to settle in Chitrakoot in 1974. At that time, Chitrakoot was a remote destination with minimal facilities, moreover an abode to some of the region's most dreaded dacoits. Choosing humility over a life of luxury, he overcame immense challenges to build one of the premier eye care institutions in the world.

Guided by the principle of *Svadharma*, he dedicated himself to his inherent duties with unwavering honesty and precision. For more than half a century, he served the Sadguru Seva Sangh Trust tirelessly, working around the clock every single day. His philosophy for success was anchored in three core pillars: *Atma Anushasan* (self-discipline), *Atma Tyag* (self-sacrifice), and *Atma Vishwas* (self-belief). He led by example, consistently being the first to arrive at the hospital and the last to leave. His legendary accessibility meant that anyone, regardless of their rank, could meet him without a formal appointment. By nurturing a genuine family environment, he established *Sadguru Netra Chikitsalaya (SNC)* as a truly unique organization. Such was his devotion that he often placed the interests of SNC above his own personal life, earning him the reverence of the entire trust as a

fatherly figure.

Deeply committed to the welfare of all beings *(Sarva-bhuta-hite-ratah)*, he maintained an unwavering focus on *Seva*. He believed that treating patients was a form of divine service, and through his tireless work, he brought hope and sight to millions by making high-quality eye care accessible to every social class. As a genuine *Karma Yogi*, he viewed his achievements as a sacred offering *(Yajna)* to God and his Gurudev. This spiritual foundation allowed him to maintain perfect equanimity *(Samatvam)*, remaining humble in times of great success and resilient in the face of challenges.

Guided by the ideals of Mahatma Gandhi, he dedicated himself to the development of rural India. He believed that the migration of rural youth to cities could be reversed if they were given the same opportunities within their own communities. He not only focused on developing the eye hospital and gave employment to the rural youth but also opened schools so that their children could receive high-quality education. He invested substantially in renewable energy and water harvesting long before these practices had gained widespread importance. His visionary approach established a global model of rural development through eye care, demonstrating how eye care can function effectively as a social industry.

Beyond founding a major institution, Dr. Jain acted as a source of motivation and inspiration that reshaped countless lives. His work represents a paradigm change in how eye care is delivered throughout India. His legacy will endure forever, sustained by his profound impact on humanity and his dedicated service to society.

Dr. Alok Sen
Sadguru Netra Chikitsalaya
Chitrakoot

Beyond the Page...

The

Newsletter of Uveitis Society (India)

goes

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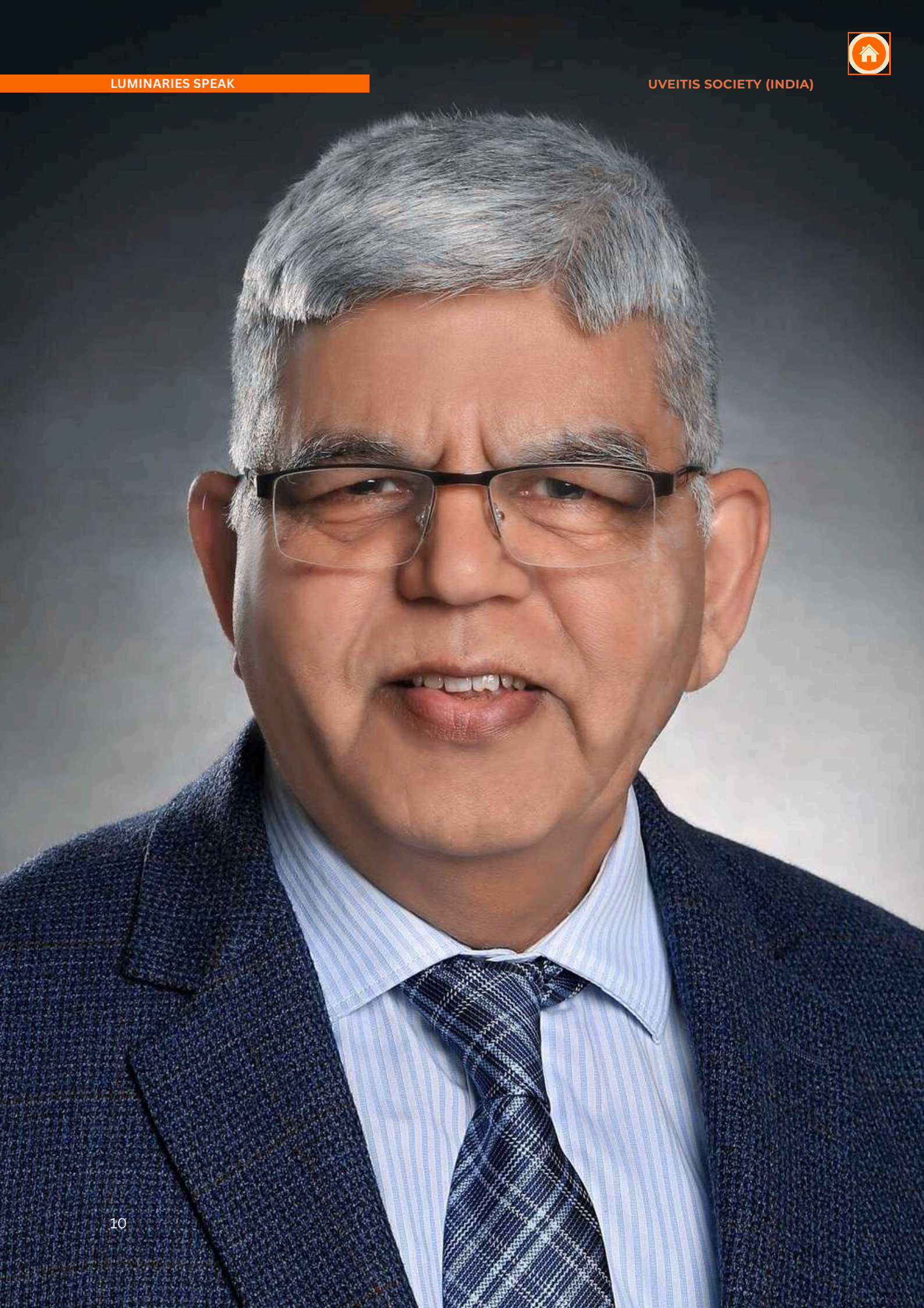


TAP HOME TO
RETURN TO CONTENTS

PROF. AMOD GUPTA

Few names in Indian ophthalmology are as deeply intertwined with the evolution of uveitis as Prof. Dr. Amod Gupta. A clinician, teacher, and visionary, his approach to uveitis has always been rooted in careful observation, intellectual honesty, and patient-centred care. In this conversation, Prof. Gupta reflects on his journey, the changing landscape of uveitis, and the principles that continue to guide his clinical practice and mentorship. His words offer not only clinical insight, but also a philosophy that has shaped generations of ophthalmologists.

Sir, you have shaped the field of uveitis in India in profound ways. What first drew you toward uveitis as a subspecialty?

Uveitis in India was, at that time, a largely virgin field—among the least understood and least discussed areas of ophthalmology. Of all subspecialties, it posed perhaps the greatest intellectual challenge. When I first began seeing uveitis patients in 1977, every case fascinated me and compelled me to think like a physician first and an ophthalmologist second. The field offered immense opportunities for documentation, interdisciplinary collaboration, and original research, all of which deeply appealed to me.

You are credited with establishing one of the strongest uveitis training ecosystems in the country. What were the guiding principles behind building this legacy?

A PRINCIPLE YOU NEVER COMPROMISE ON

Integrity

I have always believed that our patients are our best teachers, and this philosophy has been consciously passed on to the next generation. I abandoned the dogmatic approach early in my career—the written word was never the gospel truth. Careful observation, meticulous documentation, and long-term follow-up of patients formed the core of our practice, a legacy I inherited from my teacher, Prof. I. S. Jain.

Apart from him, I did not receive training from any grandmaster or institution abroad. We learned by documenting and following every patient who intrigued us. We were never afraid to share our observations and interpretations, placing them on the touchstone of peer review. Teachers and students learned together—asking questions, exploring uncertainties, and in the process developing close collaborations with allied disciplines such as rheumatology, immunopathology, and microbiology.

We followed the principles of genuine experiential learning. Unsolved clinical puzzles were never swept under the carpet; instead, they gave us sleepless nights until we found answers. Unless you see your patient again and continue to do so for years, you cannot master the art and science of medicine. This is a slow journey, but the wisdom gained has allowed us to confidently participate on platforms worldwide.

What are the "first principles" you rely on when approaching a complex or atypical case of uveitis?

My approach to every patient has been to break the case down into descriptors or keywords that best define the signs and symptoms—what we now call semantic qualifiers. Rather than performing a clinical examination as a ritual, every step is preceded by a question: Do I see keratic precipitates? If yes, what type? What is their distribution? Are there cells, flare, or synechiae?

The well-known aphorism, "The eyes don't see what the mind does not know," is profoundly true. When we approach a patient with a vacant or distracted mind, we miss what is unusual or important. One must train the mind to seek answers through a continuous series of questions, or else the most critical signs may be overlooked.

At the end of this exercise—supported by documentation using all relevant ancillary tools—you should be able to paraphrase your patient. Only then is it time to consider differentials and order appropriate laboratory investigations to rule conditions in or out. I do not believe that one is often the first person in the world to encounter a new disease. Before arriving at such a conclusion, it is essential to visit your e-library, search

the literature using carefully chosen keywords, and learn from what others have described. If gaps still remain, then it is time to build your own case series or collaborate with others to expand it.

What changes in the understanding or management of uveitis have you found most satisfying over the arc of your career?

Several developments have been particularly gratifying:

1. The categorisation of uveitis into infectious, autoimmune, and masquerade syndromes—moving away from an era when almost all uveitis was treated indiscriminately with corticosteroids, often with disastrous consequences.
2. The ability to document inflammatory pathologies using advanced imaging modalities, along with the development of diagnostic tools such as PCR and mNGS.
3. The determination to leave no stone unturned in identifying the aetiology, even when it required invasive techniques such as pars plana vitrectomy or retinal biopsy.
4. The international recognition of tuberculosis as the most common cause of uveitis in TB-endemic regions—an achievement that has been personally very fulfilling.

From clinical diagnosis to imaging-driven and immunology-based management, uveitis has transformed significantly. Which developments have had the greatest impact?

Among the most critical developments over the past five decades has been the widespread use of slit-lamp biomicroscopy, which was uncommon even in medical schools earlier. The universal adoption of the +90D lens for retinal examination, along with binocular indirect ophthalmoscopy, significantly enhanced posterior segment evaluation. The widespread availability of fundus fluorescein angiography, OCT, and multimodal imaging has now become standard practice, fundamentally transforming diagnosis and

A CLINICAL TOOL YOU RELY ON MOST

> **My slit-lamp biomicroscope with a +90D lens**

management. Equally important has been the ability to extensively document cases for discussion with experts and trainees, both offline and online—an evolution made possible largely through platforms such as Microsoft PowerPoint. Learning opportunities through local, regional, national, and international meetings have expanded enormously in India. One particularly notable initiative, owing to the foresight of Prof. N. A. Rao, was the early exposure of Indian ophthalmologists to international experts in uveitis.

"Equally important is educating physicians not to view uveitis as merely an ocular disease"

You have mentored generations of uveitis specialists. What qualities are essential in a young clinician entering this field?

First, a naturally meticulous, curious, and analytical mind that can connect the dots. Second, strong communication and teaching skills to explain the complexities of uveitis—its investigations, management strategies, cost implications, and long-term course—to patients and trainees alike. Third, the ability to collaborate effectively with colleagues from other disciplines. Fourth, patience in managing complex and often prolonged cases. And finally, resilience—the capacity to cope with failures and poor outcomes.

In your view, what are the most important challenges in uveitis care in India today?

India may now have the largest number of ophthalmologists in the world, yet most are understandably focused on cataract surgery, a largely skill-based subspecialty. Very few develop a deep interest or true expertise in uveitis. For a nation of nearly 1.4 billion people, we need many more dedicated uveitis specialists across the country. Uveitis demands an exceptionally curious, problem-solving individual, often willing to work with limited financial rewards.

Given India's vast geography and diversity of disease patterns, there is an urgent need for a nationwide uveitis database, with participation from major centres across all states. India has successfully led several international consensus initiatives, and it is now time for Indian ophthalmologists to collectively define and address issues such as the most common causes of uveitis in India—particularly TB uveitis and TB retinal vasculitis.

Advanced diagnostic tests like PCR and mNGS are currently available only in select centres, are expensive, and require standardisation. Similarly, newer pharmaceuticals, biologics, and biosimilars are limited to a handful of institutions. Strengthening communication and mutual education between ophthalmologists, rheumatologists, and physicians is essential to improve access and appropriate use of these therapies.

Equally important is educating physicians not to view uveitis as merely an ocular disease, and training comprehensive ophthalmologists in early referral practices. They must also be educated on the judicious use and tapering of

corticosteroids, the timely introduction of steroid-sparing immunomodulatory therapy, and strategies to improve long-term compliance.

If there were one meaningful shift you could influence—clinical, educational, or systemic—what would it be?

We must move from a fragmented approach to a truly systemic one. This includes establishing de-identified, open-access uveitis registries supported by AI-driven analytics for real-time surveillance, standardised diagnostics, and equitable access to therapies. A powerful example is the IRIS (Intelligent Research in Sight) registry of the American Academy of Ophthalmology, which collects large-scale data across ocular diseases.

What continues to motivate and inspire you in your clinical and academic journey?

After more than 50 years in academia, the desire to learn becomes a way of life —a lifelong pursuit. Having satisfied my own intellectual curiosity and clinical challenges many years ago, my greatest motivation today lies in seeing the next generation realise its full potential. My only wish is for them to excel and further enhance India's reputation on the global stage.

Is there a guiding principle every clinician should carry throughout their career?

Patients are our sacred texts. We exist because of our patients, not the other way around. They are the source of your name, your recognition, and any acclaim you may receive.

What final advice would you like to share with young uveitis specialists who look up to you as a role model?

There are no shortcuts in a complex specialty like uveitis. No two patients are alike. Each patient offers an opportunity to learn and to become a better version of yourself. Never compromise on your values.

Prof. Amod Gupta's reflections remind us that uveitis is not merely a subspecialty—it is a discipline that demands humility, patience, and lifelong learning. His words continue to guide not only how we treat disease, but also how we think, teach, and mentor.

OCULAR SYPHILIS

WHEN TO SUSPECT & HOW TO MANAGE

Dr. Arshee Ahmed
Senior Consultant
Uveitis & Intraocular Inflammation
Sankara Nethralaya
Chennai

Christopher Columbus is well-known for his voyages across the Atlantic leading to the Columbian-exchange; the beginning of sustained contact and trade. But he is also considered responsible for the appearance and the wide spread of a disease in sailors who accompanied him back from the first transatlantic expedition. This is one of the popular theories which abound regarding the discovery of the disease known as Syphilis which was considered to be so disfiguring and resistant to the medical arts of its time that Joseph Grunpeck, a German physician who contracted syphilis in the early 1500s wrote-

"So cruel, so distressing, so appalling that until now nothing more terrible or disgusting has ever been known on this earth."

47 yr old male with HIV since 11 yrs - non-compliant to ART

- BCVA OD 2/60,N24 , OS 6/24,N10.
- OU ground glass retinitis involving the superior,temporal quadrants
- Syphilitic lesions on his hands and scrotum +
- Recent h/o contact with a FSW (female sex worker) +
- Both RPR and TPHA +

In 1905, syphilis was identified as an infection caused by a spirochete bacterium, Treponema pallidum. Although it has been around for centuries, this disease has seen a resurgence in recent times. One reason why it continues to pose challenges is its wide and unpredictable clinical spectrum- *there are hardly any "diagnostic" clinical features* to pinpoint it. It is widely referred to as the **"Great Imitator/Masquerader"** because it can mimic a wide range of inflammatory and infectious eye conditions making diagnosis difficult even for experienced clinicians. It can present in any stage of the disease, affect any part of the eye and thus, presents with myriad diagnostic challenges to clinicians.

STAGE	PRIMARY	SECONDARY	LATENT
TIME FROM INFECTION	3-6 weeks	6 weeks - 6 months	>1 year
SYSTEMIC MANIFESTATIONS	Painless chancre at site of inoculation, regional lymphadenopathy	Fever, malaise, maculopapular rash, generalized lymphadenopathy	Asymptomatic
OCULAR MANIFESTATIONS	Rare; conjunctivitis chancres	Anterior/ intermediate/ posterior uveitis, optic neuritis, neuroretinitis	
INFECTIVITY	Highly infective	Highly infective	Early (high infectivity) Late (low infectivity)

Traditionally, untreated syphilis is known to follow through four clinical stages - primary, secondary, latent and tertiary. Ocular involvement is more commonly seen in the secondary, latent and tertiary stages of the disease. It can be unilateral or bilateral although bilateral involvement is more common. Another important variant is congenital syphilis which happens when the infection is transmitted from an infected mother to her child during pregnancy leading to significant ocular morbidity in children.

Once we have established that there is no clear diagnostic clinical marker for the disease, we can safely say that diagnostic approach to ocular syphilis should be based on a thorough clinical examination, a clear anatomical diagnosis and then appropriate laboratory testing. It is prudent to understand that testing for syphilis should be a part of the laboratory work-up for *all* patients with uveitis. The commonly used diagnostic tests are summarised here for clarity.

STAGE	TERTIARY	NEUROSYPHILIS	CONGENITAL
TIME FROM INFECTION	Years - decades	Any stage	In utero/ childhood
SYSTEMIC MANIFESTATIONS	Gummatous disease	Meningitis, Tabes dorsalis, general paresis	Hutchinson's triad, deafness, skeletal abnormalities
OCULAR MANIFESTATIONS	Chronic uveitis, optic atrophy, retinal vasculitis, interstitial keratitis	Optic neuritis, neuroretinitis cranial nerve palsies, Argyll-Robertson pupil	Interstitial keratitis chorioetinitis
INFECTIVITY	Not infectious	Variable	N/A

Two main testing algorithms are employed in practice for diagnosis - **the traditional algorithm,** where first a non-treponemal test is done for screening and only positive samples are further subjected to treponemal tests or - **the reverse algorithm**, wherein an initial screening is done with treponemal immunoassays. Both algorithms are widely accepted and choice can be based on practical considerations like laboratory resources, including staff, space, cost, test volume, and patient population being served.

NON-TREPONEMAL TESTS

VDRL (VENEREAL DISEASE RESEARCH LABORATORY)	Detects anti- cardiolipin antibodies Visualised by microscopic flocculation	Reported as titers (eg 1:32) Higher titer = active disease	False positives - (autoimmune diseases, pregnancy, TB) False negatives - late syphilis (prozone phenomenon)
RPR (RAPID PLASMA REAGIN)	Similar to VDRL; Visualised by macroscopic flocculation	Reported as titers	Less specific than treponemal tests

TREPONEMAL TESTS

TPHA / TPPA TREPONEMA PALLIDUM HAEMAGGLUTINATION / TREPONEMA PALLIDUM PARTICLE AGGLUTINATION ASSAY	Indirect agglutination assay to detect antibodies against Treponema pallidum subspecies pallidum	Reported as reactive/non-reactive	Confirmatory test Positive for life Cannot differentiate between active and treated infection
FTA-ABS FLUORESCENT TREPONEMAL ANTIBODY-ABSORPTION	Detects antibodies specific to Treponema pallidum	Reported as reactive/non-reactive	Confirmatory test Not useful for monitoring
ENZYME IMMUNOASSAY	Enzyme or chemiluminescent immunoassay for treponemal antibodies	Reported as positive/negative	Screening in reverse algorithm Positive tests require confirmation

In 1910, Paul Ehrlich introduced Salvarsan – an arsenic-based compound that became the first effective chemical treatment for syphilis. Although toxic, it remained the standard of care for several decades.

A major turning point came in 1943 when John Mahoney, a physician in the United States Public Health Service along with his colleagues reported the successful use of penicillin in treating early syphilis in humans. This was a landmark moment, as penicillin proved more effective than previous therapies and lacked the severe toxic side effects of heavy metals like arsenic or mercury which were commonly used at the time.

CSF ANALYSIS

CSF VDRL	Detects reagin antibodies in CSF	Highly specific if positive	Detects neurosyphilis Low sensitivity
CELLS COUNT & PROTEIN	Inflammatory markers	Pleocytosis Elevated protein	Supportive evidence Non-specific

DIRECT DETECTION

DARK FIELD MICROSCOPY	Direct visualization of bacteria	Reported as presence of motile spirochetes	Primary syphilis lesions Requires expertise Not for ocular disease
PCR FOR TREPONEMA PALLIDUM	Detection of treponemal DNA	Reported as positive/negative	Research/selected cases Limited availability Not standardised
IMMUNOHISTO CHEMISTRY (IHC)	Allows direct visualization of the organism within histopathological specimens by using antibodies directed against Treponema pallidum	Reported as positive/negative	Congenital syphilis High specificity Limited availability

Since that path-breaking discovery, the antibiotic of choice for syphilis remains penicillin; till date, no resistance of T.pallidum to penicillin has been documented.

The same regimen followed for the treatment of neurosyphilis is recommended for ocular syphilis. Intravenous aqueous crystalline penicillin G at a dosage of 18-24 million units/ day for 10-14 days is the treatment of choice.

However, in case of an allergy to penicillin, either desensitization or i.v ceftriaxone can be chosen at a dosage of 1-2 gm/day for 10-14 days.

Salvarsan– an arsenic based compound remained the standard of care for several decades.

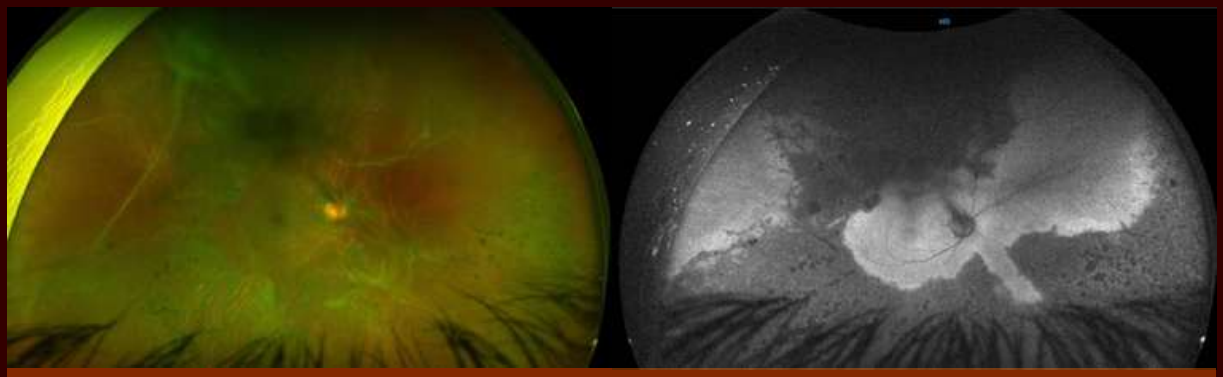
As with other forms of infectious posterior uveitis, corticosteroids should be used with extreme caution, and only under the cover of appropriate antibiotic therapy. Steroids help in controlling the ocular inflammation and are also often indicated to reduce the severity of the Jarisch-Herxheimer reaction which can occur following the initiation of treatment.

Any discussion on syphilis would be incomplete without addressing HIV –

as both are infectious diseases with a significant and well-recognized inter-dependence. All patients with ocular syphilis should be tested for HIV co-infection as the routes of transmission and at-risk patient population is identical for both. While syphilis doesn't require an immune-compromised state to progress, HIV-related impairment of cell-mediated immunity leads to a more aggressive course of the disease. Co-infected patients are more likely to have bilateral involvement, severe posterior segment disease and higher chances of cerebrospinal fluid abnormalities, regardless of CD4 counts.

The same antibiotic regimen as HIV negative patients is followed for co–infected patients but close follow–ups are required due to higher relapse rates.

Management of these patients is best done in collaboration with infectious disease specialist to ensure comprehensive care.



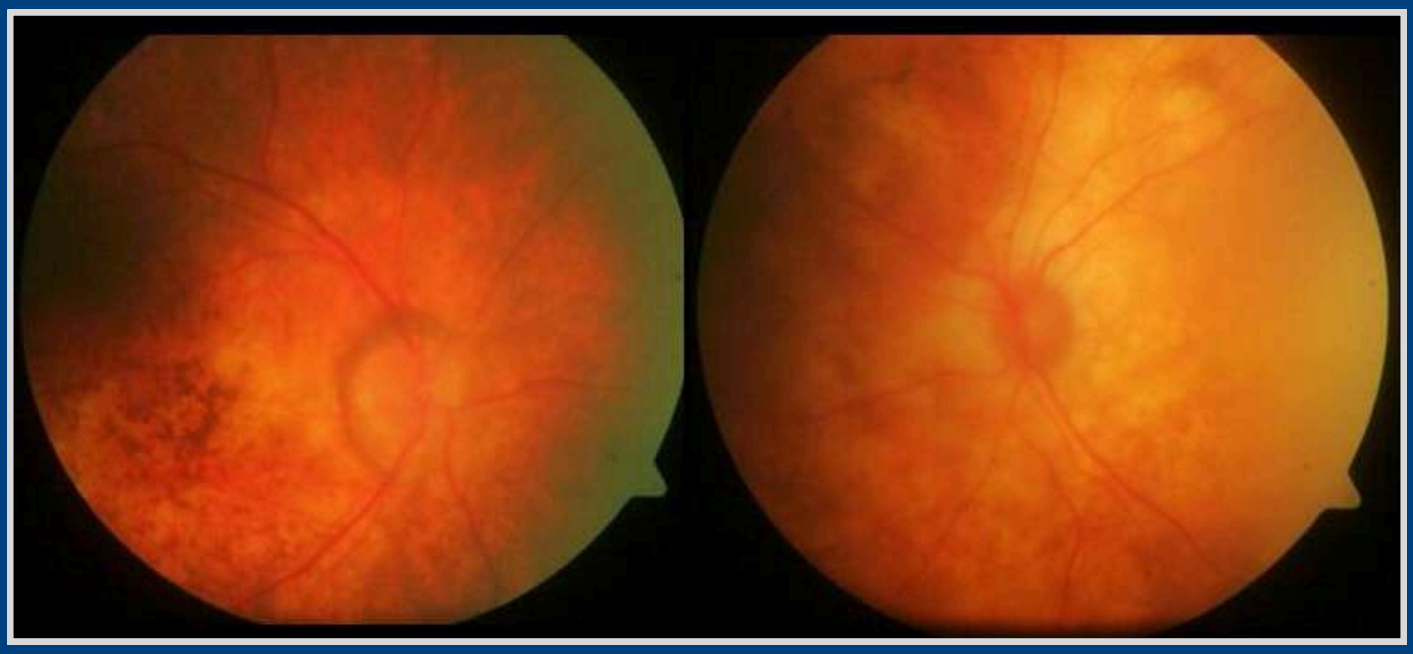
Post syphilitic retinitis – extensive loss of autofluorescence denoting gross chorioretinal atrophy

10 RED FLAGS TO SUSPECT OCULAR SYPHILIS

- **Posterior uveitis/panuveitis without other clear causes**
- **Retinal occlusive vasculitis**
- **Co-existing HIV infection**
- **Necrotizing retinitis mimicking ARN**
- **OCT : RPE - outer retinal hyperreflectivity + RPE excrescences + ellipsoid zone disruptions + choroidal thickening**
- **Acute syphilitic posterior placoid chorioretinitis (ASPPC)**
- **Poor response/ worsening with steroids**
- **High-risk sexual behavior**
- **Ground glass appearance**
- **FFA : early hypofluorescence of placoid lesions with progressive hyperfluorescence, late staining or leakage, retinal vasculitis and capillary non-perfusion.**

In conclusion, ocular syphilis remains a critical diagnosis for ophthalmologists due to its protean manifestations and potential for complete visual recovery with timely treatment. With an estimated 6 million new cases of syphilis reported globally each year, awareness of its resurgence is crucial. Routine syphilis testing in cases of unexplained uveitis, adherence to neurosyphilis treatment protocols, and early recognition of HIV co-infection are key to achieving optimal outcomes.

VOGT KOYANAGI HARADA DISEASE



Dr. Mayur R. Moreker
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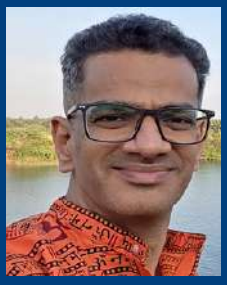


Acknowledgements: The author wishes to acknowledge Dr. Deepak Bhatt from UBM Institute, Dadar, Mumbai for his support, always, in imaging our uveitic and other ocular diseases.

Vogt in 1906, Koyanagi in 1929 and Harada in 1929 described a clinical entity independently, which was later in 1932 recognized to be the same disease process, with Babel suggesting the term Vogt-Koyanagi-Harada disease (VKH); of which, most patients are around the second and fifth decades of life with a predilection reported more in women in some of the studied populations.

My personal tryst with this disease began way back in 2005; after seeing my first patient of this enigma called "VKH" and then I began searching Indian literature on it; and back then found an analysis of 87 patients. We, then went on to contribute to literature with an analysis of our own patients.

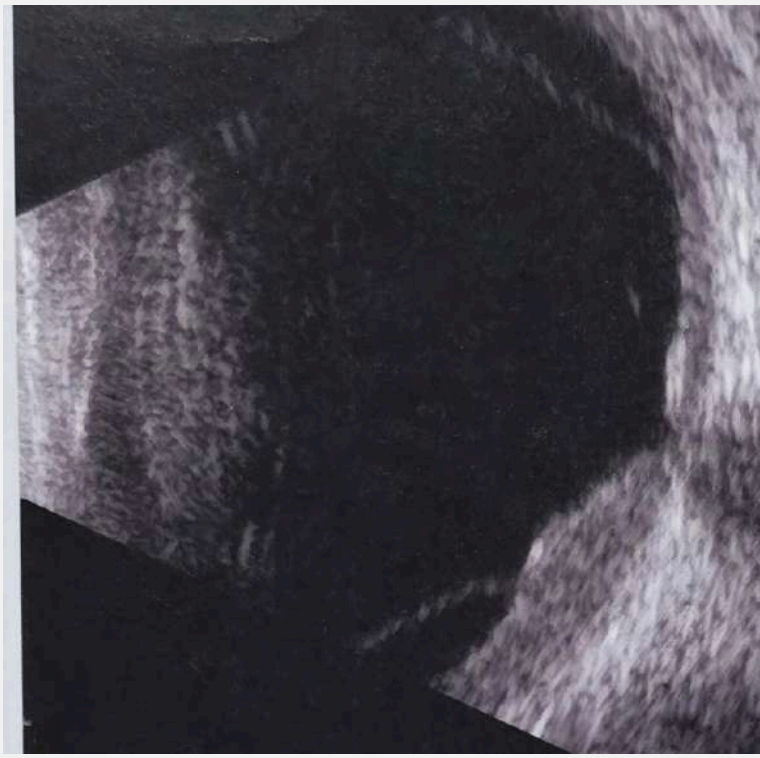
KEY CLINICAL FEATURES ACROSS STAGES

The clinical features vary, depending on stage of the disease with the **prodromal stage** being similar to a viral illness with headaches and fever for 3-5 days; which progresses to blurred vision, photophobia, redness and eye pain; i.e. the **uveitic stage;** which could be asymmetrical but often, later proves to be bilateral within 2 weeks; with signs that include elevation of the peripapillary retino-choroid layer, hyperemia and edema of the optic disc, and multiple serous retinal detachments. Eventually, the anterior chamber may get affected, with the patient developing panuveitis; which, if inadequately treated leads to the **chronic or convalescent stage**, characterized by the development of vitiligo, poliosis, and choroidal depigmentation and later a "*sunset-glow fundus*". *Sugiura sign (perilimbal vitiligo)* is the earliest depigmentation to occur. The **recurrent stage** occurs, if inadequately treated with acute exacerbations of anterior uveitis.

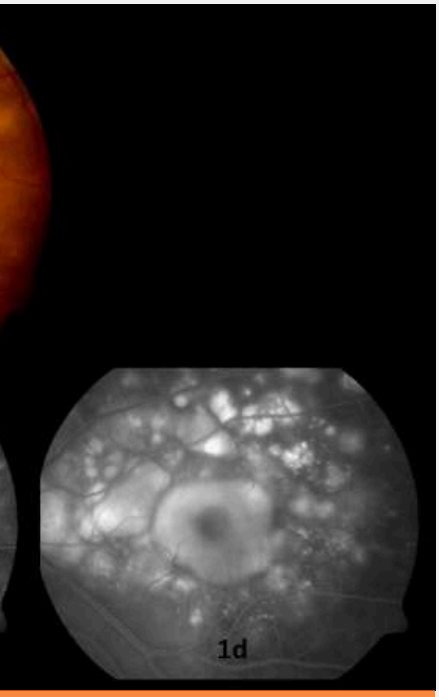
The disease is ominous for its propensity to cause vision-threatening complications cataracts, glaucoma, subretinal neovascularization amongst others.

focal areas of delay in choroidal perfusion in the early phase

multifocal areas of pinpoint leakage in the mid phase



ROLE OF IMAGING



large placoid areas of hyperfluorescence with pooling within the subretinal fluid in the late phase

Fundus Fluorescein Angiography (FFA)

During the *acute* stage, an early irregular focal or patchy fluorescence of the choroidal circulation, with various pinpoint areas of leakage at the level of the retinal pigment epithelium leading to localized hyperfluorescent spots, which increase in size, coalesce, and expand into the subretinal space in areas of serous detachment leading to a large area of leakage. The optic disc shows leakage. It is important to note that absence of early pinpoint peripapillary hyperfluorescence suggests that the disease is beyond its early stages, mandating immediate aggressive and prolonged treatment.

Indocyanine Green Angiography (ICG)

ICG shows an early choroidal stromal vessel hyperfluorescence and hypofluorescent dark spots during the early and mid-phase, distributed mainly posteriorly, and typically in excess of those seen clinically on FFA. The late phase (in *active* stage) shows hypofluorescent spots fading and being replaced by hyperfluorescent ones (representing focal sites of active choroidal inflammation); while in *chronic* stage, the hypofluorescent dark spots are seen during all the phases of ICG.

B Scan in acute stage showing vitreous echoes with marked choroidal thickening and exudative retinal elevation

Optical Coherence Tomography (OCT)

An OCT can be used to document subretinal fluid with corrugation of the RPE/choroid and also choroidal thickening.

Ultrasonography (USG Bscan)

This is *my first "go-to"* imaging modality, especially when acute VKH shows diffuse and "peripapillary" choroidal thickening with low to medium reflectivity, serous retinal detachments apart from the obvious vitreous opacities.

DIAGNOSTIC CRITERIA AND DIFFERENTIALS

The revised Diagnostic Criteria for VKH Disease were recognized at the First International Workshop on Vogt-Koyanagi-Harada disease and classify disease as **Complete** (1 to 5); **Incomplete** (1 to 3 and either 4 or 5) and **Probable** (1 to 3):

Criteria:

1. No history of penetrating ocular trauma or surgery preceding the initial onset of uveitis
2. No clinical or laboratory evidence suggestive of other ocular disease entities
3. Bilateral ocular involvement
4. Neurological/auditory findings (may have resolved by time of examination). A) meningismus and not headache alone, or B) tinnitus, or C) cerebrospinal fluid pleocytosis
5. Integumentary findings (not preceding onset of central nervous system or ocular disease). A) alopecia, or B) poliosis, or C) vitiligo

Differential Diagnosis includes amongst others, sympathetic ophthalmia, sarcoidosis, intraocular lymphoma, posterior scleritis, acute posterior multifocal placoid pigment epitheliopathy (APMPPE), lupus choroidopathy and of course central serous chorioretinopathy; especially if antecedent steroid use or in patients with chronic kidney disease.

PRINCIPLES OF MANAGEMENT

Intravenous methylprednisolone 1 gm after excluding systemic infection and contraindications and always under physician's supervision for 3 -5 days, followed by high-dose oral steroids to be tapered very slowly is the norm. Notably, reports suggest that patients receiving treatment for less than 6 months are more likely to have recurrences (58.8%) compared to those treated for 6 months or more (11.1%). Further, immunomodulatory therapy as first-line treatment has been associated with better visual acuity outcomes when compared to corticosteroid therapy alone. The immunosuppressants used are azathioprine (1-2.5 mg/kg/day) and cyclosporine A (3-5 mg/kg/day) and also, mycophenolate mofetil and rituximab.

SPECIFIC LEARNINGS FROM PUBLICATIONS FROM INDIA

VKH in the elderly:
Patients receiving systemic steroids with immunosuppressants had better visual outcomes at final follow-up compared to steroids alone.

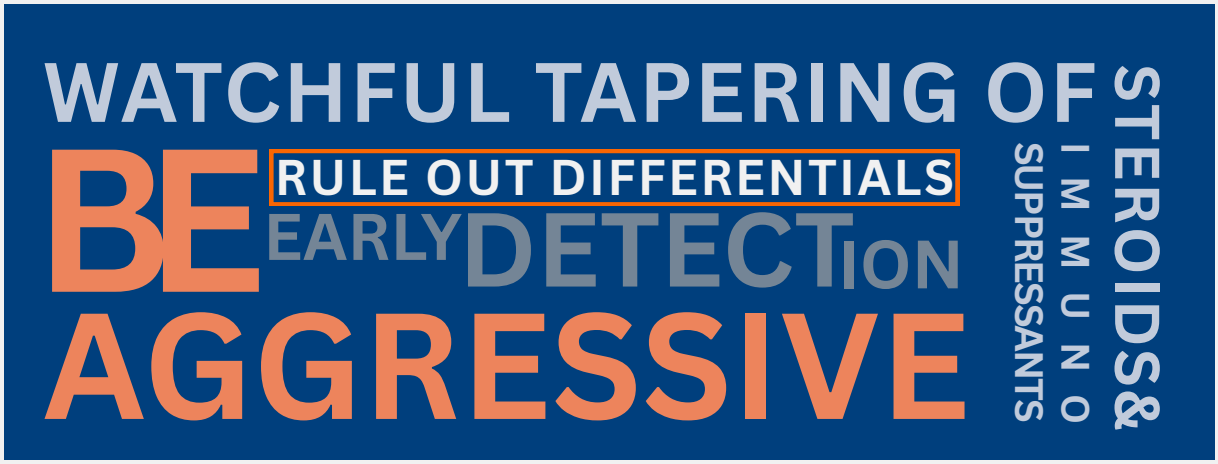
VKH in pediatric age group:
VKH in children poses a challenge due to a delayed presentation and have a worse visual acuity at the time of presentation as compared to adults. Rates of remission may be low along with high risk of complications and hence there is a need for prolonged immunosuppression.

First line therapy in VKH Disease:
Azathioprine has been documented as "first-line therapy" in patients with new onset of acute VKH disease.

Failure of primary immunosuppressive agents in VKH disease:
VKH is the most common etiologically diagnosed uveitic entity with primary immunomodulatory therapy failure and nowadays there is an increased use of tofacitinib in subsequent revisions of treatment in such patients.

VKH and pregnancy:
Pregnancy, despite being an immunomodulatory state, VKH is notorious to have a course similar to that of nonpregnant women and needs close monitoring and follow-up.

PRACTICE PEARLS



RETHINKING VOGT–KOYANAGI–HARADA DISEASE

EVOLVING IMAGING AND THERAPY

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There has been significant research and advances surrounding Vogt-Kayanagi-Harada disease (VKH), starting from its new classification into early and late stage according to SUN working group, analyses of investigative modalities like indocyanine green angiography and OCT angiography (OCTA), immuno- and genetic pathogenesis giving us a better understanding of the disease at a molecular level and even management and response to newer immunomodulatory drugs. In this article, we have mainly concentrated on the use of OCTA in VKH, how it helps us to monitor and analyse the disease severity and the use of Janus Kinase (JAK) inhibitors and biologics in such patients.

OCTA IN VKH

Other than detecting and monitoring the activity of choroidal neovascularisation, OCT angiography (OCTA) has recently gained popularity for its non-invasive detailed visualisation of the retinal and choroidal vascular structures. We know that the OCTA comprises of different vascular plexuses- superficial (SCP), intermediate (ICP), deep capillary plexus (DCP) and choriocapillaris (CC). Choroidal vascularity index (CVI) is the ratio of choroidal vascularity volume (CVV) to the total choroidal volume, thus representing a three dimensional blood

flow density and reflecting the large vessel vascular density of the choroid.[4] The en face OCTA scans at the level of the *choriocapillaris* shows *multiple, discrete, dark areas* which are variable in shape and size and widespread in distribution. Partial or complete resolution may be seen with treatment whereas persistent dark areas may indicate chronic damage. Unlike central serous chorioretinopathy, dark areas are not co-localized to the area of subretinal fluid but rather present throughout the area scanned.[5,6]

Studies showed that the *vascular density (VD)* in the SCP and DCP were lower in acute onset VKH patients and lower CC-VD was noted as well. These changes recovered with treatment. However, the *superotemporal* quadrant of the macula was found to recover first probably due to asymmetric and preferential drainage of that area through vortex veins. SCP-VD and CC-VD was found to recover much earlier than DCP-VD. This is due to VKH being a primarily *choroidal* disease. Abnormal choroidal blood flow leads to poor oxygenation and nutrition of the outer retinal layer thus causing slower recovery of the DCP and potential disruption of the outer retinal layers. Thus, DCP-VD in the macula was correlated with visual acuity.[7] It has been suggested that DCP-VD is a sensitive predictor of disease severity in VKH.[8] Macular capillary perfusion was significantly reduced both in SCP and DCP in patients with inactive VKH disease.[9]

It is suggested that in inactive VKH with sunset glow fundus, many inflammatory cells are present in the choroidal stroma causing dilation of choroidal vessels. Higher CVI than normal was found in such patients. It was also reported that patients with higher large and medium sized choroidal vessel VD at two weeks of treatment had higher risk of developing sunset glow fundus.[7] Retinal hypoxia can also lead to foveal

OCTA SLABS FOR OPTOVUE AVANTI SOFTWARE VERSION

Slab	Containing	Extent	Depth (differs according to device used)
Superficial slab	Superficial vascular plexus	ILM to IPL	3-15 microns below ILM
Deep slab	Deep vascular plexus (intermediate + deep capillary plexus)	INL to OPL	15-70 microns below IPL
Outer Retina slab	Avascular zone	OPL to ONL or RPE	70 µm below the IPL to 30 microns below RPE
Choriocapillaris	Choriocapillaris	Beneath the RPE	30 µm to 60 µm below the RPE.

avascular zone (FAZ) enlargement.[8] Another study showed that VKH patients without optic disc edema had a higher proportion of increased vessel perfusion densities of radial peripapillary capillary plexus (RPC) and retinal plexus after treatment compared to VKH patients with disc edema. This could be a compensatory response to local inflammation-induced ischemia which for some reason may be obstructed in patients without optic disc swelling. The reason is not very clear. However, CC-VD was seen to increase gradually after treatment, regardless of the presence or absence of optic disc swelling.[10]

OCTA has the potential to be a tool for monitoring inflammatory activity in Vogt-Koyanagi-Harada disease (VKH). Recovery of VD and monitoring of lesions on en-face OCTA can hint at disease severity and activity. It is recommended at patients with VKH disease in the active phase undergo monthly OCTA evaluations to monitor dynamic vascular changes and biannual monitoring should be done during quiescent phases to assess long-term vascular remodeling.[4]

JAK INHIBITORS AND BIOLOGICS IN VKH

Rapid and aggressive control of inflammation is the main goal of treatment of VKH patients. Recently, the use of JAK inhibitors and biologics in non-infectious uveitis is on the rise. Though generally not given as a first line therapy, their use has shown successful management of recalcitrant inflammation in VKH. Biologics are complex therapeutic agents derived from living cells that target specific components of the immune system, such as monoclonal antibodies or recombinant proteins designed to block key inflammatory mediators like tumor necrosis factor-alpha (TNF-α), interleukins, etc.

A study with 70 VKH patients receiving adalimumab (ADA) for more than 6 months showed it to be effective and generally well tolerated with 17.1% patients showing adverse effects.[11] ADA has also been successful in recalcitrant paediatric VKH.[12] Other than ADA, infliximab has shown good response in controlling recurrent inflammation in both adults and children.[13,14] Rituximab has also been reported to be effective in patients with VKH disease unresponsive to TNF blockers.[14] Interferon-alpha-2a has been used successfully in 2 cases of refractory VKH.[15]

JAKs are a family of cytoplasmic non-receptor protein tyrosine kinases consisting of four members, JAK1 to JAK3 and TYK2. These kinases transmit cytokine signalling through the JAK/STAT signaling pathway, a fast membrane to -

JAK INHIBITORS AND BIOLOGICS USED IN VKH - REPORTED SO FAR

Class	Drug	Mechanism
JAK inhibitors	Tofacitinib	JAK 1,3 inhibitor
	Baricitinib	JAK 1,2 inhibitor
Biologics	Adalimumab	Fully humanized antiTNF-a monoclonal antibody
	Infliximab	IgG1 chimeric monoclonal antibody against membrane-bound and soluble TNF-a
	IFN-alpha-2a	Pleiotropic cytokines which have immunomodulatory effect on T cell and dendrites

nucleus signalling module that regulates the transcription and expression of various critical mediators involved in immune responses, cancer development, and inflammatory diseases. Significant increase in the locus of JAK1 in CD4, JAK3 in monocytes, STAT4 in CD8, and STAT1 have been reported in patients with VKH disease.[16]

Use of JAK inhibitors in VKH is less reported. A study by Kawali et al[17] reported that the combination of tofacitinib and methotrexate was effectivein controlling inflammation in VKH. On the other hand, Liu et al[18] reported successful treatment of glucocorticoid intolerant naïve VKH patients with tofacitinib 5 mg twice daily dose along with peribulbar triamcinolone acetonide injection with no serious adverse events. Persistent bullous retinal detachment despite five doses of intravenous methylprednisolone and oral steroids have also shown good response to tofacitinib.[19] Successful use of baricitinib in VKH has also been reported.[20]

Upadacitinib is a reversible and selective inhibitor of JAK1 that has been approved for the treatment of numerous autoimmune diseases, such as rheumatoid arthritis, ankylosing spondylitis, psoriatic arthritis, etc. Potential use in VKH has been shown in experimental autoimmune uveitis model. However its actual use in VKH has not been reported.[16]

What is interesting and important to note is that the use of both JAK inhibitor and biologics for other disorders have led to the development of a VKH-like response.

Patients treated with interferon-alpha 2a for chronic viral hepatitis C have reported this phenomenon.[15] Use of drugs like prembrolizumab, cemiplimab in patients with malignancy have shown this response.[21,22] In fact, even the use of tofacitinib and ADA have led to VKH-like reaction. Tran et al[23] reported a male patient put on tofacitinib for treatment of vitiligo, who thereafter came to the clinic 12 months later with macular exudative retinal detachment and undulating retinal pigment epithelium in one eye. His vision improved once tofacitinib was discontinued. Gehrke et al reported a paradoxidal pro-inflammatory effect in the form of a VKH like reaction in one eye of a female patient after undergoing adalimumab treatment for hidradenitis suppurativa for 2 years.[24] In her case, ADA was discontinued and she was started on oral steroids, cyclosporine and mycophenolate mofetil instead.

So even though the use of JAK inhibitors and biologics in VKH is rapidly gaining popularity, it is imperative that ophthalmologists be aware of the paradoxical pro-inflammatory phenomenon and treat accordingly.

OCTA has emerged as a valuable, non-invasive tool in VKH, offering insights into disease activity, severity, and vascular remodeling, while aiding longitudinal monitoring. Concurrently, JAK inhibitors and biologics represent promising options for refractory cases, though their use requires careful consideration.

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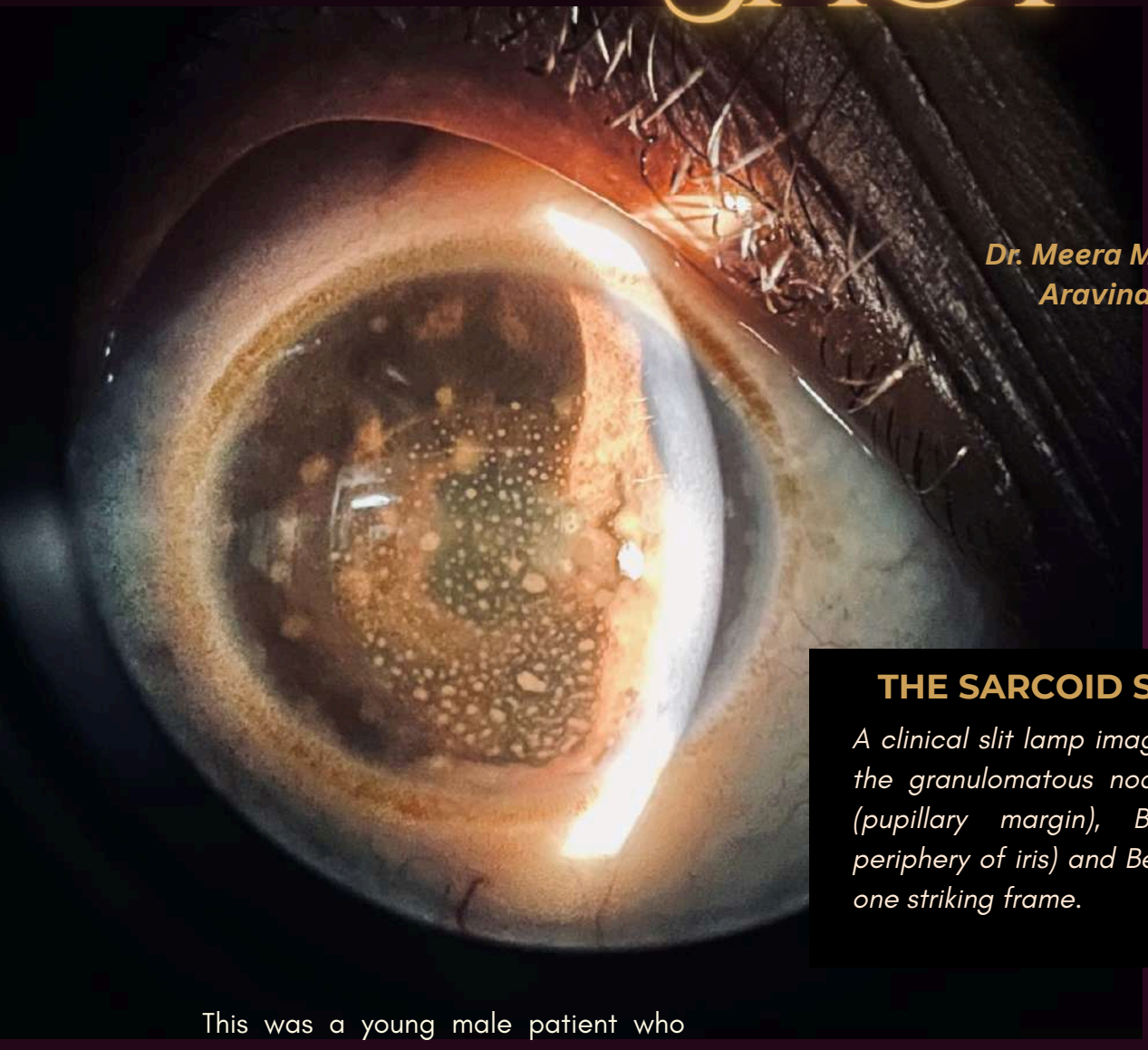
SnapSHOT



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Coimbatore

1

THE SARCOID SIGNATURE

A clinical slit lamp image depicting all the granulomatous nodules: Koeppe's (pupillary margin), Bussaca's (mid periphery of iris) and Berlin's (angle) in one striking frame.

This was a young male patient who presented with progressive visual deterioration and floaters. All relevant ocular and systemic work up were done - Mantoux was negative, serum ACE was high and HRCT chest revealed bilateral hilar lymphadenopathy. A diagnosis of ocular and systemic sarcoidosis was made in conjunction with the pulmonologist and the patient was started on oral and topical steroids.

Teaching point: Ocular tuberculosis classically is associated with muton-fat KPs and broad posterior synechiae, but in most cases, clinical features alone cannot differentiate between TB and sarcoidosis- it must be supplemented by systemic tests.

2 LUNAR SURFACE? NO, IT'S MULTIFOCAL CHOROIDITIS!

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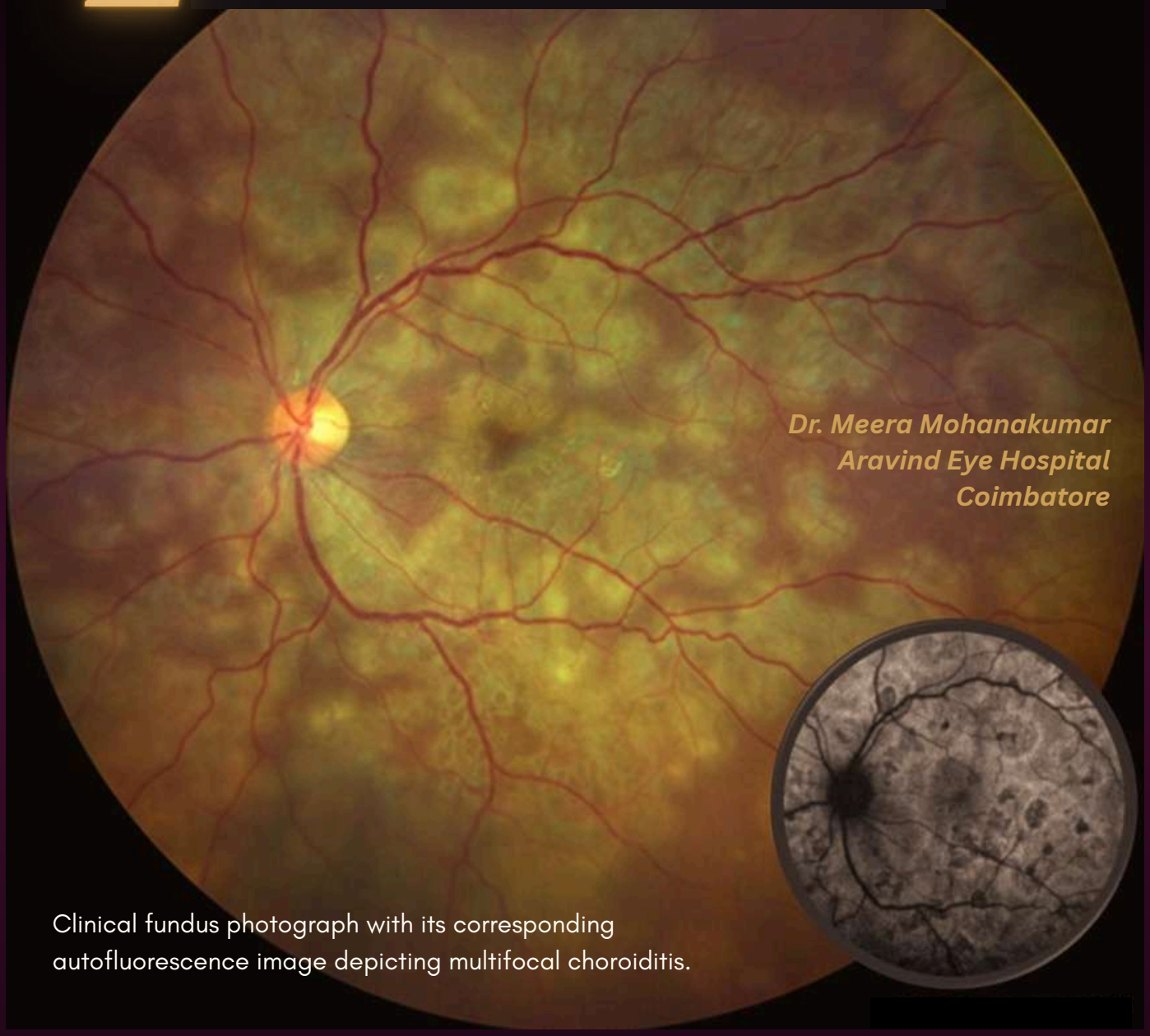
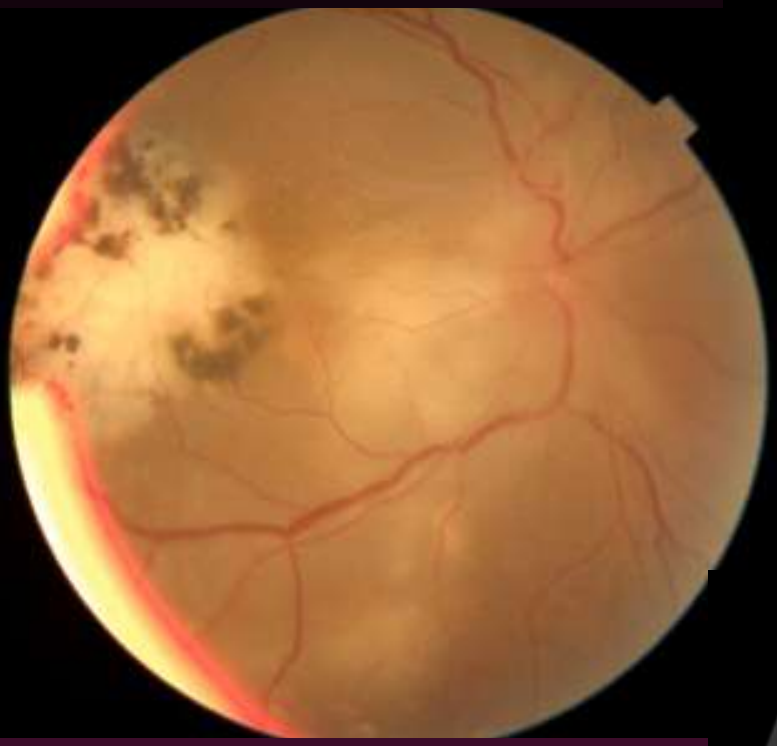


Clinical fundus photograph with its corresponding autofluorescence image depicting multifocal choroiditis.

Autofluorescence is an important imaging tool that captures the intrinsic autofluorescence from ocular tissues - and an important tool for monitoring disease activity and response to treatment with the added advantage of being noninvasive.

Teaching point: Active lesions show hyper-AF borders or patchy/diffuse hyper-AF indicating ongoing inflammation and metabolic stress, healing lesions demonstrate mixed hyper- and hypo-AF reflecting transitional RPE dysfunction, while inactive or scarred lesions appear uniformly hypo-AF, signifying RPE atrophy.

3 RECURRENT INTRAOCULAR TUBERCULOSIS



A 59-year-old gentleman was treated for right eye (OD) macular choroidal granuloma 4 years back, presented with redness and pain in OD which showed ciliary congestion and anterior chamber reaction.

Fundus examination revealed healed choroidal granuloma scar at macula with active peripapillary choroiditis.

The patient was started on a course of topical steroids and referred to a pulmonologist. He was started on a second course of anti tubercular therapy (ATT) along with oral steroids following which the recurrent choroiditis healed.

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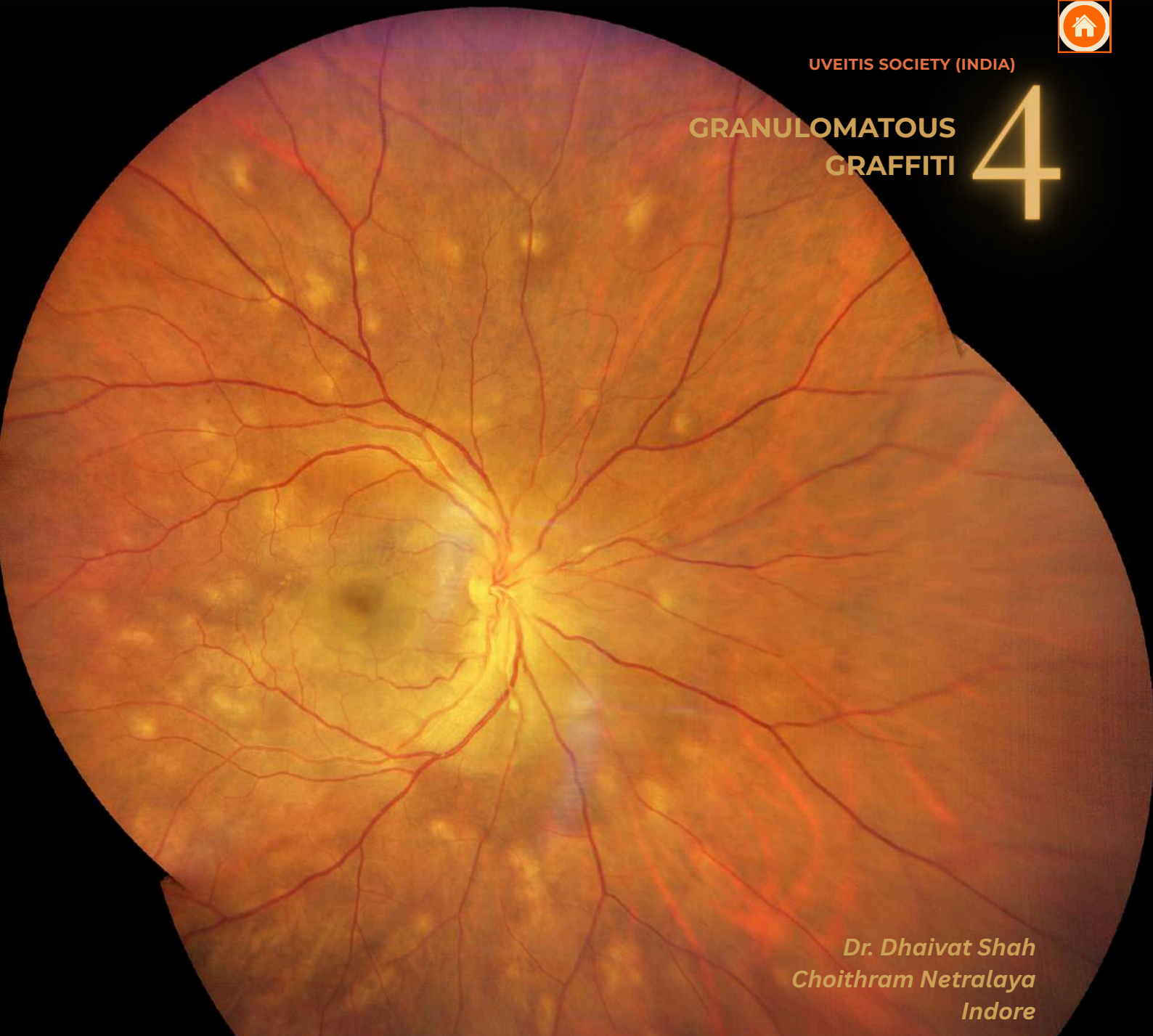
Coimbatore

Teaching point: IOTB inflammation can recur even after completing 6 months ATT. While initially presenting as posterior uveitis, anterior and intermediate uveitis presentations are common in post ATT recurrence.

GRANULOMATOUS GRAFFITI 4



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Choithram Netralaya
Indore

This wide-field fundus montage beautifully reveals Dalen-Fuchs nodules in a patient with acute Vogt-Koyanagi-Harada (VKH) disease. These creamy-yellow, sub-retinal lesions represent focal granulomatous aggregates of epithelioid cells and macrophages between Bruch's membrane and the RPE. At presentation, the involvement was clinically unilateral; the fellow eye showed a completely normal fundus examination, with no evidence of inflammation or serous detachment.

Teaching point: While the "sunset glow" fundus defines the chronic stage, Dalen-Fuchs nodules are also a critical diagnostic clue of granulomatous panuveitis. They serve as a visual signature of the autoimmune attack against uveal melanocytes. Recognizing this granulomatous pattern is vital for initiating aggressive systemic corticosteroid therapy to prevent permanent retinal damage and profound vision loss, highlighting the retina's role as a graffiti for systemic disease.

Case ARENA

CHELONAE CONUNDRUM! : A CASE OF DE NOVO NON-TUBERCULOUS MYCOBACTERIUM ANTERIOR SCLERITIS

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INTRODUCTION

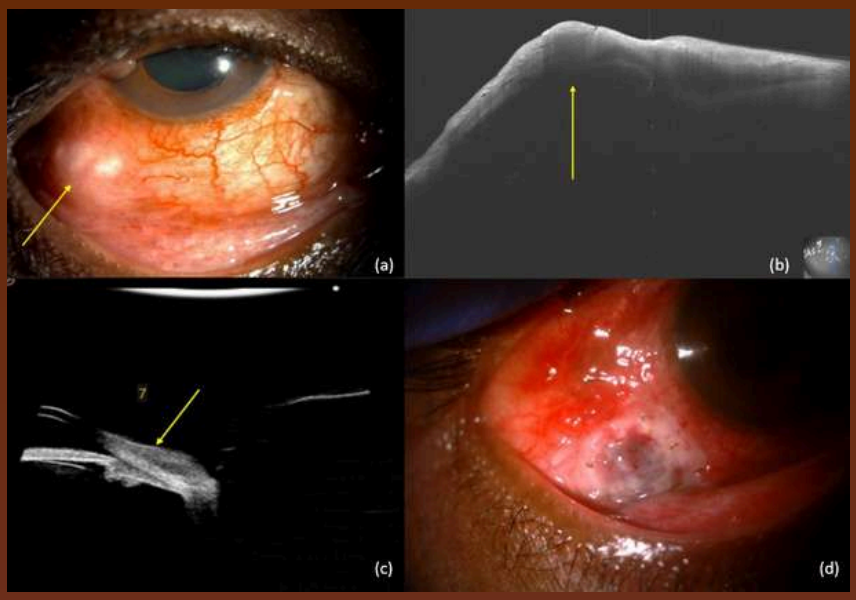
Atypical or non-tuberculous mycobacteria (NTM) are a group of acid-fast bacteria that can cause a spectrum of ocular infections including keratitis, scleritis, uveitis, endophthalmitis and orbital cellulitis. Trauma or surgery has the highest correlation with development of this infection. We describe here a case of de novo anterior scleritis, due to Mycobacterium chelonae.

CASE REPORT

A 55-year-old male presented with redness and pain in his right eye (OD) for the past 2 months. He denied any previous history of ocular trauma or surgery. He had been diagnosed elsewhere as necrotizing anterior scleritis and given a course of oral steroids, which was tapered over six weeks and stopped. On examination, his Best Corrected Visual Acuity (BCVA) was 6/9, N6 in both eyes (OU). Anterior segment OD showed a scleral nodule with pus pointing in the inferotemporal quadrant (Figure a). Fundus examination showed a normal optic disc and vessels, with a few RPE defects superotemporal to the fovea. The left eye was unremarkable. Anterior Segment Optical Coherence Tomography (AS OCT) demonstrated an elevated, hyper-reflective lesion within the sclera, with posterior shadowing (Figure b). Ultrasound Biomicroscopy (UBM) in OD revealed a staphyloma at 7 clock hours (Figure c), with trace supraciliary effusion from 6- 8 clock hours.

ESR was raised (43 mm/h). QuantiFERON TB Gold, RA Factor, ANA, c ANCA and p ANCA were negative. Right eye scleral scraping for Gram's, KOH, Acid Fast Bacilli (AFB) with 25% Sulphuric acid and PCR for M. tuberculosis IS 6110 and MPB 64 were negative. He was then planned for scleral de-roofing under local anaesthesia under oral steroid cover. AFB staining with 1% Sulphuric acid was positive for few AFB and culture identified the organism to be Mycobacterium chelonae. He was started on oral Ciprofloxacin 500 mg twice daily for 2 weeks, along with

OD showing a scleral nodule with pus pointing in the inferotemporal quadrant.



AS OCT of OD demonstrated localised increased thickness of the sclera with an elevated, hyper-reflective lesion within it, with posterior shadowing.

UBM of the OD revealed a staphyloma at 7 clock hours.

OD post scleral de-roofing, showing a healing conjunctival defect.

fortified Amikacin 2.5% eyedrops hourly. He showed good response to treatment and is under close follow-up (Figure d).

DISCUSSION

Infectious scleritis is a rare condition, accounting for 5 - 10% of all scleritis cases, often leading to diagnostic and treatment challenges and poor visual outcomes.[1] The sclera, being avascular, compact and covered by episclera and conjunctiva, makes it relatively impermeable to infection. Predisposing factors that may breach these barriers and cause microbes to invade the sclera include ocular trauma, surgery, contiguous spread from infectious keratitis or endophthalmitis, systemic immunosuppression, use of antimetabolites like mitomycin C or chronic use of topical corticosteroids.[2]

Tuberculous scleritis may occur either as a result of direct scleral invasion by Mycobacterium tuberculosis or by an immune-mediated process.[3] A scleral biopsy may reveal caseating granulomas with multinucleated giant cells and acid fast bacilli. The most common infection associated with Non-Tuberculous Mycobacteria (NTM) is keratitis, especially as outbreaks following LASIK surgery.[4] NTM scleritis, is quite rare, accounting for less than 12% of infectious scleritis, among which M. chelonae is a rapid grower and most commonly occurs post vitreoretinal surgeries.[5]

Kheir et al analysed 18 cases and observed that 94.4% were preceded by surgery, of which 77.8% had undergone scleral buckling.[6] Immunocompromised state is another predisposing factor. There have been some reports of cases that have no predisposition, where it presents as isolated scleral nodules, as in our case.

The in vivo efficacy of the drugs may be poor; thus, combination therapy is advocated. These infections often tend

to recur after cessation of therapy; therefore, therapy with at least two drugs and prolonged treatment for 4 weeks to 6 months after the resolution of clinical signs is recommended. Topical corticosteroids are best avoided.[7]

Despite its rarity, NTM scleritis is important, as its management may be challenging, both due to lack of immune protection to combat infection of the relatively avascular sclera and poor antimicrobial efficacy. Complete resolution of infection occurs in most cases, if prompt microbiologic diagnosis is made.

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NODULAR POSTERIOR SCLERITIS WITH CHOROIDITIS SIMULATING CHOROIDAL TUMOUR

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INTRODUCTION

Posterior scleritis is an uncommon and often under-recognized form of scleral inflammation, accounting for approximately 2 - 12% of all cases of scleritis.[1] In the absence of concomitant anterior scleritis, the diagnosis can be particularly challenging.[2] Based on ultrasonographic features, posterior scleritis may present as either a diffuse or nodular variant, the latter being distinctly rare.[3] Nodular posterior scleritis poses a significant diagnostic dilemma, as it can closely mimic choroidal tumours such as melanoma or metastatic lesions, as well as several other conditions presenting as nodular scleral thickening. Herein, we report a case of nodular posterior scleritis with overlying choroiditis, highlighting the diagnostic challenges and the importance of recognizing this rare entity to avoid unnecessary interventions.

CASE REPORT

A 61-year-old female presented with painful diminution of vision in the right eye noted for 20 days. She had previously undergone extensive systemic evaluation elsewhere and had been diagnosed as having choroidal melanoma, following which she sought a second opinion. Magnetic resonance imaging (MRI) of the brain with contrast was reported to have an asymmetric, enhancing T1-hyperintense thickening involving the posterior and right lateral wall of the choroidal layer of the right globe. Positron emission tomography (PET) showed corresponding asymmetric thickening along the posterior and lateral choroidal wall, while computed tomography (CT) of the chest demonstrated mediastinal lymph-adenopathy without evidence of active infection.

There was no history of fever, chronic cough, or similar episodes in the past. Best-corrected visual acuity was 6/48 in the right eye and 6/9 in the left eye, with intraocular pressures of 24 mmHg in both eyes. Anterior segment examination was unremarkable in both eyes. Fundus examination of the right eye revealed an elevated, yellowish subretinal mass at the posterior pole with surrounding subretinal fluid and trace posterior vitreous cells and a cup-disc ratio (CDR) of 0.6 [Fig. 1a]. The left eye showed a CDR. of 0.7, with the rest of the fundus findings being unremarkable.

Optical coherence tomography (OCT) of the right eye demonstrated an oval hyporeflective lesion within the choroidal

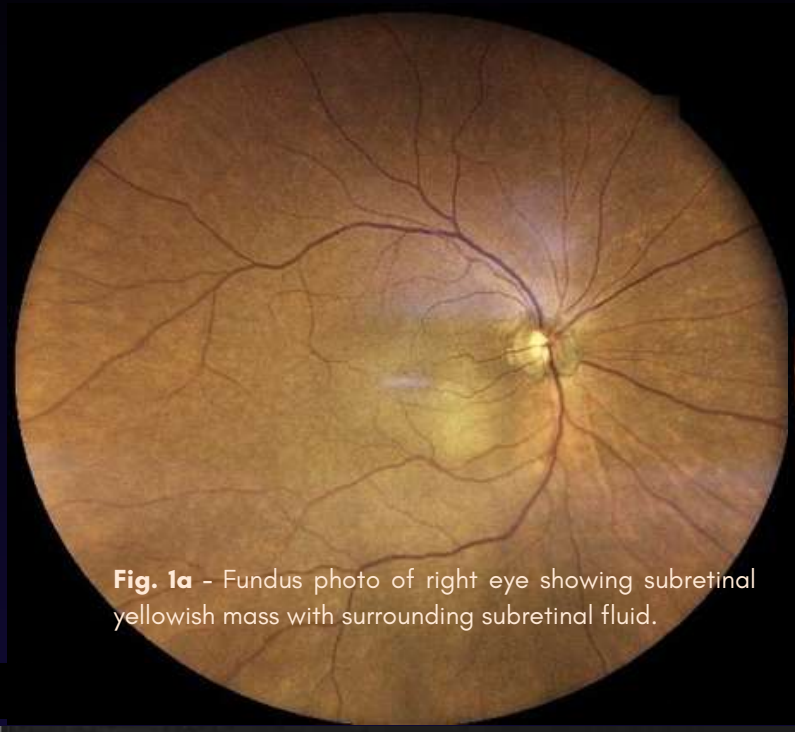

Fig. 1a - Fundus photo of right eye showing subretinal yellowish mass with surrounding subretinal fluid.

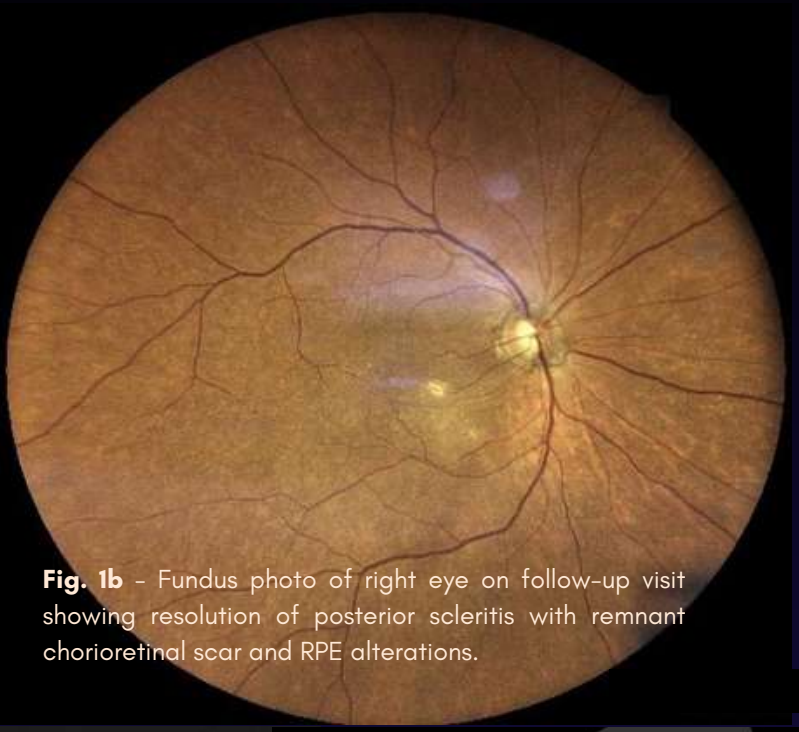

Fig. 1b - Fundus photo of right eye on follow-up visit showing resolution of posterior scleritis with remnant chorioretinal scar and RPE alterations.

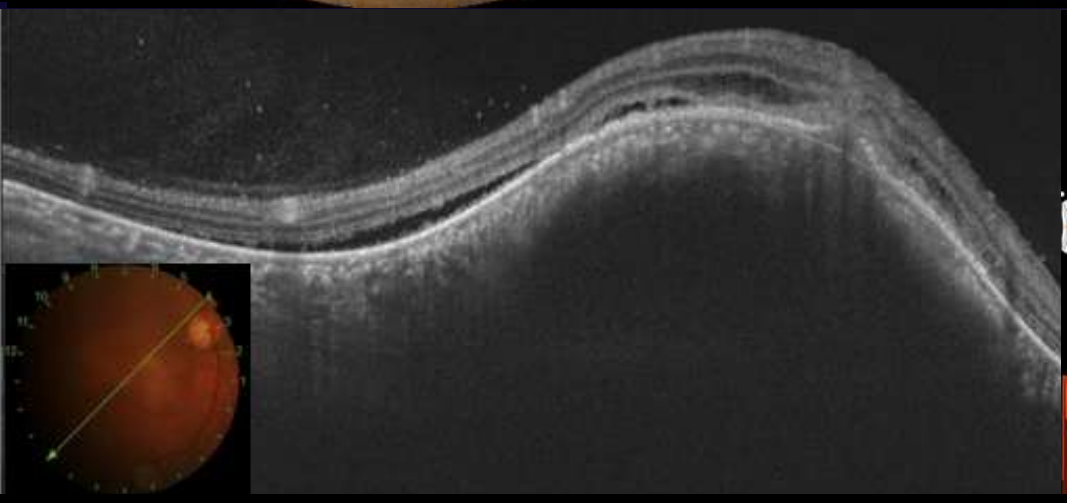

Fig. 2a - Baseline swept-source optical coherence tomography (SS-OCT) showing an oval hyporeflective lesion within the choroidal stroma suggestive of a granuloma, causing elevation of the RPE-choroidal complex with focal disorganization of the outer retinal layers and surrounding subretinal fluid.

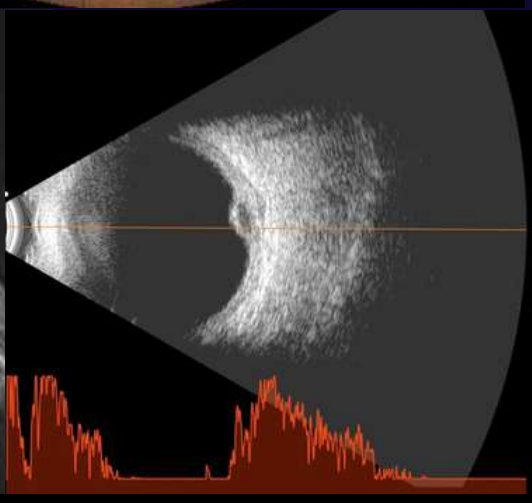

Fig. 2b - B-scan showing an elevated choroidal mass with heterogenous internal reflectivity and thickening of sclero-choroidal coats with positive T-sign.

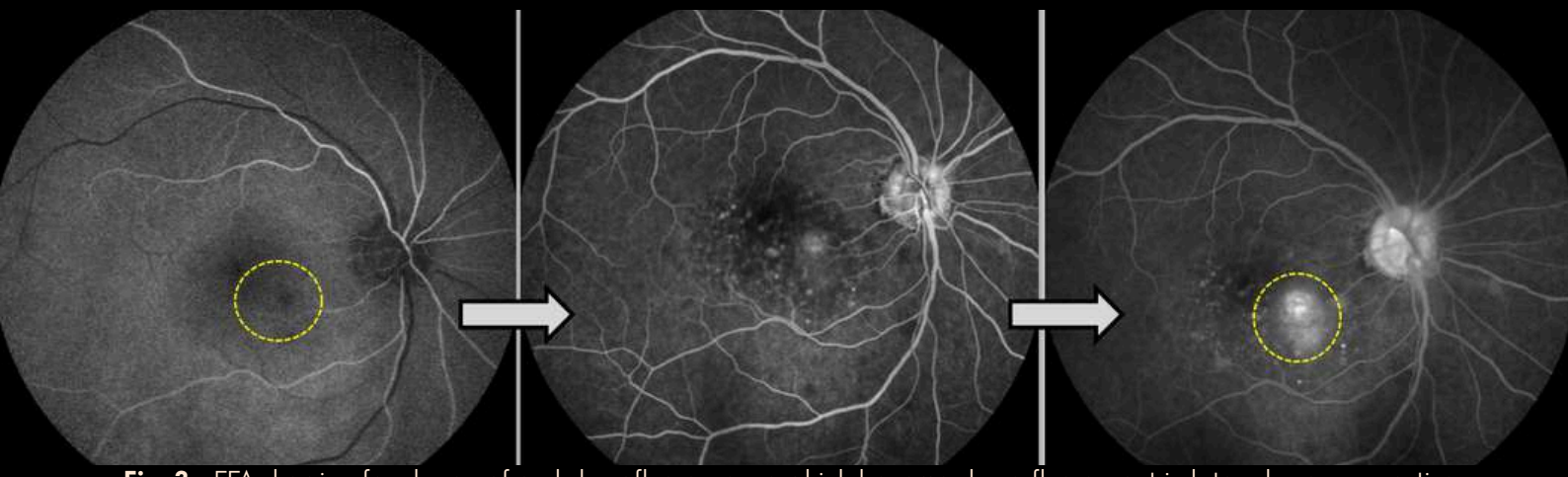

Fig. 3 - FFA showing focal area of early hypofluorescence which becomes hyperfluorescent in later phases suggestive of choroiditis/granuloma (yellow dashes) with pin-point hyperfluorescence which pools into the subretinal space.

stroma, with overlying retinal pigment epithelium disruption, inflammatory infiltration of the outer retinal layers, and an associated shallow neurosensory detachment [Fig. 2a]. B-scan ultrasonography revealed an elevated choroidal mass temporal to the optic nerve head with heterogeneous internal reflectivity, focal thickening of the sclero-choroidal coats, and a positive T-sign, without choroidal excavation or acoustic shadowing [Fig. 2b]. Fluorescein angiography revealed progressive pinpoint hyperfluorescence with late pooling and absence of dual circulation. An area of choroiditis inferotemporal to the optic disc was noted, appearing hypofluorescent in the early phase and hyperfluorescent in the late phase [Fig. 3].

Based on the multimodal imaging findings, a provisional diagnosis of nodular posterior scleritis with overlying choroiditis was made. Investigations to determine the cause of scleritis were inconclusive. ESR was elevated, while serum angiotensin-converting enzyme levels were within normal limits. Mantoux test, QuantiFERON-TB Gold test, and serological tests for syphilis and HIV were negative. Systemic evaluation by a physician did not reveal any underlying inflammatory or autoimmune disorder.

The patient was initiated on oral corticosteroids at a dose of 60 mg/day, which was gradually tapered over two months. Empirical oral antibiotics (amoxicillin 500 mg twice daily for one week) were also administered, as the etiology remained uncertain along with topical anti-glaucoma medications. At the end of three months, her visual acuity improved to 6/9p in the right eye. Fundus examination and OCT demonstrated resolution of the lesion with residual choroidal and outer retinal atrophy. She has remained clinically stable over a follow-up period of six months [Fig. 1b].

DISCUSSION

Nodular posterior scleritis is a potentially sight-threatening inflammatory condition that commonly presents with orbital pain, headache, and visual impairment. During the course of the disease, inflammation may extend contiguously to involve the choroid or vice versa, resulting in a spectrum of choroidal manifestations.[4] Histopathological evidence supports this association; biopsy specimens from enucleated eyes with posterior scleritis have demonstrated choroidal thickening with inflammatory infiltrates, as reported in seven cases by Calthorpe et al.[5]

The present case underscores the ability of posterior scleritis with associated choroiditis to closely mimic a choroidal tumour. The extensive radiological investigations performed initially were suggestive of a neoplastic etiology. However, careful attention to clinical features can aid in arriving at the correct diagnosis. Pain, a prominent symptom in our patient, strongly favors an inflammatory process, as choroidal tumours are typically indolent and painless.[6] Multimodal imaging further assists in differentiation. B-scan ultrasonography, a key diagnostic tool, characteristically demonstrates sclero-choroidal thickening with moderate-to-high internal reflectivity, absence of

acoustic shadowing, and associated subretinal fluid. On fluorescein angiography, the absence of dual circulation provides a strong argument against a choroidal tumour.

Identifying the underlying etiology of posterior scleritis is essential for guiding appropriate management. In the present case, extensive investigations failed to reveal an infectious or systemic inflammatory cause. Idiopathic nodular posterior scleritis is known to respond favorably to systemic corticosteroid therapy[7] and our patient showed a dramatic clinical and anatomical improvement following oral steroid treatment. This therapeutic response further helped in reaffirming the diagnosis.

Misdiagnosis of posterior scleritis as a choroidal tumour can lead to inappropriate management and, in some cases, irreversible outcomes such as enucleation.[8] A trial of anti-inflammatory therapy may therefore serve as a valuable therapeutic and diagnostic test, potentially sparing patients from invasive and unnecessary interventions.

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TAKAYASU ARTERITIS ASSOCIATED OCULAR ISCHEMIC SYNDROME

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INTRODUCTION

Takayasu arteritis, first described by Takayasu in 1908, is a chronic, granulomatous autoimmune inflammation of large and medium-sized vessels, particularly the aorta and its branches.

It involves all three layers adventitia, media, and intima and is mediated by CD4+ T cells, monocytes, and macrophages, leading to vascular remodeling with aortic aneurysms and intimal hyperplasia causing occlusion of branch vessels.[1]

Ocular involvement is variably reported in 8–65% of cases.[2]

Takayasu retinopathy has been classified by Uyama and Asayama into four stages:

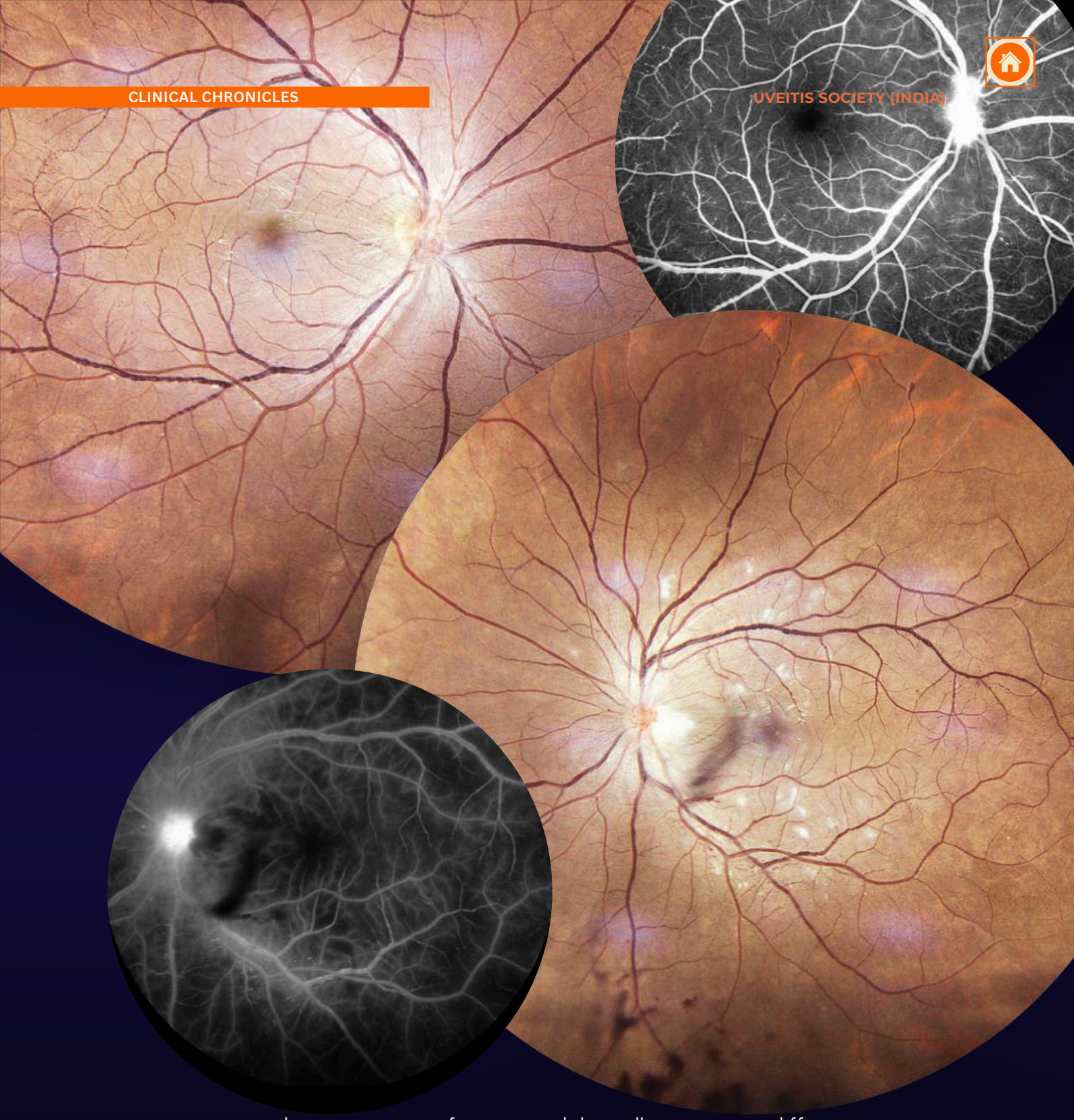
- venous dilatation
- microaneurysms
- peripheral arteriovenous anastomosis
- neovascular complications[3]

Ocular manifestations may be the presenting feature of systemic Takayasu arteritis.[1-3] Reported findings include ocular ischemic syndrome (OIS), central and branch retinal artery occlusions, anterior ischemic optic neuropathy, and hypertensive retinopathy.[4,5]

The disease is more commonly seen in females (7:1), Asians, and younger patients. We report a case of bilateral stage 4 Takayasu retinopathy with OIS as the presenting sign of Takayasu arteritis.

CASE REPORT

A 32-year-old woman presented with complaints of diminution of vision in both eyes with occasional pain and redness for the past 6 months. Systemic history was notable for anemia for which she was treated in the past. On examination, her best corrected visual acuity (BCVA) in right eye was 6/6 N6, left eye was 6/36, N36. The intraocular pressure was 13 mm Hg in both eyes. Anterior segment examination showed mild circumciliary congestion, occasional cells in anterior chamber and neovascularisation of iris in both eyes. The left eye pupil was mid-dilated and fixed. Gonioscopy was normal in both eyes. Fundus examination revealed dilated veins, multiple microaneurysms in the mid periphery, neovascularisation at the disc (NVD) in both eyes. Additionally, minimal vitreous hemorrhage was noted in the left eye (see figure). Optical coherence tomography (OCT) macula in both eyes was normal. Fundus fluorescein angiography (FFA) showed marked delay in both arm retina time (38 seconds) and arteriovenous transit (20 seconds), with capillary non perfusion in all quadrants, midperipheral dot hyper fluorescence due to microaneurysms, disc leak due to NVD, peripheral arteriovenous



anastomosis and sausage sign of veins due to hypoperfusion (see figure). Based on the typical FFA findings, a diagnosis of ocular ischemic syndrome (OIS) was made. As the patient was very young, a thorough systemic examination and investigations were ordered, along with cardiac evaluation.

Of note, carotid doppler showed bilaterally symmetric diffuse concentric wall thickening with 80-85% luminal stenosis extending throughout the entire length up to bifurcation of the right and left common carotid arteries. The internal and external carotid arteries on both sides showed luminal narrowing and reduced caliber. CT aortogram also showed luminal narrowing. Based on the above findings a rheumatology opinion

was taken and diagnosis of Takayasu arteritis was confirmed.

The patient was started on topical and systemic steroids at 1 mg/kg body weight along with methotrexate 15 mg weekly dose, as per the 2018 EULAR recommendations.[6] She underwent panretinal photocoagulation in both eyes. Systemically, she was suggested carotid endarterectomy or stenting, which she declined.

On follow-up her right eye was stable, however the vision in her left eye dropped to hand movement close to face secondary to the development of a total cataract which was possibly due to the severe anterior segment ischemia.

Cataract surgery in such cases can be complicated by intraoperative hyphema and poor visual outcomes. A preoperative intracameral or intravitreal anti-VEGF injection may reduce the risk of these complications.[7]

DISCUSSION

Ocular findings in Takayasu arteritis can be due to hypoperfusion secondary to the occlusive arteritis of the aortic arch or hypertensive retinopathy changes secondary to involvement of renal artery or supra-renal aorta.[8] Any of these manifestations in a young patient should prompt a thorough systemic examination. Bilateral brachial and radial pulse examination, variability in the blood pressure of >20 mmHg between the two arms are all diagnostic clues towards Takayasu arteritis.

As previously emphasized by Currier R D et al.,[2] ocular manifestations constitute the most common and consistent feature of this condition. The present case serves as yet another reiteration of this observation, reinforcing the importance of recognizing these findings.

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WINNER NEXTGEN 2025

RETINAL SIGNATURES IN SSPE

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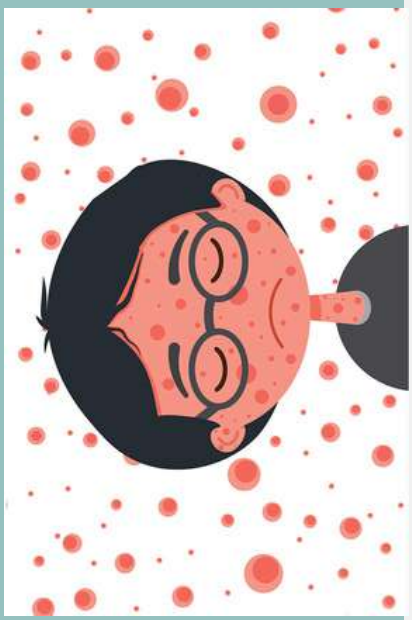

Subacute sclerosing panencephalitis (SSPE) is one of those diagnoses that most of us read about during training, maybe see a picture or two, and quietly hope we'll never encounter in real life. But every once in a while, a patient walks in and reminds you why keeping rare entities at the back of your mind really matters.

Subacute sclerosing panencephalitis is a progressive, often fatal demyelinating disease caused by persistent measles virus infection. While it is classically a neurological condition, the eye often tells the story first — sometimes long before the brain does.

Over the years, I have seen a small number of patients who taught me the same lesson repeatedly: SSPE can walk into your clinic disguised as retinal disease.

WHAT SHOULD MAKE YOU PAUSE?

Most of these patients were young — teenagers or young adults — often from a low socioeconomic background, with an unclear or unreliable immunization history. They came with painless diminution of vision, and at first glance, nothing else seemed very dramatic systemically.

On fundus examination, however, there it was — bilateral macular necrotising retinitis. The lesions were grey-white, irregular, and ill-defined, with the classic "moth-eaten" appearance that many of us associate with SSPE. What really stood out was how fast these lesions evolved. Even without any treatment, they scarred rapidly — sometimes within days to weeks. That rapid scarring itself became a major clue.

Not every patient followed the textbook. Some had atypical lesions, some were asymmetric, and a few presented at different stages in each eye. A couple even showed waxing and waning activity. The takeaway? Absence of a classic picture does not rule out SSPE.

THE DIAGNOSTIC STRUGGLE

The real challenge begins when there are no neurological signs at presentation. Understandably, SSPE doesn't top the list of differentials for most clinicians, including neurologists — especially because it is rare. These patients are often labelled as viral retinitis, inflammatory retinitis, or even demyelinating disorders like MOG or multiple sclerosis. In fact, inappropriate immunosuppression can sometimes be started before SSPE is considered.

Diagnosis ultimately rests on Dyken's criteria, incorporating clinical features, EEG findings, CSF measles antibody titers, and neuroimaging. But in several cases, it was the ophthalmic suspicion that triggered the correct neurological workup.

WHY THIS MATTERS IN 2026?

Across cases, certain patterns kept repeating — young age, bilateral macular involvement, rapid scarring of necrotising lesions, and unclear vaccination history. Sadly, despite reaching the diagnosis, outcomes remained poor. There is still no definitive cure for SSPE.

With declining vaccination coverage and periodic measles outbreaks, SSPE is far from obsolete. Nearly half of these patients may have ocular involvement, and many will present to an ophthalmologist first. While early diagno-

-sis may not change the final outcome, it does prevent mis-management, allows timely counselling, and reinforces the importance of vaccination. Sometimes, recognising what we are seeing — and having the courage to say "this doesn't fit" — is the most important intervention we can offer.

TAKE-AWAY POINTS

- Bilateral macular necrotising retinitis in young patients, especially with unknown immunization history, should raise suspicion for SSPE.
- Rapid scarring of retinal lesions without treatment is a crucial diagnostic clue.
- Ophthalmologists may be the first to suspect SSPE—early recognition and neurological referral are vital.

GENETICS & UVEITIS

WINNER NEXTGEN 2025

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Anant Bajaj Retina Institute
Amod Gupta Young Researcher Award '24
Author, Poet of "Echoes from a growing heart"

Uveitis is complex and demanding - at times unyielding and idiopathic. Inflammatory disorders of the human body including uveitis (both autoimmune and autoinflammatory) are multifactorial and have a non-linear causality. It is a wonderful yet intricate and dynamic interplay of various infections, immune dysfunctions, environmental factors, genetics and also personal factors like stress, diet, physical activity and exercise. The title of this article should not sway you to think that genetics is the basis of uveitis. Most of the time it is not. However, there could be three scenarios, wherein we could test for the genetic basis of uveitis in the clinics which I would divide as three groups:

1. Autoimmune - auto-inflammatory spectrum
2. Infections
3. Inherited retinal dystrophies (IRDs)

Starting bottom to top, **Group 3** is the least understood. They can be further subcategorised into two –

Where inherited retinal dystrophies *without any 'true' uveitis*, mimic uveitis. Here, clinical features like vitreous haze, cystoid macular edema, band-shaped keratopathy, retinal vasculitis-like changes, and vascular leakage on FFA – mimic uveitic phenotypes and we may misdiagnose it as uveitis instead of an inherited retinal dystrophy.

For instance,
-ABCA4 retinopathy autofluorescence can mimic uveitis patterns.
-Retinitis pigmentosa has cystoid macular edema and vitreous changes.
-X-linked retinoschisis (RS1), PRPH2, and CRB1 phenotypes can overlap with inflammatory maculopathies.
These can easily mislead us in clinics.

The second subgroup adds much more to the confusion. These are cases that *have true uveitis* – and often, uveitis is preceding the clinical presentation as IRDs. Here, the very genetic defects that cause retinal dystrophy are the ones that lead to structural and/or functional changes predisposing to uveitis.

For instance
-ALPK1 mutations activate the proinflammatory NF-κB pathway causing the ROSAH syndrome.
-CAPN5 mutation leads to dysfunction of innate and adaptive immune systems, causing the autosomal dominant neovascular inflammatory vitreoretinopathy (ADNIV).
-VCAN1 mutations are associated with irregularities in the vitreous modelling.

Coming to **Group 2**, we see atypical or recurrent infection that have a genetic predisposition due to defects in immune cells, mediators or pathways.

- HLA B35 predisposes progression of HIV to AIDS and Toxoplasma retinitis.
- Recurrent toxoplasma retinitis is associated with defects or dysfunctions in lymphocyte maturation and IFN-γ responses. Variations in ERCC3, MANBA, IRF4, HAVCR2, CARMIL2, CD247, MPO, C2 and CD40 genes have been seen to contribute to this.
- TLR pathway polymorphisms have been shown to predispose to toxoplasmosis and CMV infections.
- Defects in the IL-12 / IFN-γ axis, particularly IL12RB1 and IFNGR1, are seen to be associated with mycobacterial disease.
- STAT1 mutations impair Th17 cell differentiation, leading to enhanced susceptibility to fungal and viral infections.
- CARD9 deficiency shows a strong, predisposition to invasive fungal disease.
- NEMO / IKBKG mutations result in combined immunodeficiencies with a broad vulnerability to opportunistic bacterial, mycobacterial, and viral infections.

These are just a few examples to name.

Lastly, coming to **Group 1** - now, this is the group we are most comfortable with. This is the recognised group of autoinflammatory, autoimmune and mixed pattern diseases. We have all seen, read and treated seronegative spondyloarthropathies, ANCA associated vasculitis causing uveitis and scleritis. Monogenic autoinflammatory diseases like Familial Mediterranean Fever (FMF), Tumor Necrosis Factor Receptor-Associated Periodic Syndrome (TRAPS), Hyperimmunoglobulin D Syndrome (HIDS), and Blau syndrome, as well as monogenic autoimmune diseases like Autoimmune Lymphoproliferative Syndrome (ALPS) and Immunodysregulation Polyendocrinopathy Enteropathy X-linked (IPEX) syndrome, can rarely cause uveitis or ocular inflammation

TAKE-AWAY POINTS

- Uveitis may co-exist with or precede presentation of inherited retinal disorders.
- Inherited retinal disorders can mimic uveitis due to vitreous, retinal vascular changes and/or cystoid macular edema
- Genetic factors may predispose to 'atypical' or 'recurrent' infections.

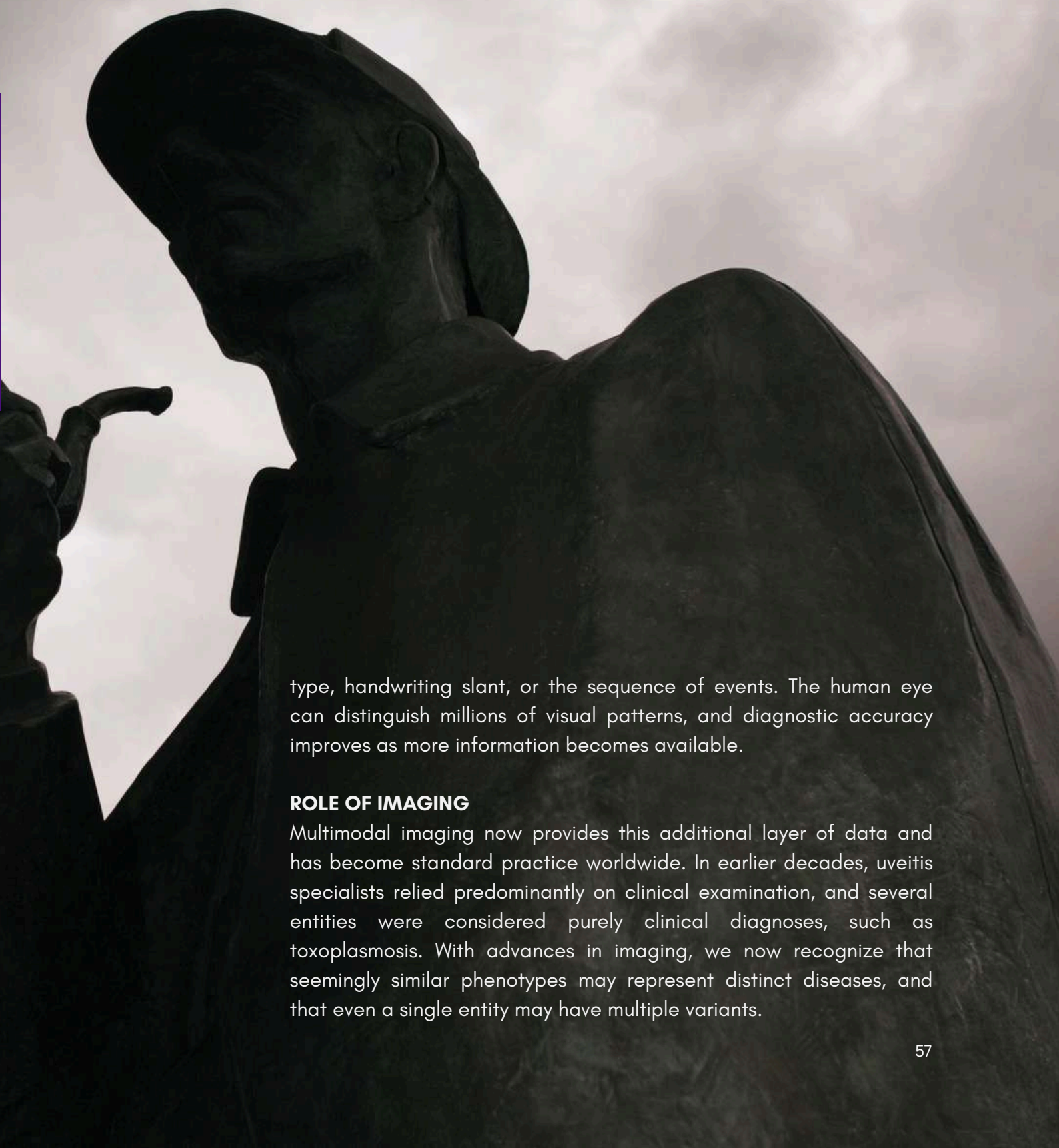
IMAGING THE INVISIBLE

Uveitis has traditionally been classified using an approach we often describe as "naming and meshing," allowing us to reach a diagnosis efficiently while minimizing unnecessary delays and investigations. Because diagnosis is largely based on pattern recognition, careful phenotyping is essential. We assess where a disease fits within a known framework, although this can be challenging when different entities share overlapping features such as hemorrhages, edema, and vasculitis. In such situations, the morphology of lesions, including their shape, color, size, location, and evolution, becomes critical in distinguishing one condition from another. At the same time, infectious causes must be excluded through appropriate laboratory testing, which can sometimes be invasive.

PATTERN RECOGNITION

Pattern recognition remains central to uveitis practice. A useful parallel can be drawn from the detective methods of Arthur Conan Doyle. In the Sherlock Holmes series, diagnosis is achieved not by a single dramatic clue but by the recognition of patterns among seemingly ordinary details. Holmes distinguishes between what appears similar to the untrained eye by focusing on subtle variations in footprint depth, cigar ash

INSIGHTS FROM THE MULTIMODAL IMAGING IN UVEITIS (MUV) PROJECT



type, handwriting slant, or the sequence of events. The human eye can distinguish millions of visual patterns, and diagnostic accuracy improves as more information becomes available.

ROLE OF IMAGING

Multimodal imaging now provides this additional layer of data and has become standard practice worldwide. In earlier decades, uveitis specialists relied predominantly on clinical examination, and several entities were considered purely clinical diagnoses, such as toxoplasmosis. With advances in imaging, we now recognize that seemingly similar phenotypes may represent distinct diseases, and that even a single entity may have multiple variants.

This concept is not unique to medicine. Classical literature often uses the device of similar looking sisters to illustrate how superficial resemblance can conceal important differences in character and identity. In Pride and Prejudice, the Bennet sisters are frequently grouped together socially, but careful observation reveals distinct personalities, values, and trajectories. The clinician's task in uveitis is analogous. At first glance, two placoid lesions may look identical. However, closer inspection of subtle features such as lesion borders, distribution, choroidal involvement, associated vascular changes, and temporal evolution reveals their true identity. Multimodal imaging functions as our "close reading" of the fundus, allowing us to move beyond surface resemblance and appreciate the deeper structural and functional differences that define each disease entity.

AI INTEGRATION

In the current era of artificial intelligence and deep learning, it is essential to integrate all available data to achieve precise diagnoses. With this objective, the International Uveitis Study Group (IUSG) undertook the task of standardizing the imaging language across six key modalities: color fundus photography (CFP), fundus autofluorescence (FAF), fundus fluorescein angiography (FFA), indocyanine green angiography (ICGA), optical coherence tomography (OCT), and optical coherence tomography angiography (OCTA). Initially, definitions and clinical utility statements were developed for five common placoid chorioretinopathies, often grouped under the misnomer of white dot syndromes.

Under the leadership of Prof. Vishali Gupta, a steering committee was formed to establish the study methodology and harmonize imaging interpretation across posterior uveitis entities. This led to the creation of the Multimodal Imaging in Uveitis (MUV) study group and its task force of disease specific experts. The group aligned its framework with the Standardization of Uveitis Nomenclature (SUN) criteria published in 2021, which are widely accepted and incorporate artificial intelligence compatible methodologies for disease classification.

The MUV steering committee established methodology, grading protocols, and disease specific expert panels to ensure reproducibility and interobserver agreement. The MUV used a structured nominal group technique (NGT), a robust methodology well tested in the literature. Using the NGT, the MUV experts proposed several consensus criteria for various conditions.

"Pattern recognition remains central to uveitis practice."

For instance, the experts agreed that serpiginous choroiditis is characterized by geographic, centrifugally spreading lesions with imaging features that highlight choriocapillaris-driven pathology (using ICGA and OCT) and allow clear distinction between active borders and inactive atrophic areas. Multifocal choroiditis and punctate inner choroidopathy show multiple discrete inflammatory lesions at the level of the retinal pigment epithelium and choroid with a high propensity for secondary neovascular complications, for which OCT, ICGA and OCTA-based assessment is particularly valuable. Birdshot chorioretinopathy demonstrates a discordance between clinically visible lesions and more extensive choroidal involvement on ICGA, along with characteristic retinal vascular leakage on FFA that serves as an important marker of disease activity.

In an orchestra, when a single instrument plays alone, the melody may be recognizable but lacks depth, harmony, and texture. As additional instruments join in, each contributes a distinct layer, rhythm, counterpoint, or tonal color. The strings provide continuity, the brass add emphasis, and the percussion defines structure. Only when all sections perform together under a common score does the full composition emerge in its intended form.

The MUV effort mirrors this orchestral model and is an ongoing process. Each imaging modality offers a different "voice," highlighting a particular structural or functional aspect of inflammation. Individual disease panels contribute their specialized interpretations, and consensus methodology provides the unifying framework. When these elements are integrated, they produce a richer, more precise understanding than any single modality or observer could achieve alone, ultimately allowing us to interpret complex uveitic patterns with greater clarity and confidence.

Dr. Aniruddha Agarwal, MD, Ph.D.

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IMMUNOLOGY AT THE CORE OF UVEITIS CARE

Current paradigms of the management and treatment of ocular inflammation, principally non-infectious uveitis, are based on the understanding of broader Immune Mediated Inflammatory Diseases (IMID). Understanding these disease pathways both broadly across the IMID spectrum and in tissue specific responses helps us bring about more personalised treatment options for our patients. To achieve this requires knowledge of systemic and regional immunology.

The field of immunology continues to uncover the immune system's broader roles, both in maintaining health and in triggering the mechanisms that, when dysregulated, lead to disease. Study of ocular immunology remains as relevant as ever (1). Throughout the last century pivotal scientific discovery has emerged.

Study of ocular immunology remains as relevant as ever.

This includes insights into phagocytosis and cellular immunity from **Metchnikoff**, deeper knowledge of humoral immunity, including discovery of anti-sera and antibodies by **Elrich**. The understanding of self vs non-self from **Burnett** and **Medawar** and the demonstration of immune privilege.

The two arms of immunity are innate (rapid response) and adaptive (selective memory, through T and B cell receptors and MHC/HLA antigens), appreciating both has been essential to explaining how vaccines work, how inflammatory diseases develop, and why transplants are rejected. Such understanding was furthered by **Steinman** and discovery of the dendritic cells and the initiation of immune responses.

CURRENT AND FUTURE INSIGHTS

Prof. Andrew D. Dick FMedSci

Director of UCL-Institute of Ophthalmology
Duke-Elder Chair of Ophthalmology
Faculty of Brain Sciences
Co-Director NIHR BRC Moorfields and UCL-Institute of Ophthalmology
Professor of Ophthalmology University of Bristol

At the time, we believed that the innate response was a rapid response to infection and danger, with little specificity, until Janeway and others described *pathogen-associated molecular patterns (PAMP)* recognised by pattern recognition receptors (PRR).

PAMP are highly conserved structures unique to microbes that are sensed by multiple membrane, cytoplasmic and nuclear receptors (including Toll-like receptors (TLR)). Their role is to generate pro-inflammatory responses to non-self and eradicate the invading micro-organisms.

Bringing together the roles of both the semi-selective innate immunity and highly selective adaptive immunity and taking it further, **Matzinger's Danger Theory** proposes that the *immune system is triggered by tissue or cell damage and stress, not just 'foreign' antigens.*

The richness of these discoveries of cellular and molecular networks of the immune system has led to treatments for IMID entities like non-infectious uveitis, transplantation and regenerative medicine. This includes treatment options used today like calcineurin inhibitors, anti-metabolites and monoclonal antibodies like inhibitors of cytokines, depletors of B and T cells and intracellular pathway inhibitors (eg. JAK-inhibitors).

Understanding how tissues protect themselves while responding to danger, how they recognise self antigens, and the discovery of pathways of immune tolerance is equally vital.

To this end **Sakaguchi's** discovery of the function of T Reg cells was essential to understand how we continuously regulate our immune responses. The notion is that while we need to protect against infection for survival, we require to regulate our immune response to prevent cellular and tissue damage.

This fine balance is influenced by our environment - as a *holobiont*, as well as specific tissue microbiome (for example the gut); all are integral to regulating immunity and T reg biology.

This leads onto a central premise - that the immune system constantly and actively regulates our tissue and cell function (which includes regulating cellular metabolism and autophagy/mitophagy as well as immune response pathways).

Pradeu has generated a *discontinuity theory of immunity* (2) where the response to the stimulus is through detection of antigenic stimuli over time so that the immune system can detect changes and respond accordingly, depending on the acuteness (kinetics) and the level of antigenic stimulus etc. Therefore, *the immune response is graded and proportional* and when antigen levels remain low and chronic, this can lead to immune tolerance, maintaining homeostasis and protecting cells and tissues from the damaging effects of persistent stimulation.

WHAT DOES THIS MEAN IN RELATION TO THE EYE?

The healthy eye possesses immune privilege which means there are anatomical barriers and immune regulatory networks allowing the organ to suppress immune responses against endogenous and exogenous antigens. The blood-retina barrier, cellular mechanisms including regulatory T cells, and cytokine mechanisms, including TGFb and IL-10 all operate to maintain homeostasis. A prevailing notion is that non-infectious uveitis begins with ocular autoantigen presentation via MHC class II molecules, activating naïve T-cells. These naïve CD4+ T-cells differentiate into TH1 and TH17 subsets, then migrate to the retina, where they release pro-inflammatory cytokines (e.g., IFNγ, IL-2, IL-17), triggering a cascade that recruits macrophages and neutrophils, producing the tissue manifestations of uveitis. This may seem reductive, but these concepts have emerged from a broader understanding of immunity and autoimmunity. As the field progresses, keeping immunology central will be essential to advancing both diagnosis and therapy. A clear example is how deeper insight into ocular immunology has allowed the field to harness the benefits of anti-TNF therapies, reducing the steroid burden placed on patients.

The understanding of our innate and adaptive immunity along with the insights from human genomic sequencing has unveiled the genetic and molecular basis of autoimmunity and autoinflammatory disorders. An example of this is the discovery of genetic variants of the disorders of innate immunity giving rise to monogenetic autoinflammatory disorders(4). These disorders are systemic syndromes that may also have ocular involvement (e.g Blau's syndrome). The mutations in pathways of innate immune activation bring further understanding of immune pathways for more common disorders as well as help develop more specifically targeted therapies.

With all this information, how does immunology facilitate management and treatments in the future? One way this will help is to move away from a phenotypic and anatomical naming of disease entities to a molecular and cellular specific nomenclature. This will be increasingly possible with the advent of exquisitely sensitive bioinformatic assisted single cell multi-omic platform analysis on biopsy samples such as aqueous or vitreous (5). This will not only support the development of new therapies, but also strengthen the case for repurposing the wide range of biologics and small molecule agents already used in treating IMIDs with a greater likelihood of success in non-infectious uveitis.

Finally, a conundrum that remains to be answered is the role of infection either as a cause of inflammation or exaggeration of autoinflammatory and autoimmune ocular inflammation (6). Metagenomic analysis will help us better define and diagnose the direct aetiological role of infectious agents. Additionally, the field is now advancing in understanding how a dysregulated tissue as well as gut microbiome may dysregulate T regulatory cells or promote T cells that cause uveitis. This is at least experimentally plausible, and the field is now moving toward greater understanding of these mechanisms in humans paving the way for a new class of treatments.

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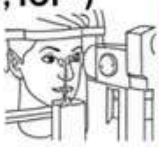
NEW INTERNATIONAL RECOMMENDATIONS FOR UVEITIC GLAUCOMA

RECOMMENDED GLAUCOMA-RELATED TESTING AND TIMING

Eye Pressure Check (Tonometry, IOP*)



- At every visit
- After initiating corticosteroids

IOP: Intraocular Pressure

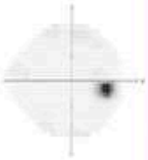
Drainage Angle Exam (Gonioscopy)

- At initial uveitis work-up
- Biannually, or if any significant change in inflammation

OCT RNFL Imaging (Optic Nerve)

- At initial IOP elevation work-up or as a baseline
- Biannually or if any suspected VF changes or poorly controlled IOP

Visual Field (VF)



- At initial IOP elevation work-up
- Every 3-6 months (active disease)
- Every 6-12 months (quiet disease)

Fundoscopy with photography*

- At initial IOP elevation work-up, to establish a baseline
- Every 6-12 months

Optic nerve disc color photographs

RECOMMENDED FREQUENCY FOR IOP MONITORING AFTER STARTING CORTICOSTEROIDS

Eye Drops

- IOP check **1-2 weeks** later
- 8-12 weeks later, sooner if increase in dosing
- If IOP-lowering therapy initiated or IOP is uncontrolled: **every 1-4 weeks** until IOP <16 mmHg, then every 3-6 months

Subtenon or Suprachoroidal Triamcinolone (Kenalog® or Xipere®)

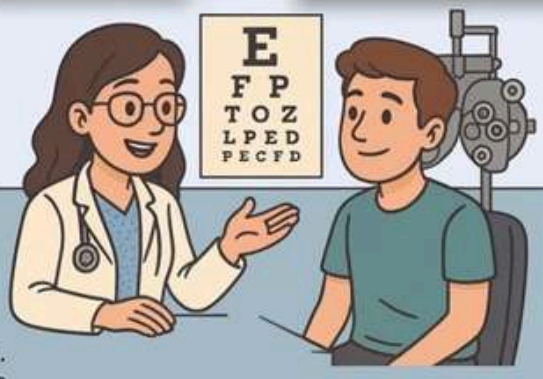
- IOP check **1-4 weeks** later, depending on your history of IOP spikes
- If IOP-lowering therapy initiated or IOP is uncontrolled: **every 1-4 weeks** until IOP <16 mmHg, then every 3-6 months

Intravitreal Injections and Implants

- IOP check **1 week** later for Kenalog® or **2 weeks** later for Illuvien®, Retisert® and Ozurdex®
- Again 2 weeks later, then every 4 weeks (months 2-6 or longer)

Important Reminders

- Target IOP is **below 16 mmHg** to better protect vision
- Consult with your eye doctor or specialist promptly for any questions or concerns

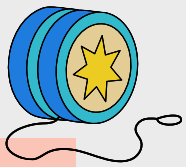


Uveitic Glaucoma Interest Group Recommendations for Uveitis-Related Ocular Hypertension and Glaucoma Management (2025). *Ocular Immunology and Inflammation.* doi: 10.1080/09273948.2025.254228

For more information about uveitic glaucoma or for further details on the new recommendations visit: **www.uveiticglaucoma.com**

FROM OZURDEX TO ILUVIEN

THE EVOLUTION OF INTRAVITREAL CORTICOSTEROID IMPLANTS IN UVEITIS



Non-infectious posterior uveitis is a chronic, relapsing disease in which treatment success is not just about quelling inflammation today, but on preventing the next flare while limiting cumulative steroid burden and clinic visits. Local therapy offers high intraocular drug levels with minimal systemic exposure. The evolution of intravitreal corticosteroid implants over the past two decades reflects a sustained effort to optimise the balance between efficacy, duration, and safety.

Before the modern implant era, local therapy relied on off-label intravitreal **triamcinolone** injections. While effective, the drug washed out within approximately three months, creating a clinical "yo-yo" effect - a therapeutic peak followed by inflammatory rebound. Repeated relapses led to cumulative photoreceptor damage and permanent visual compromise.

Sustained-release implants were engineered specifically to flatten this curve.

RETISERT® AND THE SURGICAL ERA

The **FIRST** sustained-release intravitreal corticosteroid implant

- 0.59 mg fluocinolone acetonide device
- Approved in 2005

Undeniably effective at reducing recurrence (from 40-54% in the 34 weeks before implantation to just 7-14% afterwards)

But required surgical implantation via pars plana sclerotomy

And carried a formidable adverse-effect profile, namely:

- near-universal cataract formation
- high rates of IOP elevation requiring topical therapy (50%), and a substantial proportion of patients needing glaucoma filtration surgery (35%).

OZURDEX® AND THE SHIFT TO INJECTABLE DELIVERY

- Delivered via a **22-gauge** injector
- With a biodegradable poly(lactic-co-glycolic acid) matrix
- Releasing dexamethasone over approximately **4–6 months**
- With peak pharmacological effect in the first 8–12 weeks.

- The **HURON trial** demonstrated significant improvements in vitreous haze clearance and visual acuity gains compared with sham, with benefits persisting through 26 weeks.
- The **POINT trial** subsequently confirmed non-inferiority to intravitreal triamcinolone and superiority to periocular triamcinolone for uveitic macular oedema,
- The **MERIT trial** demonstrated superiority over both intravitreal methotrexate and intravitreal ranibizumab for persistent or recurrent uveitic macular oedema.

The side-effect profile represents a marked improvement over Retisert®:

IOP elevation and cataract rates are substantially lower, and the vast majority of IOP rises are managed with topical therapy alone (Table 1).

Ozurdex's relatively short duration is both its strength and its limitation. It is well suited to acute flares, bridging while systemic immunosuppression takes effect, and as a diagnostic "steroid trial" to confirm an inflammatory component. However, for chronic relapsing disease, repeated injections every few months introduce cumulative procedural risk, treatment burden, and recurrent drug-concentration troughs.

ILUVIEN® AND SUSTAINED MICRO-DOSE DELIVERY

This fluocinolone acetonide (FAc) intravitreal insert addresses the chronicity gap!

ILUVIEN® (Alimera Sciences), originally licensed for diabetic macular oedema, delivers **0.25 µg/day of FAc** at initial release for up to **36 months** from a non-biodegradable polyimide tube injected through a **25-gauge needle**.

Phase 3 data demonstrated reductions in recurrence rates and adjunctive treatment requirements compared with sham, with significantly longer median time to first recurrence and lower rates of clinically meaningful vision loss.

12-month uveitis recurrence rates were 38% with ILUVIEN® versus 98% with sham.

The side effect profile reflects the trade-off inherent in sustained steroid delivery.

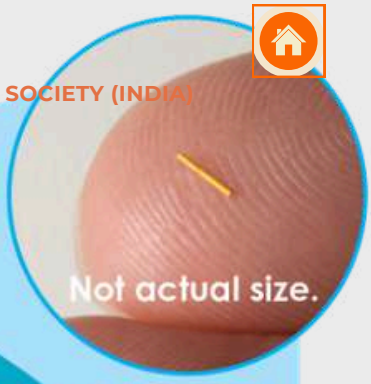
Cataract surgery remains near-certain in phakic eyes and requires explicit pre-treatment counselling.

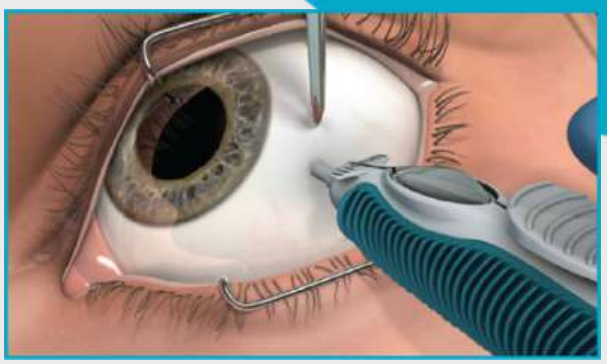
However, the IOP profile is markedly better than Retisert®, with low rates of IOP-lowering surgery and comparable mean IOP between treated and sham groups at 36 months (Table 1).

Glaucoma defined by optic nerve damage has not been systematically reported in the FAc insert trials, though the MUST Trial's cautionary long-term data with Retisert® underscore the need for indefinite IOP surveillance with any intravitreal steroid.

ILUVIEN®

(fluocinolone acetonide intravitreal implant) 0.19mg





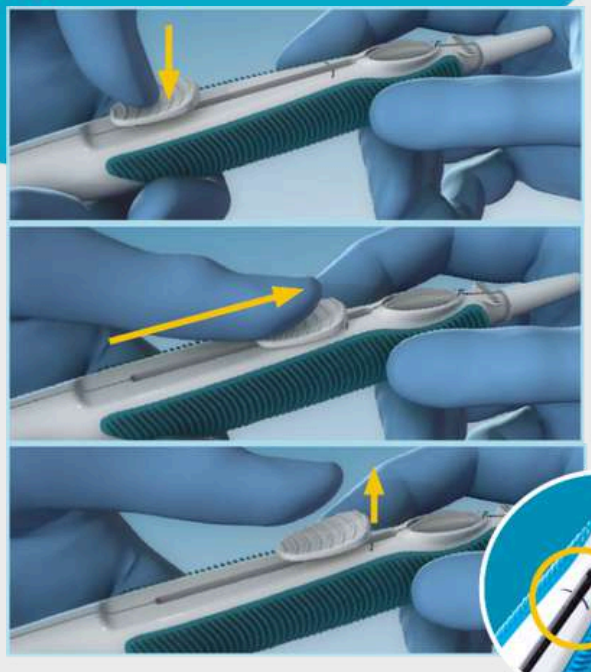


TABLE 1: COMPARISON AT A GLANCE

	Ozurdex®	ILUVIEN®
Indication	Treatment of adult patients with non-infectious uveitis affecting the posterior segment of the eye	Prevention of relapse in recurrent non-infectious uveitis affecting the posterior segment of the eye
Active agent	Dexamethasone 0.7 mg	Fluocinolone acetonide 0.19 mg
Duration	~4–6 months	Up to 36 months
Biodegradable	Yes (PLGA matrix)	No (polyimide tube)
Delivery	22G injector	25G injector
Peak effect	Weeks 4–8	Steady low-dose release (0.25 µg/day initial)
Pivotal trial efficacy	47% vitreous haze = 0 at wk 8 vs. 12% with sham (HURON trial)	28% recurrence at 6 mo vs 91% recurrence with sham (Phase 3) 38% recurrence at 12 mo vs 98% recurrence with sham (Phase 3)
Best suited to	Acute flares, bridging, steroid trial, UME	Chronic relapsing posterior uveitis
IOP elevation (⩾25 mmHg)	~7% (HURON); ~21% (systematic review)	~24% (Phase 3)
Glaucoma surgery	0% (HURON and multicentre cohort)	5.7% at 3 years (Phase 3), 2% at 3 years (Indian replication study)
Cataract surgery (phakic)	5% at 26 wks; cumulative with repeat Tx	~74% over 3 years
Re-treatment	Repeat injection every 3–6 months	Rarely needed within 3 years

Clinical Perspective

In practice, the choice between intravitreal corticosteroid implants is often best understood as a treatment sequence rather than a binary decision. In eyes with active non-infectious posterior segment uveitis, short-acting therapy with a dexamethasone implant may first be used to induce quiescence, reduce macular oedema, and assess corticosteroid responsiveness in a reversible way. This includes not only the anatomical and inflammatory response, but also tolerability, particularly intraocular pressure behaviour. In eyes that relapse after one or two dexamethasone implants, or in eyes with a known favourable prior dexamethasone response in whom recurrence is anticipated, escalation to long-acting FAc becomes a logical next step to maintain, control and reduce the burden of repeated procedures and visits. This approach reflects current expert practice, while recognising that direct comparative evidence between repeated dexamethasone implantation and earlier fluocinolone use remains limited.

Lens status should sit near the centre of this decision-making. Pseudophakic eyes, and eyes with pre-existing cataract or planned cataract surgery, are generally more straightforward candidates for long-acting FAc. Systemic context also matters. Local corticosteroid implants are not universal substitutes for systemic immunosuppression. In bilateral symmetrical disease, disease with active systemic involvement, or phenotypes in which deeper choroidal inflammation is prominent, systemic therapy often remains the mainstay of treatment, with local implants used as adjuncts to improve ocular control. In contrast, unilateral disease, bilateral asymmetrical disease, or posterior segment uveitis without systemic involvement may be particularly suitable settings for implant-led local strategies.

Underpinning all of this is the MUST Trial lesson: long-term follow-up showed that systemically treated eyes fared better than the Retisert® cohort by 1.4 lines of acuity at seven years, with fewer ocular complications. Whether lower-dose implants like FAc compare more favourably remains unknown. Local corticosteroid implants are best understood not as replacements for systemic immunosuppression, but as powerful adjuncts within a broader treatment strategy.

Dr. Panayiotis Maghsoudlou
NIHR Academic Clinical Lecturer in Ophthalmology, University of Bristol
Visiting Lecturer, University of Bath
Chair, ALSPAC Law & Ethics Committee

ADVISE TRIAL

A VISUAL SUMMARY

Standard management of non-infectious intermediate, posterior and panuveitis involves oral corticosteroids as first line agent to control acute inflammation. However, due to their well known long-term toxicities, conventional immunosuppressive drugs are important to achieve successful corticosteroid discontinuation. Commonly used initial immunosuppressive drugs to treat uveitis include antimetabolites like methotrexate, azathioprine and mycophenolate mofetil. It often requires one or more drugs to achieve long-term remission of uveitis and combination therapy is usually initiated with addition of calcineurin inhibitors like cyclosporine and tacrolimus.

Adalimumab, a fully humanized, anti-tumour necrosis factor α (anti-TNFα) monoclonal antibody, received FDA approval for use in adult non-infectious uveitis in 2016 and in children aged 2 and above in 2018.

The ***ADVISE*** **Trial (*AD*alimumab *Vs*. conventional *I*mmuno*S*uppr*E*ssion for uveitis Trial)** is a

- multicentre randomized comparative trial
- comparing adalimumab with other conventional immunosuppressive drugs
- in achieving corticosteroid sparing in treatment of non-infectious, intermediate, posterior, and panuveitis.

Primary aim: To compare the effectiveness of adalimumab and conventional immunosuppressants in achieving *steroid sparing* effect (ie inactive uveitis with prednisone ⩽7.5 mg/day or equivalent for two consecutive visits ⩾28 days) within the first *6 months*.

Secondary aim: To measure *steroid sparing* effect in the two groups at *12 months* as well as complete *steroid discontinuation* (ie inactive uveitis without oral steroids for two consecutive visits ⩾ 28 days within *6 months* as well as within *12 months*.

Inclusion criteria

- **Age ⩾13 years**
- **Active or recently active (<60 days) non-infectious uveitis**
- **Receiving prednisone (or prednisolone) of >7.5 mg/day**
- **Anticipated increase in the dosage of prednisone to >7.5 mg/day.**

Exclusion criteria

- **Active/latent TB (untreated)**
- **MS or demyelinating disease**
- **Behçet disease**
- **Concurrent use of ⩾2 CIDs**
- **Long-acting intravitreal steroid implant (⩽3 yrs)**
- **Anti-TNF-α therapy (⩽60 days)**
- **Prior adalimumab failure/intolerance**
- **Pregnancy or lactation**

227 patients

STRATIFIED RANDOMIZATION

based on
1. Number of systemic immunosuppressives at baseline AND
2. Baseline prednisone dose

Immunosuppressive = 0 Oral steroids <30 mg	Immunosuppressive = 0 Oral steroids ⩾30 mg	Immunosuppressive = 1 Oral steroids <30 mg	Immunosuppressive = 1 Oral steroids ⩾30 mg

	ADALIMUMAB (n = 114)	**CONVENTIONAL IMMUNOSUPPRESSIVES (n = 113)**	
Successful corticosteroid sparing at 6 months	69%	54%	(P = 0.029)
Successful corticosteroid sparing at 12 months	86%	77%	(P = 0.077)
Successful corticosteroid discontinuation at 12 months	55%	40%	(P = 0.028)

PRACTICE PEARLS

- Adalimumab enables faster corticosteroid sparing and a higher chance of complete steroid discontinuation within 12 months.
- Fewer cataract surgeries were needed with adalimumab compared to conventional immunosuppressive regimens. However, the percentage of pre-existing lens opacities or cataract was also higher in the conventional immunosuppressive drugs group compared to adalimumab.
- Overall systemic side effects were rare in both the groups. Discontinuation due to toxicity was observed with conventional immunosuppressives, although the number of patients discontinuing treatment was very few.
- Stepwise dose escalation, regimen heterogeneity and higher loss to follow-up with conventional immunosuppressives may have delayed or diluted its apparent efficacy. A short follow-up period of 12 months might also be a limitation considering the long-term efficacy of these drugs.
- This study was an open-label design, study. Staff and participants were unmasked to treatment routes. Differences in center-based assessments in both the groups was another limitation adding to the bias.
- Anti-adalimumab antibodies and the role of combination immunosuppression were not addressed in this study.

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Medical Retina and Uvea Consultant
Cataract Surgeon
Madhav Netralaya
Nagpur

A song of eyes and fire

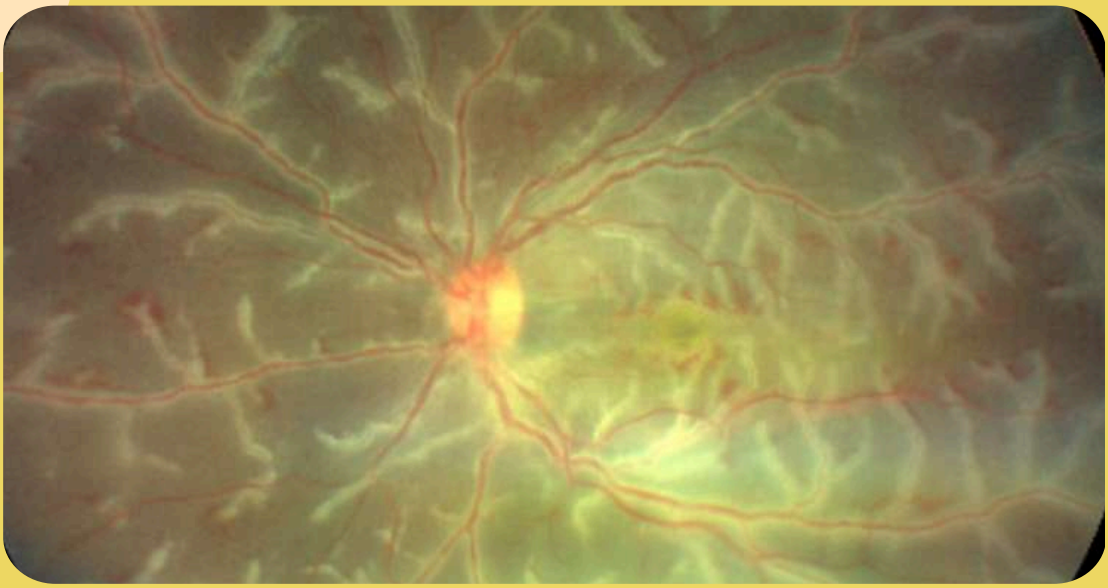
Dr. Madhuvanthi Mohan
Rajan Eye Care Hospital
Chennai

TEST YOUR UQ!

UVEITIS QUOTIENT

Dr. Anu Jospeh
Consultant
Uvea Services
Aravind Eye Hospital, Coimbatore

1 Identify the retinal manifestation



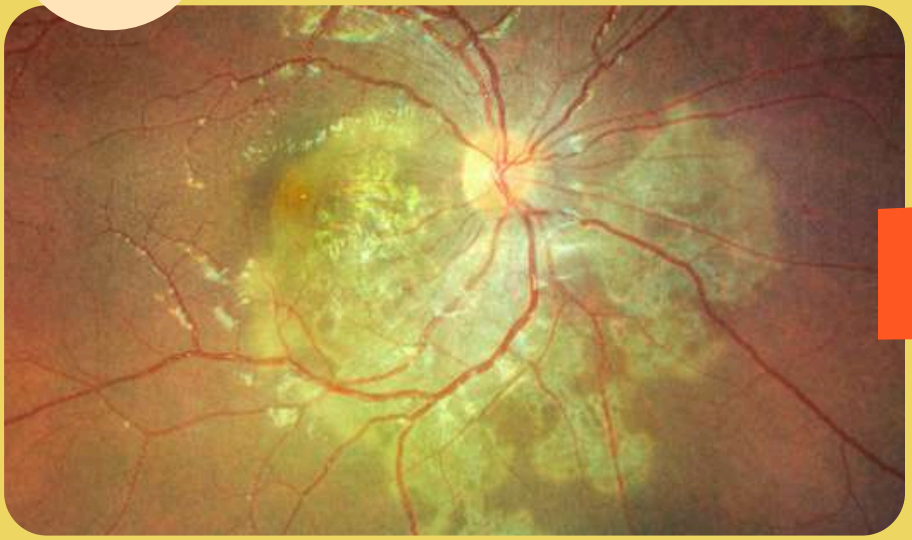
FROSTED BRANCH ANGITIS

An acute panuveitis with severe vasculitis (veins > arteries) affecting the whole retina. It is also called **diffuse acute retinal periphlebitis**.

Originally described in Japanese literature by Ito in 1976. It can be an idiopathic disorder or can be associated with ocular and systemic diseases.

Most common ocular associations include CMV retinitis, HIV retinitis and toxoplasma chorioretinitis. It has been described in systemic disorders like SLE, Crohn's disease, acute lymphocytic leukemia etc. Prompt corticosteroid response is a characteristic feature.

2 Name the multimodal imaging technique best to monitor this disease activity

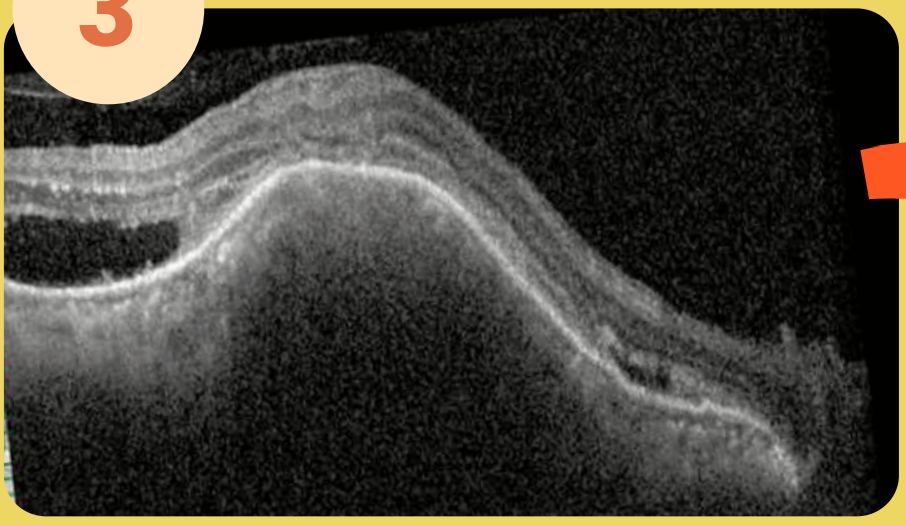


FUNDUS AUTOFLUORESCENCE

To diagnose, map and monitor disease activity in serpiginous choroiditis.

Active lesions show hyperautofluorescent border.
Inactive lesions show hypoautofluorescent dark areas reflecting irreversible atrophy of the RPE.

3 Identify the OCT sign

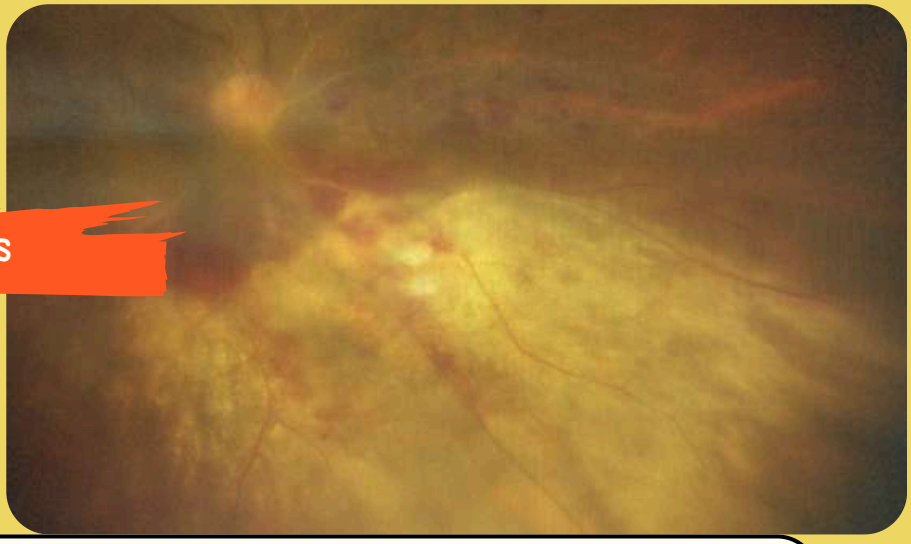


CONTACT SIGN

An adhesion between the outer retina and the retinal pigment epithelium over a choroidal lesion, often indicating a choroidal tuberculoma.

A dome shaped lesion is often seen beneath the contact sign and maybe accompanied by subretinal fluid. Monitoring this OCT sign helps track the tuberculoma's response to systemic anti-tuberculosis treatment.

4 Identify the disease



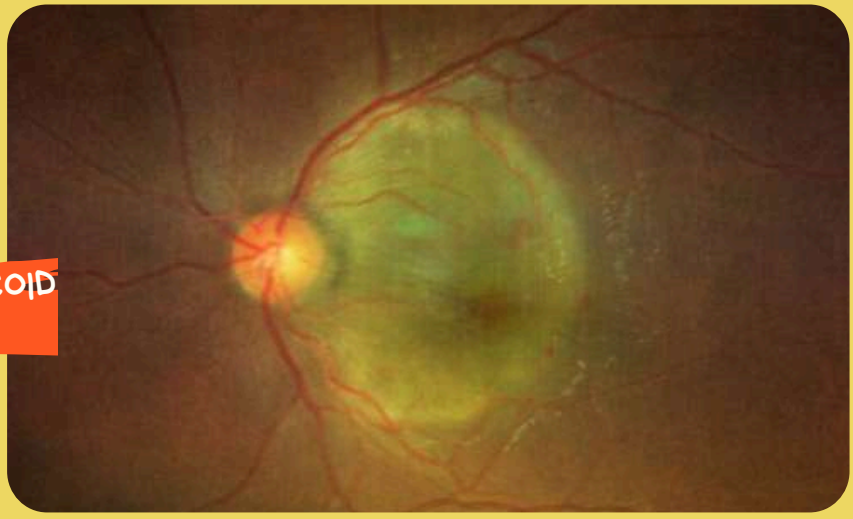
CYTOMEGALOVIRUS RETINITIS

Appears as yellowish white lesions with hemorrhages and can resemble a “pizza pie” with cottage cheese and ketchup.

On fundus examination, the lesions are mostly wedge shaped with apex pointing towards the optic disc. It can also involve the periphery, characterised by a “granular” appearance and without hemorrhages.

The choice of systemic agent is oral valganciclovir (CMV DNA polymerase inhibitor).

5 Identify this classic lesion



ACUTE SYPHILITIC POSTERIOR PLACOID CHORIORETINOPATHY

Round/oval, yellowish-white placoid lesions in the macular or paramacular region.

OCT shows outer retinal disruption (ellipsoid zone loss), nodular RPE thickening (*pitchfork sign* of elevated granular hyperfluorescent RPE lesions) and subretinal fluid.
ICGA shows characteristic hypocyanescence.
FFA shows "leopard spot" hyperfluorescence.
FAF shows shows hyper autofluorescence and is considered one of the most useful imaging modality to detect even the most subtle lesion.

EYE HUMOUR

HAHAHA

Resident: "How can you tell it's a granulomatous uveitis?"
Senior: "Simple. It comes with granuloma-level drama."

Waiter: "What will you have today, doctor?"
Retina specialist: "Just whatever's well pigmented with a smooth finish and no macular holes, please."

Fellow: "I think I'm finally understanding uveitis."
Uveitis: "Cute."

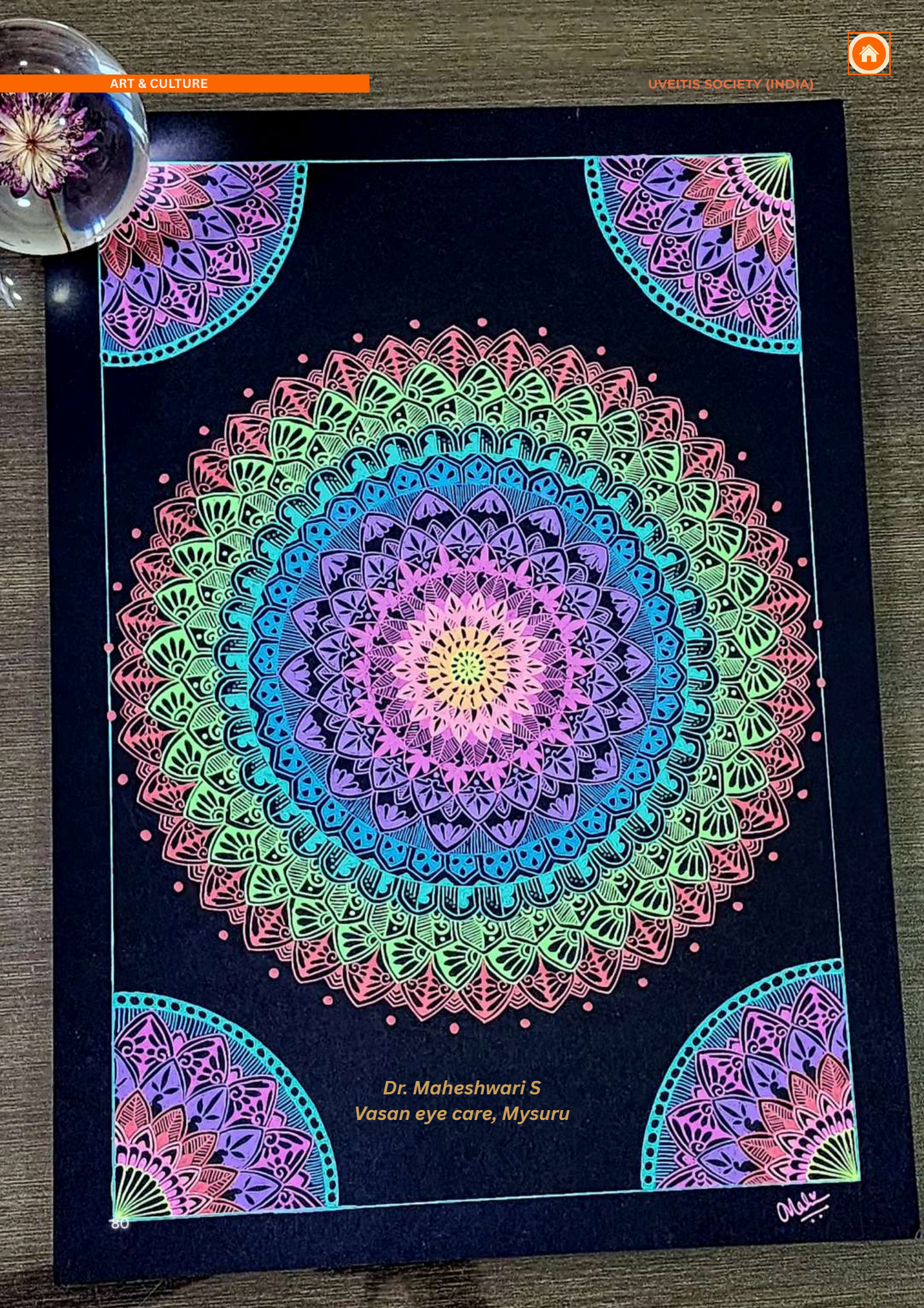
Resident: "I think it's TB uveitis."
Consultant: "Excellent. That's the diagnosis 34% of ophthalmologists make!

Patient: "I hope you don't mind, doctor... I googled my symptoms."
Doctor: "I don't mind at all. I also googled my car problem last week.
Both of us regretted it."

Fellow: "Sir, there's a new patient with pain, photophobia, and redness."
Consultant (checking watch):
"Of course! The classic Friday-evening uveitis."

"Everything is fine, doc."
— said while blinking 64 times per minute with a 3+ cell reaction.

The Fellow's Life
"My hobbies include:
- Seeing flare
- Treating flare
- Explaining flare
- Getting into flare
Basically... I live in a flare cycle."



Dr. Maheshwari S
Vasan eye care, Mysuru

Healing with Heart

In rooms where silence speaks so loud,
Where hope feels lost within the crowd,
I've seen brave hearts, though worn and tired,
Still rise again, still feel inspired.

To every patient, strong yet frail,
Who walks through storms, who does not fail—
Your courage whispers, soft and true,
"There's light ahead. Keep walking through."

To caregivers with gentle hands,
Who hold on tight, who firmly stand,
Your quiet love, both fierce and kind,
Heals wounds the world may never find.

And to the girl I used to be,
With sleepless eyes and big dreams free,
Through endless nights and doubts so deep,
You chose to rise, you chose to leap.

You asked, "Am I enough to stay
Strong in this long and winding way?"
But look at you — still here, still whole,
Still healing hearts, still healing souls.

To colleagues walking side by side,
In shared fatigue, in growing pride,
This journey shines a little more
Because of you — as your kindness soar.

And to my parents, friends so dear,
Who stayed when paths were unclear,
Your faith became my guiding light,
My strength through every fragile night.

Through tired eyes and hopeful sighs,
Through whispered prayers beneath the skies,
One truth remains, both soft and strong:
We heal with love, not skill alone.

And in this life of tests and trials,
Of heavy hearts and fragile smiles,
May compassion shape the way we see—
In every "you" and every "me."

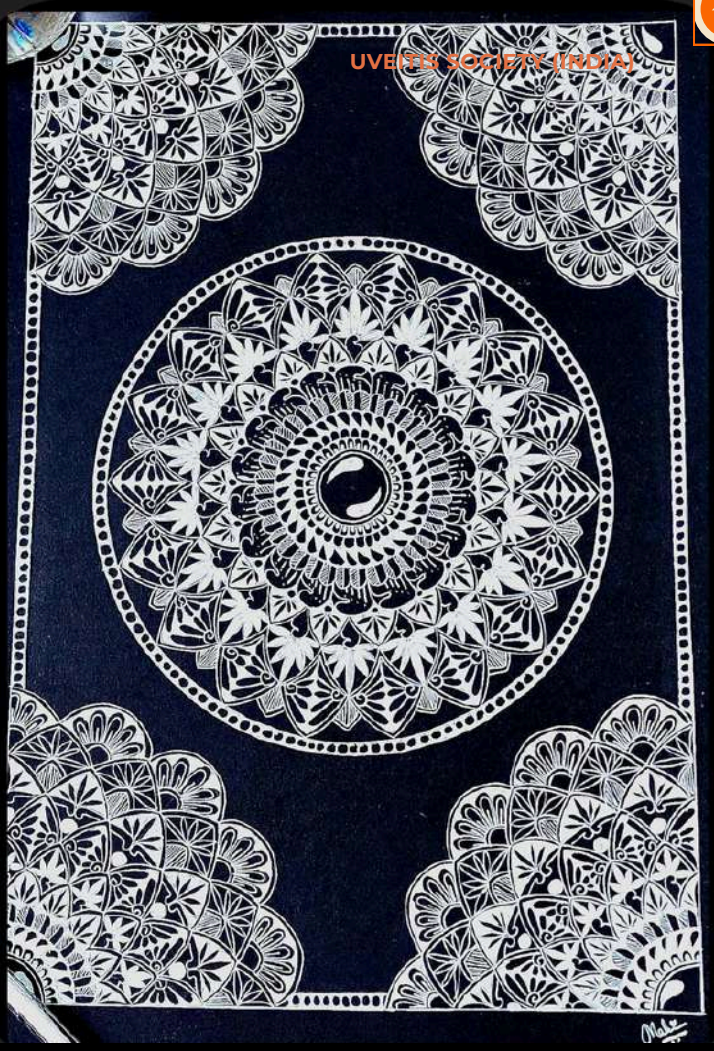
Let kindness lead where we may go,
In quiet acts, in care we show.
For when we choose to stay and care,
We light the world — just by being there.

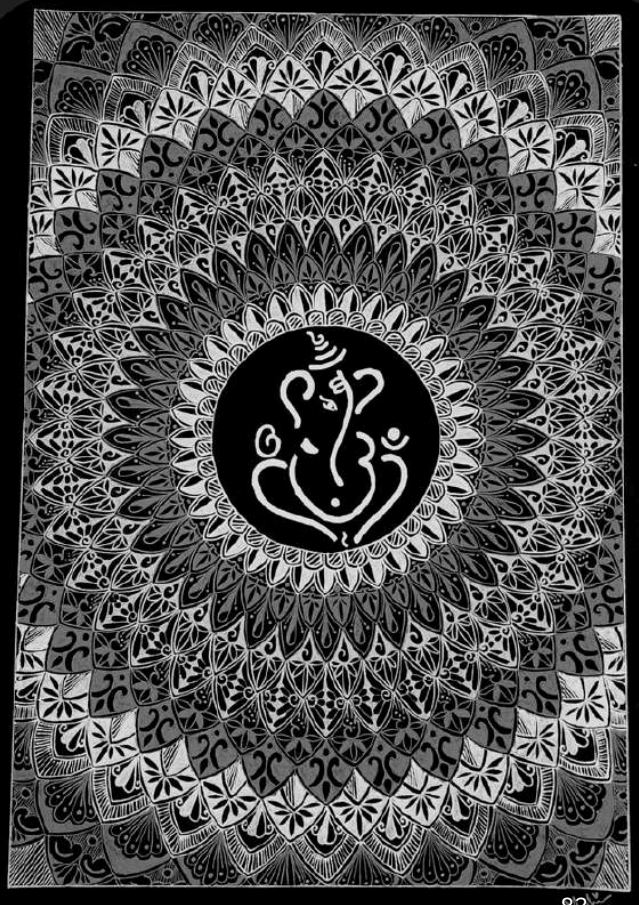
Dr. Vaibhavi Noticewala
Nirvana Netralaya
Superspeciality Retina & ROP Clinic

Meditation thr

Dr. Maheshwari S
Vasan eye care, Mysuru

With its circular symmetry and rhythmic patterns, it calms the mind, sharpens focus, and invites inner stillness. Each stroke becomes a quiet moment of balance, where art turns into a peaceful, mindful journey.





ough Creativity

A Home Divine

Dear God,
Give vision to my eyes,
for they do not see.
Give sound to my ears,
for they do not hear
Give touch to my skin,
for it seldom feels,
Give smell to my nose,
for it doesn't discern.
Give wisdom to my intellect,
And clarity to my mind,
Give sense to my senses;
And humbleness to my ego.
Make these flesh, bones and heart;
A temple of thine, a home divine.

Dr. Anup Kelgaonkar
LV Prasad Eye Intitute

Take me Home

A poem written during the tough years of residency, amidst the exhaustion and overwhelm, home is not just a place, its also the people and the experiences that make you feel safe.

Going home feels so right
It's like you've got invisible wings
Everything you've been holding in so tight
You can now let go of and fly like a kite.

Home is a place you can, of your favorite food, take an extra bite
And sleep stress-free through the entire night
It's a place your parents are always in sight
And where every hurdle can be conquered with daddy's might.

The place you'd love to do the house hold chores
And even enjoy feeling bored
And into your favourite "large mug" pour
The extra chocolate that was, specially for you, in store.

Where fighting can make you feel right
You don't have to think twice and keep quiet
Because your friends are the guiding light
Through your dark nights and sorry plights.

The anticipatory whiff of freshly brewed hot coffee
And the signature taste of mama's hand made toffee
The warmth that comes from touching your home's cold floor
And the hidden blessings you realize in visiting your favourite ice cream store
Leaves you asking for nothing more.

Dr. Kelinl Saolapurkar
Shraddha Eye Care Trust
Nethradhama Eye Hospital

From Sight to Life

AN OPTOMETRIST'S DEEPAWALI RANGOLI

The Rangoli was made by our Optometrist, Ms. Sadhana Kamble for Deepawali 2025; with contributions from our staff at Taparia Institute of Ophthalmology, Bombay Hospital Institute of Medical Sciences, Mumbai including Mrs. Suman Gangawane, Mrs. Shivani Abhishek Patil, Mr. Sujeet Wadkar, Mr. Ram Naresh Ram, Ms. Trupti Khaladkar, Ms. Jyoti Dodhi, Dr. Vaishnavi Dubale and Dr. Zahra Parekh

Dr. Mayur R. Moreker
Bombay Hospital Institute of Medical Sciences, Mumbai

My anchor as a new Mom

It's been a whole year
Of many tears, of some new fears, of losing touch with peers
A phase of life's changing gears.

A year of deeper understanding, of closer bonding, of self finding
And my flawed ways minding

A year of letting go, of going slow
A year that tested my grind
The year I became more kind
The year I saw a new person in my husband, indeed a very rare find!

It's been the hardest year yet adapting to these rapid changes ,
Yet the one thing that has remained constant through all the phases,
has been the safety and comfort you always seem to find in our embrace,
And the faith you put in us through your loving gaze

Through life's fast pace
These moments we shall always chase!
Happy birthday, my dearest!

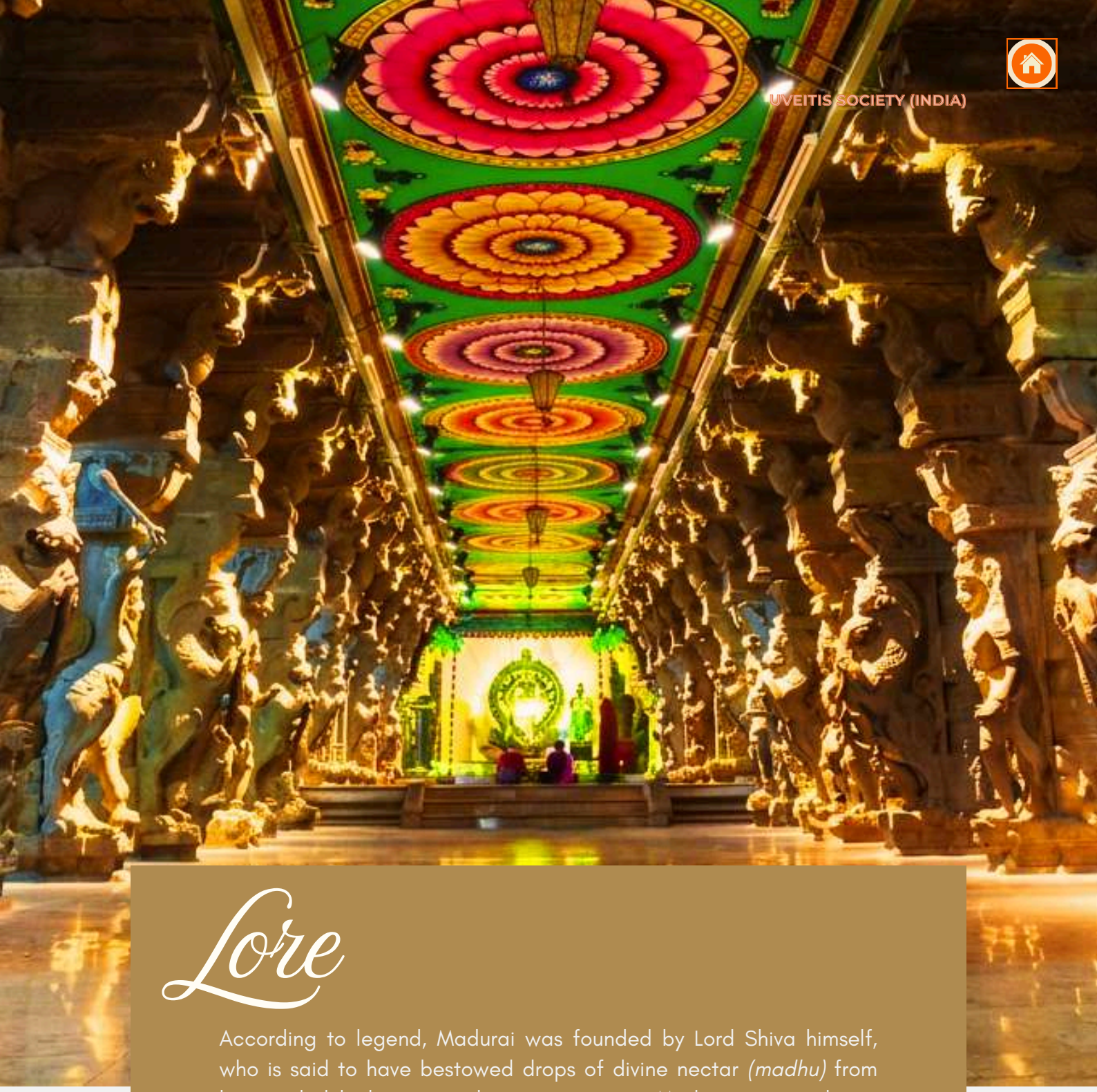
Dr. Kelinl Saolapurkar
Shraddha Eye Care Trust
Nethradhama Eye Hospital

MADURAI

WHERE HERITAGE MEETS HEALING

Dr. Lisa Sunny
Consultant
Uvea Services
Aravind Eye Hospital, Madurai

Dr. Rajshree Ezhilan
Consultant
Uvea Services
Aravind Eye Hospital, Madurai



Lore

According to legend, Madurai was founded by Lord Shiva himself, who is said to have bestowed drops of divine nectar *(madhu)* from his tangled locks, giving the city its name. Madurai was earlier a forest called *Kadambavanam*. Once, a merchant passing through the forest saw *Indra*, the King of Gods, worshipping a *Swayambhulingam* under a *Kadam* (burflower) tree. This was reported to *King Kulasekara Pandya*, who cleared the forest and built the magnificent *Sri Meenakshi Sundareswarer Temple* around the holy *lingam*.

A beautiful lotus-shaped city grew around the temple. Lord Shiva is believed to have appeared at the naming ceremony and blessed the city, following which it came to be known as "*Madhurapuri*."

Historically, Madurai rose to prominence as the seat of the *Pandya* dynasty, which patronized religion, literature, and the arts. Later rulers, including the *Nayakas*, added their own architectural and cultural layers, shaping the richly textured city seen today.

Spirituality

Madurai is synonymous with the renowned *Meenakshi Amman Temple*, a masterpiece of Dravidian architecture. Dedicated to *Goddess Meenakshi (Parvati)* and *Lord Sundareswarar (Shiva)*, the temple is famous for its towering *gopurams* adorned with thousands of colourful sculptures and the iconic *Aayiram Kaal Mandapam* (Hall of 1000 Pillars). It remains a vibrant hub of devotion, attracting thousands of devotees every day.

Ancient literature narrates the tale of *King Malayadhwaja Pandya* and *Queen Kanchanamalai*, who performed a *yajna* seeking a successor. Instead, a three-year old daughter with three breasts emerged from the flames. A divine voice foretold that she would lose the third breast upon meeting her destined husband. Raised as a warrior queen, she later met Lord Shiva in the form of *Sundareswarar* and assumed her true form as *Goddess Meenakshi*.

The iconic *St. Mary's Cathedral* and the historic *Goripalayam Dargah* further reflect the city's rich religious diversity.

The *Vandiyur Mariamman Teppakulam* in Madurai is a massive 16-acre, 17th-century temple tank built by *King Thirumalai Nayaka* in 1645. Located about 5 km from the *Meenakshi Amman Temple*, it features granite steps on all sides and a central *Mandapam* with a Ganesh temple.

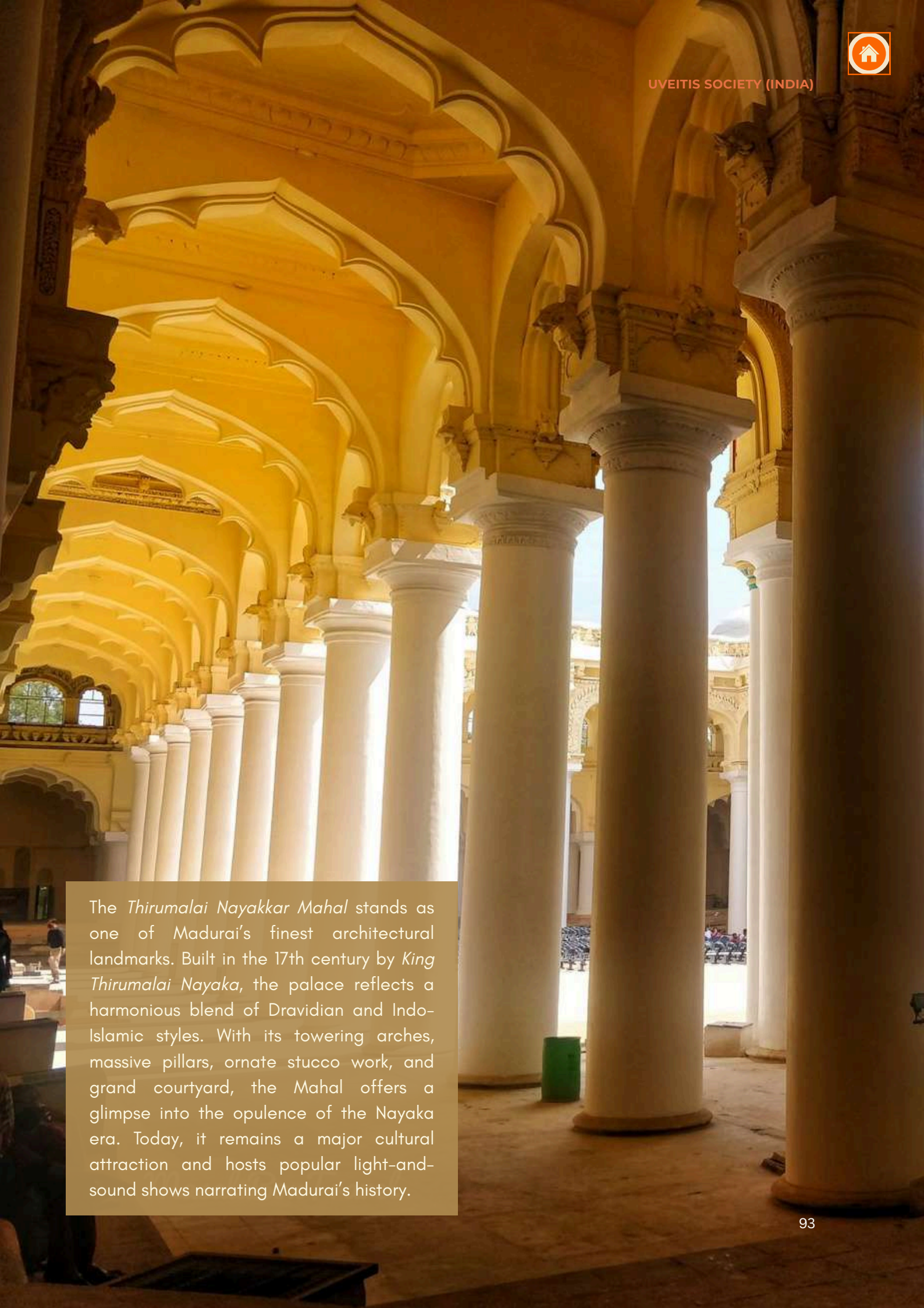
Cradle
OF TAMIL ART & LITERATURE

Madurai holds a revered place in Tamil cultural history. According to tradition, it was the seat of the Tamil Sangam, where poets and scholars composed and discussed literature under royal patronage. Tamil literature flourished here, shaping linguistic and philosophical traditions that endure to this day.

The city is also deeply connected to classical art forms. *Bharatanatyam* finds both historical roots and contemporary expression in Madurai's temples and institutions. Carnatic music continues to thrive, especially during festivals and temple celebrations.

Culinary
HERITAGE

The celebrated Madurai biryani, made with *seeraga samba* rice, is light in grain yet rich in aroma. Street food culture is vibrant and theatrical, featuring flaky *parottas* with spicy salna, sizzling *kothu parotta*, and snacks like *bonda*, *bajji*, and *sundal*. Among the city's most beloved icons is *Jigarthanda*, a unique cooling dessert drink that originated here.



The *Thirumalai Nayakkar Mahal* stands as one of Madurai's finest architectural landmarks. Built in the 17th century by *King Thirumalai Nayaka*, the palace reflects a harmonious blend of Dravidian and Indo-Islamic styles. With its towering arches, massive pillars, ornate stucco work, and grand courtyard, the Mahal offers a glimpse into the opulence of the Nayaka era. Today, it remains a major cultural attraction and hosts popular light-and-sound shows narrating Madurai's history.

Healthcare & EDUCATION

The city is home to renowned institutions such as Madurai Medical College, Government Rajaji Hospital, and several leading private hospitals and specialty centres that serve patients from across the region.

A defining feature of Madurai's healthcare landscape is the presence of the *Aravind Eye Care System*, founded in 1976 by *Dr. Govindappa Venkataswamy* (Dr. V). Headquartered in Madurai, Aravind has grown into one of the world's largest and most influential eye care networks. Through its innovative high-volume, high-quality, and affordable model, it has transformed the delivery of ophthalmic services and played a pivotal role in reducing avoidable blindness in India and across many developing nations.

What sets Madurai apart is not merely the age of its monuments or the grandeur of its festivals, but the way tradition seamlessly flows into everyday life. Flower sellers string garlands at dawn for temple rituals, classical music drifts through streets during festivals, and age-old recipes are recreated in modest kitchens and bustling eateries.

In Madurai, heritage is not merely remembered — it is *practiced, celebrated, and savoured* every day.

WARM Welcome

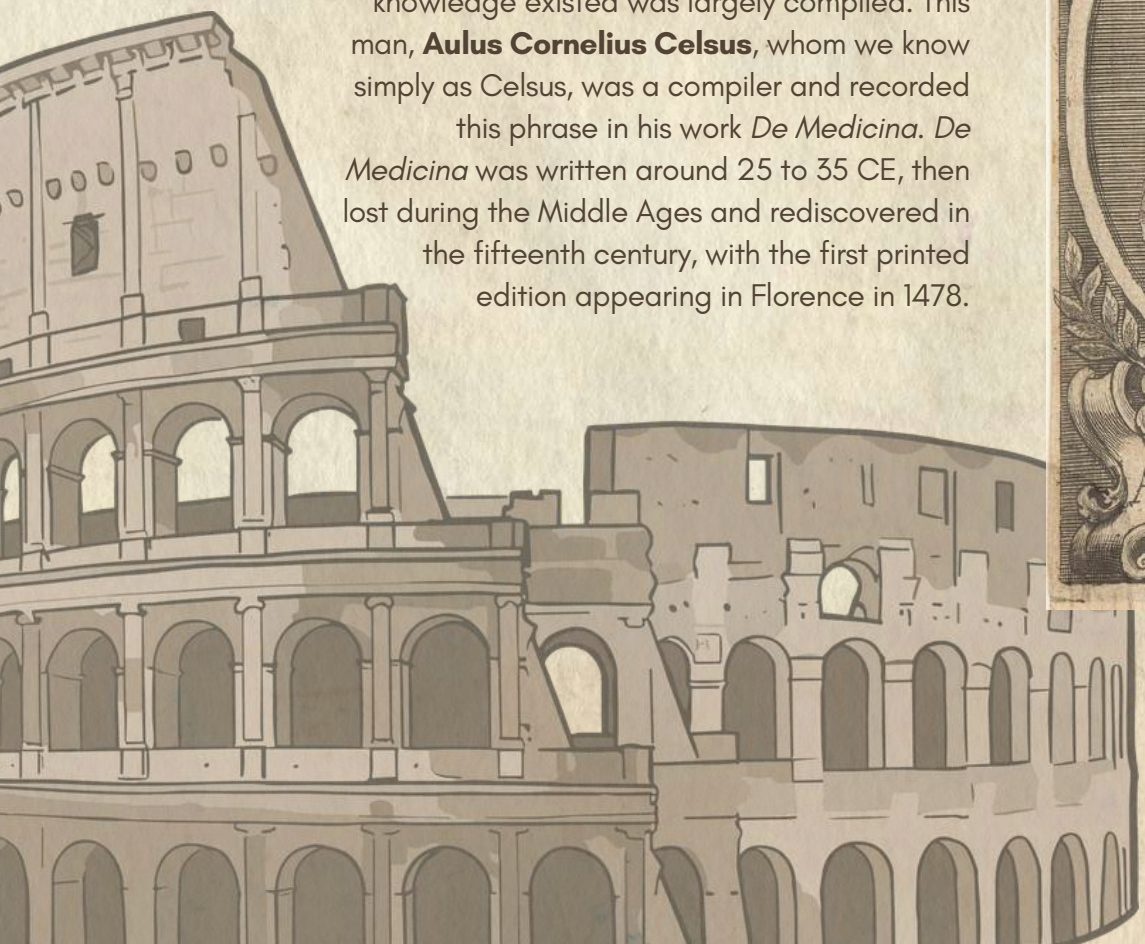
*As **Madurai** hosts the **Annual Conference of Uveitis Society (India), USICON 2026** in **September 2026**,*
the city extends a warm and heartfelt welcome to all delegates.
Beyond scientific exchange and academic enrichment, we hope your time here allows you to experience the spirituality, warmth, flavours, and timeless charm of this historic city.

CLOSING NOTE FROM THE SECRETARY

FROM CELSUS TO CYTOKINES

THE EVER-EVOLVING STORY OF INFLAMMATION

Nearly 2000 years ago, a Roman man who was not even a physician by training wrote in his book, *"Notae vero inflammationis sunt quattuor: rubor et tumor, cum calore et dolore,"* meaning the signs of inflammation are four: redness and swelling, with heat and pain. In those days there were no journals; in fact, the first scientific journal was published only in 1665, so whatever knowledge existed was largely compiled. This man, **Aulus Cornelius Celsus**, whom we know simply as Celsus, was a compiler and recorded this phrase in his work *De Medicina*. *De Medicina* was written around 25 to 35 CE, then lost during the Middle Ages and rediscovered in the fifteenth century, with the first printed edition appearing in Florence in 1478.



Much later, **Rudolf Virchow,** a nineteenth century German physician and pathologist widely regarded as the *Father of Modern Pathology*, added a fifth dimension, loss of function. It is remarkable that many years before the development of the microscope, this Roman scholar described the cardinal signs of inflammation, which still stand as the foundation of our modern understanding. Even today, when a patient walks into our clinic, before any investigation, we are still, in essence, thinking like Celsus. An uveitic eye manifests as congestion, there is swelling of the uveal tract due to inflammation, the patient presents with pain, and there is a rise in temperature; one of the finest examples is the convection currents in the anterior chamber created by the warm iris and cooler cornea, and the pupillary reaction becomes sluggish due to inflammation. No wonder the power of human observation can, in many ways, transcend even molecular level insights.

The history of inflammation remains fascinating. **Julius Cohnheim**, a student of Virchow and a pioneer of experimental pathology, performed an elegant experiment. Using a microscope on the transparent mesentery of a live frog, he demonstrated that leukocytes, or white blood cells, are not static but actively move, migrate, and exit blood vessels, a phenomenon we now call *diapedesis*.

Then came another breakthrough, the concept of immunity, and interestingly it did not begin in humans but in a starfish defending itself. **Elie Metchnikoff**, a curious mind from Russia, while experimenting with starfish larvae, inserted tiny thorns into their bodies and observed amoeba like wandering cells rapidly surrounding and attempting to engulf the foreign body, leading to the discovery of phagocytosis. Interestingly, his theory was initially dismissed by the German scientist **Paul Ehrlich**, who proposed that immunity relied on soluble factors such as antibodies in the blood serum that neutralize toxins or microbes before cells intervene. **Metchnikoff** believed antibodies were secondary and even irrelevant, while Ehrlich believed phagocytes were not the primary drivers. They were not merely disagreeing; they were describing two completely different worlds, one cellular and the other humoral. With growing evidence, science eventually united these two perspectives, and in a

remarkable turn, Metchnikoff and Ehrlich shared the Nobel Prize in Physiology or Medicine.

Physicians had always known that patients with inflammation develop fever, malaise, and systemic illness, but no one truly understood why. Many suspected the presence of a mysterious substance released by the body, an endogenous pyrogen. This substance was observed in rabbits as early as 1943 and was eventually isolated in 1977, leading to the discovery of *Interleukin 1*. Suddenly, inflammation was no longer just about cells; it became communication, with cells talking to each other through cytokines. Then came the paradox of a molecule discovered with great excitement, *Tumor Necrosis Factor*. As early as the 1890s, **William Coley** observed that bacterial infections could lead to tumor regression, with the help of a substance, termed as *Coley's toxins*, which later laid the foundation for the discovery of Tumor Necrosis Factor. The name itself was dramatic, as it could destroy tumors, but soon it became clear that the same molecule could also drive sepsis, tissue damage, and uncontrolled inflammation, where the hero became the villain.

Today, we stand at an interesting point where we block TNF, block IL 6, and modulate the immune system with precision. What Paul Ehrlich once envisioned as a magic bullet is now part of our daily practice. But here is the real takeaway: despite all our advances and the expanding armamentarium of PCR, cytokine profiling, and biologics, inflammation still poses the same fundamental challenge. *It can protect, it can destroy, and sometimes it can deceive*! *Perhaps that is why the world of inflammation remains so fascinating, because of the challenge it continues to pose.*

In many ways, this evolving story of inflammation mirrors our own journey as a community: constantly learning, questioning, and refining our understanding. It is in this spirit that we bring out each issue of this newsletter, not merely as a compilation of articles, but as a reflection of shared thought, effort, and curiosity.

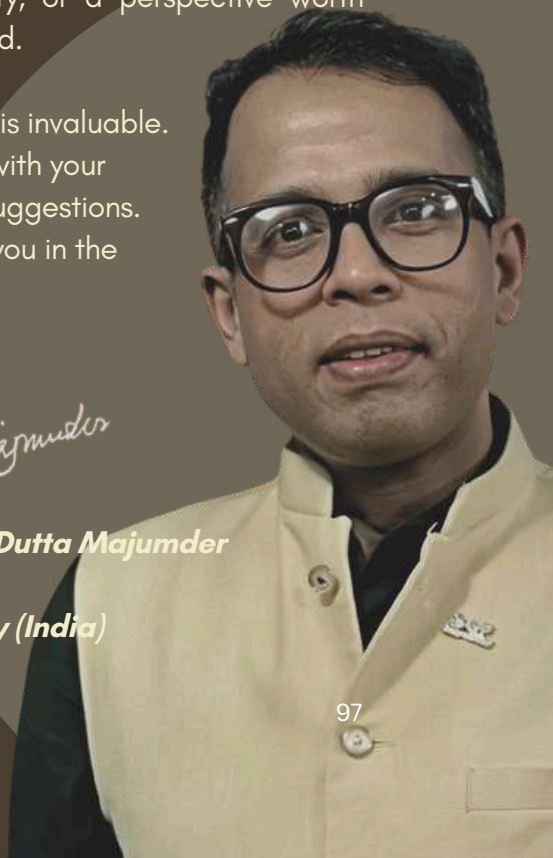
With the new team of USI, our effort has been to give the newsletter a fresh look and a new dimension. This would not have been possible without the dedicated work of *Dr. Vandana Pradeep* and *Dr. Richa Pyare*, who have put in tremendous effort to shape both its content and design.

Traditionally, a secretary's message tends to be a routine addition, often read out of obligation rather than interest. We felt this space could be reimagined. Instead of a conventional note, we hope to end each issue with something reflective, perhaps a thought, a story, or a perspective worth carrying forward.

Your feedback is invaluable. Do write to us with your thoughts and suggestions. Until then, see you in the next issue.

Best wishes

Parthopratim Dutta Majumder
Secretary
Uveitis Society (India)



Uveitis Society (India)

ORGANIZED BY

ARAVIND EYE CARE SYSTEM

USICON 2026

27th Annual Conference of Uveitis Society (India)

25th | 26th | 27th September

Venue: The Residency Signature, Madurai

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