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TAP HERE TO

**BECOME
A MEMBER**



Dear friends and colleagues,

Welcome to another edition of Eyetis!

Uveitis has always been a field driven by curiosity.

We have questioned what we know, explored new ideas, welcomed new voices and learned from perspectives beyond our own. It is this openness that keeps our field moving—and keeps our community growing.

This issue celebrates that spirit while also trying a few new things along the way. We hope you enjoy the mix of familiar voices and fresh perspectives, and that somewhere along the way, something makes you pause, wonder, question, or look at an idea a little differently.

After all, curiosity is where every good conversation begins.

Happy reading!

Dr. Vandana Pradeep
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"Is there any point to which you would wish to draw my attention?"

"To the curious incident of the dog in the night-time."

"The dog did nothing in the night-time."

"That was the curious incident," remarked Sherlock Holmes.

— Sir Arthur Conan Doyle

Ophthalmologist

Creator of Sherlock Holmes ("Silver Blaze")

Welcome back to another edition of Eyetis!

Holmes solves this case not by what he finds but rather by what is missing.

Uveitis specialists work much the same way. We fit together signs, symptoms, imaging and laboratory tests piece by piece, and the piece that isn't there often tells us as much as the ones that are. Curiosity, the willingness to keep asking why, is what holds that work together, and it is the thread running through this issue.

In this issue, we push the boundaries of our curiosity geographically with a new section, *Around the World in Uveitis*, on endemic conditions that take us beyond the familiar and into territory most of us will never meet in our own clinics.

As always, I hope you enjoy the issue.

May you stay curious!

Dr. Richa Pyare

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It gives me immense pleasure to present the winter edition of the USI Newsletter. This edition unites wisdom from our luminaries, deep clinical learning, cutting-edge science, and the voices of the next generation of uveitis specialists.

A key highlight is *Luminaries Speak*, featuring invaluable insights from Dr. Jyotirmay Biswas, drawn from his extensive experience in uveitis practice.

Our *Special Conversation* with Prof. James Rosenbaum offers a unique perspective bridging ophthalmology and rheumatology

Core Concepts revisits the essentials of acute

retinal necrosis, while *Deep Dive* explores ocular Behçet's disease—from foundational principles to a practical masterclass.

Clinical Chronicles brings learning closer to everyday practice through *Case Arena* and *SnapShot*.

In the *NextGen Corner*, we look to the future while exploring the gut-eye axis and ocular fluid analysis in uveitis.

Through the *Global Window*, we learn the exciting and rapidly evolving role of artificial intelligence and AI-led imaging in uveitis care.

The *What's New* section delves into emerging areas such as JAK inhibitors, ocular thermography, and contemporary clinical research.

For those who enjoy a clinical challenge, *Test Your UQ* offers an opportunity to reflect on and test your knowledge.

And with *Around the World in Uveitis*, we travel to Nepal to explore SHAPU, reminding us of the diverse clinical experiences and challenges within our region.

I extend my sincere gratitude to all the contributors, editors, and members of the USI team whose time and expertise have made this edition possible. I also invite all our members to continue sharing their experiences as we move forward together.

Warm regards,

Dr. Padmamalini Mahendradas
President
Uveitis Society (India)

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Beyond the Page...

Newsletter of Uveitis Society (India)

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TAP A TITLE
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LUMINA SPEAKERS

PROF.

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*Few individuals have
shaped the landscape
of uveitis in India as
profoundly as
Prof. Jyotirmay Biswas.*

*Clinician, teacher,
ocular pathologist,
researcher and mentor,
his career spans
decades of pioneering
contributions to ocular
inflammation and
infectious eye disease.
In this conversation, he
reflects on clinical
observation, pathology,
diagnostic humility, and
the lessons learned from
a lifetime in uveitis.*

Over the years, what do you think truly separates a good uveitis clinician from a great one?

A good clinician diagnoses, investigates, and manages disease effectively. However, I believe there are a few qualities that can transform a good uveitis specialist into a great one.

Uveitis is mostly a disease of young adults. Many of them do not have any previous history of blurring or difficulty in vision – and the sudden attack of uveitis with blurring of vision, pain, redness and watering comes as a shock to them. Many find it difficult to accept this new disease.

A great uveitis specialist would spend time explaining the reason for uveitis. A detailed approach to the patient's background, the impact of this disease on his personal and family life, and the financial burden should be understood. A great uveitis specialist shows empathy and provides a holistic approach. I believe every uveitic disease can be explained to the patient in these terms.

Uveitis often forces us to look far beyond the eye. How has the speciality changed the way you look at patients over the course of your career?

At the beginning of my career, we used to classify many uveitic diseases as idiopathic or of unknown etiology, but the advent of newer technology like

polymerase chain reaction (PCR), high-resolution CT chest and various other imaging modalities has transformed our practice and made precise etiological diagnosis possible. Carefully planned systemic workups and growing collaborations with colleagues from other branches of medicine including rheumatology, pulmonology and dermatology have helped us arrive at diagnoses that have been missed in the past. Uveitis constantly reminds us that the eye is not an isolated organ. To understand the disease fully, we must understand the patient as a whole.

Your work in ocular pathology has profoundly influenced generations of ophthalmologists. How has ocular pathology shaped the way you approach uveitis clinically?

My training in ocular pathology has made me a better clinician and I have enjoyed seeing uveitis patients much more since my pathology training. Since then, whenever I see mutton-fat KPs, I see macrophages and epithelioid cells touching the posterior surface of the cornea. In non-granulomatous uveitis and fibrinous anterior uveitis, I see polymorphonuclear leucocytes and fibrin material adhered to the back of the cornea. Similarly, when I see a Koeppe or Busacca nodule, I immediately recognise them as manifestations of a granulomatous inflammation. And a Dalen-Fuchs nodule brings to my mind a break in the Bruch's membrane with a collection of lymphocytes and epithelioid

cells underneath it. Pathology has also helped me differentiate infectious uveitis and noninfectious uveitis much more effectively.

About 40 years ago, I saw a case of subretinal abscess which later became a painful blind eye that eventually required evisceration. When I later examined the specimen under the microscope, I was overwhelmed to find innumerable acid-fast bacilli within the subretinal space (Figure 1).

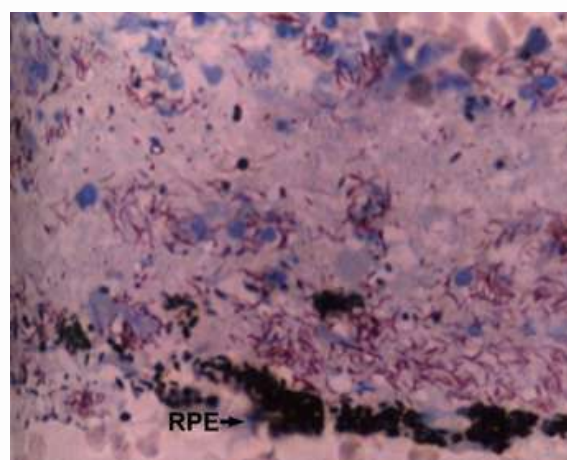


Figure 1: Myriads of acid-fast bacilli in subretinal space in Ziehl-Neelsen staining

This experience fundamentally changed my understanding about tubercular uveitis and convinced me that direct mycobacterial infection of the uveal tract is possible. Even today, when examining similar patients, these images come to mind and help me understand the disease at a much deeper level.



In an era increasingly driven by imaging and investigations, do you feel the art of clinical observation is gradually being lost?

I believe this is true to some extent.

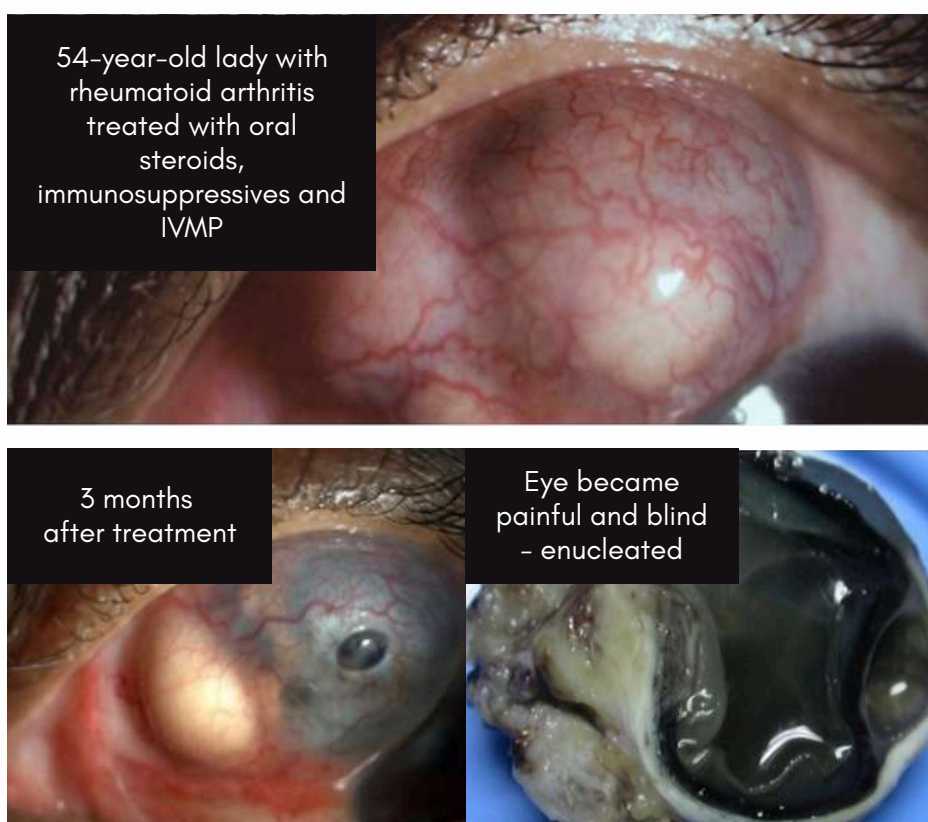
We must first be very good clinicians - examine the patient carefully, see whether it is non-granulomatous or granulomatous, infectious or non-infectious, and assess the nature and extent of tissue damage and complications that may already be present. Investigations are important, and should come later mainly to support the diagnosis. I agree, sometimes, of course, it can give a new diagnosis, but the art of clinical observation cannot be overemphasized.

Was there a moment in your career when a patient's diagnosis fundamentally changed the way you practice medicine?

Yes, there is one case that taught me a lesson I have never forgotten.

I once treated a patient with multiple nodular scleritis believing it to be of non-infectious autoimmune etiology. I treated it with high-dose steroids and immunosuppressants. The patient did not respond and subsequently was lost to follow-up. A month later, he returned with a painful blind eye and severe suppuration. Eventually, the eye had to be enucleated.

Histopathological examination revealed





“Do not fall in love with your diagnosis”

caseating necrosis and PCR was positive for *Mycobacterium tuberculosis*. This was the case that changed my thinking forever.

Henceforth, I have always considered the possibility of infectious etiology in nodular scleritis, particularly if there is a pus point, and if it is not responding to the usual anti-inflammatory agents. Tragically, the patient not only lost the eye but later succumbed to disseminated tuberculosis.

It was a deeply humbling experience and remains one of the most important lessons of my career.

What has uveitis taught you about uncertainty, patience and humility in medicine?

One of the most important lessons uveitis teaches us is that diseases do not read textbooks. They do not always follow textbook descriptions.

Patients come to the uveitis clinic with

complex systemic ailments and a clinical picture that can evolve with time. I remember a case of syphilitic uveitis that initially presented with retinal vasculitis, but then developed nodular and extensive retinal lesions which did not respond to anti-syphilitic therapy. The patient never regained useful vision.

Such cases remind us of limitations of our current treatment and understanding of disease. Despite our best efforts, we are often unable to make a precise diagnosis, control the inflammation and the patient follows a downhill course to ultimately lose vision.

Re-evaluate your patients, revisit your diagnosis and remain open to alternative explanations.

Do not fall in love with your diagnosis. Be willing to take opinions from colleagues – whether junior or senior – and learn from every mistake, so that it is not repeated.

Looking back, what aspect of your journey in uveitis gives you the greatest sense of fulfillment?

One of the most fulfilling chapters of my career was my work on ocular manifestations of HIV.

In 1995, I diagnosed two of the earliest recognised cases of AIDS in India. At that time, very little was known about ocular manifestations of HIV infection.

Clinicians were then, understandably,



apprehensive of examining these patients. I saw them patiently and participated in their treatment and management. I went on to write around 40 articles on ocular manifestations of HIV infection including a manual of ocular lesions in AIDS in India. I also prepared a clinical profile of ocular lesions in AIDS, which was later published in the American Journal of Ophthalmology.

Another work that has given me immense satisfaction is my work on Polymerase Chain Reaction (PCR) in uveitis and ocular infection. I started working with Prof. H N Madhavan in 1990 with a few cases. What started as a small poster in the International Uveitis Study Group in Yokohama, Japan, subsequently grew into a series of 22 publications.

Today, PCR has become an indispensable tool in the diagnosis of viral and various other infective uveitis. Watching this field evolve and contribute to better patient care has been deeply rewarding.

What concerns you most about the future generation of clinicians and what gives you hope?

One concern I have is that the younger generation of ophthalmologists may be depending too much on imaging technologies and electronic medical records. While these advances are undoubtedly valuable, they should never replace direct interaction with patients.

Maintaining eye contact, listening carefully, and taking the time to explain the diagnosis and treatment plan remain fundamental aspects of good clinical care. Investigations are important, but patients must never feel that they are being treated by machines rather than by doctors. We are not God, but we can do humanly as much as possible, for each and every patient of ours.

At the same time, I am very optimistic about the future too.

The younger generation of uveitis specialists is highly academic, well-read, and deeply engaged with scientific literature. The quality of presentations at meetings has improved remarkably, and many young ophthalmologists are undertaking innovative basic and translational research that I once dreamed of pursuing myself.

The Uveitis Society of India has also played a major role in disseminating knowledge through meetings, CMEs, and educational programs across the country, helping to increase awareness and interest in the field.

My message to every young ophthalmologist is this:

Uveitis is not merely a diagnosis. It is a disease affecting a human being. Treat every patient with compassion, and manage every disease with a scientific and holistic approach.



ARN

ACUTE RETINAL NECROSIS



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**RECOGNISE EARLY
TREAT PROMPTLY**



Acute retinal necrosis (ARN) is a rapidly progressive necrotizing retinitis characterized by peripheral retinal necrosis, occlusive retinal vasculitis and prominent intraocular inflammation.^{1,2} ARN is a true ophthalmic emergency. Prompt initiation of antiviral therapy can preserve vision, limit retinal destruction and reduce the risk of fellow-eye involvement.³⁻⁵

RecoGnise

BEFORE IT
BECOMES
CLASSICAL



Most ophthalmologists can recognise established ARN. The greater challenge is recognising it before it becomes classical.

Early ARN may present as only a few small peripheral satellite lesions before they

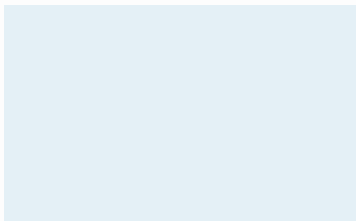
enlarge, coalesce and spread circumferentially over a matter of days.^{3,6} Maintaining a high index of suspicion is therefore critical. Consider ARN in patients presenting with unilateral anterior uveitis associated with vitritis, particularly when the clinical picture suggests herpetic disease. A recent history of herpes zoster ophthalmicus or varicella infection, unexplained ocular pain, or associated scleritis should further increase suspicion.

The anterior segment often provides valuable clues. Fresh pigmented keratic precipitates, raised intraocular pressure, sectoral iris atrophy and disproportionate vitritis should prompt a meticulous search for retinal involvement. Even when no retinal lesion is identified initially, patients with a high index of suspicion should undergo repeated dilated fundus examinations, as ARN may evolve within days.^{3,8}



DON'T StoP

AT THE ANTERIOR SEGMENT



Dense vitritis is a clue!

A meticulous dilated fundus examination is mandatory in any patient with anterior uveitis associated with dense vitritis. The diagnosis of ARN often lies in the far peripheral retina and may

be missed if the examination is limited to the posterior pole. Indirect ophthalmoscopy with careful examination of the retinal periphery—and scleral indentation when necessary—should be performed whenever infectious retinitis is suspected.

Dense vitritis is one of the commonest reasons why early ARN is overlooked. Ironically, it should increase rather than decrease suspicion of infectious retinitis. Instead of concluding that the retina cannot be

adequately visualised, repeat the examination if necessary or use ultra-widefield imaging when available. Both ARN and toxoplasma retino-choroiditis commonly present with dense vitritis, making careful peripheral retinal evaluation essential.^{8,9}

Occlusive retinal arteriolar vasculitis is another useful clue.^{1,3} Following an affected arteriole into the retinal periphery may lead directly to an early focus of retinitis that might otherwise be missed.

Think

BEFORE PRESCRIBING STEROIDS



One of the commonest pitfalls is treating unexplained vitritis with corticosteroids before excluding infectious retinitis. Missing the underlying etiology and initiating corticosteroids can have devastating consequences. Depot periocular or intravitreal corticosteroids deserve particular caution because their effects cannot be reversed once administered.^{3,8} Before escalating

immunosuppression, always examine the peripheral retina carefully and consider infectious causes such as ARN and toxoplasma retinochoroiditis. Patients referred with steroid-resistant uveitis or worsening inflammation after corticosteroid therapy should prompt immediate re-consideration of the diagnosis rather than escalation of steroid treatment.

The principle is simple:
treat the infection first,
then the inflammation.

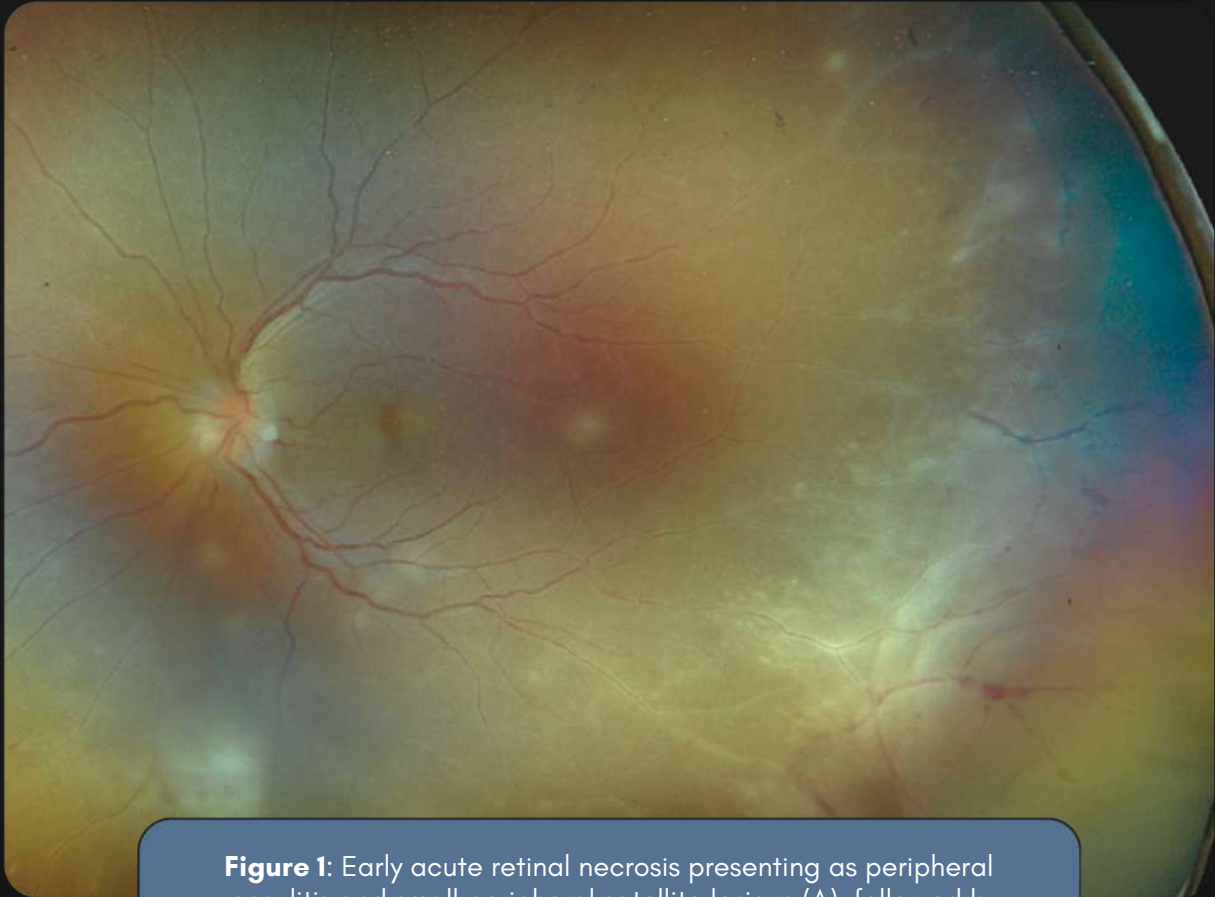
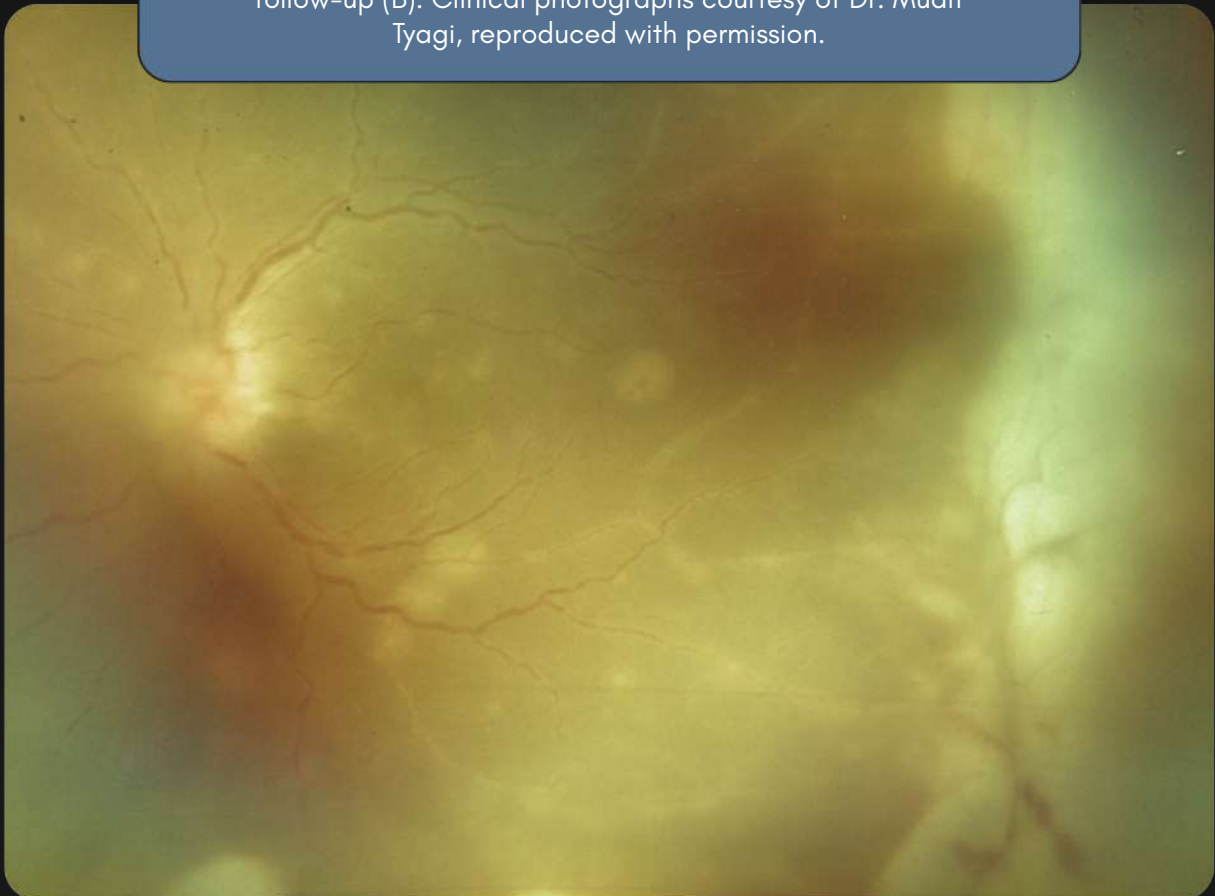


Figure 1: Early acute retinal necrosis presenting as peripheral vasculitis and small peripheral satellite lesions (A), followed by progression to confluent peripheral necrotizing retinitis at follow-up (B). Clinical photographs courtesy of Dr. Mudit Tyagi, reproduced with permission.





ARN is a

CLINICO- MORPHO- GICAL DIAGNOSIS



ARN is defined by its characteristic clinical appearance rather than by a specific pathogen. Varicella zoster virus and herpes simplex virus types 1 and 2 are responsible for the majority of cases.^{1,2,3} Rarely, other infectious agents may produce an ARN-like picture.

Polymerase chain reaction of aqueous or vitreous samples is extremely useful in confirming the etiology, particularly in atypical cases. However, treatment should not be delayed while awaiting PCR results when the clinical diagnosis is convincing.^{2,3,8}

Treatment

HAS EVOL- VED



Prompt systemic antiviral therapy remains the cornerstone of management. High-dose oral valacyclovir has emerged as an effective induction regimen for many immunocompetent patients and, in

appropriately selected cases, may avoid hospital admission.^{4,6} Intravenous acyclovir remains appropriate in patients with severe disease, inability to tolerate oral medication, poor gastrointestinal absorption or selected immunocompromised patients.⁴ Intravitreal antiviral therapy with foscarnet is an important

adjunct in rapidly progressive or sight-threatening disease.^{3,8}

Systemic corticosteroids may be introduced after adequate antiviral therapy has been established to control intraocular inflammation, preferably using oral therapy. Corticosteroids should never precede antiviral treatment.^{3,8}



Do NOT

LET IMMUNE STATUS MISLEAD YOU



Classically, ARN has been associated with immunocompetent individuals, while progressive outer retinal necrosis (PORN) is described in severely immunocompromised patients.

In reality, this distinction is not absolute. ARN can occur in patients with HIV

infection and other forms of immunosuppression. These patients may have more aggressive disease, earlier bilateral involvement and less prominent vitritis.^{8,9} Evaluation for underlying immunosuppression, including HIV testing where appropriate, should form part of the assessment.

Cause OF

VISION LOSS CHANGES WITH TIME



The causes of visual loss evolve during the course of the disease. Early visual loss is usually inflammatory and may result from dense vitritis, optic nerve involvement, macular involvement,

occlusive retinal vasculitis or, occasionally, exudative retinal detachment.^{3,8}

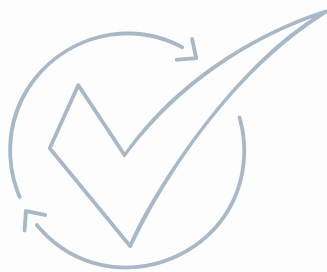
Later, retinal thinning at the junction of healthy and necrotic retina predisposes to retinal breaks and rhegmatogenous retinal detachment, which remains the commonest cause of severe visual loss.^{3,4,10} Ironically, retinal detachment often

develops just when the retinitis appears to be improving.

Patients should therefore be counselled appropriately and followed up closely even after the infection has been controlled. The role of prophylactic barrage laser remains controversial and may not be feasible in eyes with active retinitis or dense vitritis.^{8,10}



Conclusion



The greatest opportunity to preserve vision in ARN lies not in recognising advanced disease but in recognising it early. Maintaining a high index of suspicion, performing a meticulous peripheral retinal examination, avoiding premature corticosteroid therapy and initiating timely antiviral treatment can significantly improve outcomes. Even after the retinitis responds, continued surveillance remains essential because retinal detachment is often the second battle after successful control of infection.





DEEP DIVE





BEHÇET'S SYNDROME

THE SILK ROAD DISEASE



OCULAR BEHÇET'S DISEASE

THE CLUES ARE USUALLY THERE



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Behçet's disease (BD) is one of those conditions that reminds us how closely the eye is connected to the rest of the body. Although it is best known to ophthalmologists for causing recurrent uveitis and retinal vasculitis, it is actually a chronic, relapsing inflammatory disease that can involve the skin, mucous membranes, joints, blood vessels and nervous system.

The disease shows a striking geographical distribution along the historic "**Silk Road**", stretching from Turkey through the Middle East to East Asia, making it particularly relevant to ophthalmologists practising in India.¹





CLINICAL FEATURES

THE SKIN

For most patients, painful, recurrent oral aphthous ulcers are the first sign of the disease and often precede other manifestations by several years. Genital ulcers are more specific, deeper than oral ulcers and heal with scarring.

Patients may develop erythema nodosum-like lesions, papulopustular eruptions, acneiform lesions or superficial thrombophlebitis.

In countries where Behçet's disease is more common, the pathergy test is a useful clinical clue. A papule or pustule developing after minor skin trauma reflects the exaggerated inflammatory response that characterises the disease.

THE JOINTS

Joint symptoms are common and usually not destructive. Up to half of patients experience recurrent, non-erosive arthritis, most often involving large joints such as the knees and ankles. Many others complain of intermittent arthralgia that follows the same relapsing-remitting pattern seen elsewhere in the disease.

THE BLOOD VESSELS

Behçet's disease can involve both arteries and veins of any calibre. Venous disease is far more common than arterial involvement, with deep vein thrombosis being the most frequent presentation. More severe manifestations include vena caval thrombosis, pulmonary artery

aneurysms and arterial occlusions.

THE NERVOUS SYSTEM

Neurological disease (in about 5–10% of patients) is one of the more serious manifestations of Behçet's disease. Patients may present with brainstem involvement, meningoencephalitis, cerebral venous sinus thrombosis or, less commonly, optic nerve disease. Once neurological involvement develops, the overall prognosis becomes considerably less favourable.

THE EYE

The hallmark of ocular Behçet's disease is a recurrent, bilateral, non-granulomatous uveitis associated with occlusive retinal vasculitis that involves veins more frequently than arteries. Diffuse perivascular sheathing is seen during active inflammation. Occasionally, the inflammation can be so striking that it resembles frosted branch angiitis, sometimes accompanied by neuroretinitis. Inferior peripheral vitreous inflammatory precipitates arranged in a characteristic linear "pearl-like" pattern may be seen emerging a few days after the onset of inflammation and then resolve spontaneously. As the inflammation resolves, affected vessels may remain as sheathed or appear as "ghost" vessels. With repeated attacks, retinal ischaemia and capillary non-perfusion may develop, eventually leading to retinal neovascularisation and the accompanying risk of vitreous or retinal haemorrhage.



Another important consequence of inflammation is disruption of the blood-retinal barrier resulting in widespread vascular leakage involving the optic disc, macula, peripheral retina and cystoid macular oedema (CMO). CMO remains one of the leading causes of visual loss and, if recurrent, may leave permanent structural damage.

Behçet's retinitis is a distinctive posterior segment finding and usually occurs alongside active retinal vasculitis. Retinal infiltrates appear as transient, multifocal, deep white lesions, particularly around the posterior pole and retinal vessels. These infiltrates usually respond rapidly to immunosuppressive therapy and generally heal without significant chorioretinal scarring.

Hypopyon was once regarded as the classic sign of Behçet's disease, but more recent case series show that it is less common than previously believed, occurring in roughly one-third of patients. When present, it has a characteristic appearance: a mobile, non-fibrinous hypopyon that shifts with changes in head position.

Ultimately, it is the cumulative effect of repeated inflammatory attacks that determines visual outcome. Over time, persistent inflammation can lead to retinal gliosis, diffuse retinal and macular atrophy, optic disc pallor and markedly attenuated sheathed retinal vessels. At this stage, the fundus may even resemble retinitis pigmentosa, reflecting the irreversible retinal damage responsible for severe visual loss.

DIAGNOSTIC CRITERIA

A patient may present to an ophthalmologist with retinal vasculitis, a dermatologist with recurrent ulcers, or a rheumatologist with joint pain. Putting these pieces together is often the key to making the diagnosis.

The **ISG criteria**⁵ require recurrent oral ulceration occurring three or more times in one year, together with any two of the following:

- Recurrent genital ulceration
- Eye lesions (anterior or posterior uveitis or retinal vasculitis)
- Characteristic skin lesions (erythema nodosum or pseudofolliculitis)
- A positive pathergy test

Meanwhile, the **ICBD criteria**⁶ are a weighted scoring system:

- Ocular lesions (uveitis, retinal vasculitis, chorioretinitis or papillitis) – 2 points
- Recurrent oral aphthosis (at least three episodes per year) – 2 points
- Recurrent genital aphthosis – 2 points
- Skin lesions (papulopustular rash or erythema nodosum) – 1 point
- Central nervous system involvement – 1 point
- Vascular manifestations, including venous thromboembolism, superficial thrombophlebitis, arterial thrombosis or aneurysms – 1 point
- Positive pathergy test – 1 point

A total score of ≥ 4 points classifies as Behçet's disease.



Although HLA-B51 is strongly associated with Behçet's disease, it is only supportive of a diagnosis of Behçet's and is NOT a diagnostic test.

The allele is present in 40-80% of patients living in endemic regions and increases disease susceptibility more than fivefold. However, nearly half of patients with Behçet's disease are HLA-B51 negative, while 15-20% of healthy individuals in endemic populations also carry the allele.

Another genetic marker, HLA-A26, has emerged as an independent susceptibility factor and has been linked to a poorer visual prognosis.⁷

SEVERITY GRADING SCORES

Making the diagnosis is only the first step. Equally important is assessing how active the disease is, monitoring response to treatment and recognising patients who are at risk of progressive visual loss.

- The *Behçet Disease Current Activity Form (BDCAF)* and

- The *Behçet Syndrome Activity Score (BSAS)*

grade systemic activity.

- The *Behçet's disease Ocular Attack Score 24 (BOS24)*

grades ocular activity.

TREATMENT

Not very long ago, corticosteroids formed the backbone of therapy. Today, the philosophy is different.

Topical corticosteroids remain appropriate for isolated anterior uveitis.

Once the disease becomes recurrent, bilateral or involves the posterior segment these patients require systemic immunomodulatory treatment.

In such situations, intravenous methylprednisolone followed by tapering oral corticosteroids along with local corticosteroid therapy in selected patients with refractory CMO and in patients with contraindications to systemic corticosteroid therapy remains standard practice.

The 2018 European League Against Rheumatism (EULAR) recommendations advocate early and aggressive immunosuppressive therapy for patients with posterior uveitis, retinal vasculitis or panuveitis.⁹



Among conventional immunosuppressants, azathioprine and cyclosporine A have the strongest evidence base,

with one important exception to be always remembered: cyclosporine A should be avoided in patients with active neuro-Behçet disease because of its association with central nervous system involvement.

The biggest advance in recent years has undoubtedly been the introduction of biologic therapy, especially anti-TNF- α agents with clinical experience and recent studies firmly establishing their role in managing sight-threatening Behçet uveitis.¹⁰ Infliximab has an exceptionally rapid onset of action and is particularly valuable during acute vision-threatening attacks.

Adalimumab is currently the only FDA-approved biologic for non-infectious intermediate uveitis, posterior uveitis and panuveitis.

Factors that consistently predict a poorer prognosis include male sex, poor visual acuity at presentation, a diagnostic delay of more than nine months, frequent relapses (more than three attacks each year), persistent ocular inflammation, poor treatment adherence and established optic atrophy.¹²

Among these, one combination deserves

particular attention. Patients with both ocular and neurological Behçet's disease face a substantially greater risk of severe visual disability. Approximately 40% develop legal blindness compared with 25% of patients whose disease is confined to the eyes. Recognising this subgroup early and treating it aggressively can make a meaningful difference to long-term outcomes.

PAEDIATRIC BEHÇET'S DISEASE

Paediatric Behçet's disease (PBD), defined as disease onset before 16 years of age, accounts for 3–26% of all cases.¹³

Children frequently develop symptoms gradually over several years. A child may initially present with recurrent oral ulcers, while ocular, neurological or vascular involvement emerges much later.

The *Paediatric Behçet Disease (PEDBD) criteria* were developed considering six equally weighted clinical domains:

- Recurrent oral aphthosis
- Genital ulceration
- Skin lesions
- Ocular involvement
- Neurological manifestations
- Vascular involvement

The ocular picture is similar to that seen in adults. Bilateral panuveitis with retinal vasculitis remains the commonest presentation. Unfortunately, children



often present with complications that are already well established.

Treatment principles have evolved in much the same way as they have for adults. The emphasis is no longer simply on controlling acute inflammation but on preventing repeated attacks before permanent retinal damage occurs. Corticosteroids are combined early with steroid-sparing immunosuppressive agents including early introduction of biologics such as infliximab and adalimumab.

Close collaboration between ophthalmologists, paediatric rheumatologists and other specialists is essential to preserve vision while minimising the long-term effects of both the disease and its treatment on growth and development.

another cause of uveitis. Looking beyond the eye for recurrent oral ulcers, genital ulcers, skin lesions, vascular events or neurological symptoms often provides the missing piece of the diagnostic puzzle.

Ultimately, preserving vision in Behçet's disease depends on recognising the condition early, treating inflammation promptly and aggressively, and working closely with colleagues across specialties. This approach is particularly important in children, where delayed diagnosis and recurrent inflammation can have lifelong visual consequences.

CONCLUSION

Behçet's disease continues to be one of the most challenging causes of non-infectious uveitis encountered in clinical practice.

Fortunately, our understanding of the disease has advanced considerably and the introduction of effective immunomodulatory therapy, especially biologic agents has transformed the visual outlook for many patients.

For the practising ophthalmologist, perhaps the most important lesson is that Behçet's disease should always be considered as a systemic disease with ocular manifestations rather than simply





THE ART OF MANAGING

OCULAR BEHÇET'S DISEASE



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RECOGNISING THE ACTIVE ATTACK

Behçet uveitis (BU) typically presents as a recurrent, bilateral, non-granulomatous panuveitis.

- Retinal vascular involvement is predominantly occlusive and may result in arteriolar attenuation, retinal nonperfusion, and, in severe cases, retinal or disc neovascularization.
- The retinal infiltrates of BU are characteristically superficial, appearing as discrete white patches that do not obscure the underlying retinal vessels. These lesions may resolve within several days without leaving significant retinal scarring.
- A characteristic clinical finding is the development of inferior, pearl-like inflammatory deposits arranged in a linear pattern, typically appearing 4–7 days after the onset of an acute attack. They generally resolve spontaneously over weeks without sequelae.

- Vitreous haze is a sign of an active inflammation in the posterior pole. Vitritis is most prominent at the beginning of the attack.
- Anterior segment inflammation, interestingly, may be relatively mild with minimal or absent ciliary injection, diffuse endothelial dusting, and normal or low intraocular pressure. Mutton fat keratic precipitates, persistent anterior chamber cells and chronic high grade flare are uncommon in BU.
- Occasionally, a mobile hypopyon (due to absence of fibrinous exudate, allowing it to move freely with gravity) may be noted.
- Recurrent or severe BU can result in irreversible structural damage, including retinal and optic nerve atrophy, diffuse retinal atrophy and gliosis, and marked attenuation and sheathing of retinal vessels, producing a characteristic cord-like appearance.

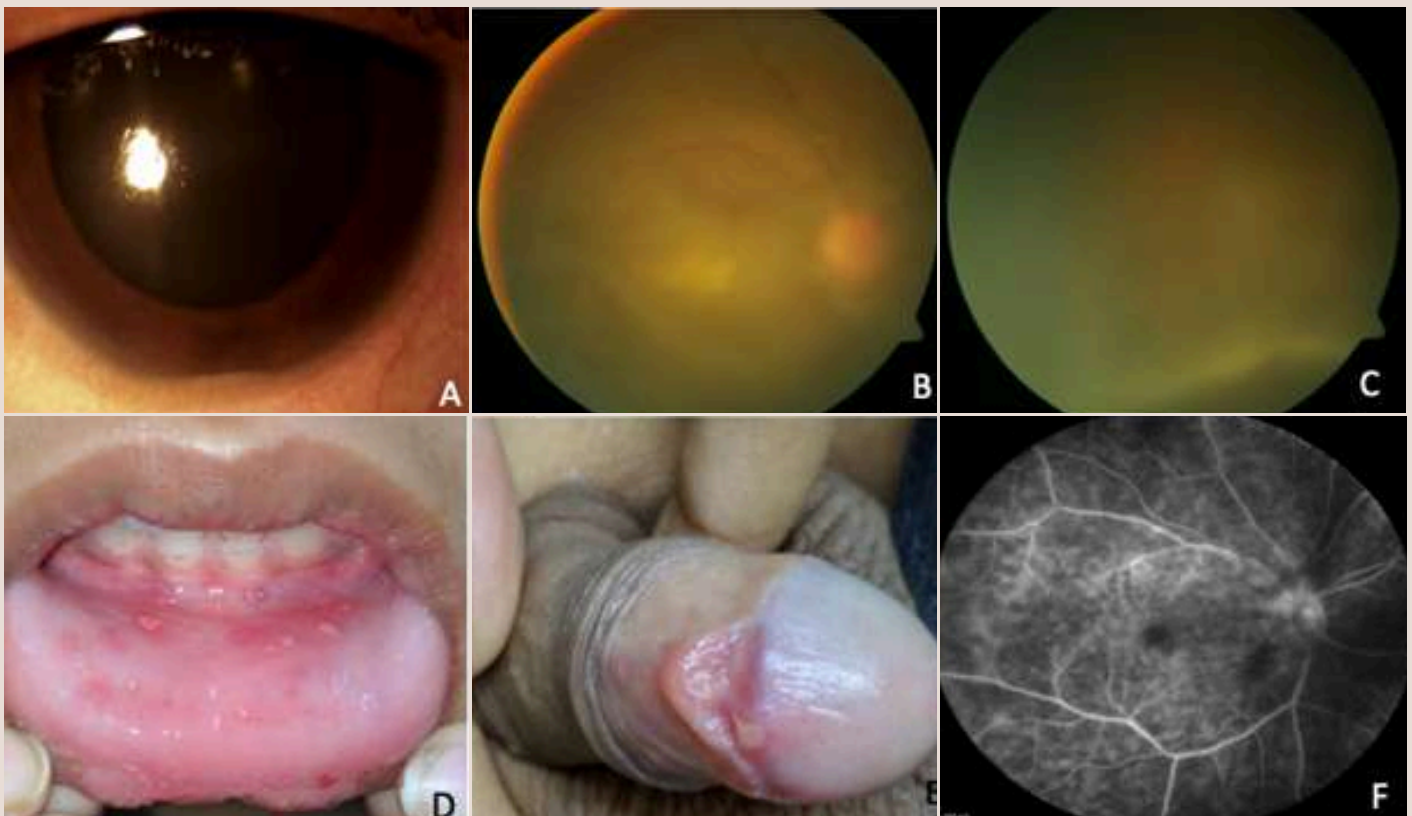


Figure 1: Composite photograph showing hypopyon (A), vitritis with retinitis (B), inferior string of pearls (C), oral aphthous ulcers (D), genital ulcers (E) and fern-like leakage on fluorescein angiography (F).



MULTIMODAL IMAGING TO ASSESS ACTIVITY

OPTICAL COHERENCE TOMOGRAPHY

Spectral-domain optical coherence tomography (SD-OCT) provides high-resolution structural assessment of the retina and vitreous.

Active retinal infiltrates appear as focal areas of hyperreflective retinal thickening, with optical shadowing without associated thickening of the underlying choroid.

Resolution of retinal infiltrates may be followed by localized retinal nerve fiber layer defects and thinning.

SD-OCT may also demonstrate localized vitreous condensations with a characteristic “smoking volcano” configuration, which can provide an additional imaging correlate of active inflammation.

FLUORESCEIN ANGIOGRAPHY

Fluorescein angiography (FA) remains a cornerstone in the assessment of BU. The characteristic fern-like pattern of peripheral vascular leakage reflects active vasculitis and provides an

objective measure of disease activity. FA is particularly valuable for documenting the extent of vascular involvement, detecting retinal nonperfusion, and monitoring response to treatment.

Ultra-widefield angiography can further improve visualisation of the peripheral retina and may facilitate more accurate detection of peripheral vascular inflammation and nonperfusion, thereby contributing to treatment decisions.

LASER FLARE PHOTOMETRY

Laser flare photometry provides an objective quantitative assessment of anterior chamber flare and, where available, can complement clinical examination in monitoring ocular inflammatory activity. However, its clinical use remains limited in many regions because of restricted availability.

Ultimately, multimodal imaging in ocular Behçet's plays an important role in documenting inflammatory activity, establishing the diagnosis and extent of disease, identifying structural damage, and monitoring treatment response.

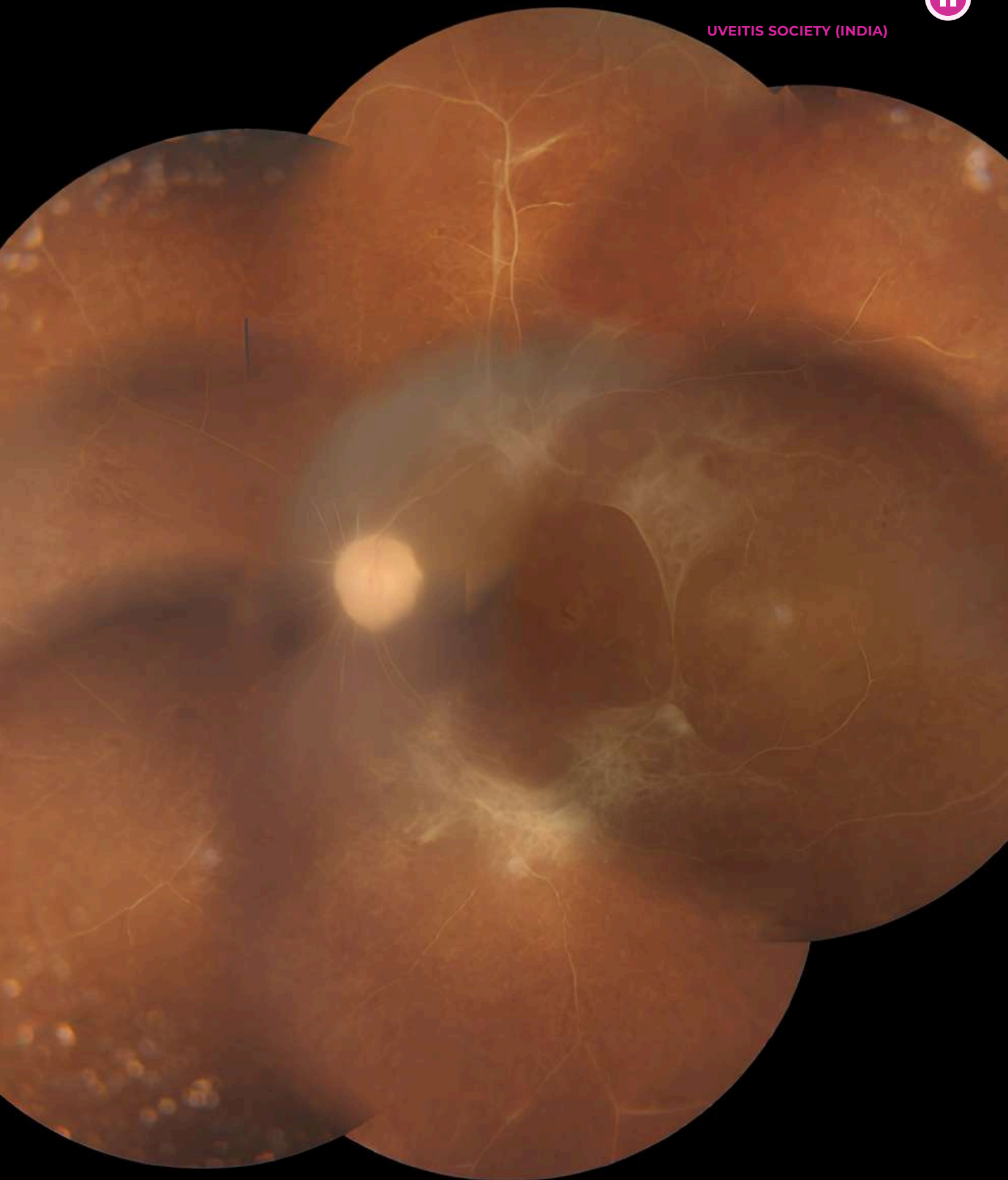


Figure 2: Fundus photograph of the left eye showing optic atrophy, sclerosed white cord-like vessels and extensive retinal gliosis.



TREATMENT OF OCULAR BEHÇET'S DISEASE

MANAGING THE ACUTE ATTACK

Acute exacerbations of BU require prompt and aggressive treatment to suppress inflammation and prevent irreversible damage. Systemic corticosteroids remain an important component of treatment for acute, sight-threatening inflammation, although their use should generally be accompanied by appropriate immunomodulatory therapy in patients with recurrent or severe disease.

MOVING BEYOND CORTICOSTEROIDS

Among conventional immunosuppressive agents, azathioprine and cyclosporine A have the strongest evidence and recommendations for the management of posterior uveitis associated with BD. The choice of immunomodulatory therapy should be individualised according to the severity, frequency, and pattern of ocular inflammation, as well as the presence of systemic disease.

THE ROLE OF BIOLOGICALS

Anti-tumour necrosis factor- α (anti-TNF- α) agents, particularly adalimumab and infliximab, have substantially expanded the therapeutic options for patients with refractory or recurrent BU and are widely used as first- or second-line steroid-sparing therapies in severe disease. Interferon- α has also demonstrated efficacy in refractory BU, although its use remains limited in our clinical settings.

More recently, targeted biologic and small-molecule therapies have emerged as potential options for refractory disease. Tocilizumab has demonstrated benefit in selected patients, particularly those with persistent ocular inflammation despite conventional immunosuppression. Janus kinase (JAK) inhibitors have also shown encouraging results in refractory BU, although evidence remains relatively limited compared with that for established biologic therapies. Further studies are required to define their optimal positioning within the treatment algorithm.



The decision to start immunosuppression goes beyond selecting the appropriate drug.

The assessment of infection risk is particularly important before initiating and while receiving systemic immunomodulatory therapy, especially high-dose corticosteroids and anti-TNF- α therapy, or other potent immunosuppressive agents.

In regions where tuberculosis is prevalent, such as India, evaluation for latent or active tuberculosis is an essential component of treatment planning. Social and living conditions may further influence infection exposure and the feasibility of infection surveillance. Overcrowded and poorly ventilated living environments can increase exposure to tuberculosis and respiratory infections. Assessments should include known tuberculosis exposure, chronic cough among household contacts, compliance with medications and infection monitoring.

There is also a higher risk of infection with higher doses of biologics. Using 5 mg per kg, with escalation to 7–10 mg per kg dosing of infliximab can lead to the development of disseminated infections.

These factors should be incorporated into the overall risk assessment when planning immunosuppressive therapy, particularly with biologic agents.

CONCLUSION

- Behçet uveitis remains one of the most vision-threatening manifestations of the disease.
- Early recognition of active inflammation, prompt initiation of appropriate steroid-sparing therapy, and meticulous long-term monitoring are essential to prevent irreversible retinal and optic nerve damage.
- Recognition of characteristic clinical features, combined with multimodal imaging, facilitates early diagnosis, objective assessment of disease activity, and timely treatment.
- The therapeutic landscape of BU has evolved considerably, with conventional immunosuppressive agents complemented by biologic therapies and, more recently, targeted small-molecule agents.
- Ultimately, a multidisciplinary and individualised approach remains central to achieving inflammatory control and preserving vision in patients with ocular Behçet's disease.





SPECIAL CONVERSATIONS

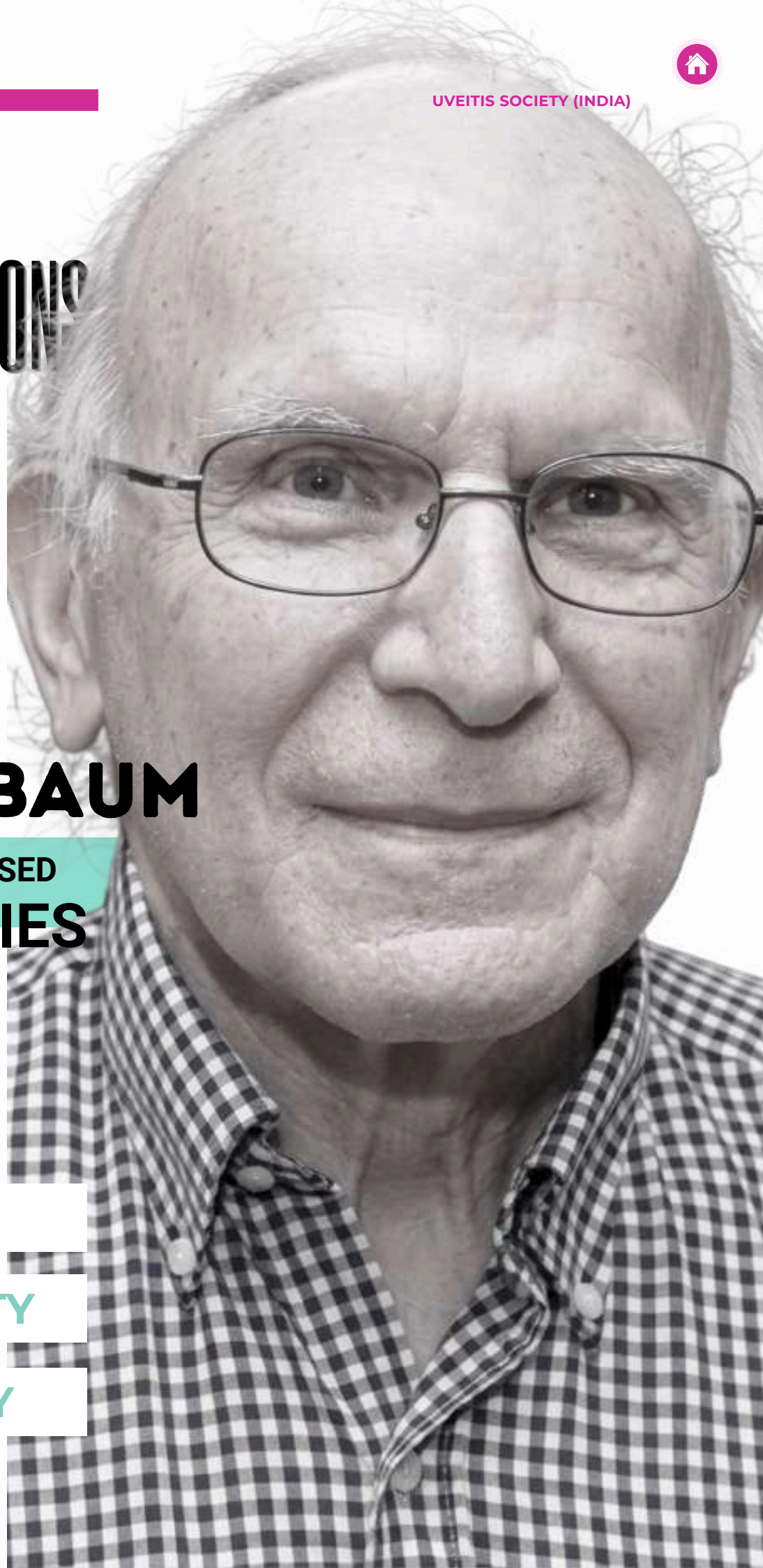
DR. JAMES ROSENBAUM

A LIFE THAT CROSSED
BOUNDARIES

CURIOSITY

UNCERTAINTY

SERENDIPITY





“The Best Experiments Are
Not Those That Prove Your
Hypothesis.”

A rheumatologist who became Chief of Ophthalmology, a scientist whose work has shaped our understanding of uveitis, and a researcher who believes some of the best experiments are those that prove us wrong. In this conversation with Dr. Parthopratin Dutta Majumder, Dr. James Rosenbaum reflects on serendipity, scientific curiosity, diagnostic uncertainty and the importance of following one's passion.

THE ACCIDENTAL OPHTHALMOLOGIST

You trained as a rheumatologist but went on to become Chief of Ophthalmology — an extraordinary crossing of traditional speciality boundaries. Looking back, was this journey carefully planned, or was it driven more by curiosity and opportunities that you chose not to ignore?

My interest in ophthalmology was accidental and not a plan. I always fumbled dismally with an ophthalmoscope. I had difficulty spelling “ophthalmology”. My best friend in medical school advised me to learn the mnemonic T-E-O-N to guarantee I would know all the ophthalmology necessary to succeed. The mnemonic stands for two eyes, one nose.



Medicine increasingly rewards specialization, yet your career seems to demonstrate the value of crossing boundaries. Do you think we are becoming too specialized—and, in doing so, sometimes losing sight of the patient as a whole?

Like so many things in life, the answer is yes and no. If I needed a corneal transplant, I'd like to go to the doctor who has done the most transplants with the most success. But if I had unexplained symptoms, I would want a doctor with broad knowledge. In the ideal world, we would have specialists, super specialists, and generalists.

SCIENCE, SERENDIPITY AND THE UNKNOWN

Your 700 publications are an extraordinary scientific journey. But if you had to choose one question you pursued, not necessarily your most cited paper, that gave you the greatest intellectual satisfaction, what would it be, and why?

Since 1978, I have been trying to

understand the mechanism by which HLA-B27 predisposes to anterior uveitis and spondyloarthritis. I wish I could say that my research solved the puzzle. I hope I contributed, but another group gets the most credit. And like all good science, the answers allow us to ask more questions.

Uveitis is a field in which uncertainty is almost unavoidable. After decades of studying and treating inflammatory diseases, has your relationship with diagnostic uncertainty changed? Are you more comfortable saying, "I don't know," today than you were as a young physician?

In some medical fields like ophthalmology and dermatology, diagnostic uncertainty exists but it is minimal compared to rheumatology, in which the correct diagnosis and the optimal therapy often have elements of uncertainty. Empowering the patient to participate in the decision-making is the approach most internists exercise to deal with these uncertainties. But this is a delicate course because a doctor also needs to encourage the patient to be confident in the expertise of the physician. With decades of



experience, I think that I have less uncertainty, but I am constantly humbled by how much I don't know. I am comfortable saying "I don't know", but I am more likely to say, "Let's consider getting a second opinion."

You described endotoxin-induced uveitis, an observation that became an important experimental model of ocular inflammation. Do you remember the question or observation that originally led you down that path? Was it hypothesis, curiosity, serendipity — or a mixture of all three?

I was a postdoc in Hugh McDevitt's lab at Stanford in Palo Alto, California. McDevitt and Baruj Benacerraf discovered that immune responses were under genetic control and predicted that these immune response genes would affect how likely one was to develop an immune-mediated disease. Ankylosing spondylitis was the first disease to confirm the hypothesis in 1973. My post-doc fellowship began 5 years later. I reasoned that an animal model would provide a tool to understand the relationship between HLA-B27 and disease. Since infections like Salmonella and Shigella can

trigger HLA-B27-related reactive arthritis, I began injecting the footpad of rats with killed Gram-negative bacteria. They were supposed to develop arthritis (they did not), but they did get uveitis, so serendipity was aided by an incorrect hypothesis and curiosity.

Incidentally, the best experiments are not those that prove your hypothesis. The best experiments usually are those that disprove your hypothesis and the results are reproducible. Disproving your hypothesis forces you to reshape how you understand a problem and leads to novel ideas.

BEYOND MEDICINE

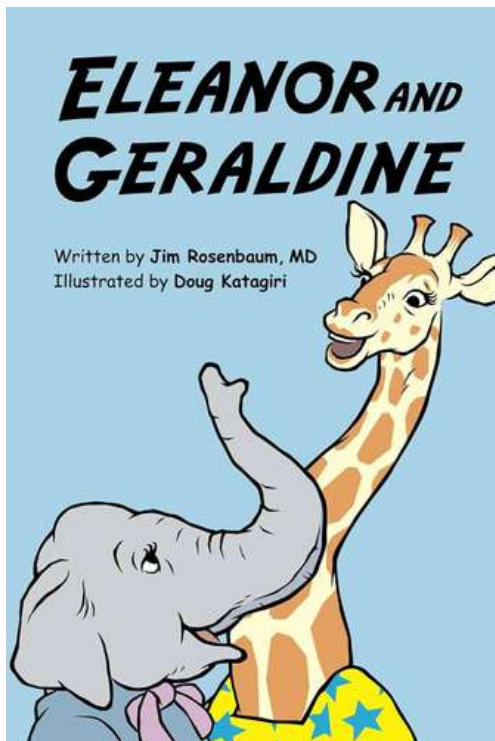
Your career includes Science, Nature, NEJM and hundreds of scientific papers—but also an illustrated children's book, Eleanor and Geraldine. What does writing for children give you that scientific writing cannot? And is creativity an important quality in a scientist?

Eleanor and Geraldine was based on a story that I made up for my daughters over 40 years ago. My



grandfather had made up stories for me and my brothers as we grew up. So storytelling and writing a children's book were just ways to preserve an oral history with the hope that it is worth preserving.

Creativity is a valuable quality for a scientist. But there are many better scientists than I am, and most have not written a children's book. My wife and I have two daughters. Both are physicians. Both are successful writers, one as the National Correspondent for the New England Journal of Medicine and one as a novelist who writes under a pseudonym.



You come from a remarkable family of physicians, and your father famously wrote “A Taste of My Own Medicine” after experiencing medicine from the patient’s side. Has your family’s experience of medicine influenced the kind of physician you became?

Of course, I did not get to choose my parents, but I am very fortunate. My father, Edward E Rosenbaum, was definitely a role model. He founded the division of rheumatology as a volunteer at what was then the University of Oregon Medical School; his autobiography, A Taste of My Own Medicine, became the 1991 Disney film The Doctor, starring William Hurt.

LOOKING BACK, LOOKING FORWARD

If you could meet the 30-year-old James Rosenbaum just beginning his academic career, what would you tell him to pursue more vigorously – and what would you tell him not to worry so much about?



Everyone should follow their passion. If going to work feels onerous, you should probably think about a different career.

I tell myself not to worry about many, many things. I give patients

the same advice. Too bad that we don't have the ideal pill to reduce anxiety and make worry go away. But on the other hand, that same anxiety plays a huge role in motivating us to solve the challenges of life.



The Rosenbaum family celebrating Ed Rosenbaum's 80th birthday, Portland, Oregon, 1995. The pictured group includes 7 MDs, 2 lawyers, 2 MDs to be, and one lawyer to be. Six in the group have authored books or monographs.



Snapshot

A BULLOUS SYMPHONY

1

Fundus photo showing striking bilateral bullous exudative retinal detachments with ILM folds radiating from the posterior pole in a 24-year-old lactating woman presenting with severe headache, vomiting, and bilateral visual loss of 15 days duration. OCT confirmed multiple serous retinal detachments with diffuse choroidal thickening, and a diagnosis of acute Vogt-Koyanagi-Harada (VKH) disease was established after excluding infectious and neurological causes. Prompt treatment with high-dose corticosteroids, immunomodulatory therapy, and antiglaucoma medications was initiated.

*Dr. Abhigna Nagendra
Sankara Eye Hospital
Bengaluru, Karnataka*

Teaching point: Acute VKH can present during lactation with a prodrome of severe headache. Bilateral bullous exudative retinal detachments are a distinctive diagnostic clue, and recognising this pattern even after delayed presentation allows timely immunosuppression with the potential for excellent visual recovery.



2

**WHEN THE SERPENT
REACHES THE MACULA**

*Dr. Dhaivat Shah
Dr. Nicey Roy Thomas
Ahalia Eye Care, Abu Dhabi
United Arab Emirates*

A 30-year-old male presented with a history of multiple recurrent attacks of serpiginous choroiditis. Unfortunately, by the time he reached us, the disease had already involved the macula, resulting in significant photoreceptor and outer retinal damage. Ultra-widefield Optos imaging revealed bilateral chronic chorioretinal lesions extending from the optic disc in a classic serpiginous (snake-like) pattern. Optical coherence tomography (OCT) demonstrated outer retinal loss, while fundus autofluorescence (FAF) showed dense hypo-autofluorescence, indicating irreversible retinal pigment epithelium (RPE) and photoreceptor loss. He was treated with pulse-dose corticosteroids and subsequently maintained on immunosuppression with mycophenolate mofetil 500 mg twice daily. He has remained stable for 2 years with follow-up every 3 months, focusing on early detection of reactivation and prevention of further progression.

Teaching point: Early diagnosis and aggressive immunomodulation are crucial to prevent macular involvement. Multimodal imaging helps differentiate active inflammation from healed destructive lesions. Long-term surveillance is essential due to the recurrent nature of serpiginous choroiditis and the risk of irreversible outer retinal damage.



Case ARENA

A SUBRETINAL WINDOW TO SYSTEMIC MAYHEM

Dr. Deepanshu Agrawal

Dr. Mehendi Nagda

Dr. Arun Bhargava

Retina Speciality Hospital, Indore

INTRODUCTION

Ocular metastasis is the most common intraocular malignancy in adults and usually results from hematogenous dissemination of an advanced systemic cancer.¹ Although uncommon in routine ophthalmic practice, its recognition is of immense clinical importance as it may be the first manifestation of an otherwise occult primary malignancy. The rich vascular supply of the uveal tract, particularly the choroid, makes it the predominant site of metastatic involvement, accounting for nearly 85-90% of all intraocular metastases.

CASE REPORT

A 62-year-old male presented with unilateral painless diminution of vision in the left eye. The patient was diagnosed with choroidal tuberculoma owing to a positive Mantoux and QuantiFERON TB Gold test elsewhere and presented for a second opinion. Visual acuity was 20/20 and 20/200 in the right (OD) and left (OS) eyes, respectively. OD was clinically

unremarkable. Fundus examination of OS revealed a large subretinal mass. Fundus fluorescein angiography revealed early hypofluorescence with late hyperfluorescence and associated pinpoint leakage along the margin of the lesion. SD-OCT through the lesion showed involvement of the choroidal stroma along with outer retinal layer irregularities and retinal pigment epithelium thickening. B-scan demonstrated a lesion in the superotemporal quadrant measuring 15.84 mm × 5.72 mm horizontally and vertically.

With suspicion of ocular metastasis, considering the abnormal morphology of the lesion for a tuberculoma, radiological imaging was advised. HRCT chest revealed a soft tissue density lesion with subtle post-contrast enhancement noted in the left upper lobe measuring about 25 × 24 mm. Also noted were subcentimetric-sized mediastinal lymph

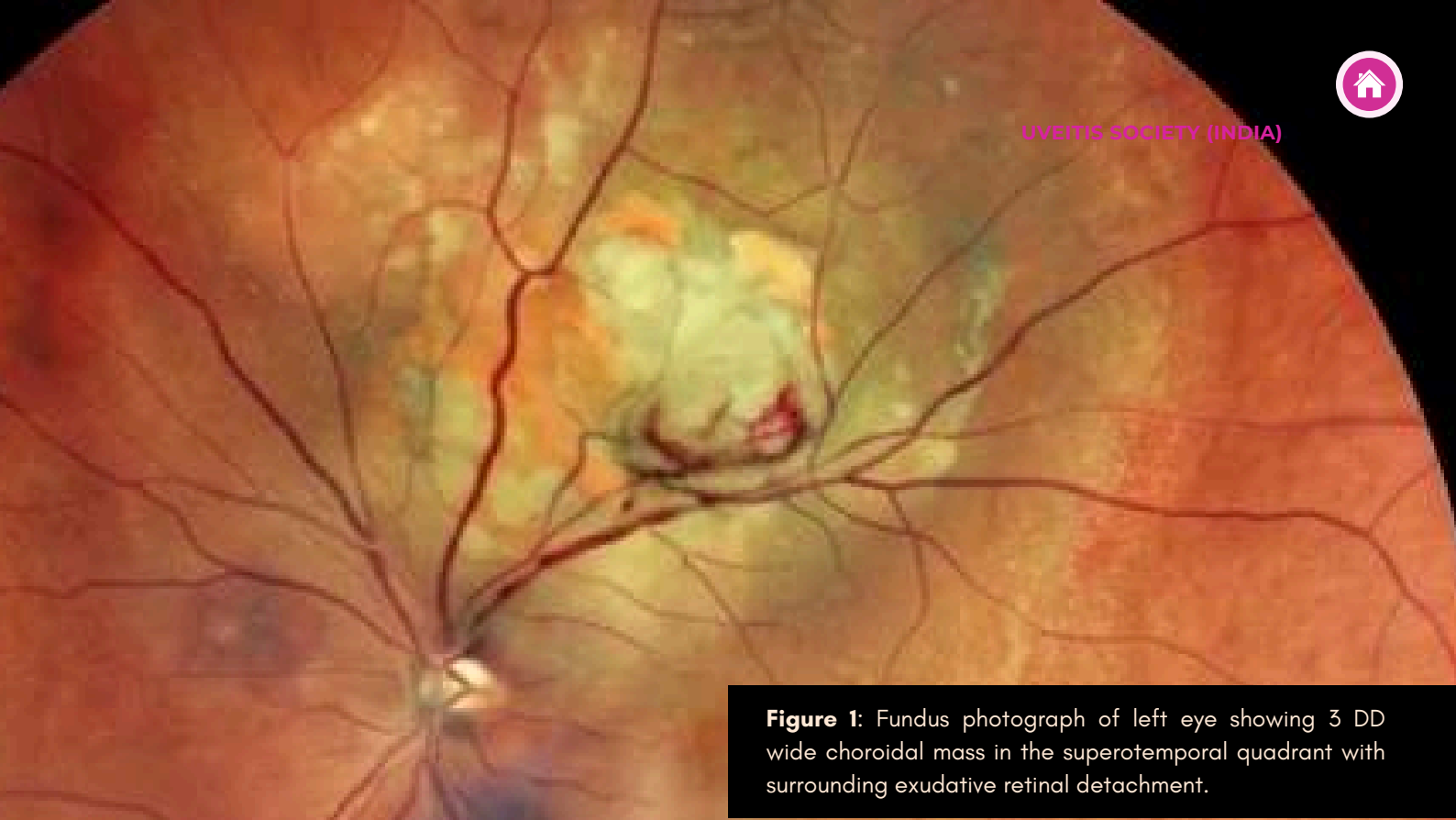


Figure 1: Fundus photograph of left eye showing 3 DD wide choroidal mass in the superotemporal quadrant with surrounding exudative retinal detachment.

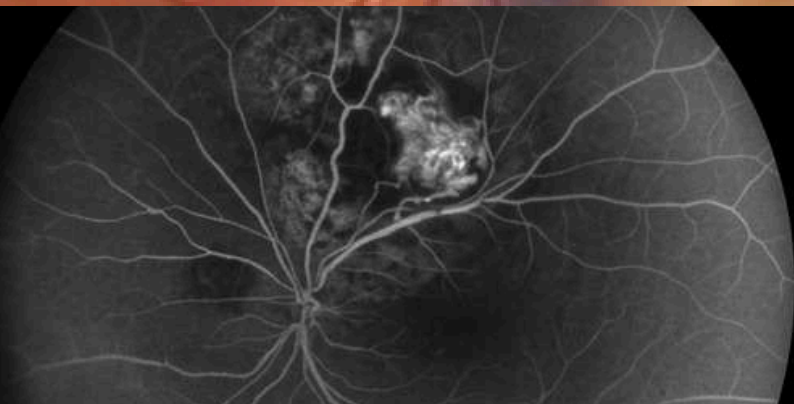


Figure 2: FFA - Early hypofluorescence with late hyperfluorescence, along with pinpoint leakage along the margin of the lesion.

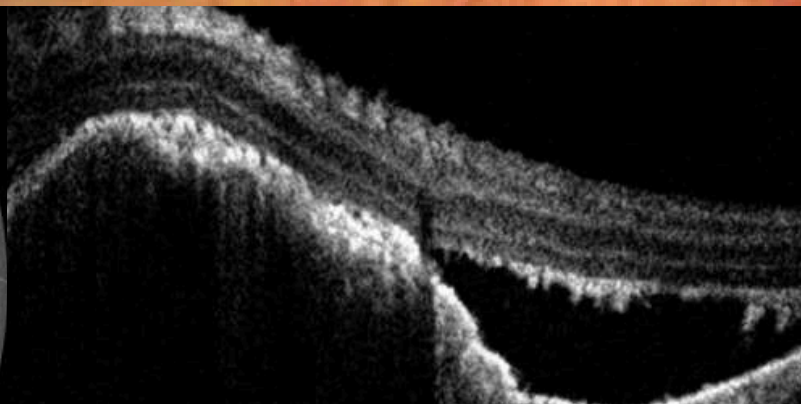


Figure 3: SD-OCT passing through the lesion shows involvement of choroidal stroma, outer layer irregularities with associated RPE thickening and mounting.

nodes and multiple randomly distributed soft tissue density nodules bilaterally. Following this, PET-CT was performed, which showed FDG-avid uptake in the left lung with variable uptake by the lung nodules, mediastinal lymph nodes, left eyeball lesion, and a bone marrow lesion in the right humerus. Pulmonology and oncology opinions were taken. Left lung lesion biopsy on histopathology was suggestive of non-small cell carcinoma. On immunohistochemistry, TTF1, Napsin A, and EGFR markers were positive, and

a diagnosis of adenocarcinoma of the lung was made. The patient is currently undergoing chemotherapy.

DISCUSSION

Ocular metastases occur in approximately 8-10% of patients with systemic cancer. Among intraocular metastatic lesions, the choroid is involved in approximately 88% of cases, followed by the iris (about 9%) and ciliary body (approximately 2%). The preferential involvement of the choroid



is attributed to its dense vascular network supplied by the posterior ciliary circulation. Breast carcinoma is the leading primary tumour responsible for ocular metastasis overall, accounting for approximately 40–50% of cases, followed by lung carcinoma, which contributes around 20–30%.² An important clinical aspect is that ocular metastasis may precede the diagnosis of the primary malignancy.

Lung carcinoma deserves particular attention because of its aggressive biological behaviour and propensity for early hematogenous spread. Adenocarcinoma is the most common histological subtype associated with ocular metastasis, followed by small-cell lung carcinoma.³ Ocular metastasis from lung cancer may occur even in patients with minimal respiratory symptoms, making ocular examination the first clue to disseminated disease.

Consequently, every patient presenting with a suspicious choroidal lesion without a known primary malignancy should undergo a thorough systemic evaluation, including chest imaging, as lung carcinoma remains one of the most common occult primary sources.

This case emphasises that lung carcinoma may present to the ophthalmologist in diverse ways, either as true ocular metastasis or as a neoplastic ocular masquerade syndrome. In tuberculosis-endemic countries, positive immunological tests such as Mantoux or QuantiFERON-TB Gold frequently indicate latent infection and should not be considered diagnostic of ocular tuberculosis in isolation. Consequently, supportive laboratory and radiological investigations should always be interpreted alongside the clinical presentation, multimodal ocular imaging, and response to treatment.





SUPRACHOROIDAL FLUID ON SWEEP SOURCE AS-OCT: DIAGNOSING POSTERIOR SCLERITIS MASQUERADING AS PRESEPTAL CELLULITIS

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INTRODUCTION

Posterior scleritis is an uncommon but potentially vision-threatening inflammatory disease that frequently masquerades as episcleritis, orbital cellulitis, acute angle closure attack or other ocular disorders, often delaying diagnosis. Although B-scan ultrasonography remains the cornerstone of diagnosis, advances in multimodal imaging, including swept-source anterior segment OCT (SS AS-OCT), have enabled detailed visualization of scleral and choroidal changes; however, incidental detection of suprachoroidal fluid on AS-OCT has rarely been reported. We report a case of diffuse anterior and posterior scleritis in which AS-OCT incidentally demonstrated suprachoroidal fluid, providing an additional imaging clue that complemented conventional multimodal imaging.

CASE REPORT

A 30-year-old woman presented with recurrent redness, periocular swelling and pain on ocular movements in the left eye for five days. She had experienced similar episodes over the preceding three months and had initially been diagnosed

elsewhere as episcleritis and later as presumed preseptal cellulitis based on magnetic resonance imaging (MRI) findings. The patient was treated with topical corticosteroids for episcleritis and then systemic antibiotics for suspected preseptal cellulitis after which the symptoms subsided and inflammation resolved as per records. One month later the patient had a recurrence of redness and pain in the left eye. Extensive systemic evaluation including HIV, treponema pallidum hemagglutination assay (TPHA), antinuclear antibody (ANA), anti-neutrophil cytoplasmic antibodies (ANCA), rheumatoid factor and serum angiotensin-converting enzyme (ACE) were all non-contributory. Mantoux was positive with a negative interferon-gamma release assay (IGRA).

At presentation, best corrected visual acuity (BCVA) was 6/6 in both eyes. Examination revealed mild episcleral congestion with localised superior scleral tenderness. Initial B-scan ultrasonography was unremarkable, and she was treated with topical loteprednol. Five days later, she returned with worsening pain and reduced vision (6/18) in the left eye (Figure 1). Fundus examination demonstrated optic disc

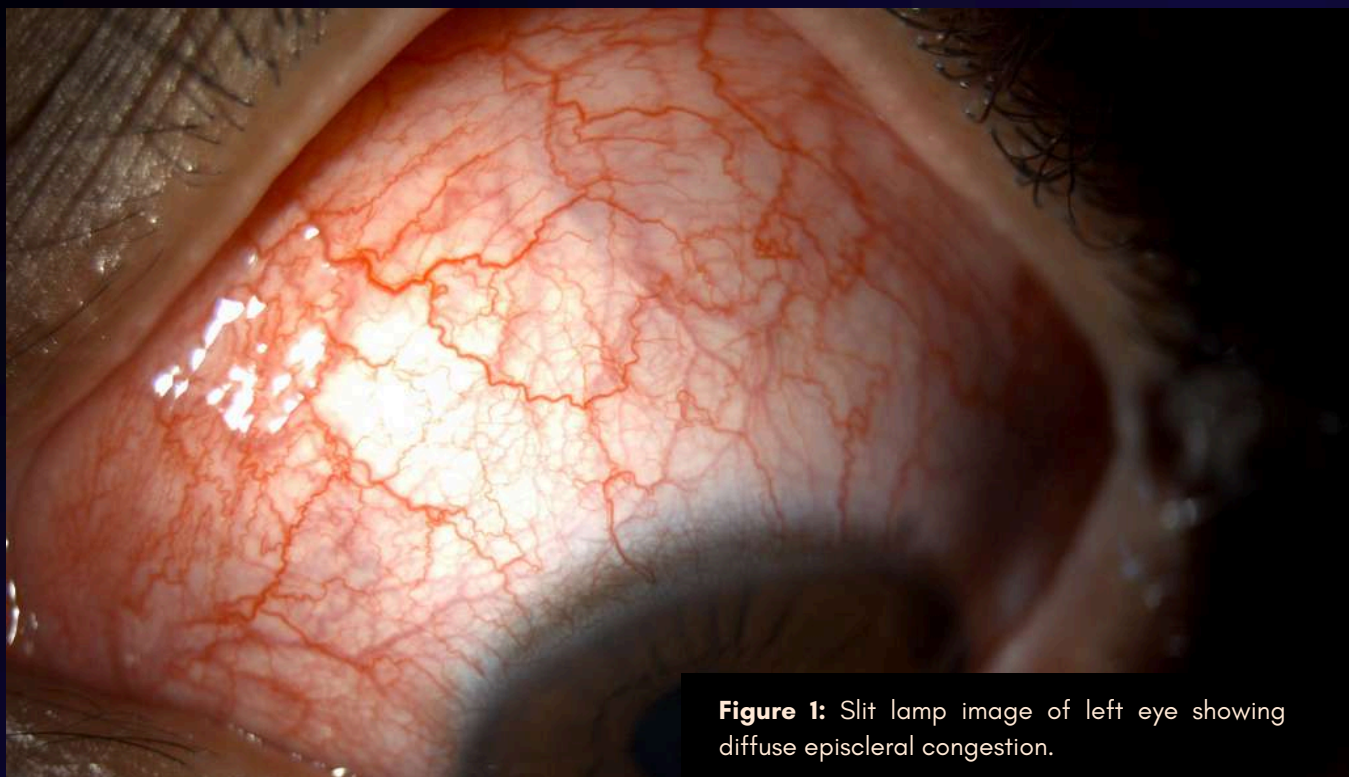


Figure 1: Slit lamp image of left eye showing diffuse episcleral congestion.

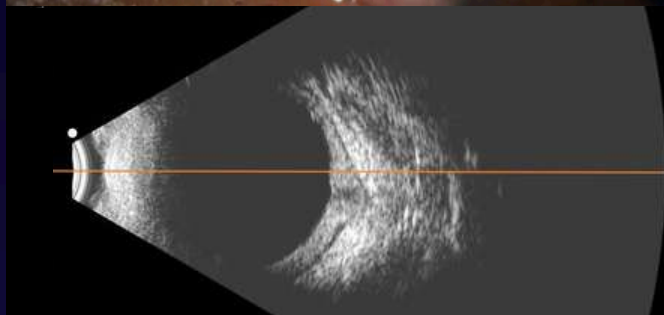


Figure 2: B-scan of left eye demonstrating sub-Tenon's fluid (T-sign) and increased retinochoroidal-scleral thickness (2.2 mm).

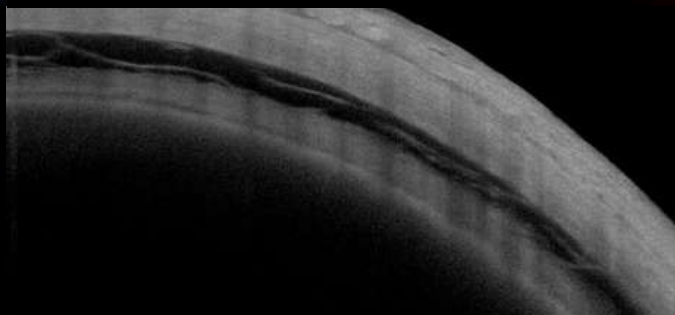


Figure 3: Anterior segment OCT performed over the superior sclera demonstrating intralamellar hyporeflective clefts within the thickened sclera together with a distinct hyporeflective space immediately beneath the sclera, consistent with suprachoroidal fluid accumulation.

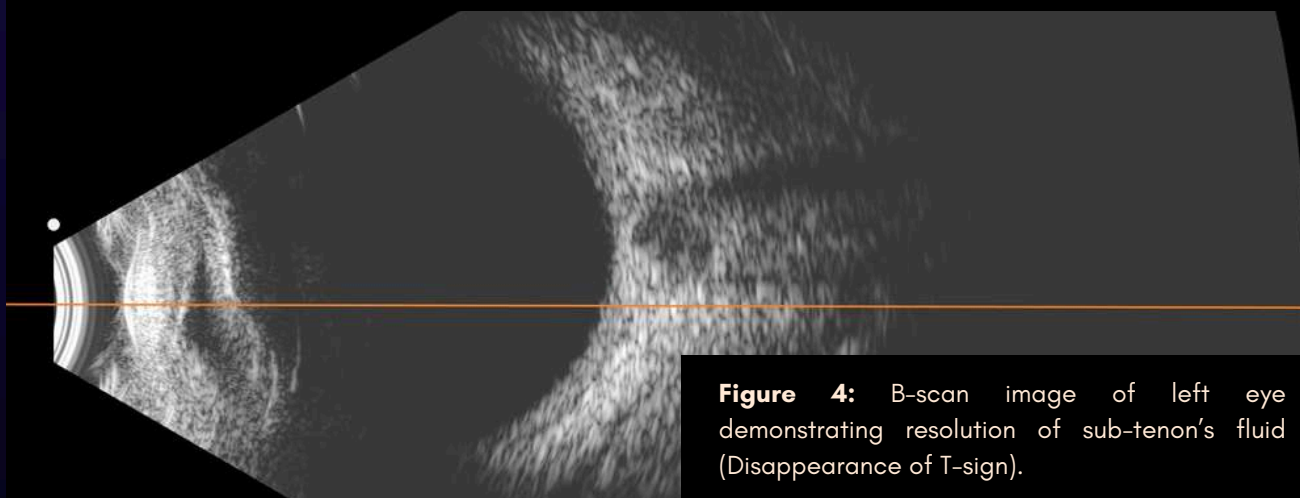


Figure 4: B-scan image of left eye demonstrating resolution of sub-tenon's fluid (Disappearance of T-sign).

hyperemia with nasal internal limiting membrane folds. OCT showed diffuse choroidal thickening (subfoveal choroidal thickness approximately 675 μm) without subretinal fluid. Repeat B-scan demonstrated sub-Tenon's fluid (T-sign) and increased retino-choroidal-scleral thickness (2.2 mm) (Figure 2).

Swept-source anterior segment OCT performed over the superior and temporal sclera demonstrated intralamellar hyporeflective clefts within the thickened sclera together with a distinct hyporeflective space immediately beneath the sclera, consistent with suprachoroidal fluid accumulation (Figures 3 and 4). These findings were noted incidentally while evaluating anterior scleral inflammation. Combined with the clinical findings and ultrasonography, a diagnosis of diffuse anterior and posterior scleritis was established.

The patient was treated with intensive topical prednisolone acetate 1% and oral analgesics. One week later, pain resolved completely, visual acuity improved to 6/6, episcleral inflammation subsided, choroidal thickness reduced, and repeat ultrasonography demonstrated complete resolution of sub-Tenon's fluid. Oral corticosteroids were therefore avoided.

DISCUSSION

Posterior scleritis continues to represent a diagnostic challenge because its presenting features frequently overlap with orbital inflammatory disease, cellulitis and isolated episcleritis, resulting in delayed diagnosis.^{1,2} B-scan

ultrasonography remains the imaging gold standard, although characteristic findings may be absent during the early phase of disease, as demonstrated in our patient.³ Recent studies have shown that anterior segment OCT can objectively demonstrate scleral oedema, increased scleral thickness and treatment response in anterior scleritis.⁴ In our patient, SS AS-OCT imaging additionally revealed a discrete hyporeflective suprachoroidal space that corresponded to inflammatory suprachoroidal fluid, a finding that has rarely been highlighted in the literature. Recognition of this imaging sign, together with intralamellar scleral oedema, may provide an additional clue toward diagnosing posterior scleritis, particularly when ultrasonographic findings are equivocal or evolving.⁵ This case also demonstrates that multimodal imaging can prevent repeated misdiagnosis and unnecessary antibiotic therapy while facilitating prompt anti-inflammatory treatment with excellent visual recovery.⁶

CONCLUSION

This case underscores the potential role of SS AS-OCT as an adjunctive imaging modality in posterior scleritis, with incidental visualisation of suprachoroidal fluid serving as a novel diagnostic clue.



THE GUT — AXES —

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"All disease begins in the gut."

*-Hippocrates of Kos
(Hippokrátēs ho Kóos: c. 460–c. 370 BCE)*

Twenty-four centuries later, science is now testing how far that claim reaches.

The human gut microbiome, often referred to as "the second genome of the human body", comprises a myriad of microorganisms including bacteria, viruses, protozoa, fungi and archaea, with bacteria being dominant in terms of number, functions as well as genetic entities inhabiting the gastrointestinal tract.

Having co-evolved with the human host over a billion years of colonization, the gut microbiome carries out three core

physiological functions:

- (1) conferring structural defence against pathogenic colonization,
- (2) facilitating vital nutrient acquisition, and
- (3) maintaining a dynamic, reciprocal dialogue with the host immune system.

It is this third role that is now redrawing the map of ocular immunity.



Recent work describes a “*gut-eye axis*”¹:

a bi-directional communication channel
through which gut microbes act as a
primary gatekeeper of ocular immune
homeostasis.

For uveitis specialists, it supplies a long-missing piece of the puzzle: a plausible reason why so many cases flare without any identifiable trigger.

THE MECHANISMS OF OCULAR DYSBIOSIS

In healthy individuals, the gut microbiota ferments dietary fibre into beneficial short-chain fatty acids (SCFAs), such as butyrate and propionate, which strengthen the intestinal barrier and promote the maturation of anti-inflammatory regulatory T cells (Tregs).¹ When this balance is disrupted, a state of dysbiosis occurs, SCFAs fall, and pro-inflammatory Th17 and cytokines rise, leaving patients vulnerable to systemic inflammation via four complementary mechanisms:

1 Molecular Mimicry: Structural cross-reactivity between retinal self-antigens and microbial peptides results in generations of autoreactive T cells in the gut prompting effector T cells to traffic from the intestine straight into the uveal tissue.²

2 Immune Imbalance: Loss of protective bacteria leads to upregulation of pro-inflammatory Th1, Th17 cells and IL-17 and a corresponding downregulation of anti-inflammatory Tregs and IL-10, collectively fostering aberrant immune response.²

3 The “Leaky Gut”: Increased intestinal permeability allows highly immunogenic bacterial components, such as lipopolysaccharides (LPS), to escape into the systemic circulation, driving downstream ocular inflammation.²

4 Loss of protective metabolites: A significant drop in SCFA-producing bacteria strips the host of critical localized immune tolerance mechanisms.

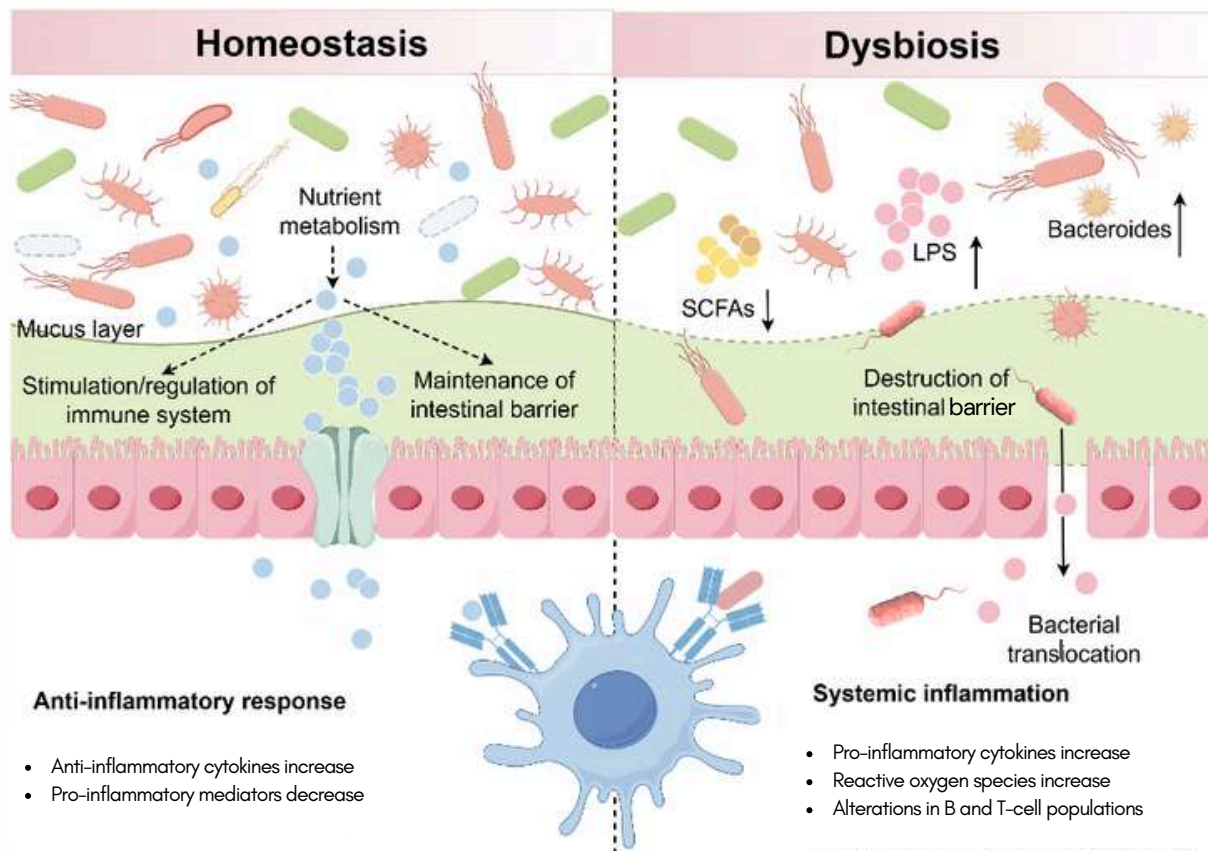


Figure 1: Intestinal microbiota in states of homeostasis and dysbiosis. (LPS-lipopolysaccharide, SCFAs-short-chain fatty acids) (Figure taken from Gao et al., 2026)

INSIGHTS FROM THE INDIAN COHORT

Pioneering microbiome mapping conducted on a South Indian patient cohort provided the first definitive evidence of distinct dysbiosis in Indian uveitis patients compared to healthy human subjects.³ This emerging paradigm holds profound clinical relevance for clinicians practising in the Indian subcontinent.

The study demonstrated a sharp reduction in microbial diversity and a notable depletion of protective, anti-inflammatory genera—such as *Faecalibacterium*, *Bacteroides*, *Lachnospira*, and *Ruminococcus*—alongside an enrichment of pathogenic, pro-inflammatory organisms like *Prevotella* and *Streptococcus*.³

This distinct structural shift suggests that deep-seated alterations in the gut profile may directly exacerbate or potentially trigger idiopathic intraocular inflammation within the unique clinical population.⁴

TRANSLATIONAL HORIZONS: RESTRUCTURING THE GUT TO PROTECT THE EYE

Many autoimmune conditions including Vogt-Koyanagi-Harada disease, Behçet's disease, systemic sarcoidosis, HLA-B27-associated uveitis, ankylosing spondylitis (AS), multiple sclerosis (MS), and inflammatory bowel disease (IBD) have now been linked to gut microbiome dysbiosis. Experimental studies suggest that resetting a maladaptive dysbiosis



can significantly quieten intraocular inflammation. Recognising the gut-eye axis highlights an exciting focus towards novel preventive and therapeutic

approaches alongside the conventional corticosteroid and systemic immunosuppressive therapies.

Targeted Strategy	Proposed Mechanism of Action	Clinical Relevance / Impact
Dietary Intervention and Fiber	Boosts microbial fermentation, rapidly elevating internal protective SCFA pools	Restores natural intestinal barrier integrity, diminishing baseline "leaky gut" leaks
Exogenous Probiotics	Corrects localized microbial imbalances while reinforcing the mucosal tight junctions	Restores structural baseline health, mitigating the systemic tracking of toxic LPS
Immunomodulators	Directly suppress ocular inflammation and exert indirect therapeutic effects by modulating the gut microbiome	Effective in non-infectious uveitis cases by specific modification of gut microbiome
Selective Antimicrobials	Selectively targets and removes specific pro-inflammatory, cross-reactive bacterial lines	Modulates T-lymphocyte subsets in extra-intestinal lymphoid tissues
Fecal Microbiota Transplant (FMT)	Introduces complete, stable, functional microbial communities from healthy donors	Effectively re-establishes long-term, systemic immune tolerance

These evolving therapies are changing the way we think about uveitis. In the future, evaluating a patient's overall gastrointestinal wellness, metabolic health, and dietary profile may soon

become just as routine as ordering an HLA-B27 test or a chest X-ray.

The next frontier in uveitis may not lie inside the eye—but inside the gut.





OCULAR FLUID BIOMARKERS IN UVEITIS

LOOKING BEYOND THE
CLINICAL FINDINGS

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A patient with recurrent panuveitis does not respond to corticosteroids. Is this really autoimmune uveitis—or are we missing an infection or even lymphoma?

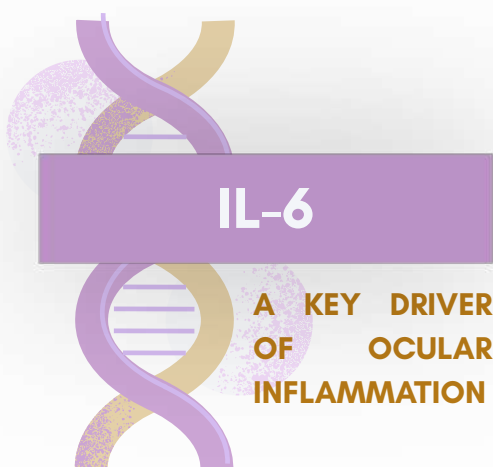
Increasingly, the answer lies not in another blood test, but in a few drops of aqueous humour.

Ocular fluid biomarkers are increasingly changing the way we diagnose and manage uveitis. The aqueous and vitreous humour act as a "liquid biopsy" of the eye, providing a direct window into the local inflammatory environment through analysis of cytokines, antibodies, nucleic acids, proteins and other molecules that are often not reflected by systemic investigations alone.

Interleukin-6 (IL-6) contributes to blood-retinal barrier breakdown, inflammatory cell recruitment and persistent ocular inflammation.

Elevated aqueous IL-6 levels have been consistently reported in Behçet's uveitis, Vogt-Koyanagi-Harada (VKH) disease, HLA-B27-associated uveitis and other non-infectious uveitis.

The success of IL-6 inhibitors such as tocilizumab further highlights its importance as both a biomarker and therapeutic target.





TNF - α

A BIOMARKER
THAT BECAME A
THERAPEUTIC
TARGET

Tumour necrosis factor-alpha (TNF- α) has been demonstrated to have a central role in ocular inflammation. Elevated intraocular levels have been noted in Behçet's uveitis, VKH disease and other active non-infectious uveitis.

The effectiveness of adalimumab in non-infectious uveitis highlights the role of TNF- α as both a biomarker of disease activity and a therapeutic target.



IL-8 , MCP

MARKERS OF
INFLAMMATORY
CELL
RECRUITMENT

Interleukin-8 (IL-8) is a potent neutrophil chemoattractant and reflects acute neutrophilic inflammation. It is consistently elevated in Behçet's uveitis, HLA-B27-associated acute anterior uveitis, and cytomegalovirus (CMV) anterior uveitis.

Monocyte chemoattractant protein-1 (MCP-1) on the other hand, recruits monocytes and macrophages, reflecting chronic monocyte-driven inflammation. It has been shown to be increased in Vogt-Koyanagi-Harada disease, juvenile idiopathic arthritis-associated uveitis, idiopathic intermediate uveitis, and ocular toxoplasmosis.



PCR

TRANSFORMING
THE DIAGNOSIS
OF INFECTIOUS
UVEITIS

Not all ocular biomarkers are inflammatory proteins. **Polymerase chain reaction (PCR)** analysis of aqueous or vitreous samples for detecting pathogens such as herpes simplex virus, varicella zoster virus, cytomegalovirus and *Toxoplasma gondii* has become an invaluable tool for diagnosing infectious uveitis. In selected cases, Goldmann-Witmer coefficient testing and pathogen-specific antibody analysis provide complementary evidence, enabling timely diagnosis and targeted antimicrobial therapy.



IL-10

WHEN "UVEITIS"
ISN'T UVEITIS

Perhaps no biomarker has changed clinical decision-making as dramatically as **Interleukin-10 (IL-10)**.

Primary vitreoretinal lymphoma produces high intraocular IL-10 levels, and an increased IL-10/IL-6 ratio strongly supports the diagnosis of this important masquerade syndrome while helping distinguish it from chronic inflammatory uveitis.

PROTEOMICS

FINDING DISEASE-
SPECIFIC
MOLECULAR
SIGNATURES

Proteomics has expanded the search for novel ocular biomarkers through large-scale protein analysis of aqueous and vitreous humour. For instance in Vogt-Koyanagi-Harada (VKH) disease, increased expression of complement proteins C1QB and C1QC suggests activation of the complement pathway.

Although these findings remain largely confined to research settings, they are improving our understanding of disease mechanisms and may eventually aid diagnosis, prognostication and treatment selection.

EXOSOMES

TINY
MESSENGERS
WITH ENORMOUS
POTENTIAL

Exosomes are nano-sized vesicles present within the aqueous and vitreous humour that carry proteins, lipids, messenger RNA and microRNA, making them valuable indicators of intraocular immune environment.

They are therefore being investigated as biomarkers for disease activity, treatment response and early detection of relapse and represent one of the most promising areas in ocular biomarker research.



Biomarker	Clinical relevance
IL-6	Active inflammation
TNF-α	Therapeutic target
IL-8	Acute inflammation
MCP-1	Chronic inflammation
PCR	Infectious uveitis
IL-10	Primary vitreoretinal lymphoma

When Should You Consider Ocular Fluid Analysis?

- **Diagnostic uncertainty**
- **Treatment-resistant uveitis**
- **Suspected infectious uveitis**
- **Chronic or recurrent uveitis**
- **Suspected masquerade**

Cytokine profiling, PCR-based pathogen detection and IL-10 analysis are increasingly being incorporated into routine clinical practice. The next generation of ocular fluid biomarkers is expected to move beyond individual cytokines towards disease-specific molecular signatures, including complement proteins (C1QB/C1QC), exosomal proteins and microRNAs, and metabolomic profiles. These emerging biomarkers, identified through multiomics approach, may allow earlier diagnosis, more accurate disease stratification, prediction of treatment response and truly personalised management of uveitis.



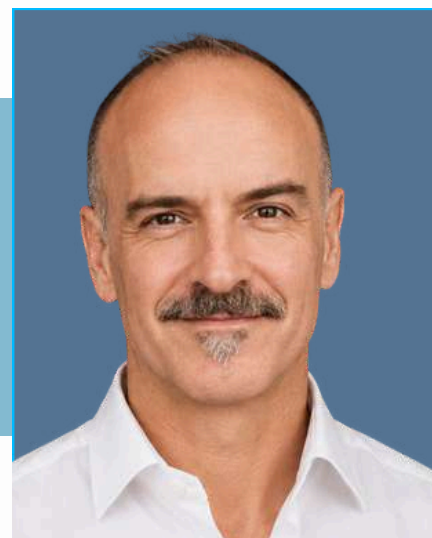


THE AUGMENTED EYE

HOW ARTIFICIAL
INTELLIGENCE,
MULTIMODAL IMAGING
AND
PREDICTIVE ANALYTICS
ARE REDEFINING THE
FUTURE OF UVEITIS CARE



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Artificial intelligence is no longer waiting outside the uveitis clinic. It is already converting images into measurements, patterns into probabilities, and longitudinal records into actionable trajectories.

A GLOBAL CLINIC IS THE PERFECT TEST

The question is no longer whether AI will enter uveitis practice, but

whether uveitis specialists will shape it with the clinical discipline this heterogeneous disease demands.

Practising uveitis in Abu Dhabi offers an unusually clear window into both the promise and the limitations of artificial intelligence. On the same clinic list, I may see inflammatory eye disease shaped by different genetic backgrounds, infectious exposures, healthcare systems, and treatment histories. Tuberculosis, toxoplasmosis, Behçet disease, sarcoidosis, viral anterior uveitis, and immune-mediated disease do not occur in equal proportions everywhere. An algorithm trained in one region cannot be assumed to understand them all.



What excites me is not a machine that offers a faster label. It is a system that helps us detect change earlier, ask better questions, and protect vision with greater consistency across very different patients.

This matters because uveitis is not a single disease. It is a family of inflammatory conditions in which the same visible sign can point to infection, autoimmunity, malignancy, trauma, or a masquerade syndrome. The experienced clinician does more than recognise a pattern. We consider timing, laterality, anatomy, systemic clues, prior treatment, and what is unexpectedly absent. AI should be judged by whether it improves that reasoning, not by whether it can confidently label an image.

WHEN IMAGES BECOME MEASUREMENTS

For more than two decades, I have watched ophthalmic imaging evolve from documentation to decision-making.

OCT made macular oedema measurable. Enhanced-depth and swept-source imaging revealed a new view of the choroid. Widefield angiography changed how we think about peripheral vascular leakage.



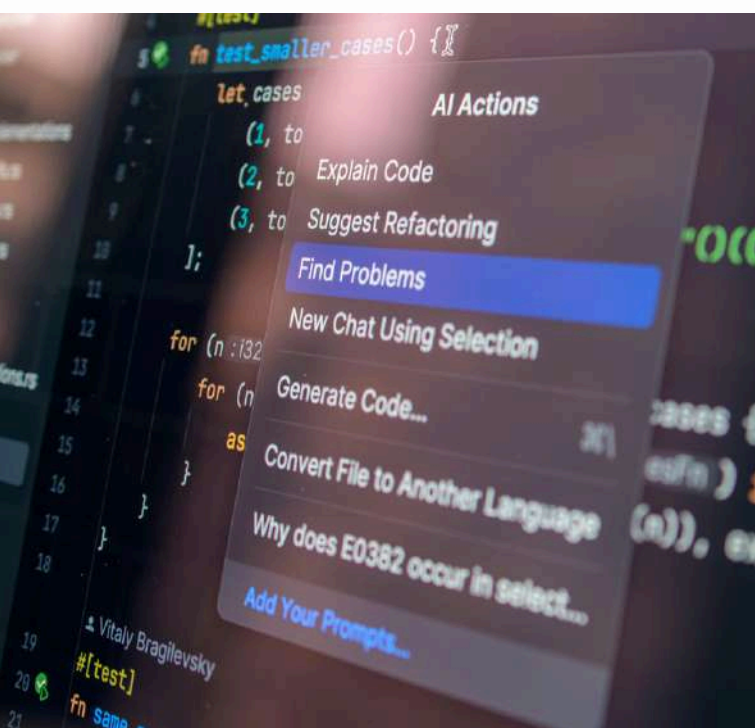


AI is the next step, but it is different. Earlier technologies gave us better pictures. AI can interrogate those pictures, compare them over time, and detect changes that may not yet be obvious to us.

Objective measurement offers the most practical opportunity. Anterior chamber cells and vitreous haze remain central to uveitis care, yet grading varies between observers and even within the same observer on different days. AI-assisted analysis of anterior-segment OCT can count hyperreflective particles and provide a repeatable estimate of anterior chamber inflammation [1]. OCT analysis can also quantify vitreous signal intensity as an objective marker of vitreous inflammation [2].

That may sound technical, but the clinical question is simple.

Is the eye genuinely quieter, or does it merely look quieter today?



A machine-readable measure could help us detect subtle responses, identify early recurrence, and distinguish inflammatory activity from permanent damage. In trials, it could make treatment effects easier to detect. In practice, it could support safer, more confident decisions.



ONE PATIENT, MANY IMAGES, ONE STORY

Uveitis is already a multimodal specialty. We combine OCT, fundus autofluorescence, fluorescein angiography, indocyanine green angiography, colour photography, ultrasound, and sometimes anterior-segment imaging. The problem is no longer a lack of information. It is the effort required to synthesise all that information and follow it over time.

The real advance will not be another isolated detector. It will be a system that recognises images as belonging to the same person, aligns them across visits, measures macular fluid, vascular leakage, choroidal change, vitreous signal, and structural loss, and then presents a trajectory rather than a stack of scans. That is where AI can become genuinely useful. It can remember every visit, while the clinician decides what the change means.

AI is a disciplined
second reader.

CLASSIFICATION HELPS, BUT IT IS NOT DIAGNOSIS

The SUN Working Group showed how computational methods can bring order to a complex field. By combining expert consensus with machine learning across 5,766 cases, the group developed classification criteria for 25 uveitides, achieving low overall misclassification in validation datasets [3].

This was an important achievement, demonstrating that expert-defined patterns could be converted into clear and testable rules.





Yet classification criteria are designed to create consistent research groups. They are not a substitute for diagnosis in an individual patient. A model may choose the most likely answer among the diseases it has learned and still fail when it encounters an unusual infection, a treatment-modified presentation, two diseases at once, or a masquerade syndrome. The risk is not simply a wrong answer. The greater risk is a confident answer that closes the diagnostic process too early.

I see AI as a disciplined second reader. It should rank possibilities, flag features that do not fit, retrieve relevant history, and show what informed its conclusion. A useful system might say that an image resembles Behçet uveitis while also warning that the age, tempo, or unilateral course is atypical. Such disagreement can sharpen clinical judgment. A black-box label cannot.

THE CLINICAL COPILOT

Now imagine opening a follow-up visit to find a concise timeline compiled from years of fragmented records. The system has matched serial scans, measured inflammatory markers, identified the corticosteroid dose at each turning point, and noted that two prior tapers below the same threshold were followed by recurrence. It suggests a monitoring interval and flags a missing safety test before immunomodulatory treatment. You can inspect every source and reject every inference.

This is where natural language processing and generative AI may add real value. Imaging is only one layer of the decision-making process. Clinical notes, laboratory results, medication exposure, molecular data, systemic disease, and patient-reported symptoms all contribute to the story. A well-designed copilot could summarise them, identify gaps, and reduce the risk that an important clue is buried in the record.

It may also help beyond specialist centres. Tele-imaging systems could identify patients who need urgent review, facilitate communication with referring clinicians, and monitor symptoms or adherence between visits. The goal is not to create a cheaper version of specialist care. It is to extend specialist judgment without pretending that every patient, camera, or population is the same.

CAN AI TELL US WHAT HAPPENS NEXT?

Prediction is the more ambitious frontier. We would all welcome an early warning of relapse, macular oedema, glaucoma, structural loss, or treatment toxicity. We would also value better guidance when choosing among local treatment, conventional immunosuppression, and biologic therapy. AI may eventually help estimate those risks and compare likely responses.

Longitudinal imaging could provide part of the answer. Small changes in



choroidal architecture, vascular leakage, vitreous signal, ellipsoid-zone integrity, or lesion autofluorescence may appear before conventional evidence of relapse. Combined with treatment exposure and laboratory trends, these signals might warn against tapering in an apparently quiet eye.

We must remain cautious. A prediction is useful only if it changes a decision and improves an outcome. Electronic records also contain hidden biases. The treatment an expert selects is not necessarily the best, and patients with more severe disease are treated more aggressively. Prospective studies must demonstrate that AI-guided care preserves vision, reduces toxicity, improves quality of life, or saves meaningful time. Accuracy alone is insufficient. The same principle applies to clinical trials, where AI could screen for eligibility, quantify endpoints, and identify patients most likely to progress or respond.

**Efficiency is
welcome, but
responsibility
must remain
visible.**

WHAT I NEED BEFORE I TRUST AI

First, the data must be multicentre, longitudinal, and reviewed by clinicians who understand the disease. A large archive of uncertain labels does not become reliable simply because it is large. Infectious diseases and masquerade syndromes must be represented, not treated as inconvenient exceptions.

Second, an algorithm that performs brilliantly in one hospital may struggle with another population or imaging device. It should be tested across centres, demographic groups, imaging clarity, disease prevalence, and uncommon presentations. I am more persuaded by prospective performance in the real clinic than by another impressive retrospective accuracy figure.

Third, if AI provides an answer, I want to know why it reached that answer, how confident it is, and what it may have missed. Human oversight is not a temporary compromise as the technology improves. In inflammatory eye disease, it is part of safe design.

Finally, privacy, cybersecurity, data ownership, evolving algorithms, and accountability require clear answers. A system that evolves after deployment must be monitored like any other clinical intervention. Efficiency is welcome, but responsibility must remain visible.



THE FUTURE STILL NEEDS A SPECIALIST

AI will make measurement, comparison, retrieval, and documentation easier. It will not make uveitis simple. As familiar patterns become easier to recognise, specialist value will concentrate on the difficult cases with conflicting evidence, rare phenotypes, systemic complexity, incomplete data, or serious consequences of error.

The uveitis specialist of the AI era must understand both inflammation and inference. We need to recognise an artifact, know when a biomarker reflects damage rather than activity, question a high-probability answer that is biologically implausible, and recognise when the model has never truly encountered a patient like the one in front of us. We also need to help design the datasets, endpoints, and clinical questions. Otherwise, technology will optimise what is easy to count rather than what matters to patients.

My conviction is clear:

"AI will not replace the uveitis specialist. It will sharpen the specialist's vision, amplify clinical judgment, and reveal what the human eye alone cannot yet see."





OCULAR THERMOGRAPHY

A NEW WINDOW INTO OCULAR INFLAMMATION

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Infrared thermography, once largely confined to military surveillance and industrial engineering, is gradually finding a niche in ophthalmology as a non-contact method for assessing ocular physiology and inflammation.

All tissues emit infrared radiation proportional to their temperature. Thermal cameras convert this radiation into colour-coded maps that reflect the surface temperature of tissues. In the eye, subtle changes in ocular surface temperature (OST) may provide clues to underlying inflammatory or vascular pathology.



The roots of ocular thermography date back to the 1950s and 1960s, when contact thermometry was initially used to estimate corneal temperature. A major advance came when Mapstone introduced infrared ocular thermography using a non-contact bolometer in 1968, demonstrating characteristic thermal patterns in anterior uveitis and other ocular disorders.¹

The ocular surface represents a dynamic thermal interface influenced by tear film stability, eyelid blinking, vascular perfusion, and intraocular inflammation. The central cornea is normally cooler because of its avascularity, while the nasal canthus tends to be warmer owing to rich vascular supply. Healthy OST usually ranges from 32–36°C, with minimal inter-ocular asymmetry (0.1–0.3°C; usually <0.6°C).² (Figure 1)

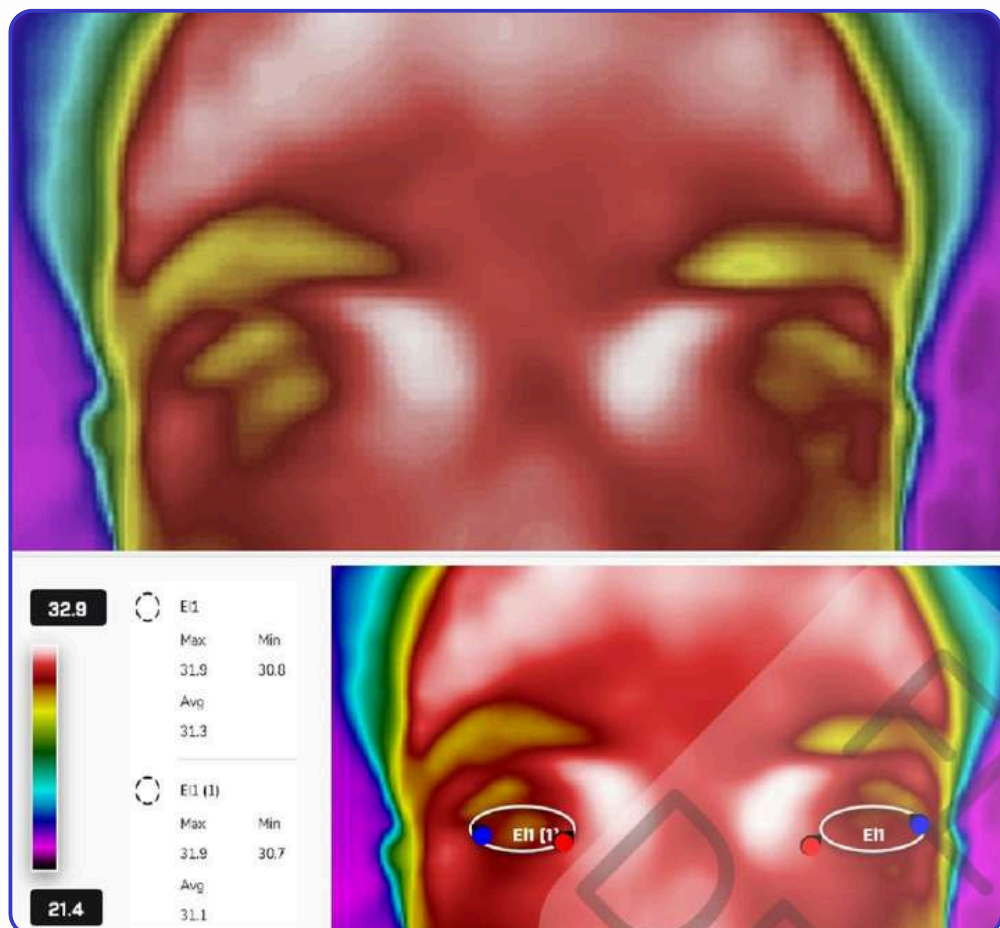


Figure 1: Ocular thermography image of a healthy 47-year-old volunteer demonstrates a symmetric thermal pattern with no significant interocular temperature difference. The average OST was 31.3°C in the right eye and 31.1°C in the left eye, consistent with normal physiological thermal distribution.



A temperature difference $>0.6^{\circ}\text{C}$ between eyes may suggest pathology.

Inflammation alters this thermal balance. Altered blood flow and increased metabolic activity can lead to measurable changes in the OST.

Earlier studies demonstrated elevated corneal temperatures in anterior uveitis and anterior scleritis, while more recent work has expanded its applications to posterior segment inflammation and orbital disease.^{3,4}

THE STAR APPLICATION

Among these, posterior scleritis is perhaps one of the most promising applications of ocular thermography.³ (Figure 2) Posterior scleritis can be diagnostically challenging because the anterior segment examination can be normal, fundus findings can mimic exudative retinal detachment, central serous chorioretinopathy, idiopathic choroidal folds, or choroiditis, and scleral thickening may at times be difficult to appreciate on B-scan ultrasonography.

In such situations, thermography can reveal increased OST compared with the fellow healthy eye, even when overt clinical signs are subtle. Interestingly, thermographic changes could also precede clinical relapse, suggesting a potential role in monitoring subclinical disease activity.⁵

The difference in thermal behaviour between scleral and choroidal tissues is also particularly intriguing. In an experimental model, choroidal tissue demonstrated faster cooling, whereas scleral tissue retained heat for longer durations.⁶ This could explain why posterior scleritis tends to produce more sustained elevation of OST, while primary choroidal inflammations may show relatively transient thermal changes.⁶

These observations suggest that thermography may potentially aid in differentiating clinically overlapping entities such as posterior scleritis and choroiditis, although larger validation studies are still needed.

BEYOND SCLERITIS

Beyond uveitis and scleritis, thermography has shown utility in inflammatory orbital disease, particularly thyroid eye disease,^{4,7} where active disease demonstrates increased temperatures in the caruncle and conjunctiva that decline following corticosteroid therapy, correlating with disease activity scores.⁷

Conjunctival benign reactive lymphoid hyperplasia has been shown to appear cooler than surrounding tissues, whereas active scleritis demonstrates focal hyperthermia.³ Corneal ulcers and graft rejection episodes have also demonstrated increased local temperatures proportional to inflammatory severity.⁹

Thermography may also have relevance in ocular vascular disorders. Reduced ocular temperatures have been

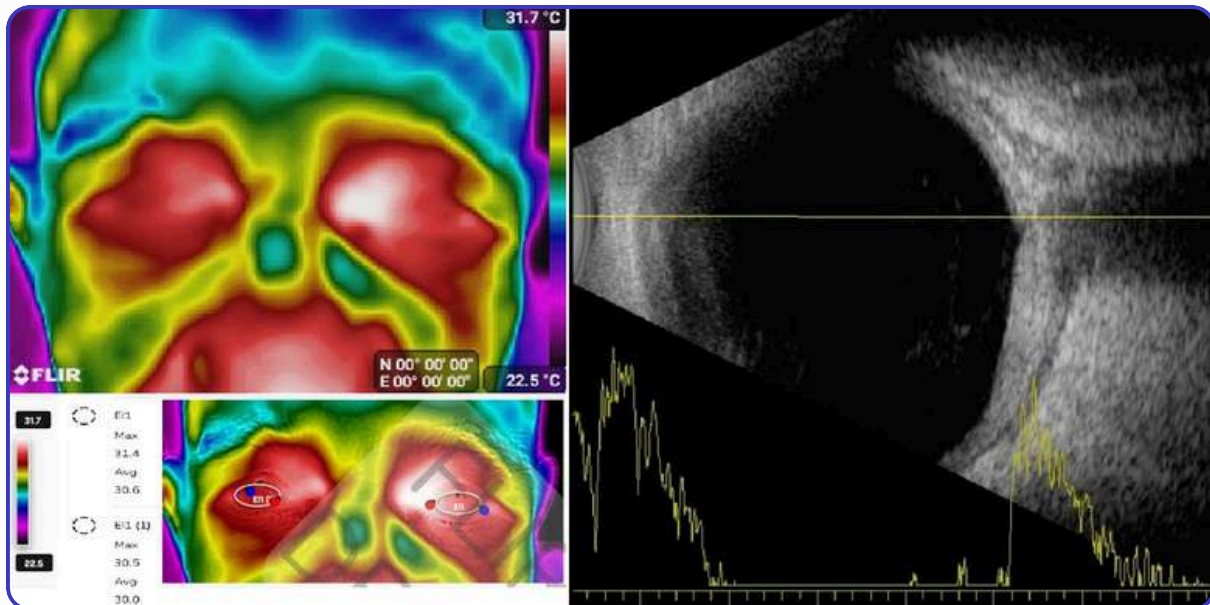


Figure 2: A 76-year-old male, known case of rheumatoid arthritis with bilateral anterior scleritis on immunomodulatory therapy, had been under regular follow-up for over 1 year before discontinuing methotrexate due to pulmonary symptoms and low blood counts. He presented with complaints of left eye pain (OS) and was started on topical steroids for relapse of mild anterior scleritis. He returned after 10 days with

persistent pain despite topical steroids. Anterior segment and fundus examination were unremarkable in both eyes. Thermography revealed increased OST in OS compared to OD. (OST OD: 30.0°C, OS OST: 30.6°C) Subsequent B-scan revealed diffuse scleral thickening with sub-Tenon's fluid, confirming the first episode of posterior scleritis in a long-term follow-up of anterior scleritis.

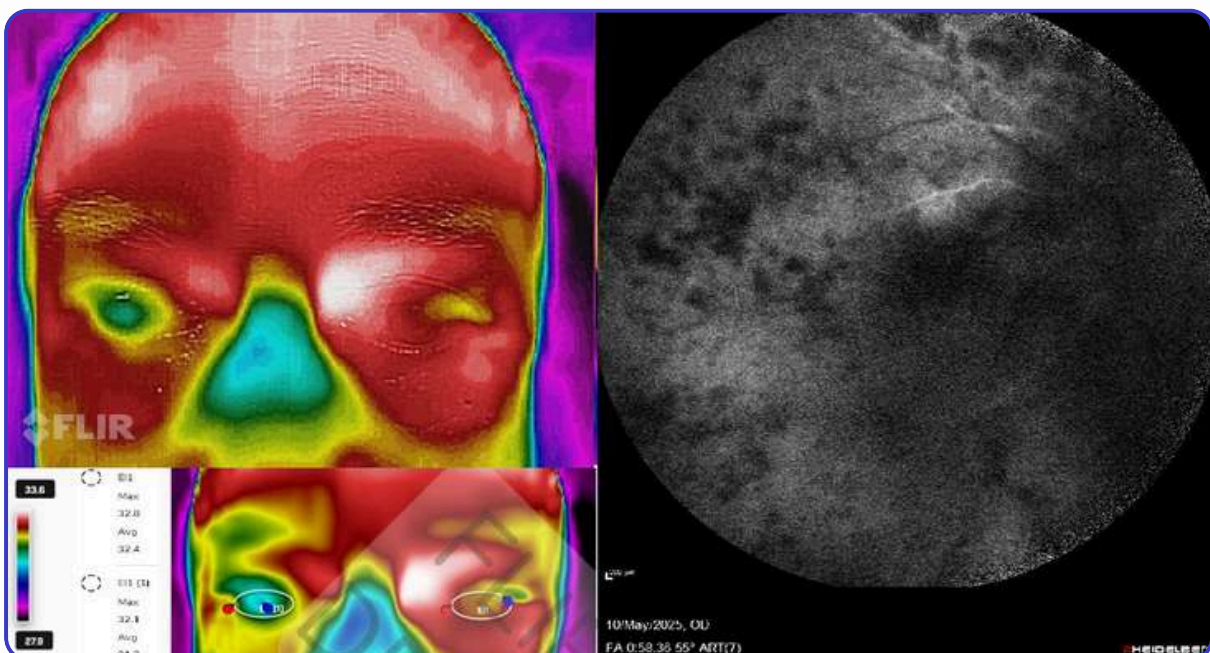


Figure 3: A 69-year-old male, known case of old branch retinal vein occlusion, was referred to the Uvea department for newly developed keratic precipitates with 1+ anterior chamber cells. Fundus examination revealed superior sectoral laser marks, hemorrhages and sclerosed vessels. Thermography revealed cold OD. (OST OD: 31.3°C, OS: 32.4°C) Fundus fluorescein angiography showed prolonged arm-retina time of 58 seconds, suggestive of ocular ischemic syndrome.



described in carotid artery stenosis and retinal vascular occlusions due to compromised perfusion. In systemic vasculitides such as Takayasu arteritis, where ocular ischemia and hypoperfusion may occur, thermography could theoretically provide a rapid, non-invasive assessment of altered ocular perfusion, although dedicated studies remain limited.¹⁰ (Figure 3)

CHALLENGES

Despite its promise, ocular thermography comes with its own set of challenges. OST is highly sensitive to environmental as well as internal conditions, including room temperature, humidity, airflow, blink dynamics, and physiological fluctuation of body temperature. Tear film evaporation alone can significantly alter readings.

Several practical precautions should therefore be considered: measurements should be obtained after the patient has rested in a controlled room for several minutes, before contact procedures such as B-scan ultrasonography, and with consistent camera distance and fixation. Simultaneous comparison with the fellow eye or with a relatively thermally stable reference point, along with repeated dynamic measurements, may improve reliability.^{3,6}

Another limitation is the lack of standardised acquisition protocols and normative databases. Different devices, thermal sensitivities, colour scales, and analysis methods make inter-study comparisons difficult. Additionally,

thermography measures surface temperature rather than direct intraocular inflammation, making interpretation indirect and potentially confounded by dry eye disease, meibomian gland dysfunction, or ambient conditions.

FUTURE WATCH

Nevertheless, the future appears encouraging. Portable smartphone-based thermal cameras, artificial intelligence-assisted pattern recognition, and dynamic thermal mapping may eventually allow objective quantification of ocular inflammation in outpatient clinics.

For now, ocular thermography remains an under-utilised but intriguing technology, perhaps a complementary tool to conventional imaging modalities, offering clinicians a unique physiological perspective –

the eye's very own "heat signature"!





JAK INHIBITION IN UVEITIS

THE EVIDENCE SO FAR

A NEW CLASS, BORROWED FROM ELSEWHERE

JAK inhibitors did not arrive with the eye in mind.

Ruxolitinib was the first in the class, approved for myelofibrosis. Use of this class subsequently expanded across specialties including rheumatology, gastroenterology and dermatology for treating various immune-mediated diseases.



However, no JAK inhibitor has so far been licensed for uveitis.

Oral administration and their ability to modulate multiple cytokine pathways have made them attractive candidates for treating ocular inflammation.

Recent trials have begun providing answers. Adult studies have shown encouraging results, while the first paediatric trials are still less convincing. In this article, we take a sneak peek into the relevant data on JAK-pathway inhibition in uveitis.

ADULT UVEITIS AND JAK-I

A lot of what we know about JAK inhibitors and the eye has come from trials designed to answer a different question.

The most useful of these borrowed datasets is a post hoc analysis by [Poddubnyy and colleagues](#), pooling five phase 3 *upadacitinib* trials in psoriatic arthritis and axial spondyloarthritis:

- 1,789 patients on upadacitinib 15 mg
- 1,095 on placebo and
- 429 on adalimumab

Uveitis was recorded as a treatment-emergent adverse event and was numerically less frequent with upadacitinib than placebo.

One observation that stood out was that

of the 28 uveitis events on upadacitinib, 75% occurred while Ankylosing Spondylitis Disease Activity Score (ASDAS) was below 2.1 and 79% while Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) was below 4.0, with normal CRP.

The message?

Controlling the joints may not reliably control the eye

emphasising that ocular disease activity needs to be assessed independently rather than inferred from systemic disease control.

HUMBOLDT

HUMBOLDT is the first published randomised trial of a JAK inhibitor in adult non-infectious intermediate, posterior and panuveitis patients.

Patients were treated with *filgotinib* 200 mg or placebo for 52 weeks.

After 24 weeks, treatment failure occurred in 37.5% patients on filgotinib compared to 67.6% on placebo. Time to treatment failure was also significantly longer with filgotinib.

The results need more context. The

anterior chamber cell grade improved



but there was no difference between the groups in terms of vitreous haze, macular oedema or visual acuity.

Also, the trial was stopped at 30% of its planned enrolment for commercial reasons, limiting the strength of its conclusions.

NEPTUNE

Neptune explored *brepocitinib*, an oral TYK2/JAK1 inhibitor in patients with active non-infectious intermediate, posterior or panuveitis.

26 patients received either 45 mg or 15 mg brepocitinib after a prednisolone burst that tapered off by week 8.

At week 24, treatment failure had occurred in 29% of the 45 mg arm vs 44% of the 15 mg arm.

Of those with baseline macular oedema, 43% of the 45 mg arm had resolution.

On wide-field fluorescein angiography, the ASUWOG scores for retinal vascular leakage also decreased by a mean of 4.4 points in the 45 mg arm (compared to 0.5 points in the 15 mg arm).

NEPTUNE, however, was a

small, dose-ranging
study without a placebo
arm.

The figures released are from company press releases and an EURETINA presentation, and a peer-reviewed publication is still pending.

CLARITY

CLARITY is a phase 3 study now under way, randomising 371 patients to *brepocitinib* 45 mg or placebo, with time to treatment failure as the primary endpoint.

Patient enrolment is complete and results are due imminently. If positive, it will be the first adequately powered placebo-controlled evidence supporting a JAK pathway inhibitor in adult uveitis.

PAEDIATRIC UVEITIS AND JAK-I

JUVE-BRIGHT

Juve-BRIGHT is the first trial of a JAK inhibitor in childhood uveitis. It compared *baricitinib* with adalimumab in a small, open-label Bayesian trial involving children with JIA-associated uveitis or chronic ANA-positive anterior uveitis who had an inadequate response to methotrexate or biologic therapy.

The trial did not meet its primary endpoint.

8 of 24 children on baricitinib (33.3%) achieved a two-step reduction in anterior chamber cells at week 24,



resulting in a 1.03% probability that the true response rate exceeded the pre-specified 57% benchmark, well below the 80% required for the trial to be considered positive.

In the randomised group, 4 out of 5 children treated with adalimumab achieved response compared to 1 of 5 treated with baricitinib.

Among those refractory to both methotrexate and a biologic, 7 out of 19 (36.8%) responded to baricitinib.

The results, therefore, do not currently support baricitinib as an alternative to established TNF inhibition in childhood uveitis.

The small sample, open-label design and incomplete randomised comparison mean that these findings need to be interpreted with great caution.

WHERE THIS LEAVES US

JAK inhibitors are generating genuine interest in uveitis, particularly in adults. Adult data is encouraging but sparse, while the only paediatric trial to date was negative in a highly refractory population.

For now, JAK inhibitors remain investigational in uveitis. Their oral administration and distinct safety profile warrant further evaluation, but appropriately designed uveitis trials are still needed to establish their place alongside current biological therapies in uveitis.



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ADJUST

CAN WE SAFELY STOP ADALIMUMAB IN JIA-ASSOCIATED UVEITIS?

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Adalimumab (ADA) is currently the only FDA-approved biologic agent effective in achieving and maintaining quiescence in JIA-associated uveitis.

However, prolonged therapy increases cost, treatment burden, and long-term safety concerns, including infections, malignancy and demyelinating disease.

ADJUST
(ADalimumab in
Juvenile idiopathic
arthritis
associated Uveitis
Stopping Trial)

ADJUST is a multi-centre, double-masked, randomised controlled trial, conducted across the USA, UK, and Australia and is the first randomised controlled trial designed to evaluate whether adalimumab can be safely discontinued in children with controlled JIA-associated uveitis.



THE BIG PLAN

UVEITIS SOCIETY (INDIA)



Inclusion criteria

- Age > 2 years
- JIA-uveitis (onset <16 years)
- Controlled uveitis and controlled arthritis on ADA for ≥ 12 months
- >180 days of stable dose of systemic ADA: biweekly ADA 20 mg (if <30 kg body weight) and 40 mg (if ≥ 30 kg body weight)
- ≥ 90 days of stable dose of concomitant immunomodulatory therapy



Exclusion criteria

- H/O acute anterior uveitis
- H/O ocular surgery within the last 90 days or planned within 12 months
- ≥ 90 days of chronic hypotony (<5 mmHg) in either eye
- Corticosteroid use (systemic/intraocular/intra-articular) within past 12 months

87 patients



STRATIFIED
RANDOMISATION

stratified by country and concomitant immunomodulatory therapy (i.e., MTX, MMF, AZA, and leflunomide) use

Group A
ADA
(43 patients)

Group B
Placebo
(44 patients)

Treatment
Failure

Follow-up until
48 weeks

Treat as per
medical
judgement

Treatment
Failure

Follow-up until
48 weeks

Restart ADA

PRIMARY OUTCOME:

TIME
to
"treatment failure"

Treatment failure was defined as any one of the following in at least one eye

- 3+ anterior chamber (AC) cells in a single visit
- >0.5+ anterior chamber (AC) cells for ≥ 28 days
- 2-step increase in AC cells observed at two separate visits ≥ 7 days apart
- 0.5+ vitreous haze
- Active retinal or choroidal inflammation or macular oedema observed at a single visit
- Recurrence of joint inflammation



BUT PLANS CHANGE

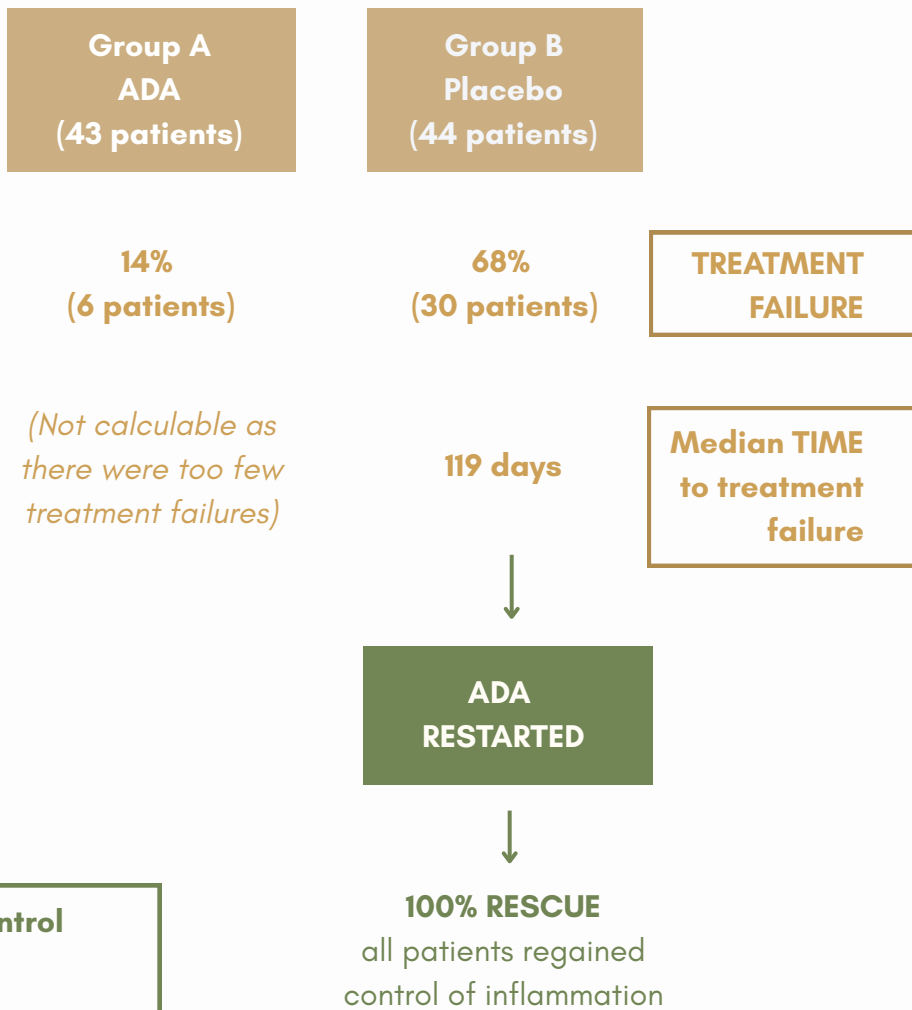
UVEITIS SOCIETY (INDIA)

During the trial, a pre-planned interim analysis of the accumulating data by an independent committee showed that there was already a significantly higher rate of treatment failure in the placebo group, meeting the predefined criteria for “**early termination of the study**”.

HAZARD RATIO = 8.7

i.e., children who discontinued adalimumab had a significant 8.7-fold higher instantaneous risk of treatment failure during follow-up.

(95% CI 3.6–21.2; $p < 0.0001$)



PRACTICE PEARLS

Stopping ADA increases the chance of relapse from 14% to 68%. ADJUST supports continuing ADA to maintain remission in children with well-controlled JIA-associated uveitis.

Median time to treatment failure after stopping ADA was approximately 4 months (119 days). Hence, the decision to stop ADA should be individualised and requires close follow-up, especially for the first 6 months.

Every patient who relapsed regained control after restarting ADA, with a median of 105 days to re-establish control.

ADJUST demonstrates a substantially increased risk of treatment failure after adalimumab withdrawal in children with controlled JIA-associated uveitis. If discontinuation is considered, patients require close monitoring, particularly during the first 6 months





TEST YOUR UVEITIS QUOTIENT

The author wishes to acknowledge Dr. Padmamalini Mahendradas, Dr. Sanjana Vinod and Dr. Rohan Dedhia for their valuable help with procuring the clinical images.

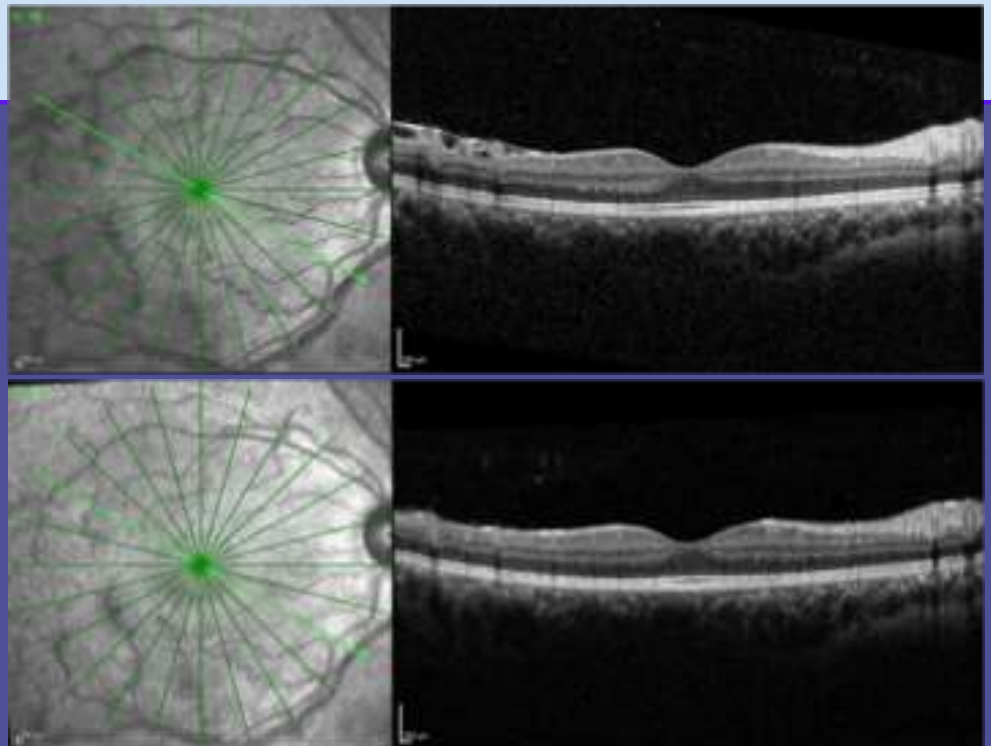
Dr. Aditya Patil
Consultant Ophthalmologist
and Uveitis Specialist
Ojas Maxivision Eye Hospital
Bandra West, Mumbai

**1**

Identify this non-infectious condition causing retinal infiltrates, mouth ulcers and occlusive vasculitis.

BEHÇET'S UVEITIS

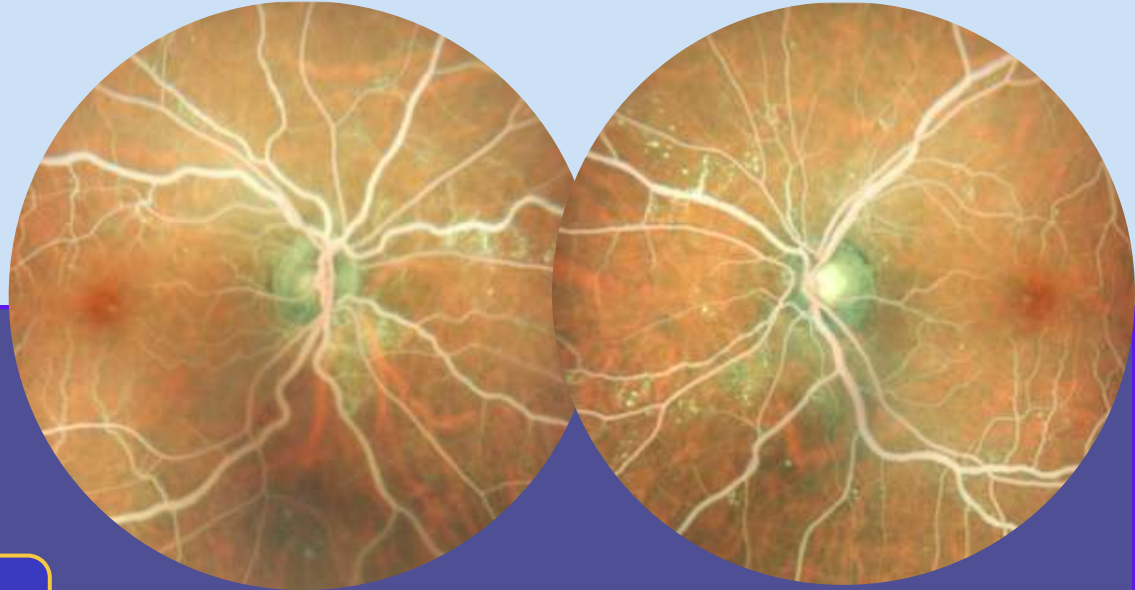
OCT in Behçet's uveitis shows focal thickening and hyper-reflectivity of inner retinal layers which resolves with thinning or loss of retinal nerve fibre layer after treatment with corticosteroids and biologics.



**2**

Identify this retinal manifestation that may mimic uveitis.

UVEITIS SOCIETY (INDIA)

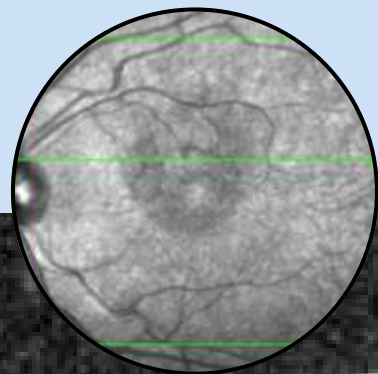
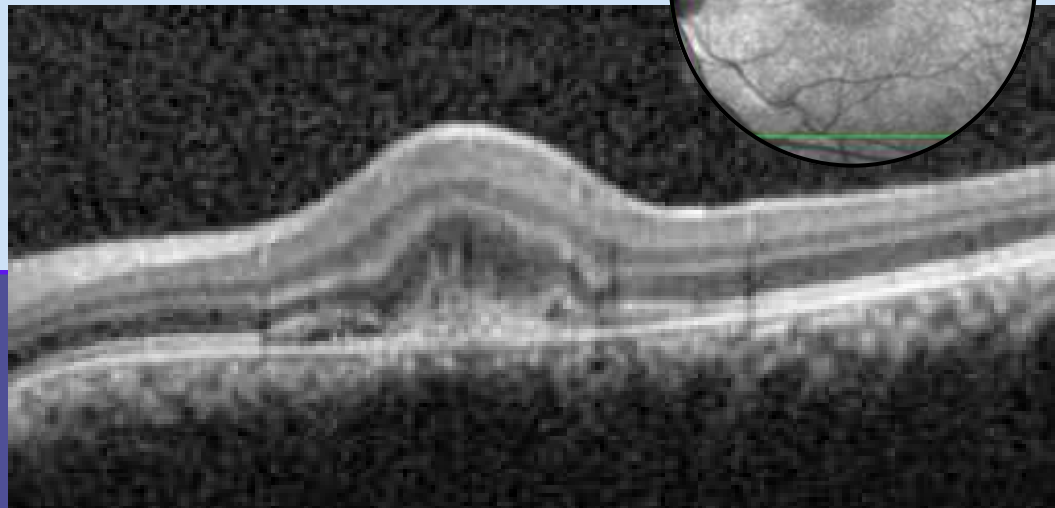


LIPEMIA RETINALIS

Lipemia retinalis is a rare retinal manifestation of severe hypertriglyceridaemia (usually triglycerides $>2,000$ - $2,500$ mg/dL), characterised by creamy white retinal vessels and a salmon-pink fundus. It is usually asymptomatic, reversible with lipid reduction, and signals an increased risk of acute pancreatitis.

3

Identify this OCT sign.



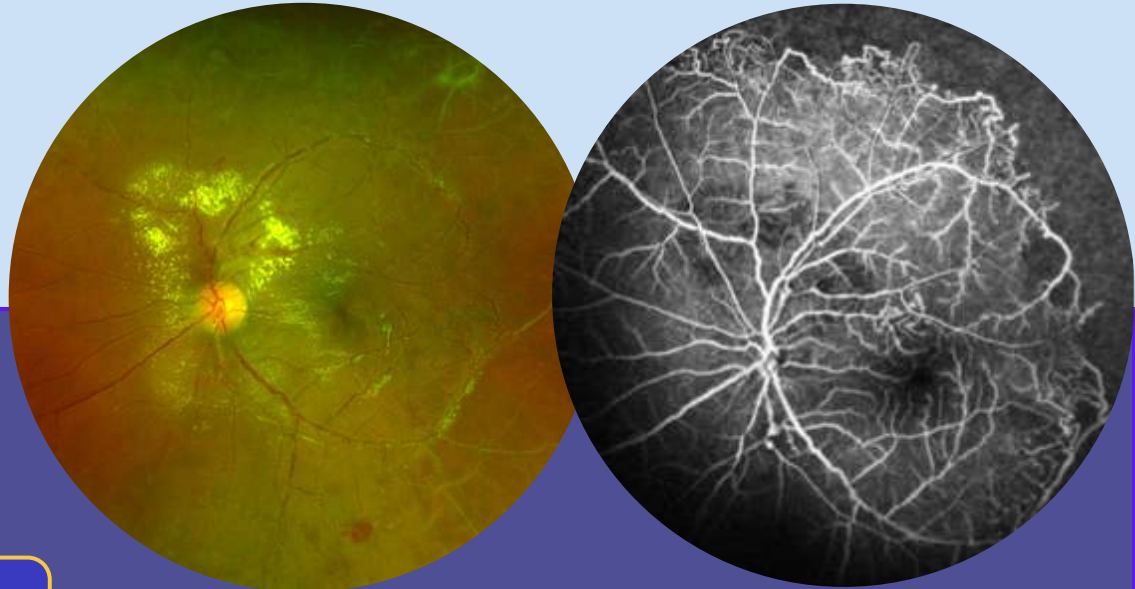
PITCHFORK SIGN

Pitchfork sign is characterised by subretinal hyperreflective material with multiple vertical finger-like projections extending anteriorly into the outer retina. It is often seen in inflammatory CNVM and is suggestive of activity.

**4**

An 18-year-old male with diminution of vision. Identify the condition.

UVEITIS SOCIETY (INDIA)

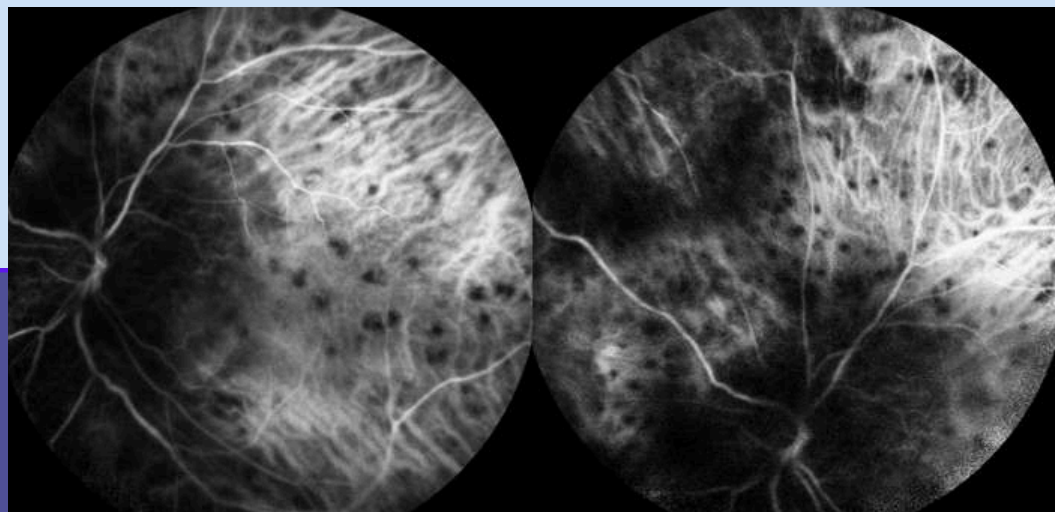


IRVAN SYNDROME

Idiopathic retinal vasculitis and neuroretinitis (IRVAN) syndrome is characterized by occlusive retinal vasculitis, exudation, aneurysmal dilation at arterial bifurcations and peripheral capillary non-perfusion. Early panretinal photocoagulation is essential to prevent complications.

5

Hypofluorescent dark dots (HDDs) on Indocyanine green angiography are suggestive of?



CHOROIDAL GRANULOMAS / SCAR TISSUE

HDDs are areas of non-perfusion of indocyanine green dye and represent foci of choroidal stromal inflammatory cell infiltration or choroidal scar tissue. They are often seen in VKH syndrome, sympathetic ophthalmia, birdshot chorioretinopathy.



EYE HUMOUR

HAHAHA

Doctor: "Any systemic symptoms?"

Patient: "Doctor, I only came for my eye..."

Doctor: "Welcome to uveitis."

Patient: "Doctor, will this inflammation ever end?"

Doctor: "That depends..."

Patient: "On what?"

Doctor: "Which follow-up visit we're talking about."

Patient: "Sir, is this infectious or autoimmune?"

Doctor: "Yes."

Patient: "How long will the treatment last?"

Doctor: "Let's first define 'long.'"

Patient: "Doctor, I googled my symptoms."

Doctor: "And?"

Patient: "Apparently I have seventeen diseases."

Doctor: "That's actually fewer than I expected."

Uveitis teaches patience...

Mostly to the doctor.

Patient: "My eye feels perfectly fine now."

Doctor: "That's wonderful."

Patient: "So I stopped all treatment."

Doctor: "...and that's why I drink coffee."

Doctor: "Do you have old reports?"

Patient: "Yes."

Doctor: "Wonderful."

Patient: "They're at home."



AROUND



THE WORLD IN UVEITIS



SHAPU

NEPAL'S SEASONAL BLINDING UVEITIS



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Seasonal Hyperacute Panuveitis, (SHAPU) remains one of ophthalmology's most striking geographic curiosities: a fulminant, blinding uveitis of children that, across more than five decades of documentation, has been confirmed mostly in Nepal (except one report from Bhutan).¹⁻⁴ It is characterised by sudden onset of unilateral red eye with minimal pain, no discharge, but intense intraocular exudation giving the appearance of white pupillary light reflex (Figure 1).

First reported among school children of Pokhara in 1975, it targets an immunocompetent child who develops painless, unilateral visual loss that can progress from normal vision to no perception of light within 24-48 hours.⁵⁻⁷ Two-thirds of patients present with blindness at first contact, and in a matter of hours to days, malignant hypotony, retinal detachment, and permanent loss of eyesight occurs, consequently leading to phthisis bulbi

and cosmetic disfigurement.^{1-3,8} SHAPU is different from other uveitis conditions like endophthalmitis, Behçet's disease, ophthalmia nodosa and HLA-B27-associated uveitis due to its unilateral seasonal occurrence, devastating rapidity of progression of the disease, localisation in the specific regions of Nepal, with absence of trauma, surgery and a discernible systemic illness, and progression of inflammation from anterior to posterior chamber.^{1,2} Among the paediatric population in Nepal, idiopathic uveitis is the most common, followed by SHAPU and toxoplasmosis.⁹



Figure 1: Clinical photograph of a SHAPU child with unilateral "White pupil in a Red Eye"



WHY “SHAPU”?

The disease was named as Seasonal Hyperacute Panuveitis (SHAPU) after years of outbreak observation by Prof. Madan Prasad Upadhyay. Prof OM Krishna Malla named it *seasonal endophthalmitis*,^{5,6} but SHAPU remained in use as each word had its own meaning:



Seasonal (S): Typically occurs with clock-like precision as an outbreak in the post-monsoon season (autumn and winter) of the English calendar in alternate years. Sometimes, a smaller outbreak may occur in the summer season of even years. The first outbreak occurred in 1975, then in 1977, 1979 and so on.



HyperAcute (HA): The intraocular inflammation progresses with astounding speed and visual loss often sets in within hours of onset. The cytokine storm within the aqueous and vitreous cavities confirms the hyperacute nature of destruction within the eye, justifying the use of the term hyperacute.



PanUveitis (PU): There is progression of inflammation from anterior to posterior uveal tract, leading to severe anterior chamber fibrinous reaction with iritis, dense vitreous exudation with vitritis and posterior segment involvement with retinitis, causing posterior uveitis. Hence, the term 'panuveitis' was used due to pan-uveal involvement. Leukocoria represents severe exudation in the anterior and posterior segments, and malignant hypotony indicates cyclitis with ciliary shutdown.



Figure 2: Close-up view of female *Gazalina chrysolopha* showing white body with black striations and brown tail (above)

Figure 3: High population of *Gazalina chrysolopha* moths during the outbreak season at the mid-hilly regions of Nepal (below).

CONNECTION WITH ECOLOGY

There is a strong and in-depth connection between SHAPU and the ecological and epidemiological factors. Exposure to white moths was found to be the strongest risk factor (Odds ratio 6.89) for the development of SHAPU.⁷ *Gazalina chrysolopha*, a moth belonging to the family Notodontidae (order Lepidoptera), has been found as the primary host insect (Figure 2).^{2,10} They remain inactive during the day, resting in gardens, tree bark, leaves, lawns, and walls and emerging at night, attracted by bright light.^{8,9} Similarly, *Alnus nepalensis* (uttis/alder tree) has been identified as the primary host plant of *G. chrysolopha*.¹⁰ Ecological regions between 1,000 and 2,000 metres, particularly the mid-hill regions of the subtropical and alpine zones, with average temperatures of 20-30 °C, suitable relative humidity and rainfall, and Uttis forests, support the life cycle of the moth and the occurrence of SHAPU in these regions of Nepal.¹⁰





Hence, SHAPU is an ocular consequence of complex interactions between human hosts, environmental triggers such as insect vectors and host plants. This proves the role of the “one health” paradigm for SHAPU care.

AETIOLOGY

The exact aetiology is still an enigma; there are two schools of thought. The first school of thought suggests that SHAPU is an inflammatory and hypersensitivity response to release of harmful toxins, proteins or antigens from the setae or exuviae of female *G. chrysolopha* moths onto the ocular surface, triggering a cytokine storm, leading to hypersensitive, hyperacute and fulminant ocular inflammation. The second school of thought is that these moths may serve as a vector to carry microbes like bacteria or viruses from their habitat to the ocular surface, causing hyperacute uveitis.^{2,8}

STAGES

The disease starts as anterior uveitis and finally progresses to panuveitis. It is classified into three stages as follows:

Mild SHAPU: Occurs in the first 48 hours of symptom onset, resembles a mild form of non-granulomatous acute anterior uveitis, where there is $\leq 1+$ cell/flare. The vitreous and retina are usually normal.

Moderate SHAPU: Occurs during day 3–7 after symptom onset, has features of anterior and intermediate uveitis with moderate to severe anterior chamber

reaction with more than 1+ cells or flare, fibrinous exudates with or without hypopyon. The exudates surround the lens and lead to leukocoria. The fundal glow may still be appreciable, and the retina is usually normal. The B-scan usually reveals hyperechoic shadows in the anterior and mid-vitreous with a thickened retinochoroidal complex.

Severe SHAPU: Occurs after day 7 with posterior segment involvement.⁷ Dense vitreous exudates with no retinal view leading to total leukocoria, creating the “white pupil in a red eye” appearance. B-scan usually depicts hyperechoic shadows in the anterior vitreous, mid vitreous, and posterior vitreous and increased thickness of the retinochoroidal complex.

TREATMENT

Prompt recognition and aggressive intervention remain the cornerstones of care. In the absence of a definitive aetiology, the patients receive a blanket therapy of antimicrobials and steroids via periocular and systemic routes.² Recently, the combined use of 0.1 mL intravitreal injection of triamcinolone, dexamethasone and moxifloxacin has shown a favourable outcome.¹¹ Early core vitrectomy in moderate and severe cases is done to remove inflammatory debris and possible inciting agents. But the outcomes remain variable. Finally, SHAPU is the end result in an end-organ (eye), of a process that began some time ago, a process whose origin lies outside the eye.





CLOSING NOTE FROM THE SECRETARY

Curiosity, CONVERSATIONS AND CONFERENCES

Dr. Parthopratin Dutta Majumder
Secretary
Uveitis Society (India)



Welcome to
USICON 2026

*the 27th Annual Conference of the
Uveitis Society of India*



**Dear friends and colleagues,**

Every year, this gathering remains a focal point for uveitis enthusiasts from India and the surrounding regions. I consciously use the word “enthusiasts” rather than experts, because uveitis is one of those fascinating branches of medicine where the more we learn, the more we realise how much remains to be understood.

The real essence of such a meeting lies beyond the lecture auditorium – in critical thinking, challenging ideas, meetings of minds, and the bonhomie that often continues over a cup of coffee or a casual corridor conversation. But what brings these minds together and makes these gatherings so powerful? Perhaps it is curiosity, the simple urge to ask –

**why, why not,
and what if?**

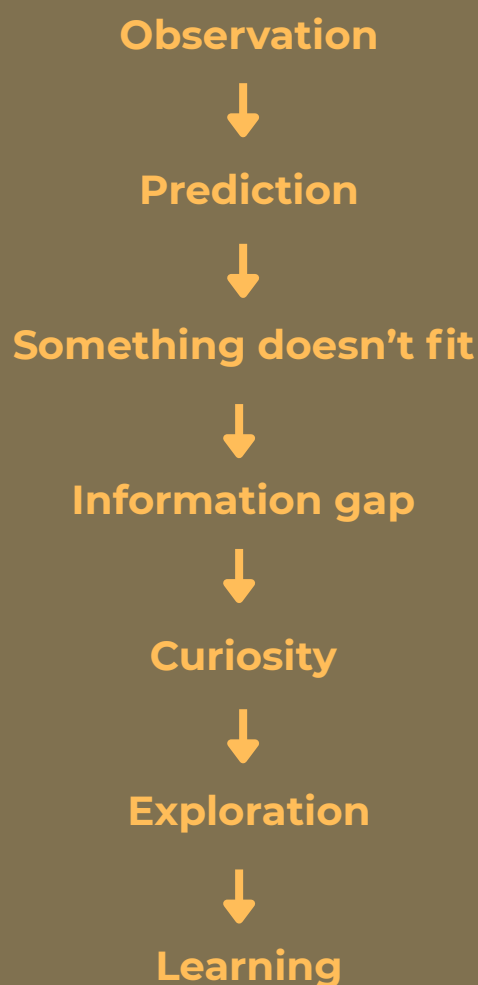
Curiosity is not merely a personal trait. It is also a desire to acquire information. It begins when an observation challenges our expectations and creates an information gap. The brain automatically compares what we see with previously acquired patterns and knowledge and immediately, the brain seems to talk to itself: “I know what this resembles...but something doesn’t fit.”

Science has always progressed by watching, questioning, borrowing, testing, and improving upon the ideas of others. The purpose is not to imitate what others are doing, but to understand why they are doing it, and then ask whether it can make

our own practice better: the curiosity to know what others are doing. How are they approaching the same problem? Are they seeing something that I am missing? Can their experience change the way I practise?

At first glance, this may look like the academic version of FOMO: the fear of missing out. But perhaps it is something more meaningful: the fear of missing an idea-FOMI.

One important trigger is the recognition of a discrepancy between what we know and what we want to know.





Perhaps that is the real power of curiosity:

it makes us ask the question, motivates us to search for the answer, and helps us remember what we discover.

In 1972, Herbert Boyer and Stanley Cohen met at a conference on bacterial plasmids. Their laboratories had been working on different pieces of what would become the same puzzle: Boyer on restriction enzymes capable of cutting DNA at specific sites, and Cohen on plasmids and methods for introducing them into bacteria. Their conversations continued beyond the formal sessions, and they realised that the techniques from their respective laboratories could be brought together. The result? Their subsequent collaboration produced recombinant DNA experiments that helped lay the foundations of modern genetic engineering. What began as a conference conversation became an idea that changed biology.

It is not only agreement that moves science forward; disagreement can be equally powerful.

The 1927 Solvay Conference brought together 29 leading scientists, 17 of whom were or would become Nobel laureates, including Einstein, Bohr, Marie Curie, Heisenberg, Schrödinger, Dirac, and Pauli. The meeting became famous for the intense debate between Albert Einstein and Niels Bohr over the foundations of quantum mechanics. Their arguments continued over breakfast and during walks between the hotel and the meeting venue.

Perhaps that is why conversations over coffee, dinner, or in a conference corridor should never be considered merely a break between scientific sessions. Sometimes, that is where the real science begins.



Einstein and Bohr debated quantum mechanics over breakfast and during walks between the hotel and the venue – a reminder that the best ideas at a conference are not always born inside the conference hall

Great meetings do not merely exchange knowledge, they challenge it.

I hope all of you have a fantastic USICON 2026, filled with excellent discussions, challenging questions, insightful wisdom, new friendships, and, above all, curiosity. May we return home not only with more knowledge, but with more questions, more ideas, and renewed energy to pursue them.

Dr. Parthopratiim Dutta Majumder
Secretary
Uveitis Society (India)





FURTHER READING

Core Concepts

Acute Retinal Necrosis ↩

1. Holland GN. Standard diagnostic criteria for the acute retinal necrosis syndrome. *Am J Ophthalmol*. 1994
2. SUN Working Group. Classification criteria for acute retinal necrosis. *Am J Ophthalmol*. 2021.
3. Schoenberger SD, et al. Diagnosis and Treatment of Acute Retinal Necrosis. *Ophthalmology*. 2017.
4. Tibbetts MD, et al. Treatment of Acute Retinal Necrosis. *Ophthalmology*. 2010.
5. Palay DA, et al. Decreased probability of bilateral ARN syndrome with acyclovir therapy. *Am J Ophthalmol*. 1991.
6. Taylor SRJ, et al. Valacyclovir in the treatment of acute retinal necrosis. *BMC Ophthalmol*. 2012.
7. Butler NJ, et al. Acute Retinal Necrosis: Presenting Characteristics and Clinical Outcomes. *Am J Ophthalmol*. 2017.
8. Kalogeropoulos D, et al. Diagnostic and therapeutic challenges in acute retinal necrosis: an update. *Eye*. 2024.
9. Kawali A, et al. Primary treatment failure in acute retinal necrosis. *Indian J Ophthalmol*. 2024.
10. Risseuw S, et al. Risk of rhegmatogenous retinal detachment in acute retinal necrosis with and without prophylactic intervention. *Am J Ophthalmol*. 2019.

Deep Dive - Foundations

Ocular Behçet's - The Clues Are Usually There ↩

1. Takeuchi M, Mizuki N, Ohno S. Pathogenesis of non-infectious uveitis elucidated by recent genetic findings. *Front Immunol* (2021) 12:640473. doi: 10.3389/fimmu.2021.640473
2. Zouboulis CC, Keitel W. A historical review of early descriptions of Adamantiades-Behçet's disease. *J Invest Dermatol* 2002;119:201-5.
3. Zając H, Turno-Kręcicka A. Ocular Manifestations of Behçet's Disease: An Update on Diagnostic Challenges and Disease Management. *J Clin Med*. 2021;10:5174.
4. Tugal-Tutkun I, Gupta, V.; Cunningham, E.T. Differential Diagnosis of Behçet Uveitis. *Ocul. Immunol. Inflamm*. 2013, 21, 337-350.
5. Criteria for diagnosis of Behçet's disease. International Study Group for Behçet's Disease. *Lancet*. 1990 May 5;335(8697):1078-80. PMID: 1970380.

6. International Team for the Revision of the International Criteria for Behçet's Disease (ITR-ICBD). The International Criteria for Behçet's Disease (ICBD): a collaborative study of 27 countries on the sensitivity and specificity of the new criteria. *J Eur Acad Dermatol Venereol*. 2014 Mar;28(3):338-47. doi: 10.1111/jdv.12107. Epub 2013 Feb 26. PMID: 23441863.
7. Lin S, Xu Z, Lin Z, Xie B and Feng J (2023) Advances in pathogenesis and treatment of ocular involvement in Behçet's disease. *Front. Immunol*. 14:1206959.
8. Tanaka R, Murata H, Takamoto M, Ohtomo K, Okinaga K, Yoshida A, Kawashima H, Nakahara H, Fujino Y, Kaburaki T. Behçet's disease ocular attack score 24 and visual outcome in patients with Behçet's disease. *Br J Ophthalmol*. 2016 Jul;100(7):990-994. doi: 10.1136/bjophthalmol-2015-307362. Epub 2015 Nov 9. PMID: 26553921.
9. Hatemi, G.; Robin, C.; Bang, D.; Bodaghi, B.; Celik, A.F.; Fortune, F.; Gaudric, J.; Gul, A.; Kötter, I.; Lemesse, P.; et al. 2018 update of the EULAR recommendations for the management of Behçet's syndrome. *Ann. Rheum. Dis*. 2018, 77, 808-818.
10. Uke, P.; Gorodkin, R.; Beare, N. Biologic therapy for Behçet's uveitis: A systematic review. *Br. J. Ophthalmol*. 2020, 104, 1045-1051.
11. Chan VTT, Mak ACY, Agrawal R, et al. International consensus and guidelines on managing ocular Behçet's disease by the Academy of Asia-Pacific Professors of Ophthalmology (AAPPO), the Asia-Pacific Vitreo-Retina Society (APVRS), the Asia-Pacific Society of Ocular Inflammation and Infection (APSIOI) and the Academia Retina Internationalis (ARI). *Asia Pac J Ophthalmol*. 2025;14:100261
12. Kaçmaz RO, Kempen JH, Newcomb C, et al. Ocular inflammation in Behçet disease: incidence of ocular complications and of loss of visual acuity. *Am J Ophthalmol*. Dec 2008;146(6):828-836
13. Azri C, Dusser P, Eid L, Barreau E, Kone-Paut I, Borocco C, Galeotti C, Saad S, Labetoulle M, Rousseau A. Ocular involvement in pediatric Behçet's disease: is it different than in adults? (a short case series and mini review). *BMC Ophthalmol*. 2023 Nov 21;23(1):474. doi: 10.1186/s12886-023-03197-5. PMID: 37990307; PMCID: PMC10664259.

Deep Dive - Masterclass

The Art Of Managing Ocular Behçet's Disease ↩

1. Hatemi G, et al. EULAR recommendations for the management of Behçet syndrome: 2025 update. *Ann Rheum Dis*. 2026.
2. Tugal-Tutkun I. Uveitis in Behçet disease—an update. *Current Opinion in Rheumatology*. 2023 Jan 1;35(1):17-24.
3. Lin S, Xu Z, Lin Z, Xie B, Feng J. Advances in pathogenesis and treatment of ocular involvement in Behçet's disease. *Frontiers in Immunology*. 2023 Sep 29;14:1206959
4. Tugal-Tutkun, I.; Gupta, V.; Cunningham, E.T. Differential diagnosis of Behçet Uveitis. *Ocul. Immunol. Inflamm*. 2013, 21, 337-350.
5. Tugal-Tutkun I, Herbot CP, Khairallah M, Angiography Scoring for Uveitis Working Group (ASUWOG). Scoring of dual fluorescein and ICG inflammatory angiographic signs for the grading of posterior segment inflammation (dual fluorescein and ICG angiographic scoring system for uveitis). *International ophthalmology*. 2010 Oct;30(5):539-52.



6. Zając H, Turno-Kręćicka A. Ocular Manifestations of Behçet's Disease: An Update on Diagnostic Challenges and Disease Management. *J Clin Med*. 2021 Nov 5;10(21):5174.
7. Sachdev N, Kapali N, Singh R, Gupta V, Gupta A. Spectrum of Behçet's disease in the Indian population. *Int Ophthalmol*. 2009;29(6):495-501. doi:10.1007/s10792-008-9273-8.
8. Rao VK, Kodali RS, Patil A, et al. Clinical profiling, treatment characteristics and outcome in Behçet's disease: a retrospective cohort study from Karnataka Rheumatology Association. *Clin Rheumatol*. 2024;43:3223-3230.
9. Rohatgi J, Singal A. Ocular manifestations of Behçet's disease in Indian patients. *Indian J Ophthalmol*. 2003;51(4):309-313.
10. Ramdoss J, Singh P, Ahmed AS, Biswas J. Clinical profile and management with immunosuppressants and biologics in Behçet's uveitis: a cohort of 25 patients from a tertiary eye care center in South India. *Indian J Ophthalmol*. 2023;71(5):1972-1976. doi:10.4103/IJO.IJO_1439_22.

CaseArena

A Subretinal Window to Systemic Mayhem ↩

1. Francis JH. Choroidal metastases. In: Sadda SR, Schachat AP, Wilkinson CP, Hinton DR, Wiedemann P, Freund KB, Sarraf D, editors. *Ryan's Retina*. 7th ed. Philadelphia: Elsevier; 2022. p. 2700-2707.
2. Konstantinidis L, Damato B. Intraocular metastases—A review. *Asia Pac J Ophthalmol (Phila)*. 2017;6(2):208-214.
3. Maller B, Salvatori S, Tanvetyanon T. Outcomes of intraocular metastasis from lung cancer in the era of targeted therapy: a systematic review and pooled analysis. *Clin Lung Cancer*. 2022;23(8):e519-e525.

Diagnosing Posterior Scleritis Masquerading as Preseptal Cellulitis ↩

1. Robertson R, Ansari FA, Gafoor S, Idolor ON, Amakye D. Posterior Scleritis: A Case Report and Literature Review of the Management of the Condition. *Cureus*. 2024 Jun 2;16(6):e61537. doi: 10.7759/cureus.61537. PMID: 38831917; PMCID: PMC11146444.
2. Preetam Peraka R, Murthy SI. Role of anterior segment optical coherence tomography in scleral diseases: A review. *Semin Ophthalmol*. 2023 Apr;38(3):238-247. doi: 10.1080/08820538.2022.2112700. Epub 2022 Aug 22. PMID: 35996334.
3. YUNIASIH, kristina; DEWI, Nadia Artha; SOFIA, Ovi. DIAGNOSTIC AND THERAPEUTIC CHALLENGES OF POSTERIOR SCLERITIS MIMICKING CHOROIDAL MELANOMA. *International Journal of Retina, [S.l.]*, v. 7, n. 1, p. 85, feb. 2024. ISSN 2614-8536.
4. Desai S, Biswas J, Ganesh SK, Bhatt D. Anterior segment optical coherence tomography: A monitoring tool in anterior scleritis. *Oman J Ophthalmol*. 2025 Jun 24;18(2):155-161. doi: 10.4103/ojo.ojo_376_24. PMID: 40666783; PMCID: PMC12258829.
5. Mehrdad Motamed Shariati. et al, Posterior localized nodular scleritis mimicking malignancy, a case report and clinical approach, *Oxford Medical Case Reports*, Volume 2024, Issue 11, November 2024, omae146,
6. Alsarhani WK, Abu El-Asrar AM. Multimodal Imaging of

Nodular Posterior Scleritis: Case Report and Review of the Literature. *Middle East Afr J Ophthalmol*. 2020 Jul 20;27(2):134-138. doi: 10.4103/meajo.MEAJO_115_20. PMID: 32874049; PMCID: PMC7442082.

NextGen Corner

The Gut-Eye Axis ↩

1. Gao Q, Xiong M, Zhou S, Lu J, Ren B, Peng Q, Zeng M, Song H. Gut microbiota and uveitis: exploring novel mechanisms of inflammatory ocular diseases via the gut-eye axis. *Frontiers in Microbiology*. 2026 Apr 22;17:1749111.
2. Samalia, P. D., Solanki, J., Kam, J., Angelo, L., and Niederer, R. L. (2025). From dysbiosis to disease: The Microbiome's influence on uveitis pathogenesis. *Microorganisms* 13:271. doi: 10.3390/microorganisms13020271.
3. Kalyana Chakravarthy S, Jayasudha R, Sai Prashanthi G, Ali MH, Sharma S, Tyagi M, Shivaji S. Dysbiosis in the gut bacterial microbiome of patients with uveitis, an inflammatory disease of the eye. *Indian journal of microbiology*. 2018 Dec;58(4):457-69.
4. Shivaji S. A systematic review of gut microbiome and ocular inflammatory diseases: Are they associated?. *Indian journal of ophthalmology*. 2021 Mar 1;69(3):535-42.

Ocular Fluid Biomarkers in Uveitis ↩

1. Shirane M, Yawata N, Sonoda KH. Current understanding of ocular fluid analysis in uveitis. *Taiwan Journal of Ophthalmology*. 2025;15(2):196-202.
2. Reeg S, Klefter ON, Subhi Y, Vorum H, Honoré B, Cehofski LJ. Proteomics analysis of aqueous and vitreous humor in uveitis: a systematic literature review. *Clinical Proteomics*. 2025.
3. Yu X, Duan R, Jiang L, Wang T, Li Z, Zhang B, Su W, Lin Y. Interleukin-6 in non-infectious uveitis: Biology, experimental evidence and treatment strategies. *Biochemical Pharmacology*. 2024;230:116605.

Global Window

The Augmented Eye ↩

1. Sorkhabi MA, Potapenko IO, Ilginis T, Alberti M, Cabrerizo J. Assessment of anterior uveitis through anterior-segment optical coherence tomography and artificial intelligence-based image analyses. *Transl Vis Sci Technol*. 2022, 11(4), 7. doi:10.1167/tvst.11.4.7.
2. Keane PA, Karampelas M, Sim DA, et al. Objective measurement of vitreous inflammation using optical coherence tomography. *Ophthalmology*. 2014, 121(9), 1706-1714. doi:10.1016/j.ophtha.2014.03.006.
3. Standardization of Uveitis Nomenclature Working Group. Development of classification criteria for the uveitides. *Am J Ophthalmol*. 2021, 228, 96-105. doi:10.1016/j.ajo.2021.03.061.
4. Nakayama LF, Ribeiro LZ, Dychiao RG, et al. Artificial intelligence in uveitis: a comprehensive review. *Surv Ophthalmol*. 2023, 68(4), 669-677. doi:10.1016/j.survophthal.2023.02.007.

What's New?



Ocular Thermography ↩

1. Mapstone, R. CORNEAL THERMAL PATTERNS IN ANTERIOR UVEITIS. *Brit. J. Ophthalmol.* 1968; 52, 917.
2. Purslow C. Wolffsohn J S. Ocular Surface Temperature- A Review. *Eye & Contact Lens* 2005 Vol. 31, No. 3.
3. Kawali A, Khanum A, Mishra SB, Sanjay S, Mahendradas P, Shetty R. Quantitative and comparative analysis of thermography parameters in posterior scleritis, choroiditis, and controls. *Indian J Ophthalmol.* Published online March 8, 2024. doi:10.4103/IJO.IJO_2830_23
4. Dave TV, Patodi P, Richhariya A, Dave VP. Thermal Imaging of the Ocular Surface in Thyroid Eye Disease: A Comparison between Active, Inactive and Healthy Eyes. *Curr Eye Res.* 2021;46:1482-1488.
5. Kawali A, Patil A, Mishra SB, Mahendradas P, Shetty R. Ocular thermography - A novel tool to suspect and monitor posterior scleritis. *Indian J Ophthalmol Case Rep* 2024;4:208-10.
6. Kawali A, Khanum A, Shettigar A, Mohan A, Sanjay S, Rajendran J, Mahendradas P, Shetty R; Thermal conduction in posterior scleritis and choroiditis. *Invest. Ophthalmol. Vis. Sci.* 2022;63:4097 - F0061.
7. Chang, TC, Hsiao Y L, Liao S L. Application of digital infrared thermal imaging in determining inflammatory state and follow-up effect of methylprednisolone pulse therapy in patients with Graves' ophthalmopathy. *Graefes Arch. Clin. Exp. Ophthalmol.* 2008, 246,45-49.
8. Kawali AA. Thermography in ocular inflammation. *Indian J Radiol Imaging.* 2013;23:281-3.
9. Sniogowski, M.C.; Erlanger, M.; Olson, J. Thermal imaging of corneal transplant rejection. *Int. Ophthalmol.* 2018, 38, 2335-2339.
10. Morgan PB, Smyth JV, Tullo AB, et al. Ocular temperature in carotid artery stenosis. *Optom Vis Sci* 1999;76:850-854.

ADJUST - Can We Safely Stop Adalimumab in JIA-Associated Uveitis? ↩

1. Acharya N, Ramanan A, Coyne A et al. Stopping of adalimumab in juvenile idiopathic arthritis-associated uveitis (ADJUST): a multicentre, double-masked, randomised controlled trial. *Lancet.* 2025;405(10475):303-313.
2. Maghsoudlou, P., Acharya, N.R. & Ramanan, A.V. The ADJUST trial and its implications for biologic discontinuation in juvenile idiopathic arthritis-associated uveitis. *Eye* 40, 288-289 (2026).
3. Schmeling H, Minden K, Foeldvari I, Ganser G, Hospach T, Horneff G. Efficacy and safety of adalimumab as the first and second biologic agent in juvenile idiopathic arthritis: the German Biologics JIA Registry. *Arthritis Rheumatol.* 2014 Sep;66(9):2580-9. doi: 10.1002/art.38741. PMID: 24942886.
4. Zholobova E, Galstian L, Nikolaeva MN, Loskutova OY. Effectiveness of adalimumab in the treatment of juvenile idiopathic arthritis associated with uveitis. *Pediatr Rheumatol Online J.* 2014 Sep 17;12(Suppl 1):O5. doi: 10.1186/1546-0096-12-S1-O5. PMCID: PMC4184154.

Around the World in Uveitis

SHAPU - Nepal's Seasonal Blinding Uveitis ↩

1. Kharel Sitaula R, Khatri A, Karki P, et al. Classification of

Seasonal Hyperacute Panuveitis (SHAPU). *Ocul Immunol Inflamm.* 2022;30(7-8):1751-1755.

doi:10.1080/09273948.2021.1944646

2. Goh YY, Sitaula RK, Rojas-Carabali W, et al. Half a Century's Quest for Seasonal Hyperacute Panuveitis (SHAPU): From Academic Curiosity to Public Health Concern. *Ocul Immunol Inflamm.* Published online January 13, 2025:1-8. doi:10.1080/09273948.2024.2444510
3. Upadhyay M, Kharel Sitaula R, Shrestha B, et al. Seasonal Hyperacute Panuveitis in Nepal: A Review over 40 Years of Surveillance. *Ocul Immunol Inflamm.* 2019;27(5):709-717. doi:10.1080/09273948.2018.1439643
4. Tamang S, Jayanna S, Tshering SC, et al. Seasonal Hyperacute Panuveitis (SHAPU) in Bhutan. *Ocul Immunol Inflamm.* 2024;32(2):203-209. doi:10.1080/09273948.2022.2164512
5. Upadhyay MP, Rai NC, Ogg JE, Shrestha BR. Seasonal hyperacute panuveitis of unknown etiology. *Ann Ophthalmol.* 1984;16(1):38-44.
6. Malla OK. Endophthalmitis probably caused by Tussock moth. In: Report of the Proceedings of the First National Seminar on Prevention of Blindness. Vol 44. 1978.
7. Upadhyay MP, Kharel Sitaula R, Manandhar A, et al. The Risk Factors of Seasonal Hyperacute Panuveitis. *Ophthalmic Epidemiol.* 2021;28(3):250-257. doi:10.1080/09286586.2020.1820533
8. Upadhyay MP, Shrestha BR. SHAPU: Forty years on Mystery Persists. *Nepal J Ophthalmol.* 2017;9(1):13-16. doi:10.3126/nepjoph.v9i1.17527
9. Manandhar A. Patterns of Uveitis and Scleritis in Nepal: A Tertiary Referral Center Study. *Ocul Immunol Inflamm.* 2017;25(sup1):S54-S62. doi:10.3109/09273948.2016.1161804
10. Bhusal DR, Kharel R, Shrestha BR, Baral S, Pandey B, Pathak P, Kumar B, Karki P, Joshi SN, Sharma AK, Upadhyay MP. Landscape Heterogeneity and Changing Bioclimatic Parameters Link to Potential Expansion of *Gazalina chrysolopha* (Lepidoptera; Notodontidae) in Nepal: A Possible Bioagent for Seasonal Hyperacute Panuveitis (SHAPU) in South Asia. *Ecol Evol.* 2025;15(10):e72325.
11. Kharel Sitaula R, Karki P, Gurung H, et al. A New Ray of Hope in the Treatment of Seasonal Hyperacute Panuveitis: Could Intravitreal Triple Combination Therapy of an Antibiotic with Dual Steroids Play a Pivotal Role? *Ocul Immunol Inflamm.* 2025;33(3):418-422. doi:10.1080/09273948.2024.2414918.

Closing Note from the Secretary

Curiosity, Conversations and Conferences ↩

1. Gruber MJ, Ranganath C (2019) How Curiosity Enhances Hippocampus-Dependent Memory: The Prediction, Appraisal, Curiosity, and Exploration (PACE) Framework. *Trends Cogn Sci* 23:1014-1025.
2. Kang MJ, Hsu M, Krajcich IM, et al (2009) The wick in the candle of learning: epistemic curiosity activates reward circuitry and enhances memory. *Psychol Sci* 20:963-973.



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