



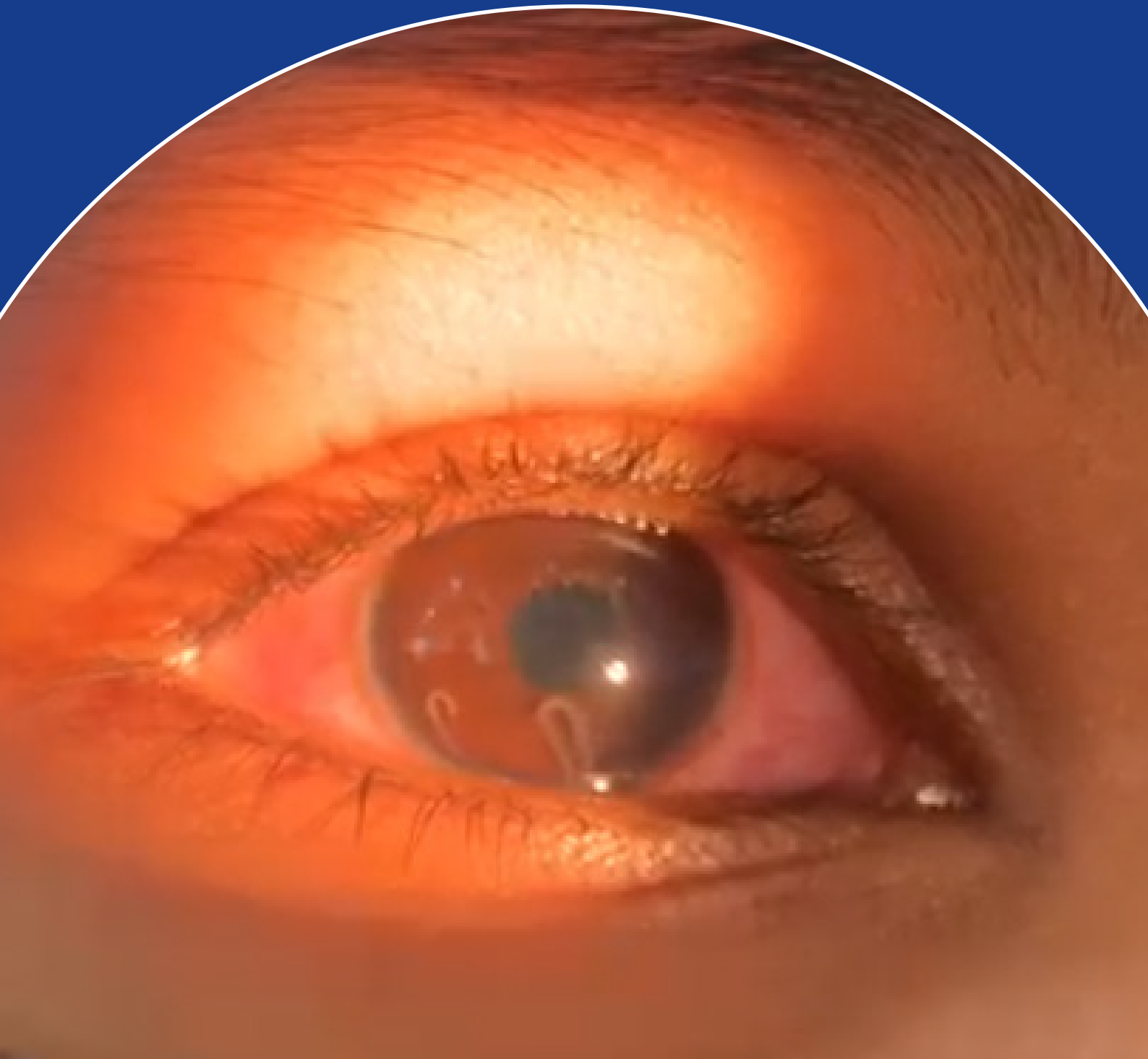
Uveitis Society (India)

EDITION-01

NEWSLETTER

VOLUME - 1 | FEBRUARY 2022

PARASITIC UVEITIS



From the Desk of **PRESIDENT**



Dear Members of USI,

Wishing you all a very safe, healthy and blessed 2022. It is indeed my pleasure and honour to write this foreword for the first USI letter of this year. These Newsletters from USI that is the initiative started by current executive committee have become a useful resource for young practicing Uveitis experts as well as comprehensive ophthalmologists. The main reason for its appeal is the simple way these facts are put together with lots of practical advice and pearls of wisdom from our senior members and we are indeed very thankful to all the members of our society for their contribution.

The theme of this Newsletter is 'Parasitic Uveitis'; an entity that we all deal with and yet miss it so many times. This newsletter is an excellent compilation of articles, cases, discussions on various types of parasitic uveitis and I am sure you all will enjoy reading it.

Once again, there is tremendous amount of hard work that goes into a project like this and the whole credit for this goes to Dr. Bala Murugan who works extremely hard with unmatched sincerity and dedication to bring out these Newsletters to you on time. I would also like to thank all the members of the Scientific Committee as well as authors and discussants who take the time out of their busy schedules to contribute to the newsletter.

Sincerely hope that you all enjoy reading it.

Dr. Vishali Gupta

MD, Professor Retina
Vitreous and Uvea Advanced Eye Centre
Post Graduate Institute of Medical Education and
Research, Chandigarh



From the Desk of **VICE PRESIDENT**



Dear Members,

Wish you all a very Happy New year 2022.

One of the educational initiatives of the Uveitis Society of India is the publication of the newsletter, focusing on specific topics in uveitis. Each newsletter is designed to give an overview, preferred practice patterns among experts and important publications on the specific topic. For a busy practitioner, this compilation of important information provides valuable assistance in dealing with cases of uveitis in their clinical practice.

The focus of this edition of the newsletter is "Parasitic uveitis". The topics discussed, range from different infections encountered, clinical tips on their diagnosis and management, the practice patterns among experts in this field, important publications, and an interesting crossword to tickle your brain cells.

On behalf of the USI, I thank all the authors for their valuable contributions. I also congratulate Dr. Balamurugan S, our editor and his team including Mrs. vaidehi for doing an exemplary job in bringing out this newsletter. We hope you enjoy reading this as much as we have enjoyed bringing this to you.

Dr. Kalpana Babu

Prabha Eye Clinic and Research Centre, Bengaluru



From the Desk of **USI SECRETARY**



Dear Colleagues,

Wishing you all a very safe and healthy 2022 !

We had a very successful virtual annual conference with some of the best uveitis experts both national and international. There was an excellent participation by our young budding uveitis specialists in various competitive sessions including some of the best free papers presented this year. The executive encouraged more participation this year by giving an increased number of prizes. The US(I) members directory was also released and hoping that all the members would have received their copy.

We are happy to share with you the first newsletter of the year 2022 on 'Parasitic Uveitis.' The newsletter covers all aspects of parasitic uveitis and our Editor in Chief – Dr. Bala Murugan along with his team and all the contributors have put in great efforts to compile it in the most interesting way for all of us to enjoy reading it. Hoping that all the members shall make the best use of this newsletter which is a treat to read.

Our website has been revamped and members of the society who are wanting to access the previous newsletters or annual conference recording, may login at indianuveitis.org and access them at their convenience. Please encourage your students/fellows to become a member of US(I) so that they too may gain access to all the academic material archived on the society website.

We have all gone through some of the most turbulent times due to the pandemic and therefore a physical conference was not possible for the last two years. Though difficult to predict, but we hope to meet you all in person during the forthcoming annual US(I) conference at Hyderabad. Requesting all the members to actively participate and make it a grand success.

I thank our president Dr Vishali Gupta for all her guidance and helping us to work as a strong team to take the work of the society forward.

Regards

Dr. Manisha Agarwal

Head of Vitreoretina Services
Dr. Shroff's Charity Eye Hospital
New Delhi



Uveitis Society (India)

From the Desk of **USI JOINT SECRETARY**



Dear friends,

Wishing you a very happy 2022, with good health and spirits. It is a proud moment for the Uveitis Society (India) to release the fourth edition of newsletter. The tremendous and constant hard work put in by Dr Balamurugan has made it possible every year ever since its release, along with the meticulous team effort by the Scientific Committee members. We thank all the contributors for their timely and precious work.

The current issue covers a topic with various common as well as uncommon entities seen in our uveitis clinics, which pose diagnostic or therapeutic challenges. We hope you all enjoy reading this edition. We look forward to your inputs and comments. Your suggestions are of immense value to the team for making the edition better every time. We also hope this newsletter encourages more and more readers who would be welcome to contribute anytime in future.

I am thankful to Dr Vishali Gupta, Dr Kalpana Babu and Dr Manisha Agarwal for their constant support and guidance.

We look forward to see you in person for our next annual meeting this year.

Stay safe and enjoy reading.

Dr. Reema Bansal

Professor, Uveitis and Vitreo-Retina Services
Advanced Eye Centre, PGIMER, Chandigarh





Uveitis Society (India)



Dr. Narsing A Rao
Patron-in-Chief



Dr. Vishali Gupta
President



Dr. Kalpana Babu
Vice President



Dr. Manisha Agarwal
Secretary



Dr. Reema Bansal
Joint Secretary



Dr. Sudharshan S
Treasurer



Dr. B Manohar Babu
Immediate Past President



Uveitis Society (India)

Executive Committee Members



Dr. Amala E George



Dr. Shishir Narain



Dr. Padmamalini Mahendradas



Dr. Somasheila Murthy



Uveitis Society (India)

Advisors



Prof. Carl Herbort



Prof. Amod Gupta



Dr. Jyotirmay Biswas



Dr. S R Rathinam



Dr. Virender S Sangwan



Dr. Sudha K Ganesh



Uveitis Society (India)

Scientific Committee



Dr. Padmamalini Mahendradas
Scientific Committee
Chairperson



Dr. Reema Bansal
Scientific Committee
Co - Chairperson



Dr. Salil Mehta
Member



Dr. Soumyava Basu
Member



**Dr. Parthopratin Dutta
Majumder**
Member



Dr. S Bala Murugan
Member
Editor-in-Chief



Uveitis Society (India)

Zonal Representatives



Dr. Subina Narang - North



Dr. Debashis Das - East



Dr. Navneet Mehrotra - West



Dr. Ankush Kawali - South



Dr. Samendra Kumar Karkhur - Central

From the USI



Pleasure is mine to be a part of the elegant editorial stalwarts in the field of uveitis who have actively contributed for this wonderful edition of USI newsletter on 'Parasitic uveitis'. To me, this topic is so special because it propelled me to opt for this niche area of Ophthalmology when my mentor motivated me in completing my thesis dissertation which was a road not oft travelled.

We in developing countries have an abundant research opportunities in this wide area of parasitic uveitis. The original researchers in this field from India had the right aptitude and attitude in converting the obstacles into opportunities which is worth emulating beyond Ophthalmology too!

The challenging era of COVID-19 is far behind us and we need to focus single mindedly at the modifiable risk factors in our life to make it an useful energetic voyage of life. We strongly believe that collective human endeavors shall bear triumphant fruits for sure like in this journey of USI newsletter. I sincerely thank all the motivating leaders, contributors, readers, critics for propelling us to improve and refine our sincere efforts. All the bouquets pertaining this newsletter are surrendered to the almighty. All the brickbats concerning this newsletter are valuable pearls for us to refine and define our selves in the future. Understand that you ask me why....It's because otherwise it would have been a lucid garland in the lotus feet of Almighty and make you smile....

Dr. S Bala Murugan

Chief of Uveitis services,

Aravind Eye Hospital, Pondicherry-605007

Email: drbalamuruganms@gmail.com



CONTENTS

Click on the icon to view the article...



Parasitic uveitis- Are there something new?

Dr. Dipankar Das

14



Pharmacology Of Drugs Used In Parasitic Uveitis

Dr. Bhavana Y, Dr. Yogish Subraya Kamath

18



Panel Discussion

Dr. Rathinam S R, Dr. J Biswas, Dr. Dipankar Das

25



Trematode induced uveitis

Dr. Rathinam S R, Dr. Vedhanayaki R, Dr. S Bala Murugan

36



Crossword Parasite puzzle

Dr. Vinaya Kumar Konana, Dr. Kalpana Babu

44



Ocular Toxocariasis – An update

Dr. Nivedita Nair, Dr. Shilpa I N, Dr. Jyotirmay Biswas

47



Parasitic Uveitis : Review of literature

Dr. Yogesh Yadav, Dr. Rakesh Verma, Dr. Manisha Agarwal

56



Diffuse Unilateral Subacute Neuroretinitis (DUSN)

*Dr. Anamika Patel, Dr. Ketan Saoji, Dr. Jyothirmay Manasa
Dr. Avinash Pathengay*

73

CONTENTS

Click on the icon to view the article...



Recent Advances in Parasitic uveitis

Dr. Nikitha Ayyadurai, Dr. Reema Bansal

83



CYSTICERCOSIS

*Dr. Padmamalini Mahendradas, Dr. Sai Bhakti Mishra
Dr. Ankush Kawali, Dr. Namita Dave, Dr. Srinivasan Sanjay*

95



Rewards and Recognition

107



USI Directory 2021

122



USI Academic Programms in 2021

123



Revamped USI website

141



Crossword Puzzle Answers

142



USICON 2022 - Hyderabad

145

Parasitic uveitis- Are there something new?



Dr. Dipankar Das

HOD: Uveitis & Ocular Pathology Services Department of Uveitis,
Uveitis and Neuro-ophthalmology Sri Sankaradeva Nethralaya
96 Basistha Road, Beltola, Guwahati, Assam, INDIA

Parasitic uveitis- Are there something new?

Parasites are free living organisms which infect human beings causing different systemic manifestations including uveitis. ^[1,2,3,4] The story of parasites and intra-ocular infections in human population started long during the emergence of civilization. ^[1,2,3,4] Uveitic parasites are seen in different parts of the World including India. Various protozoa's such as Cystoidea, trematodes, tissue flagellates, protozoa, hydatidosis, paragonimiasis, toxoplasmosis, onchocerciasis, toxocariasis, ophthalmomyiasis and other zoonoses were seen causing uveitis and adnexal parasitosis. ^[1,2,3,4]

In India, before the appearance of tubercular uveitis, toxoplasma retinochoroiditis was the commonest posterior uveitis entity seen in different pattern of uveitis studies from different parts of India. ^[1,2,3,4] Prof. Biswas from Chennai reported *Gnathostoma spinigerum* from India followed by various other case reports from different corners of the country including North east India. ^[1,2,3,4] Prof. Rathinam had done extensive study on trematode infections from swimming pool in Madurai and had number of research publications in that aspect. ^[2] Author(s) had found a unique method of detecting the live parasites under the objective of compound microscope where internal structures of various parasites were documented rapidly for the diagnosis. ^[3,4] Author(s) have also extended their studies with a new staining method by Fluorescein for better characterization of parasitic diagnosis and compared them with wet mount preparation. ^[5]

Some of the parasitic infections and research were very interesting in the past when, 2015 Nobel Prize in physiology or medicine was awarded to Prof. Youyou Tu, Prof. William C. Campbell and Prof. Satoshi Omura for their enormous contribution in the drug therapies for filariasis and malaria thereby reducing larger burden of morbidity and mortality by those two parasitic diseases. ^[6]

It has been observed that some idiopathic diseases that cause uveitis can have infectious etiologies like parasitic disease(s). ^[7] Sarcoidosis is one of the idiopathic diseases where infectious etiologies including parasitic diseases can be triggering factors. ^[7] Paragonimiasis is a parasitic infectious disease caused by lung fluke was discovered in one of the states of North east India where pulmonary abnormalities were seen by parasitic migration and this pulmonary disease is endemic in Southeast Asia. ^[8] Whether the parasitic infection can simulate sarcoid like picture and induced uveitis by various immunological mechanisms is just a thought process to explore. ^[7, 8]

It is very interesting to think whether cancer can occur in parasitic protozoans, blood parasites and obligate intracellular such as *Toxoplasma gondii* ? ^[9,10] In such case, cancer affected protozoa residing in different parts of the body including retinal pigment epithelial cells of the retina may harbor carcinogenic agent/cells inducing cancers at different sites which require extensive knowledge about the parasites and (?) cancer causing by them. Few parasites like

Clonorchis sinensis and *Opisthorchis felinus* are known to cause cholangiocarcinoma in human and experimental animals. Plasmodia species such as *Plasmodium falciparum* and *Plasmodium vivax* indirectly known to cause non-endemic Burkitt's lymphoma. [9,10]

In ocular toxoplasmosis, altered immunobiology in viable tachyzoites and tissue cysts may be found in superficial layers of infected retina and intense mononuclear inflammatory cell reaction seen in the involved retina and the adjoining structures. [11] In HIV infected individual with toxoplasmosis, there may be severe inflammatory reaction in the active stage of the disease and they are generally bilateral as well as multifocal. [1, 2, 3, 4, 11] It is not clearly known or studied about the immuno-biology of bradyzoites and tachyzoites getting change with co-infection of HIV (?). In some parasitic diseases having fulminant viral infections, the parasites should have some altered biochemical change within them which need to be seen in the experimental animals. Co-infections can even translate into genomic alteration of those infected parasites and may behave or act at the retichoroidal sites differently.

Filaria worms sometimes exhibit *Wolbachia* (a Rickettsia) living symbiotically in a coats of microfilaria in a pattern similar to mitochondria which plays an important role in the fertility of the female filarial worm. [12] Recent advances point out that therapies targeting wolbachia with antibiotics including few weeks course of Tablet Doxycycline in onchocerciasis kill the parasites as well. [12,13] Onchocerciasis is an important WHO targeted preventable disease prevailing in central, east Africa, smaller part of Sudan, central and South America. About 20 million people are affected by the disease mostly in the savannah areas. [12,13]

To sum up, ocular parasites and their manifestations are very important for infectious disease understanding. We need to abreast the newer concepts of uveitic parasitology for the better management of the parasitic diseases in general and parasitic uveitis in particular.

References:

1. Biswas J, Narain S, Das D, Ganesh SK. Pattern of uveitis in a referral uveitis clinic in India. *Int Ophthalmol* 1996-1997;20:223-8.
2. Rathinam SR, Namperumalsamy P. Global variation and pattern changes in epidemiology of uveitis. *Indian J Ophthalmol* 2007;55:173-83
3. Das D, Bhattacharjee H, Bhattacharyya PK, Jain L, Panicker MJ, Das K, et al. Pattern of uveitis in North East India: A tertiary eye care center study. *Indian J Ophthalmol* 2009;57:144-6.
4. Das, D., Ramachandra, V., Islam, S., Bhattacharjee, H., Biswas, J., Koul, A., Deka, P., & Deka, A. (2016). Update on pathology of ocular parasitic disease. *Indian journal of ophthalmology*, 64, 794–802.

5. Das D, Deka P, Bhattacharjee H, Deshmukh S, Gupta P, Deka A, Sultana S. Fluorescein dye as a novel cost-effective approach for staining raw specimens in ophthalmic pathology. *Indian J Ophthalmol.* 2020;68):2175-2178.
6. MLA style: Tu Youyou – Facts. NobelPrize.org. Nobel Prize Outreach AB 2021. Tue. 9 Nov 2021. <<https://www.nobelprize.org/prizes/medicine/2015/tu/facts/>
7. Jamilloux Y, Valeyre D, Lortholary O, et al. The spectrum of opportunistic diseases complicating sarcoidosis. *Autoimmun Rev* 2015;14:64–74.
8. Kim TS, Han J, Shim SS, et al Pleuropulmonary paragonmiasis: CT findings in 31 patients. *AJR* 2005;185:616-621
9. Fernandes R, Alves H, Botelho MC. The cancer hygiene hypothesis: from theory to therapeutic helminths. *Curr Cancer Ther Rev.* (2019). doi: 10.2174/1573394714666181003143717.
10. Subauste CS, Remington JS. Immunity to *Toxoplasma gondii*. *Curr Opin Immunol.* 1993;5:532–7.
11. Luft BJ, Remington JS. Toxoplasmic encephalitis in AIDS. *Clin Infect Dis.* 1992;15:211–22.
12. Onchocerciasis (also known as river blindness). *Parasites.* CDC. 21 May 2013.
13. Iturbe-Ormaetxe, S.L.O” Neill. *Wolbachia* – Host interactions: Connecting phenotype to genotype. *Curr Opin Microbiol*;10:221-224



Pharmacology Of Drugs Used In Parasitic Uveitis



Dr. Yogish Subraya Kamath

Professor, Department of Ophthalmology
Kasturba Medical College, Manipal

Dr. Bhavana Y

Kasturba Medical College, Manipal

PHARMACOLOGY OF DRUGS USED IN PARASITIC UVEITIS

Introduction

Intraocular inflammation may be caused by various endoparasites, including protozoa and helminths. Protozoal diseases such as Toxoplasmosis, Leishmaniasis, Giardiasis and Malaria have manifestations involving the posterior segment and optic nerve. Other protozoal diseases including Acanthameba infections, Chagas disease, and Microsporidiosis, usually affect the outer coats of the eye. Pharmacotherapy forms the mainstay of treatment in protozoal diseases. Although the definitive treatment of helminthic ocular infections is surgical removal of the worm, pharmacotherapy with anthelmintic agents, may be used as an adjuvant or in cases where isolation is not possible. However, the resulting hypersensitivity reaction to the antigens from the cell wall, or toxins released, from the dead parasites needs additional steroid cover. This is especially important in cases where there are high chances of coexisting neurological involvement, as in cases of cysticercosis.

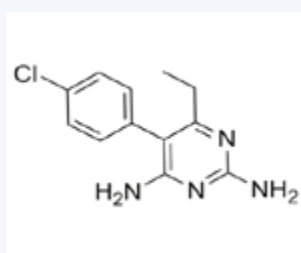
The drugs used in the major forms of parasitic uveitis are discussed in the sections below.

PHARMACOLOGY OF PROTOZOAL UVEITIS

Toxoplasmosis

Toxoplasmosis is one of the most common aetiologies of parasitic posterior uveitis. It is caused by *Toxoplasma gondii* and it is most commonly congenital but can be acquired through inhalation of oocysts from cat waste or ingestion of contaminated uncooked lamb or pork. The following drugs below are used in the treating *Toxoplasmosis*.

1. Pyrimethamine



Mechanism of action: It inhibits bacterial dihydrofolate reductase, thereby inhibiting production of folic acid.

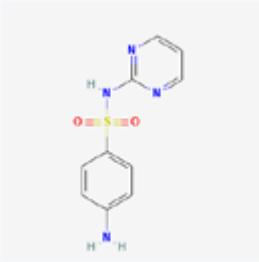
Dosage: 100mg on Day 1, followed by 25mg twice daily.

[in people less than 70 kg, 50 mg on Day 1, followed by 25 mg once daily]

Adverse effects: Bone marrow suppression needs to be monitored with weekly blood counts. Other adverse effects include megaloblastic anaemia, nausea, vomiting, diarrhoea, rash, seizures, ataxia, tremors, atrophied glossitis, fever.

Folinic acid (Leukovorin) supplementation weekly is essential to prevent hematological toxicity.

2. Sulfadiazine



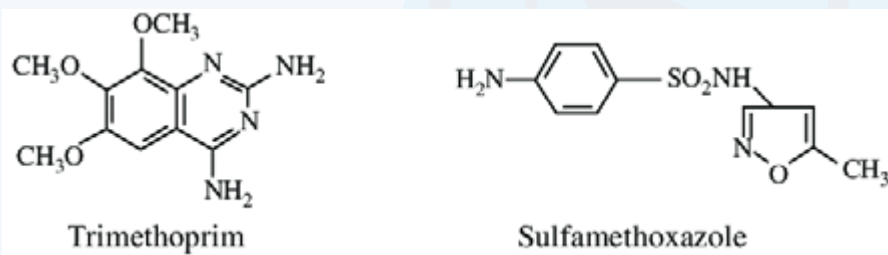
Mechanism of action: They competitively inhibit the enzyme dihydropteroate synthase, which is absent in mammalian cells, causing the inhibition of production of folic acid in the pathogen.

Dosage: sulphadiazine- 0.5-1 g four times a day

Adverse effects: Bone marrow suppression, nausea, vomiting, headache, tinnitus, convulsions, nephropathy.

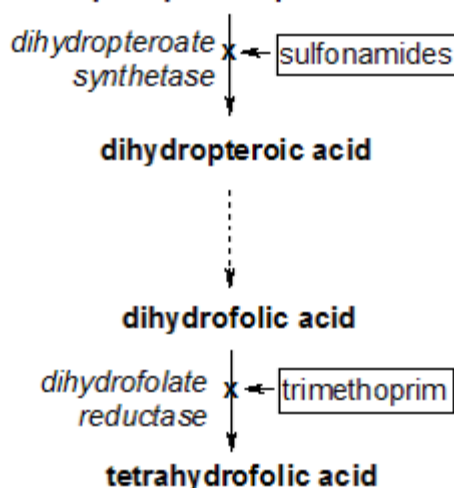
Sulfadiazine is usually given along with Pyrimethamine and Folinic acid supplementation.

3. Trimethoprim-Sulfamethoxazole [Co-trimoxazole]



Mechanism of action: Sulfamethoxazole inhibits dihydropteroate synthase; and Trimethoprim inhibits dihydrofolate reductase, thereby having a better efficacy when used in combination.

dihydropteroate diphosphate + p-aminobenzoic acid (PABA)



Dosage: Trimethoprim- 160 mg twice a day

Sulfamethoxazole- 800 mg twice a day

Adverse events: Nausea, vomiting, anorexia, rash, urticaria, bone marrow suppression.

4. **Azithromycin**

It is a semisynthetic derivative of erythromycin.

Mechanism of action: they inhibit bacterial protein synthesis by reversibly binding to bacterial 50s ribosomal subunit and inhibiting the translocation of aminoacyl site to peptidyl site.

Dosage: oral- 500 mg once daily.

Adverse effects: mild gastric upset, abdominal pain, headache, dizziness, cardiac arrhythmias, QT prolongation.

It is better absorbed on empty stomach. It has excellent tissue penetration and a long half-life which makes it suitable for once daily administration.

Spiramycin is another macrolide class of drug used in the treatment of toxoplasmosis during pregnancy. It is given in the dosage of 1000 mg q 8 hourly, throughout pregnancy or till fetal infection if any, is established.

5. **Clindamycin**

It is derived from lincomycin.

Mechanism of action: it binds to 50s ribosomal subunit and inhibits protein synthesis by interfering with formation of initiation complexes.

Dosage: 300 mg four times a day.

Adverse effects: pseudomembranous colitis, rash, nausea, vomiting, bone marrow suppression, cardiopulmonary arrest (rare).

Cross resistance of clindamycin and azithromycin may occur due to similar site of action in the protein synthesis cycle.

Clindamycin may also be used intravitreally, in dose of 1 to 1.5 mg/0.1 ml, in whom systemic therapy for ocular toxoplasmosis is not tolerated.

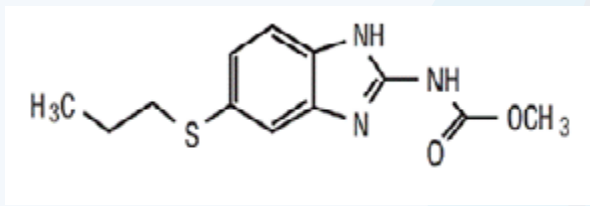
Leishmaniasis

A combination of systemic anti-leishmanial agent with steroids are the mainstay of therapy in uveitis due to leishmaniasis. The severe inflammation is presumed to be due to both, infection and an immune reconstitution type of reaction occurring due to increased antigen load after with the use of systemic anti-leishmanial therapy. Topical, subconjunctival steroids under the cover of intracameral amphotericin B and meglumine antimoniate 20mg/day IM for 2 months is recommended.

PHARMACOLOGY OF UVEITIS DUE TO HELMINTHS

The frequently used drugs in helminthic uveitis include Albendazole, Praziquantel, Ivermectin and Diethylcarbamazine. Albendazole is preferred in larva migrans and round worms such as Ascaris and Ancylostoma. Ivermectin is the drug of choice in Onchocerciasis, and Strongyloides infestation. It may also be used in Filariasis and Loaiaasis, where DEC is the drug of choice. Trematodes are treated using Praziquantel. Albendazole is also the drug of choice in cysticercosis.

1. **ALBENDAZOLE**



Chemical name: Benzimidazole carbonate.

Pharmacokinetics: For anthelmintic activity in the tissues, it is taken with fatty meal, which enhances absorption. It achieves good concentration in the tissues, including the cerebrospinal fluid.

Mechanism of Action: After absorption, it undergoes first pass metabolism in the liver to its active metabolite, albendazole sulfoxide. It inhibits polymerisation of microtubule and glucose uptake resulting in worm immobilisation and death. It also has larvicidal activity in cysticercosis and hookworm infestation. Dosage: Varies depending on the type of worm. It is used in doses of 400 mg stat and after 2 weeks for hookworm infestation; 800 mg per day for 2 to 3 weeks in cysticercosis.

Adverse effects: headache, dizziness, fever, alopecia, jaundice, and neutropenia. Long term use can cause bone marrow suppression.

When used in suspected central nervous system or ocular infestation, steroids need to be administered additionally, to avoid severe inflammatory response to the dying organisms.

USES: ascaris, trichuriasis, cysticercosis, strongyloidiasis.

2. IVERMECTIN

It is derived from *Streptomyces avermitilis*. It is a semisynthetic hydrogenated macrocyclic lactone.

Pharmacokinetics: it is well absorbed orally, and does not enter CNS (Central Nervous System). It is metabolised by CYP3A4.

Mechanism of action: it causes paralysis of the microfilaria by activating the glutamate gated chloride channel causing hyperpolarisation and then phagocytosis to eliminate them from skin and ocular tissue.

Dosage: Oral 150 microg/kg single dose.

Side effects: ocular side effects include conjunctivitis, punctate corneal opacities and rarely blindness. Systemic effects include mild pruritis, giddiness, nausea, abdominal pain, constipation, fever, arthralgia, lethargy, and transient ECG changes. It is to be avoided in pregnancy and children below 5 years.

Drug interactions: ivermectin should not be given with agents that increase GABA activity like barbiturates, benzodiazepines, valproate.

USES: drug of choice in Onchocerciasis. *W.Bancrofti*, Strongyloidiasis, Ascaris, trichuriasis, enterobiasis.

3. DIETHYLCARBAMAZINE CITRATE (DEC)

It is a derivative of synthetic piperazine

Pharmacokinetics: It is immediately absorbed from the GI (Gastro Intestinal) tract, uniformly distributed except for the fat, metabolised in the liver, and excreted within 30 mins.

Mechanism of Action: It acts by altering the organelle membrane of the microfilaria so that they become susceptible to phagocytosis. It also causes muscular paralysis of microfilaria and adult worms so that they are expelled alive.

Dosage: 6 mg per kg body weight per day in three divided doses, administered orally for 3 weeks.

Adverse effects: Nausea, vomiting, diarrhoea, loss of appetite, headache, weakness, and dizziness are common ones. A febrile reaction called Mazzotti reaction is seen where rash, pruritus, enlargement of lymph nodes, bronchospasm and fall in BP may occur due to allergic reaction to dead microfilaria. Leukocytosis and mild albuminuria are usually infrequent.

USES: filariasis, filariasis prophylaxis, tropical eosinophilia, larva migrans.

4. PRAZIQUANTEL

It is a synthetic isoquinoline pyrazine derivative.

Pharmacokinetics: Praziquantel is rapidly absorbed from intestines; Absorption is enhanced if it is ingested with carbohydrate food. It has high first pass metabolism in liver.

Mechanism of action: It is active against adult as well as juvenile and larval stages of tapeworms. At low concentration causes spastic paralysis in susceptible helminths and vacuolization of parasite tegument at higher concentration due to increased cell membrane permeability to calcium.

Dosage: 20mg/kg per dose: twice or thrice at 6-hour intervals, for schistosomiasis.

Adverse effects: headache, dizziness, nausea, vomiting, itching, urticaria, rashes, fever and bodyache. Katayama fever when treated with praziquantel may cause hallucinations and psychotic symptoms.

Drug interactions: Phenytoin, carbamazepine and dexamethasone induce praziquantel metabolism and further decrease its bioavailability.

USES: it is the drug of choice in Schistosomiasis. Also given in taeniasis, hydatid disease.

References

1. Contini C. Toxoplasmosis. In: Conn's Current Therapy 2021. Elsevier; 2019.
2. Braakenburg A., Rothova A. (2016) Advances in Ocular Toxoplasmosis. In: Biswas J., Majumder P. (eds) Uveitis: An Update. Springer, New Delhi. https://doi.org/10.1007/978-81-322-2295-8_1
3. Rathinam S.R., Kamath Y. (2016) Ocular Parasitic Infections. In: Biswas J., Majumder P. (eds) Uveitis: An Update. Springer, New Delhi. https://doi.org/10.1007/978-81-322-2295-8_13
4. Beauduy CE, Winston LG. Tetracyclines, Macrolides, Clindamycin, Chloramphenicol, Streptogramins, Oxazolidinones, & Pleuromutilins. In: Katzung BG, Vanderah TW. eds. Basic & Clinical Pharmacology, 15e. McGraw Hill; 2021. Accessed November 22, 2021. <https://accessmedicine.mhmedical.com/content.aspx?bookid=2988§ionid=250601804>
5. Mignot, Guillaume; Bhattacharya, Yagnaseni; Reddy, Aravind Ocular Leishmaniasis - A systematic review, Indian Journal of Ophthalmology: May 2021 - Volume 69 - Issue 5 - p 1052-1060 doi: 10.4103/ijo.IJO_2232_20
6. Rosenthal PJ. Pharmacology of the Antihelminthic Drugs. In: Katzung BG, Vanderah TW. eds. Basic & Clinical Pharmacology, 15e. McGraw Hill; 2021. Accessed November 24, 2021. <https://accessmedicine.mhmedical.com/content.aspx?bookid=2988§ionid=250603258>

PANEL DISCUSSION



Dr. S R Rathinam

Principal, Head of uveitis service, Aravind Eye Hospital & PG. Institute of Ophthalmology, Madurai, Tamil Nadu, India



Dr. Dipankar Das

Senior Consultant & HOD: Uvea-Ocular Pathology
Department of Ocular Pathology, Uveitis & Neuro-Ophthalmology Services
Sri Sankaradeva Nethralaya, Guwahati, Assam, INDIA



Dr. Jyotirmay Biswas

Director of Uveitis & Ocular Pathology Department,
Sankara Nethralaya, Chennai

1. What are the commonest parasitic uveitis they have encountered in clinical practice?

● **Dr. S R Rathinam**

Toxoplasmosis, Toxocara, DUSN.

● **Dr. Dipankar Das**

Toxoplasmosis, Toxocariasis, Cysticercosis amongst first three causes of parasitic uveitis seen in my clinical practice.

● **Dr. Jyotirmay Biswas**

Commonest parasitic uveitis I have encountered is toxoplasmic retinochoroiditis. In fact, it was the most common posterior uveitis in my study of pattern of uveitis published in 1995, Indian Journal of Ophthalmology (Das D, Biswas J and Ganesh S Indian Journal of Ophthalmology 1995; 43(3): 117-121.

2. Do we need serology to probe uveitis following toxoplasmosis. If not, why? If so, in which situations?

● **Dr. S R Rathinam**

In pure ocular Toxoplasmosis without systemic signs or symptoms of Toxoplasma, IgG serology is unlikely to be positive. If importance is given to serology you can miss the diagnosis .

Again toxoplasma IgG can be positive in patients from endemic areas . I would give importance to rising titres of IgG and IgM with reduction in the titres with treatment in doubtful cases.

● **Dr. Dipankar Das**

Usually, we do not require it but in a diagnostic dilemma, sometimes the serum ELISA (IgM, IgG, and Avidity) is done to correlate the findings. Negative test is also important in establishing other infectious uveitis.

● **Dr. Jyotirmay Biswas**

I routinely do ELISA toxoplasmosis however I do not wait for the serology result and I start treatment based on my clinical diagnosis of toxoplasmic retinochoroiditis which is often very characteristic with focal retinochoroiditis and localized vitreous haze. I however see that the serology confirms my diagnosis or not. If toxoplasma IgG or IgM antibody is negative, I like to revisit and see whether I am missing some other diagnosis like viral or syphilitic retinitis. If so in which situation? . If the lesions are multifocal if the vitreous haze is dense or if there is no vitreous haze, I try to rule out other condition mimicking toxoplasmic retinochoroiditis like viral, fungal and syphilitic retinitis.

3. Would you monitor serology in chronic / recurrent toxoplasma retinitis to decide duration of the therapy?

● **Dr. S R Rathinam**

No we do not monitor serology in chronic or recurrent cases to plan treatment duration . Treatment is given for 6 weeks or till clinical resolution of the lesion is achieved.

● **Dr. Dipankar Das**

Not really.

● **Dr. Jyotirmay Biswas**

No, I do not do repeat serology in chronic or recurrent toxoplasmic retinochoroiditis because if IgG is positive, it remains positive.

4. Monotherapy Vs poly therapy in systemic toxoplasmosis treatment. Which is your choice. Please substantiate.

● **Dr. S R Rathinam**

We prefer polytherapy for ocular Toxoplasmosis to prevent development of resistance. In my clinical practice, I prefer to choose Tablet Azithromycin as a mono-therapy for active toxoplasma retinochoroiditis. Most of them respond with it in combination of oral steroid. In non responding cases, I would prefer poly-therapy.

● **Dr. Dipankar Das**

In my clinical practice, I prefer to choose Tablet Azithromycin as a mono-therapy for active toxoplasma retinochoroiditis. Most of them respond with it in combination of oral steroid. In non responding cases, I would prefer poly-therapy.

● **Dr. Jyotirmay Biswas**

I prefer to give polytherapy in systemic toxoplasmosis. I give Clindamycin in adult, 300 mg one tablet four times daily and in the children 150 mg one tablet four times daily along with tablet Bactrim DS one tablet two times daily. I also give oral prednisolone (1mg per kg by body, weight, 48 hours after antitoxoplasma treatment. The duration of the treatment for six to eight weeks.

5. How intravitreal clindamycin use benefit in sight threatening toxoplasmosis retinochoroiditis

● Dr. S R Rathinam

We use intravitreal clindamycin in extensive ocular toxoplasma retinitis and when lesions are close to the disc or threatening the macula. Deposition of the drug close to the site of lesion is beneficial in ensuring rapid resolution .

Also useful in patient who cannot tolerate oral medications.

Useful in pregnancy where sulpha drugs and trimethoprim are contraindicated.

● Dr. Dipankar Das

Very useful though it is an ophthalmic off level intra-vitreous drug.

● Dr. Jyotirmay Biswas

I give intravitreal clindamycin with dexamethasone for the quick resolution of inflammation in macular threatening toxoplasmic retinochoroiditis or if the patient is intolerant to clindamycin tablet like developing diarrhea following its use.

I also give intravitreal clindamycin in case of pregnant patients.

6. What is the approach of treatment in live worm in DUSN?

● Dr. S R Rathinam

If the live worm can be well localized retinal laser to the head of the worm .If this fails T Albendazole for 21 days with oral steroids.

● Dr. Dipankar Das

If the worm is live, we prefer to do laser photocoagulation. In dead worm, we could give a course of Tablet Albendazole (400 mg daily for 3-4 weeks) along with oral steroid.

● Dr. Jyotirmay Biswas

If the live worm is seen in the vitreous cavity, we try to remove it by vitreous surgery.

If it is in the subretinal space, we give photocoagulation on the parasite and around the parasite along with oral steroids to suppress inflammatory response due to dying parasite.

7. Which is the commonest presentation of ocular toxocariasis in your practice?

● **Dr. S R Rathinam**

Peripheral granuloma with traction band extending from the granuloma to the disc.

● **Dr. Dipankar Das**

Peripheral granuloma ± TRD.

● **Dr. Jyotirmay Biswas**

The commonest presentation of ocular toxocariasis in my practice is peripheral granuloma with vitreous membrane and folds.

8. What will be the management of trematode infections in eye-algorithm?

● **Dr. S R Rathinam**

Topical steroids and cycloplegics.

Oral corticosteroids may be added

Granuloma excision/aspiration if not resolving with medical management

● **Dr. Dipankar Das**

I do not have personal experience treating trematode infection or trematode granuloma in eye.

● **Dr. Jyotirmay Biswas**

I have not encountered any trematode infection in the eye so far. I am unable to comment on this

9. In the management of Ocular cysticercosis do you prefer/ do any new option in the surgical approach?

● **Dr. S R Rathinam**

Prefer surgical management. Remove the cyst in toto without rupture of cyst

MRI brain to rule out CNS involvement

● **Dr. Dipankar Das**

Pars plana vitrectomy is now a good option to remove the scolex and the worm in cysticercosis from retina-vitreous sites practiced by retina colleague in our Institute.

● **Dr. Jyotirmay Biswas**

We try to remove the cysticercus in toto as much as possible. In case if it is not possible we try to chew it up during vitrectomy.

10. How would you manage recurrent CNVM due to toxoplasma scar in absence of inflammation?

● **Dr. S R Rathinam**

OCT,FFA

Intravitreal anti VEGF with systemic antitoxo medications and oral corticosteroids for inflammatory control and antiparasitic cover.

● **Dr. Dipankar Das**

Intra-vitreous Anti VEGF and Clindamycin.

● **Dr. Jyotirmay Biswas**

The management of CNVM following toxoplasma, I give anti-VEGF injection, if the toxoplasma lesion is inactive.

11. Do you advise anti-helminth therapy along with steroids in the treatment of toxocara? And how long do you continue (in children and in adults)?

● **Dr. S R Rathinam**

Would prefer to give antihelminths along with steroids especially if there is significant vitritis and patient is symptomatic and if the granuloma looks clinically active.
Duration 21 days.

● **Dr. Dipankar Das**

No.

● **Dr. Jyotirmay Biswas**

I don't give anthelmintic therapy with steroids in the treatment of toxocara. I feel the parasite is already dead, when lesions are seen in the eye. However if there is larva migrants we give antitoxocara treatment.

12. When do you advise surgical intervention in toxocara? Would you advise surgical excision of toxocara granuloma which requires prolonged steroid therapy or is recurrent despite adequate steroid therapy?

● **Dr. S R Rathinam**

Surgical treatment for indolent granuloma and band causing macula traction.

● **Dr. Dipankar Das**

If extensive TRD, and mostly under oral steroid coverage

● **Dr. Jyotirmay Biswas**

When there is tractional retinal detachment or presentation of endophthalmitis like picture, we do vitreoretinal surgery to remove the vitreous opacity and the membrane. Yes. We advise surgical excision of toxocara granuloma in such cases.

13. Do you routinely advise stool examination in parasitic uveitis?

● **Dr. S R Rathinam**

No.

May be useful in cysticercosis where man is the definitive host. I do not advise stool examination routinely in parasitic uveitis.

● **Dr. Dipankar Das**

Not really. But it can be sometimes useful for routine screening

● **Dr. Jyotirmay Biswas**

I do not advise stool examination routinely in parasitic uveitis.

14. How do you confirm the diagnosis of intraocular filariasis? How do you treat intraocular filariasis? What is your drug of choice?

● **Dr. S R Rathinam**

AC tap for cytology for eosinophils and for the microscopic examination of the worm. Drug of choice is diethylcarbazipine citrate for 21 days

● **Dr. Dipankar Das**

If the worm is live, we can bring it out in toto and examine under objective of compound microscopy and then do the wet mount preparation. This would give detail morphological identification of the worm. Ivermectin works well in some of filarial worms.

● **Dr. Jyotirmay Biswas**

We remove the filaria parasite and see under the microscope which often has typical appearance with sheathed parasite. You can see eosinophils in the intraocular fluid. We make all attempt to remove the filaria worm.

Diethylcarbazine (DEC) is the drug of choice, in case if we are unable to remove it or if there is peripheral smear in the night blood showing microfilaria.

15. What is the role of anti-parasitic treatment in toxocariasis.

● **Dr. S R Rathinam**

Most of the time the worm is dead and antiparasitic treatment may not be necessary. However would use antiparasitic treatment if the granuloma looks clinically active and if there is dense vitritis in addition.

● **Dr. Dipankar Das**

I do not use anti microbial in toxocariasis. I use only oral steroid

● **Dr. Jyotirmay Biswas**

I feel antiparasitic treatment in toxocariasis has no role.

16. When will you perform vitreoretinal surgery in toxocariasis.

● **Dr. S R Rathinam**

Tractional retinal detachment .
ERM with macula pucker
Media clearing

● **Dr. Dipankar Das**

In TRD.

● **Dr. Jyotirmay Biswas**

We perform the vitreoretinal surgery if there is endophthalmitis like picture or there is retinal detachment involving the posterior pole.

17. What is your approach towards using oral corticosteroids in parasitic infections.

● **Dr. S R Rathinam**

Corticosteroids are indicated in parasitic infections to control inflammation. Dose 1-1.5 mg / kg body . Anti helminthics can be added after 2 days after starting corticosteroids

● **Dr. Dipankar Das**

Intraocular Cysticercosis: Surgery to remove the scolex followed by Tablet Albendazole/ Praziquantel and oral steroid: In orbital or eyelid cysticercosis- Albendazole/ Praziquantel can be started in first instance along with corticosteroid.

● **Dr. Jyotirmay Biswas**

I do not use the oral corticosteroids routinely in cysticercosis but if there is degenerative cysticercosis causing inflammation, I give oral Prednisolone at 1 mg/kg of the body weight.

18. How do you go about systemic investigations in cysticercosis?

● Dr. S R Rathinam

MRI Brain

MRI orbit

CT brain for demonstrating cerebral calcifications.

Complete blood count -eosinophilia.

Absolute eosinophil count

CSF analysis for antigen

Stool examination

● Dr. Dipankar Das

ELISA for cysticercosis (IgG and IgM) is available in some referral Labs in India. MRI brain and orbit for neuro-cysticercosis and eosinophil and absolute eosinophil count (AEC) can be done.

● Dr. Jyotirmay Biswas

If the patient is having neurological symptoms, I do get MRI of the brain but not routinely.

19. What dietary advice do you suggest in patients with cysticercosis?

● Dr. S R Rathinam

Avoid undercooked pork,

Wash hands thoroughly with soap and water after handling meat.

Vegetables to be washed and skin peeled well .

Prefer cooked vegetables to salads

Consume only boiled water or freshly bottled water.

● Dr. Dipankar Das

All vegetables, fruits for salads etc should be thoroughly cleaned; Open/ outside displayed vegetables/meat, fish should be avoided; Raw pork, beef etc should not be taken . They need to be cooked adequately. Hand washing before meal is very important habit to avoid any sort of infections.

● Dr. Jyotirmay Biswas

I advise to avoid eating pork and undercooked meat and proper washing of vegetables.

20. In the transport of worm from anterior or posterior segment what are the precautions to be taken from OR to the laboratory?

● **Dr. S R Rathinam**

Live worm can be transported in saline .

Dead worms can be transported in formalin

● **Dr. Dipankar Das**

Rapid transport, Live worm should be put in a vial containing normal saline; Dead worm can be transported in 10 % neutral buffered formalin. If you plan to do scanning electron microscopy of the worm, you need to fix them in 2% Gluteraldehde.

● **Dr. Jyotirmay Biswas**

I put the parasite in the distilled water and bring the parasite from the operation theater to the laboratory and see as soon as possible in a wet mount preparation. Subsequently, I try to take microphotograph and try to see the head and tail end of the parasite and internal structures to identify the parasite as well as its sex.

21. When do you consider Polymerase Chain Reaction[PCR] test for parasites?

● **Dr. S R Rathinam**

Not required but can be done for ocular toxoplasmosis when the diagnosis is in doubt.

● **Dr. Dipankar Das**

First requisite-we need to have the primer to see the particular parasite; Second, in diagnostic dilemma and where other mode of diagnosis is inadequate; and third, we can do PCR from dead worm-DNA can be seen for academic/research purpose.

● **Dr. Jyotirmay Biswas**

Polymerase chain reaction can be done in the toxoplasmosis in case of HIV infection from the aqueous or vitreous fluid.

Also, In case of dirofilaria species nowadays polymerase chain study of the histopathologic section from the DNA of the parasite can be done.

Trematode induced uveitis



Dr. S R Rathinam

Principal, Head of uveitis service, Aravind Eye Hospital & PG. Institute of Ophthalmology, Madurai



Dr. Vedhanayaki R

Uvea Consultant, Aravind eye care system, Madurai



Dr. S Bala Murugan

Chief of Uveitis services,
Aravind Eye Hospital, Pondicherry

Trematode induced uveitis

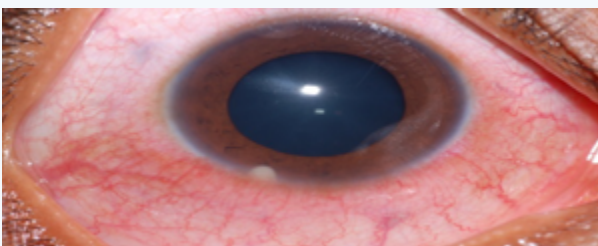
Introduction:

Infection can be the common cause of uveitis in the developing world and the etiology varies from one geographical location to another

1. The infectious etiologies may be secondary to bacteria, viruses, fungus or parasites .
2. Parasites are organisms that acquire food from the hosts they live on. Uveitis secondary to parasites is more common in the tropical countries due to the geographical variation, environmental conditions and socio-economic status of the population.
3. Ocular parasitosis is an ocular complication, when humans are infected by parasites. The ocular inflammation may be primarily due to the direct damage by the parasite and its released toxic products, or secondary to the reaction hosted by the body's immune system.
4. Trematodes are a group of flatworms and they belong to the helminth class. These are multicellular organisms that are generally visible to naked eye in the adult stage. They can cause food and water borne infections.

One such water borne infection by trematode is River water granuloma. It infects children bathing in the river contaminated with trematode infected snails and poses threat to their vision. Trematode granuloma causes ocular inflammation after exposure to river water in these children, but the uveitis responds well if the etiology is identified earlier and the inflammation treated promptly.

Picture: 1



Legend 1: Digital photo showing river water granuloma at 7 O' clock position in the anterior chamber angle.

Epidemiology:

Parasitic infections are more common in developing countries. The contributing factors for infectious diseases in these countries being lack of hygiene, poor public health education, and restricted medical access. The Southeast Asian and Indian subcontinent in particular are at risk for exposure to several foodborne and waterborne infections. But there is no specific screening programs or public awareness. They also lack epidemiologic investigations on parasitic diseases.

The occurrence of any infectious disease depends on the habitat of the infective organisms, their hosts and also the cultural status and the habits of the population. Trematode granuloma is caused by a water borne trematode which infects the snails in the fresh water. Humans are accidental hosts in their life cycle. The children usually present with ocular inflammation which starts after exposed to the contaminated fresh water ponds in the river belts. It also worsens after taking bath in the pond every time. Sometimes these children present in groups with similar history.

Trematode granuloma cases were reported in several south Indian villages in Tamilnadu and Kerala as early as 2001 by Rathinam et al 5,6 and the etiological agent has been proven with molecular evidence in 2012 as *Procerovum varium* 7. Children with granulomatous anterior uveitis after swimming in the river was reported from different geographical locations along the basin of river Nile by R.M.Amin et al from Egypt 8,9. Here again the etiological agent was proven by molecular diagnostics to be trematode. A single case report from Thailand reported in 2006, a worm from conjunctiva removed and proved to be *Philophthalmus* sp., a trematode that parasitizes the eyes of birds 10. Similar case report of ocular infection by *Philophthalmus* was reported in Mexico also 11.

Life cycle:

Several reports have identified adult worms in the conjunctival sac of birds 12. The parasite was identified and reported as a trematode belonging to *Procerovum Varium*. When these birds come in contact with water, the adult worms release the eggs. The eggs hatch into miracidia, and infect the snails, which act as intermediate host. These miracidia mature to form cercariae in the intermediate host. Humans may become accidental host if infected with the cercariae. Several other species of trematode (*F. hepatica*, *Schistosoma* sp. etc) infection has also been reported in human eye 13. The cercariae of *Schistosoma* sp parasites are known to induce dermatitis in patients exposed to infested ponds or rivers. These cercariae die after they penetrate the skin; their death causes a localized inflammatory reaction.

Systemic manifestations:

After exposure to the snail infested water, patients may develop generalised reaction like itching all over the body with swelling of the buccal and genital mucosa. Usually, these

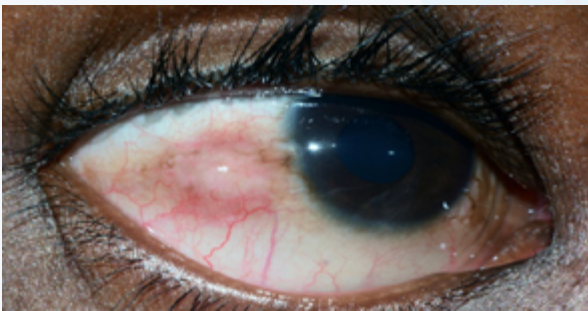
symptoms precede the ocular manifestations. The systemic manifestation is of shorter duration and self-limiting.

Ocular manifestations:

The cercariae of the trematode usually penetrates deep into the episclera and then develop subconjunctival granuloma along with granulomatous uveitis. In few cases these cercariae can enter the anterior chamber through the limbal structures and develop anterior chamber granuloma.

Mostly it is a unilateral presentation but, in few patients, it may be bilateral involvement also. They may present with one or more nodule and these are usually 2-5 mm in diameter. The common location is in the retro-corneal surface in the inferior quadrant. There may be associated granulomatous uveitis causing Keratic precipitates and anterior chamber reaction. The granuloma is well circumscribed and white in colour. In case of sub conjunctival granuloma usually the lesion is in the palpebral area. Few patients may even present with granuloma in their lid mimicking a chalazia. Usually there is no associated vision loss except in cases where the persistent inflammation can lead to complicated cataract or retro corneal membrane. The vision loss may also be secondary to the large AC granuloma which reaches the visual axis. Long term use of steroid use can cause cataract and glaucoma in few of these children.

Picture:2



Legend 2: Digital photo showing subconjunctival granuloma in the temporal region of the interpalpebral fissure.

The disease progression:

If identified earlier and treated with topical and oral steroids, usually smaller lesions resolve completely. But larger lesions even after complete surgical removal can leave behind retro-corneal scars. Patient with recurrent inflammation or in cases where treatment is not adequate, complicated cataract may develop leading to vision loss. The untreated large nodules in the long run can cause recurrent inflammation and may lead to phthisis of the eye.

Differential Diagnosis:

Even if there is a history suggesting occurrence of inflammation in association with taking bath in infected ponds, all the infectious and non-infectious causes of granulomatous uveitis have to be ruled out. These include ocular tuberculosis, sarcoidosis, foreign body granuloma, fungal granuloma and Xanthelasma¹⁴. In few cases tubercular granuloma may closely mimic trematode granuloma. When there is no temporal association with swimming or when there is strong history or systemic association with tuberculosis, molecular diagnostics and/ or histopathology maybe done to rule in or rule out tubercular etiology.

Evaluation:

Complete blood count, Differential count, Mantoux, Erythrocyte Sedimentation Rate, serum ACE are done as a routine based on the clinical picture. Systemic imaging of chest and abdomen is done in cases when needed.

In larger granuloma where lesion is aspirated or excised, the aspirate has to be sent for ruling out infectious causes. Hemotoxylin –Eosin staining, Periodic acid Schiff and Ziehl-Neelson staining are usually done along with culture of bacteria and Fungus. The conjunctival nodule on histopathological analysis may show a necrotizing granuloma containing a tegument of trematode. Splendore–Hoepli reaction can be seen in few cases where eosinophilic material can be seen surrounding or adjacent to the trematode¹⁵. It is not possible to visualize the teguments in all cases may be because the parasite structures disintegrate rapidly secondary to localised inflammatory reaction. Histopathology of the trematode granuloma usually reveals an inflammatory process made up of lymphocytes, neutrophils, and eosinophils mixed with histiocytes.

Molecular diagnostics can be done in suspected cases. The recent advancement can identify the trematode up to the species level. The aspirate has to be sent also for ruling out tuberculosis and fungal infections by performing nested PCR that targets the MPB64 and 28S rDNA genes.

Treatment:

The smaller nodules (< 3 mm) usually resolve with topical treatment using steroids in tapering doses. When the lesion is not resolving and if other differentials are ruled out, these children can be started on oral steroids at the dose of 1mg /kg body weight. The oral steroids are tapered weekly.

Surgical excision is the treatment of choice in larger granulomas (> 3mm). In cases with such larger granuloma, we can either excise or aspirate the lesion depending on the location. Subconjunctival granuloma has to be excised in toto. In case of anterior chamber granuloma, it can be aspirated through a paracentesis wound.

It is a simple procedure with no complications, except that the disease being most common among children, it has to be done under general anaesthesia. These patients are then continued on oral and topical steroids till the inflammation settles.

Role of Geographic Information System (GIS) in presumed trematode induced uveitis.

Geographic Information System is a computer software system for analysis of geographic data on wide varied health care systems. It was widely employed for varied parasitic diseases

The classical epidemiological triad Agent [=Soil map], Host [=Population density map], environment [=Drainage map] were analysed. It revealed clustering of this specific pediatric uveitis around transitional zones of soil where one soil type merges with the other soil type. It could point the pH of the water, soil type, osmotic effect, presence of an inhibitional factor or an organism in the sea habitat that needs further detailed study. Interestingly the child with maximal worm load with maximum duration of water related activities had the maximal granuloma load resulting in lowest visual acuity and highest intraocular pressure. The hypothesis that pond carrying specific snails¹⁶ were contributory for the pathogenesis of this trematode induced uveitis. A positive correlation between patient's residencies and the location of the ponds. A satellite based remote sensing was applied to attempt finding a parameter characteristics of the pond with snail habitat. It was proven that ponds carrying the risk factors can be delineated from others by analysis of spectral surface properties. This novel pond classification system propelled by field visits helped the researchers in collecting the snails from the potential snail habitats. Further molecular analysis using polymerase chain reactions (PCR) helped in identifying the specific organism as *Procerovum varium*¹⁷

Prevention:

To educate the patients and their attenders regarding the chance of recurrence or worsening of clinical picture on indulging in swimming in same infected pond. This usually provides good response in reduction of the occurrence of cohort of new cases in the same belt.

References:

1. Rathinam SR, Cunningham ET Jr. Infectious causes of uveitis in the developing world. *Int Ophthalmol Clin* 2000;40:137-52.
2. Sabrosa NA, Cunningham ET Jr, Arevalo JF. Ocular nematode and trematode infections in the developing world. *Int Ophthalmol Clin* 2010;50:71-85
3. Hoti SL, Tandon V. Ocular parasitoses and their immunology. *Ocul Immunol Inflamm* 2011 Dec;19(6):385-96.
4. Nimir AR, Saliem A, Ibrahim IA. Ophthalmic parasitosis: a review article. *Interdiscip Perspect Infect Dis* 2012;2012:587402.
5. Rathinam S, Fritsche TR, Srinivasan M, Vijayalakshmi P, Read RW, Gautom R, et al. An outbreak of trematode-induced granulomas of the conjunctiva. *Ophthalmology* 2001;108:1223-9.
6. *Rathinam SR, Usha KR, Rao NA. Presumed trematode-induced granulomatous anterior uveitis: a newly recognized cause of intraocular inflammation in children from south India. *Am J Ophthalmol* 2002;133:773-9.
7. Arya LK, Rathinam SR, Lalitha P, Kim UR, Ghatani S, Tandon V. Trematode Fluke *Procerovum varium* as Cause of Ocular Inflammation in Children, South India. *Emerg Infect Dis* 2016 ;22:192-200.
8. *Amin RM, Goweida M, Bedda A, Kamel A, Radwan A. Clinical Patterns and Causes of Intraocular Inflammation in a Uveitis Patient Cohort from Egypt. *Ocul Immunol Inflamm* 2019;27:859-867.
9. El Nokrashy A, Abou Samra W, Sobeih D, Lamin A, Hashish A, Tarshouby S, et al. Treatment of presumed trematode-induced granulomatous anterior uveitis among children in rural areas of Egypt. *Eye (Lond)*. 2019;33:1525-1533.
10. Waikagul J, Dekumyoy P, Yoonuan T, Praevanit R. Conjunctiva philophthalmosis: a case report in Thailand. *Am J Trop Med Hyg* 2006;74:848-9.
11. Sapp SGH, Alhabshan RN, Bishop HS, Fox M, Ndubuisi M, Snider CE, et al. Ocular Trematodiasis Caused by the Avian Eye Fluke *Philophthalmus* in Southern Texas. *Open Forum Infect Dis* 2019;6:ofz265.
12. Umadevi K, Madhavi R. Observations on the morphology and life-cycle of *Procerovum varium* (Onji & Nishio, 1916) (Trematoda: Heterophyidae). *Syst Parasitol* 2000;46:215-25.

13. Dalimi A, Jabarvand M. Fasciola hepatica in the human eye. *Trans R Soc Trop Med Hyg* 2005 ;99:798-800.
14. Rathinam SR, Babu M. Algorithmic approach in the diagnosis of uveitis. *Indian J Ophthalmol*. 2013;61:255-62.
15. Read RW, Zhang J, Albini T, Evans M, Rao NA. Splendore-Hoeppli phenomenon in the conjunctiva: immunohistochemical analysis. *Am J Ophthalmol* 2005;140:262-6.
16. Jayakumar Duraisamy, Sivakumar Kavitha, Sivakumar Rathinam, Gnanam Vasanthi. Geomapping of trematode-induced granulomatous anterior uveitis- a newly identified cause of blindness among children in the Pudukkottai district of the Tamil Nadu State, India. *Geospatial Health* 4(1), 2009;55-63
17. Novel Etiological Agent: Molecular Evidence for Trematode- Induced Anterior Uveitis in Children Sivakumar Rathinam, Lalan Kumar Arya, Kim R Usha .*Arch Ophthalmol*. 2012;130(11):1481-1484.



Parasite puzzle



Co Author

Dr. Kalpana Babu

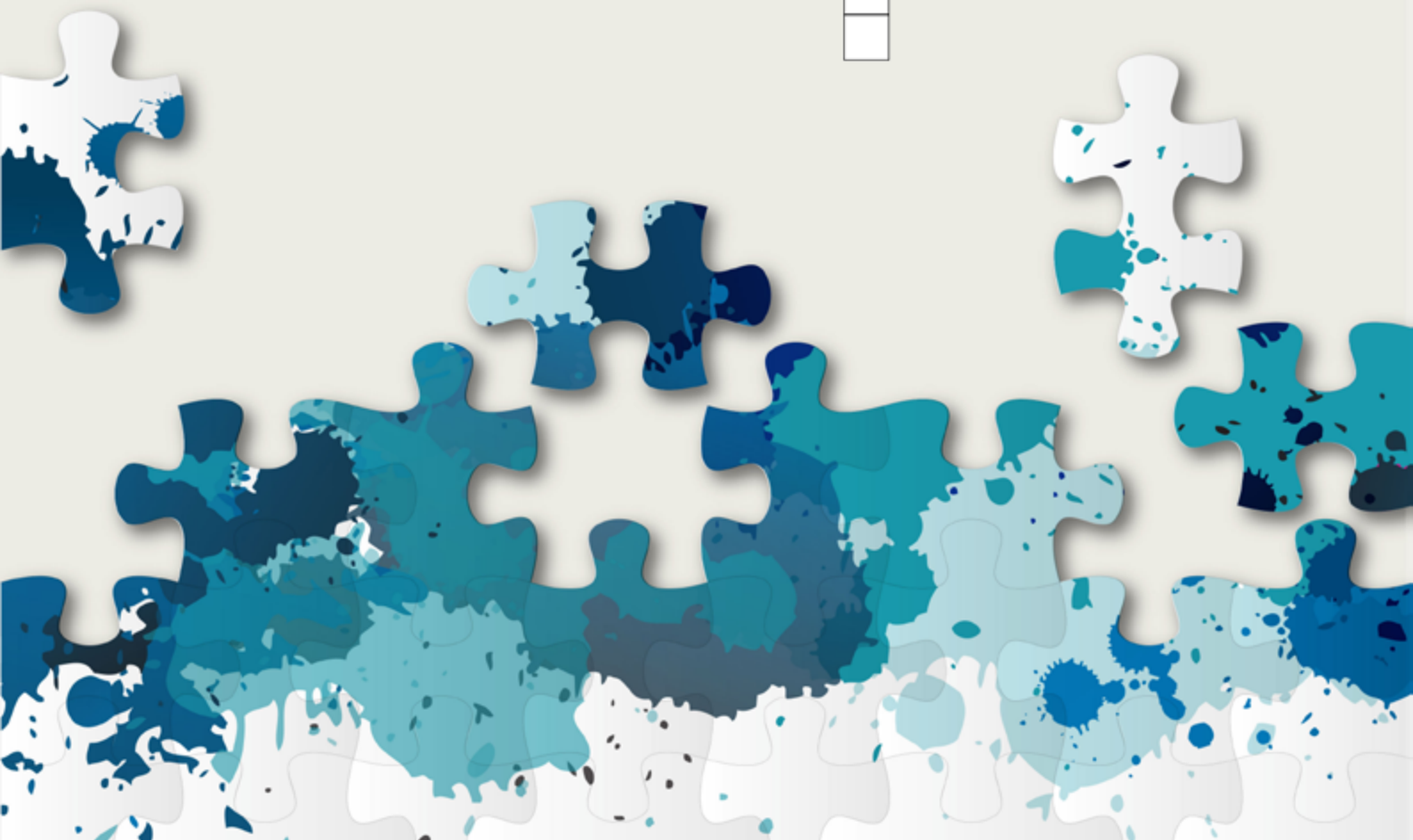
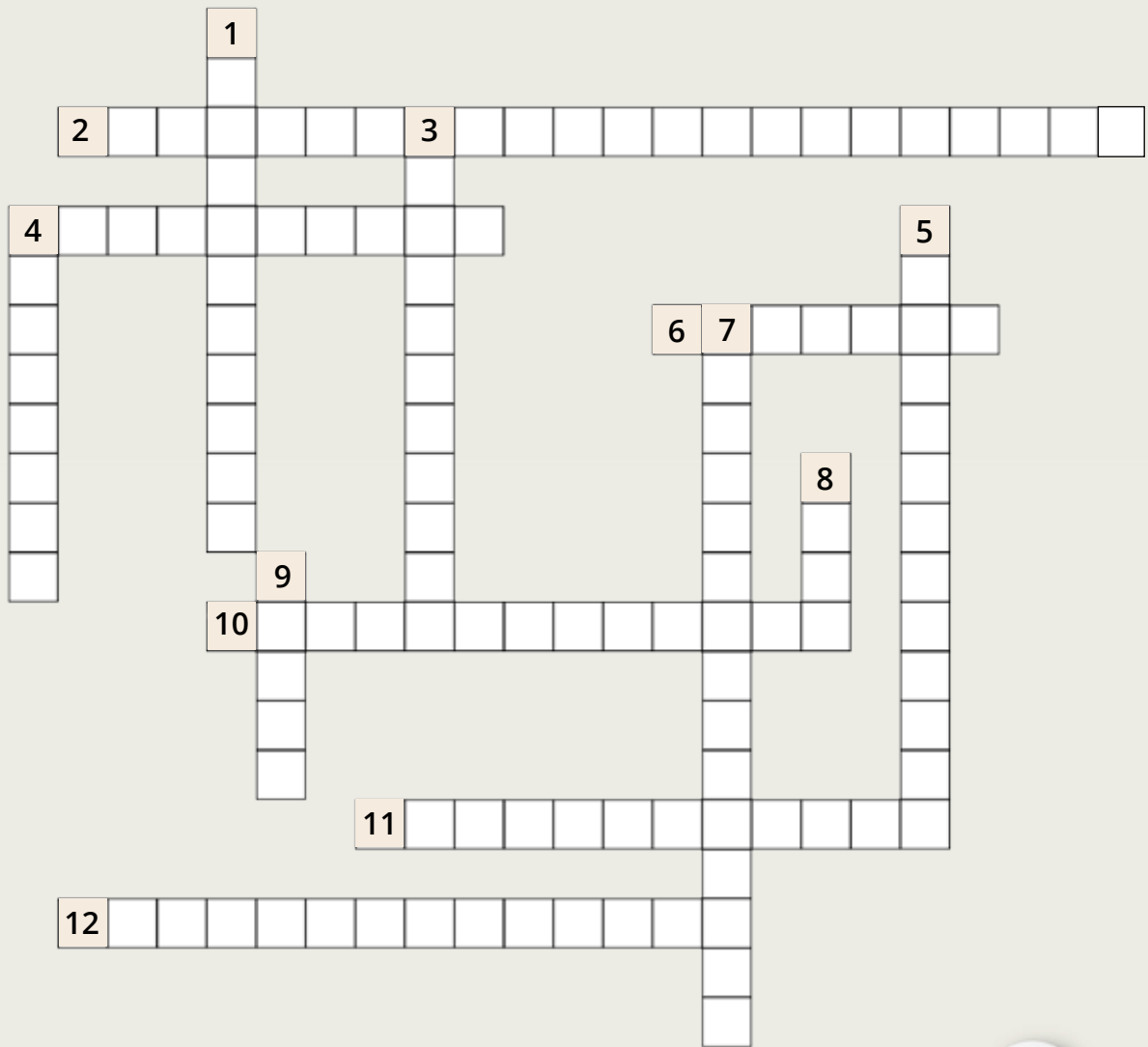
Prabha Eye Clinic and Research Centre, Bengaluru



Dr. Vinaya Kumar Konana

Vittala International Institute of Ophthalmology
Vitreoretina services

Parasite Puzzle



Across	Down
<p>2. First to recognize T gondii histopathologically in necrotic retinochoroidal lesions of adults(21)</p>	<p>1. Disease caused by oriental eyeworm(11)</p>
<p>4. Safest anti-toxo drug during pregnancy(10)</p>	<p>3. Intravitreal drug used in treatment of ocular toxoplasmosis(11)</p>
<p>6. Chronic disease caused by filarial parasite, Loa loa which is endemic to rain forests of Africa(7)</p>	<p>4. Onchocerciasis is caused by the parasite Onchocerca volvulus and transmitted by fly _____(8)</p>
<p>10. Gold standard dye test for toxoplasmosis(13)</p>	<p>5. Disease with anterior uveitis as most common manifestation of systemic infection. Humans get infection by the bite of sand flies(13)</p>
<p>11. _____ can be confused with retinoblastoma(12)</p>	<p>7. It is an insect-mediated ocular disorder caused by botfly larvae(15)</p>
<p>12. Pediatric presumed trematode infection has been reported in patients from South India who were exposed to pond water. Causative parasite is(14)</p>	<p>8. Unilateral wipe out syndrome is the other name of(4)</p>
	<p>9. Person who first described congenital toxoplasmosis with ocular involvement(5)</p>



Ocular Toxocariasis

An update



Dr. Nivedita Nair

Junior Consultant, Department of Uvea and Ocular Immunology,
Chaithanya Eye Hospital and Research Institute, Trivandrum, Kerala



Dr. Jyotirmay Biswas

Director, Department of Uvea and Ocular Pathology
Medical Research Foundation, Sankara Nethralaya, Chennai, Tamilnadu

Dr. Shilpa I N

Fellow, Department of Vitreoretina, Sankara Nethralaya, Chennai, Tamilnadu

Ocular Toxocariasis – An update

Department of Uvea, Sankara Nethralya, Chennai

Ocular toxocariasis results from hematogenous invasion of the eye by the second or third stage larva of the dog roundworm, *Toxocara canis* or *Toxocara cati*. Humans may be infected through accidental ingestion of eggs in soil contaminated by dog or cat feces or by consumption of undercooked meat from an animal infected with *Toxocara* larvae. The eye involvement occurs as a part of phenomenon of systemic migration of parasite known as visceral larvae migrans (VLM).^[1]

Ocular toxocariasis (OT) or ocular larvae migrans (OLM) syndrome manifests as intermediate, posterior, or panuveitis. Chorioretinitis, retinal granuloma, and vitritis are the common morphological forms of presentation. Characteristic fundus findings consist of posterior or peripheral chorioretinal granuloma, focal chorioretinal lesions, or endophthalmitis.^[1-3]

Epidemiology

Toxocariasis is one of most common zoonotic infections worldwide. The seroprevalence of toxocara antibody varies from 2.4% to 76.6%.^[2] In a study from North India, antibodies to *Toxocara* were detected in 11/68 (17%) subjects less than 15 years old and three (4%) subjects more than 15 years of age using enzyme-linked immunosorbent assay (ELISA).^[4] Ocular toxocariasis has been seen mainly in children, but recent reports have documented a relatively high prevalence in adults, especially in Asians.^[3,5]

In a recently published metaanalysis, the distribution pattern of OT demonstrated the highest rates in African and the lowest rates in Mediterranean territories. The authors showed that exposure to soil, contact with dogs or ownership of dogs or cats as well as consuming raw/undercooked meat are among the risk factor of OT. They recommended regular administration of anti-helminthics to the pets and cleaning and removal of pet feces from public areas especially children's play area to prevent toxocara infection.^[6]

Transmission to human beings occurs primarily from ingestion of embryonated eggs which are present in the contaminated soil. Human infestation with the second or third stage larvae of *Toxocara canis* is known as VLM. However in vast majority of patients with the systemic disease, ocular involvement is not seen. After ingestion, the eggs hatch in the human small intestine and the larvae then migrate into the mucosa to reach the portal circulation. They migrate to the liver, follow vascular channels to the lungs and then enter the systemic circulation to reach numerous organs, notably the liver, lung, brain, and the eye. The larva which may remain in circulation for weeks to months induces eosinophilic granulomatous reaction.^[1-3]

The ocular involvement is not universal in patients with VLM. The exact association between VLM and ocular involvement is not well elucidated. The possibility of ocular involvement by the parasite was first considered by Wilder,^[7] who noted that certain eyes enucleated for suspected retinoblastoma and other conditions contained a granulomatous inflammatory process with eosinophilic abscesses. The organism was later identified by Nichols as *Toxocara canis*.^[8]

Clinical Manifestation

Ocular toxocariasis is typically unilateral affecting young children. Although rare, bilateral and adult subjects with infection have been reported. The child may present with ocular inflammation or may be asymptomatic and diagnosis may be made on evaluating for strabismus due to poor vision. A white pupillary reflex (leucocoria) can also be one of the presentations, often leading to a suspicion of retinoblastoma.

- **Posterior retinochoroiditis[1]**

Ocular toxocariasis can manifest as posterior pole retinochoroiditis often involving the macula. The retinochoroiditis lesion appears clinically as a hazy, ill-defined white lesion, with overlying inflammatory cells in the vitreous (Figure-1). Once the acute inflammatory reaction subsides, the lesion appears as a well-defined, elevated white mass ranging from one-half disc diameter to four disc diameters in size.

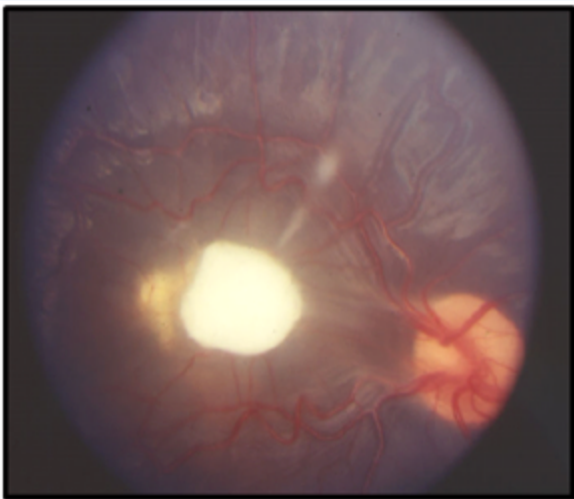


Figure-1

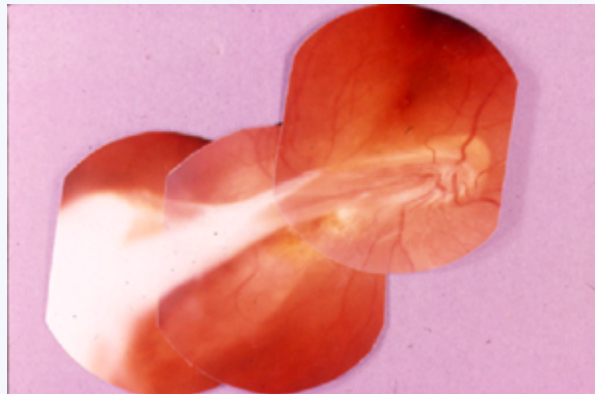
Color fundus photograph of a case of ocular toxocarasis showing the posterior pole granuloma

- **Peripheral retinochoroiditis[1]**

Ocular toxocarasis can occur as an acute inflammatory process in the peripheral retina and ciliary body region. It appears as a hazy white reaction in the peripheral fundus. In the late stages, dragging of the retina toward the inflammatory mass produces a falciform fold (Figure-2). The traction may lead to heterotopia of the macula resulting in severe loss of vision in some cases.

Figure-2

Montage Color fundus photograph showing peripheral granuloma with traction (falciform fold) in a case of ocular toxocariasis



- **Papillitis[1]**

An isolated optic disc inflammation may be seen in few cases which is characterized by an elevation of the optic disc with telangiectasia of the blood vessels with or without subretinal exudation. A secondary retinal artery occlusion can occur in certain cases when there is severe disc edema. The papillitis resolves leaving behind a peripapillary fibrous tissue.

- **Endophthalmitis[1]**

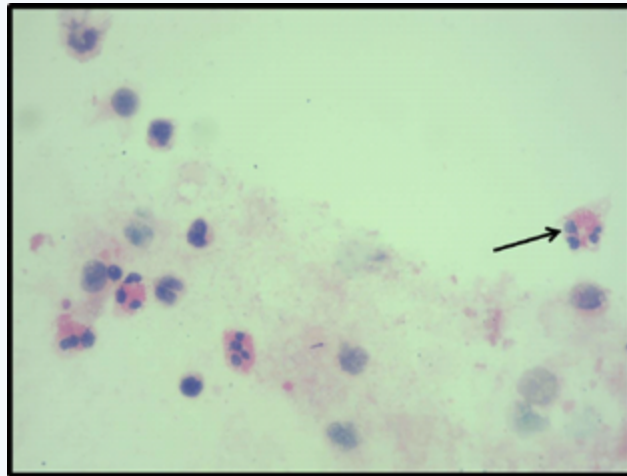
The most common and the well described manifestation of OT is endophthalmitis. It is characterized by a yellow-white mass, retinal detachment and vitreous cells, similar to case of retinoblastoma. The child usually is asymptomatic, on examination granulomatous anterior uveitis may be seen. The anterior vitreous usually shows dense inflammation with white inflammatory cells which obscures the fundus view.

Through the vitreous haze a yellow white mass in the peripheral retina may be seen in majority of cases, which closely resemble an endophytic retinoblastoma. Severe forms of the disease can cause exudative retinal detachment. In advanced cases a retrolental membrane may develop due to cyclitic membrane formation. Secondary glaucoma or phthisis bulbi can occur as complication of chronic inflammation.

Gross examination of an enucleated eye with nematode endophthalmitis characteristically shows intraocular disorganization, often with a total retinal detachment. The detached retina may show a focal thickening in the periphery, due to an inflammatory mass. The subretinal fluid is usually very gelatinous due to the presence of lipoprotein similar to the reaction seen with Coats' disease. A dense cyclitic membrane may be seen retrolentally incorporating anterior detached retina. A focus of granulomatous inflammation composed of numerous eosinophils, forming an eosinophilic abscess is usually seen on pathology (Figure-3). Surrounding the eosinophilic abscess, epithelioid cells, and inflammatory granulation tissue infiltrated by eosinophils, lymphocytes, and plasma cells are present. In the center of the abscess, a well-developed larva or the remnants of a degenerated larva or their residual hyaline capsule may be picked up infrequently.^[1]

Figure-3

Hematoxylin and eosin staining from vitrectomy specimen showing many eosinophils (arrow) in a case of toxocara uveitis. (x 200)



In a study from India, OT was identified in 27% cases of paediatric endogenous endophthalmitis. [9] Diagnosis of toxocara endophthalmitis was confirmed in this series by ELISA for anti-toxocara IgM antibodies in serum of 20% and vitreous sample of 6.7% subjects.

Other rare forms of presentations are nematode tracks seen in the fundus due to wandering larvae, diffuse unilateral subacute neuroretinitis (DUSN) like presentation, keratitis, conjunctivitis and lens involvement.

The most common and most important differential diagnosis to consider and duly to be ruled out is that of retinoblastoma. Other common differentials include Coats' disease, persistent hyperplastic primary vitreous, retinopathy of prematurity, toxoplasmosis and familial exudative vitreoretinopathies, among others. [1,2,10]

It is often very difficult to differentiate retinoblastoma and OT. Few clinically relevant differentiating features include later age of onset in OT and absence of calcification within the lesion in OT. Other findings suggestive of retinoblastoma are a non-inflamed eye without cataract (which may form as a result of inflammation in OLM) and vitreous cells with soft and round morphology, as opposed to smaller inflammatory cells in OLM. [10] Steward et al noted that the primary causes of vision loss in OT were vitritis (52.6%), cystoid macular edema (47.4%), and traction retinal detachment (36.8%). [11]

Diagnosis

Ocular toxocariasis is a clinical diagnosis and is almost always a presumptive diagnosis. Definite confirmation of diagnosis is by demonstrating the larvae in the eye. Slit lamp examination, indirect ophthalmoscopy and ultrasound B scan aid in arriving at a clinical diagnosis. [1,2]

Enzyme linked immunosorbent assay (ELISA)

An ELISA to detect IgG antibodies against *Toxocara* excretory–secretory antigen has become the reference immunological diagnostic test for toxocariasis, with sensitivity of 91% and specificity of 86%.^[3] However the absence of serum antibodies does not rule out the diagnosis of OT. In such cases the diagnosis of OT can be confirmed based on ancillary tests like elevated serum Ig E levels and systemic eosinophilia.

In a study of adult patients with OT, evaluation of total IgE level or eosinophil cationic protein (ECP) level in serum was more helpful than the blood eosinophilia test for diagnosis of OT when combined with anti-*Toxocara* antibody test.

ELISA from Intraocular fluid (aqueous/vitreous)

ELISA analysis of intraocular fluids may be more sensitive in diagnosis of OT. However, using the same cutoff value as with serum antibody, the positive rates of ELISA on vitreous fluid was as low as 33%.^[2] Further investigation on determining a suitable cutoff value for the detection of OT has to be done before the test can be used clinically to establish diagnosis. A very recent study proposes novel diagnostic cutoff values of serum and intraocular fluid anti-toxocara IgG for OT, which is 8.2 U and 1.8 U, respectively. Sensitivity increased to 80.2%, and specificity was 94.0% with the recommended serum cutoff. The sensitivity was as high as 88.4%, and specificity was 96.4% with the newly recommended intraocular fluid *Toxocara* Ig G cutoff.^[12]

Ocular imaging

Fundus photograph

Li et al described the role of ultra wide field (UWF) imaging in diagnosis of OT. They noted that UWF fundus imaging had far superior sensitivities in diagnosis of posterior pole and peripheral granuloma of OT. Other features like tractional retinal detachment, retinal folds and vitreous strands were all better identified on UWF imaging.^[13,14]

Optical coherence tomography (OCT) and fundus fluorescein angiogram

There are only few case reports describing the role of OCT or angiogram in diagnosis of OT. In OCT images, the macular granuloma appears as a highly reflective mass protruding above the retinal pigment epithelium showing dye leakage by angiography. Post treatment, the lesion is longer exudative, is less elevated, and is covered by the retinal pigment epithelium. There was reticular hyperfluorescence surrounded by a hypofluorescent rim in the angiograms. This appearance closely resembles idiopathic choroidal neovascularization (CNV) and should be included in the differential diagnosis of the idiopathic CNV.^[15]

OCT can be quite valuable for diagnosing epiretinal membrane formation, macular thickening, focal macular retinal detachments. The traction upon the retina can be documented clearly and the response to treatment can be monitored by serial OCTs.^[14]

Ultrasound B-scan

Ultrasound imaging is a valuable tool in eyes with opaque media in which the cause of vitreous inflammation and opacification is unclear. B scan can detect granuloma formation, the location and extent of vitreous membranes, and the presence of retinal detachments.^[1,14]

Ultrasound Biomicroscopy (UBM)

Few authors have assessed the role of UBM in identification of OT. Chen et al noted peripheral granulomas (84.6%), as the primary imaging feature on UBM. These peripheral granulomas were adjacent to the nasal or temporal side of horizontal meridian in 78.5% eyes and in 79.7% they were located on the surface of the ciliary body, presenting as olivary foci on radial section and botuliform foci on coronal section. Posterior pole granulomas in addition to peripheral granuloma (combined type) were noted in few cases. Other UBM findings included vitreous strands (67.9%), peripheral tractional retinal detachment (52.6%) and tractional cyclodialysis(41.0%). They emphasised the role of UBM in detection of peripheral granulomas for diagnosis of OT.^[16]

Treatment

Corticosteroids, topical and/or systemic are the main stay of treatment of OT. Corticosteroids reduce the intraocular inflammation and membrane formation. The role of anti-helminthics in treatment of OT is controversial as there have been no randomized controlled trials on the use of anti-helminthic agents for OT.

In a recent study the authors suggests steroids and albendazole^[17] an effective treatment as they noted favourable responses in more cases (64.3%) than the steroid alone group (20.0%).^[3] The recurrence rates were also less in the combination therapy group. Albendazole(400 mg given twice a day for 7–14 days) is the recommended standard drug for systemic toxocariasis. Other agents like thiabendazole (given at 50 mg/kg/day for 3–7 days) and diethylcarbamazine (given at 3–4 mg/kg/day for 21 days, starting at 25 mg/day for each adult patient and increasing the dose progressively) were also found to be effective for the treatment of systemic toxocariasis.^[2]

Retinal detachment, epiretinal membrane, and persistent vitreous opacity are common indications for vitreoretinal surgery performed in eyes with OT. Giuliari et al reported good anatomic and functional outcome of surgical treatment in 45 patients with OT.^[18] In another series, 32 out of 101 patients (31.7%) required surgical treatment. Successful surgical outcome was achieved in 68.4%, 88.9%, and 50% of patients with epiretinal membrane, vitreous opacity, and retinal detachment, respectively.^[19] Zhang et al reviewed 56 consecutive patients who underwent vitrectomy for OT, they noted an unfavourable outcome especially in patients with macular involvement.^[20]

Nd:YAG laser was used to photofracture the live motile larva in rare circumstances. The laser burn must be sufficiently strong to destroy the nematode completely as partially killed larva can induce intense inflammation.^[10]

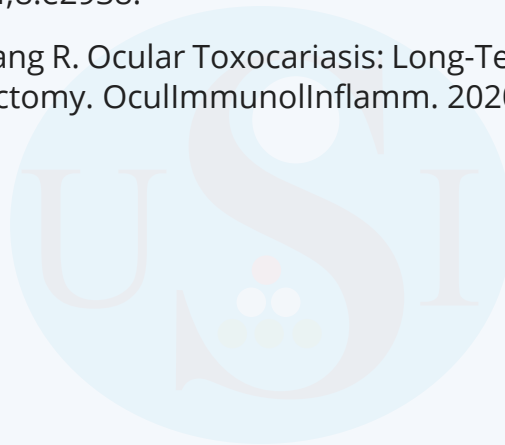
Conclusion

The current understandings of the diagnosis, treatment, and prevention of human toxocariasis and ocular involvement are limited, although it is one of the most common zoonotic infections worldwide. OT can be clinically diagnosed with specific signs. Diagnostic tests need improvement for better detection and prompt diagnosis of OT. The control of inflammation is the primary goal of treatment to reduce the sequela and preserve vision in affected children.

References

1. Shields JA. Ocular toxocariasis. A review. *SurvOphthalmol*. 1984;28(5):361-381.
2. Ahn SJ, Ryoo NK, Woo SJ. Ocular toxocariasis: clinical features, diagnosis, treatment, and prevention. *Asia Pac Allergy*. 2014;4(3):134-141.
3. Jee D, Kim KS, Lee WK, Kim W, Jeon S. Clinical Features of Ocular Toxocariasis in Adult Korean Patients. *OculImmunolInflamm*. 2016;24(2):207-216.
4. Mirdha BR, Khokar SK. Ocular toxocariasis in a North Indian population. *J Trop Pediatr*. 2002 Dec;48(6):328-30.
5. Fan CK, Hung CC, Du WY, Liao CW, Su KE. Seroepidemiology of Toxocaracanis infection among mountain aboriginal school children living in contaminated districts in eastern Taiwan. *Trop Med Int Health* 2004;9:1312-8.
6. Badri M, Eslahi AV, Olfatifar M, et al. Keys to Unlock the Enigma of Ocular Toxocariasis: A Systematic Review and Meta-analysis [published online ahead of print, 2021 Apr 28]. *OculImmunolInflamm*. 2021;1-12.
7. WILDER HC. Nematode endophthalmitis. *Trans Am AcadOphthalmolOtolaryngol*. 1950;55:99-109.
8. NICHOLS RL. The etiology of visceral larva migrans. I. Diagnostic morphology of infective second-stage Toxocara larvae. *J Parasitol*. 1956;42(4 Section 1):349-362.
9. Maitray A, Rishi E, Rishi P, et al. Endogenous endophthalmitis in children and adolescents: Case series and literature review. *Indian J Ophthalmol*. 2019;67(6):795-800. doi:10.4103/ijo.IJO_710_18
10. Schneier AJ, Durand ML. Ocular toxocariasis: advances in diagnosis and treatment. *IntOphthalmolClin*. 2011;51(4):135-144.
11. Stewart JM, Cubillan LD, Cunningham Jr ET. Prevalence, clinical features, and causes of vision loss among patients with ocular toxocariasis. *Retina*. 2005;25:1005-1013.
12. Huang L, Sun L, Liu C, et al. Diagnosis of Ocular Toxocariasis by Serum and Aqueous Humor IgG ELISA. *Transl Vis Sci Technol*. 2021;10(8):33.

13. Li S, Sun L, Liu C, et al. Clinical features of ocular toxocariasis: a comparison between ultra-wide-field and conventional camera imaging. *Eye (Lond)*. 2021;35(10):2855-2863.
14. Campbell JP, Wilkinson CP. Imaging in the diagnosis and management of ocular toxocariasis. *IntOphthalmolClin*. 2012;52:145-153.
15. Higashide T, Akao N, Shirao E, Shirao Y. Optical coherence tomographic and angiographic findings of a case with subretinal toxocara granuloma. *Am J Ophthalmol*. 2003;136(1):188-190.
16. Chen Q, Gu J, Jiang R, Zhou M, Chang Q. Role of ultrasound biomicroscopy in diagnosis of ocular toxocariasis. *Br J Ophthalmol*. 2018;102(5):642-646.
17. Barisani-Asenbauer T, Maca SM, Hauff W, et al. Treatment of ocular toxocariasis with albendazole. *J OculPharmacolTher*. 2001;17:287-294.
18. Giuliari GP, Ramirez G, Cortez RT. Surgical treatment of ocular toxocariasis: anatomic and functional results in 45 patients. *Eur J Ophthalmol* 2011;21:490-4.
19. Ahn SJ, Woo SJ, Jin Y, et al. Clinical features and course of ocular toxocariasis in adults. *PLoS Negl Trop Dis* 2014;8:e2938.
20. Zhang T, Guo D, Xu G, Jiang R. Ocular Toxocariasis: Long-Term Follow-Up and Prognosis of Patients following Vitrectomy. *OculImmunolInflamm*. 2020;28(3):517-523.



Parasitic Uveitis: Review of literature



Corresponding Author

Dr. Manisha Agarwal

Head of Vitreoretina Services, Dr. Shroff's Charity Eye Hospital
Daryaganj, New Delhi

Dr. Yogesh Yadav

Vitreoretina Fellow, Dr. Shroff's Charity Eye Hospital
Daryaganj New Delhi

Dr. Rakesh Verma


Vitreoretina Fellow, Dr. Shroff's Charity Eye Hospital
Daryaganj, New Delhi

Parasitic Uveitis: Review of literature

Introduction: The aim of this tabulated compilation on Parasitic Uveitis is to highlight the recent literature on Parasitic Uveitis. A Pubmed search of recent literature using terms Parasitic, uveitis, ocular Toxocariasis, ocular cystisercosis, Leishmania, DUSN was done. Review articles, original research and few interesting case reports/case series with novel descriptions are included. The articles are tabulated under the following headings: Authors, Journal and year of publication with relevant summary of the article. We are excluding the literature on Toxoplasmosis as the previous newsletter was focussed on it.

Authors	Journal and Year	Title	Summary
TOXOCARIASIS			
Li Huang, Limei Sun, Chengxi Liu ¹ , Songshan Li, Ting Zhang, Xiaoling Luo, and Xiayan Ding	Transl Vis Sci Technol. 2021	Diagnosis of Ocular Toxocariasis by Serum and Aqueous Humor IgG ELISA	<ol style="list-style-type: none"> 1. Clinical characteristics of Ocular Toxocariasis cannot serve as a standard diagnostic tool 2. This study confirmed the diagnostic value of serum anti-toxocara IgG using the units, which was (sample absorbance Å~ 10)/cut off value. A value of 8.2U yielded moderate sensitivity (80.2%) and high specificity (94.0%). 3. This Study was the first to focus on the cut off value of intraocular fluid anti-toxocara IgG. 4. Intraocular production of toxocara antibodies can be assessed by comparing serum and aqueous humor samples obtained from the same patient. 5. Limitations: this was a retrospective study with a limited sample size due to the rarity of OT

<p>I Hernanz , A Moll-Udina , Belles V. Llorenç & Civera A. Adan</p>	<p>Ocular Immunology and Inflammation 2020</p>	<p>Ocular Toxocariasis: Beyond Typical Patterns through the New Imaging Technologies</p>	<ol style="list-style-type: none"> 1. ELISA is the gold standard for diagnosis of human toxocariasis by detecting anti-toxocara antibodies But negative serology would not exclude ocular toxocariasis 2. Another common finding supporting systemic toxocariasis is serum hyper-eosinophilia and hyper IgE, but it used to be within normal limits in most OT patients. Microscopy lacks sensitivity to find the scarce or single parasite in the eye 3. Typical fundus images suggesting OT such as posterior pole or peripheral retinal granuloma are key findings for the diagnosis of this entity 4. Motile subretinal larva and migrating granuloma have been reported in the literature as pathognomonic findings of OT
--	--	--	--

<p>Ting Zhang, Diwen Guo, Gezhi Xu & Rui Jiang</p>	<p>Ocular Immunology and Inflammation 2020</p>		<p>This was a retrospective study which reviewed the outcomes of surgical treatment in patients with OT and identify prognostic factor.</p> <p>OT typically presents as chronic endophthalmitis with peripheral or posterior retinal granuloma. Corticosteroids are the mainstay of treatment for active endophthalmitis due to OT, whereas pars plana vitrectomy is generally reserved for patients with complications such as persistent vitreous opacification or vitreoretinal traction.</p> <p>The clearance of vitreous opacities might help reduce the incidence of recurrent inflammation, supporting the role of early vitrectomy for the treatment of toxocariasis associated chronic endophthalmitis.</p> <p>Vitrectomy without granuloma resection might be a less-damaging strategy to preserve residual vision in patients with OT and recurrent inflammation could be managed with topical or systemic corticosteroids.</p> <p>Surgical treatment improved the visual outcomes of patients with OT, but their prognosis was still relatively poor. The decision to perform surgery should be made with care, based on preoperative findings.</p>
--	--	--	---

<p>N.F. Abd El-Aal, M.A.A. Basha and A.M. Eid</p>	<p>Journal of Helminthology 2018</p>	<p>New insight into the diagnostic cut-off value of serum anti- Toxocara IgG for ocular toxocariasis in uveitis patients</p>	<p>OT diagnosis is mainly dependent on clinical confirmation and serology, as biopsy is risky.</p> <p>OT patients with positive Toxocara ELISA IgG were commonly infected as a result of consumption of raw beef, mainly raw liver, and contact with pets.</p> <p>This study detected a positive correlation between serum anti-Toxocara IgG ELISA and OT, in which patients with OT had higher serum anti-Toxocara IgG results than non-OT uveitis patients.</p> <p>Sensitivity, specificity and positive predictive value of ELISA test in OT cases were 93.9%, 90.2% and 77.8%, respectively in this study.</p> <p>Application of a cut-off value of 0.258 yielded 100% specificity. Thus, IgG ELISA is a promising diagnostic tool.</p>
<p>Abdolmajid Fata, Seyedeh Maryam Hosseini , Se Joon Woo , Mohammad Zibaei , Fariba Berenji , Bibi Razieh Hosseini Farash , Elham Moghaddas</p>	<p>Iran J Parasitol 2021</p>	<p>Frequency of Toxocara Antibodies in Patients Clinically Suspected to Ocular Toxocariasis, Northeast of Iran</p>	<p>OT diagnosis is based on typical clinical findings and laboratory tests. Intraocular parasites or its tissue is not observed by ophthalmoscopy.</p> <p>Because of behavioural food habits and tendency to eat perfectly cooked meat in Iran, the prevalence of OT might be low</p> <p>Patients who had long time history of direct contact with a dog, they had prolonged and severe uveitis, unilateral old posterior pole and peripheral granuloma (which was detected during vitrectomy) with positive anti-Toxocara IgG and negative anti-Toxoplasma IgG.</p> <p>IgG inside intraocular fluids (aqueous or vitreous humour) can be detected at higher rate than with blood serum screening.</p>

<p>Kang Yeun Pak , Sung Who Park, Ik Soo Byon and Ji Eun Lee</p>	<p>BMC Infectious Diseases (2016)</p>	<p>Ocular toxocariasis presenting as bilateral scleritis with suspect retinal granuloma in the nerve fiber layer: a case report</p>	<p>A patient with history of eating unheated meal presented with bilateral scleritis and retinal granuloma. Specific past history, positive serologic tests for toxocara, and treatment responses suggested that scleritis were manifestations of OT. Scleritis should be considered as one of manifestation of OT, and was managed with the standard anti-toxocariasis medication. Serologic exams for toxocariasis would be considered not only in typical features but also in other uveitis or scleritis, particularly when the patient has a related history.</p>
<p>Izabella Karska-Bastaa, Agnieszka Kubicka- Trząskaa, Michał Chrzęszczb Weronika Pociej-Marciaka, Bożena Romanowska- Dixon</p>	<p>Case Rep Ophthalmol 2019</p>	<p>Toxocara Optic Disc Granuloma: Deep Range Imaging Optical Coherence Tomography Findings</p>	<p>In most cases, it is difficult to establish the diagnosis of OT based on clinical manifestations only, and it may be confused with retinoblastoma in children. Due to the fact that the clinical signs of OT are not specific, a differential diagnosis should consider other parasitic diseases, other causes of posterior uveitis, and the possible occurrence of retinoblastoma in childhood. Laboratory tests include the detection of specific IgG in the serum (ELISA, 90% specificity, 91% sensitivity) or intraocular fluids. However, approximately 10% of patients with clinical signs of OT present negative results with the ELISA method from ocular fluid samples.</p> <p>Use of DRI-OCT was the most adequate non-invasive imaging method for assessment of the evolution of retinal changes, vitreous traction, and signs of vitritis during the follow-up period and for analysing the response of the optic disc granuloma to therapy. DRI-OCT imaging may offer a simple and practical diagnostic tool for the evaluation of posterior pole lesions due to OT</p>

CYSTISERCOSIS

<p>Reddy S, Panchal B, Pathengay A</p>	<p>BMJ Case Rep 2020;</p>	<p>Relationship between scolex, shape of the cyst and timing of surgery in subretinal cysticercosis</p>	<p>Cysticercosis is a parasitic infestation caused by larval form of cestode tapeworm <i>Taenia solium</i>. The parasite may get access to posterior segment of the eye through the choroidal circulation and reaches subretinal space. The larvae may stay there or reaches vitreous cavity after perforating through overlying retina.</p> <p>The diagnosis of intraocular cysticercosis is mainly clinical where a live cyst can be seen as a translucent white cyst in the vitreous cavity or subretinal space. They observe the spherical shape corresponds to a free-floating mobile cyst and the oval shape relates to a fixed cyst. when the scolex is fixed at one end, it pushes the intracystic fluid to opposite end making it oval and when the scolex is in the centre, intracystic fluid is distributed all around it making it spherical. A free-floating mobile cyst in subretinal space can migrate transretinal into the vitreous cavity. Thus, knowing the shape of cyst may help in better surgical planning and avoiding intraoperative mishaps. This may help in surgical planning of the cyst removal, as a fixed cyst is easier to locate and remove compared with a free-floating mobile one.</p>
--	-------------------------------	---	---

<p>Sudha K Ganesh, Priyanka</p>	<p>Ocular Immunology and Inflammation. 2018</p>	<p>Analysis of Clinical Profile, Investigation, and Management of Ocular Cysticercosis Seen at a Tertiary Referral Centre</p>	<p>Majority of patients in this study were young males, and all cases were unilateral, bilateral presentations have been rarely reported.</p> <p>Cystercerci most commonly found in the skeletal muscle, subcutaneous tissue, eye, and brain. This study revealed higher prevalence of Extraocular 71% than Intraocular 29%. Medial rectus muscle (26%) was the most frequent location in extraocular cases.</p> <p>Ultrasonography (USG) is an effective and economical alternative to MRI and CT for the detection of the intra/extra-ocular cysticerci.</p> <p>A cysticercus cyst with the scolex attached to the inner wall composes the “hanging drop sign.” Imaging with CT scan.</p> <p>MRI provides detailed images of living and degenerating cysticerci. Reports suggest that MRI is more sensitive than CT in imaging non-calcified lesions and can pick up the scolex better than the CT scan. While a positive ELISA for the detection of antibody to cysticerci confirms the diagnosis, negative ELISA does not rule out the diagnosis.</p> <p>Extracellular is effectively managed with a combination of albendazole and steroids and most of them recovered good vision. Amongst the Intraocular, posterior segment cysticercosis is reported to be more common than anterior segment cysticercosis. The cyst located mostly in the vitreous (56.6%) followed by subretinal cysts (36.6%) and only 6.6% of cysts in anterior chamber. The treatment of anterior chamber cysticercosis is essentially surgical.</p>
---------------------------------	---	---	--

			<p>Anterior chamber cysticercosis may be associated with the anterior uveitis which may be due to the toxin and heterologous protein leakage through the vesicle wall of the cysticercosis in the vesicular stage of its development. A live cyst could also induce inflammation and that could worsen the prognosis by causing a traction or combined traction-rhegmatogenous retinal detachment. Therefore prednisolone is used in all patients with evidence of intraocular inflammation.</p>
<p>Ramanuj Samanta, Gitanjali Sood, Shalaka R Waghmare, Nisheet Patnaik, Ajai Agrawal</p>	<p>Indian J Ophthalmol 2020</p>	<p>Submacular cysticercosis in two cases: Course and outcome</p>	<p>Cysticercosis of the posterior segment of the eye can involve vitreous cavity or subretinal space. It lodges in the subretinal space after entering the eye through choroidal circulation. It can further migrate into the vitreous cavity through a retinal hole, which is generally obscured by an inflammatory chorioretinal scar. The submacular space is more predisposed due to high vascular supply and carry a worse prognosis as compared to other subretinal locations due to physical damage of photoreceptors, underlying RPE atrophy or inflammatory effects from liberated toxins. In surgical management</p> <p>transscleral approach of cyst removal is advocated for cysts located anterior to equator, while transvitreal approach is employed for intravitreal and subretinal cysts posterior to the equator. No significant difference in outcome was reported when the cysts were removed in-toto versus cysts engulfed rapidly in vitreous cavity with cutter</p> <p>The other key step of surgery is ensuring complete removal of posterior hyaloid to avoid post-operative contraction of vitreous.</p>

LEISHMANIA

<p>Guillaume Mignot, Yagnaseni Bhattacharya, Aravind Reddy</p>	<p>Indian J Ophthalmol. 2021</p>	<p>Ocular Leishmaniasis - A systematic review</p>	<p>The retinal manifestations of Leishmaniasis consist of self-resolving hemorrhages secondary to thrombocytopenia. Two main uveitis aetiologies have been identified: uveitis in the context of active Leishmanial infection (associated with immunosuppression) and uveitis occurring as an immune reconstitution syndrome. Topical steroids are of little use in keratitis (systemic antileishmanials being the cornerstone of treatment). However, these are essential in cases of uveitis, with or without concomitant systemic antileishmanial therapy.</p>
<p>Krishna Pandey, Biplab Pal, Roshan Kamal Topno, Chandra Shekhar Lal, Vidya Nand Rabi Das, Pradeep Das</p>	<p>Rev Soc Bras Med Trop. 2020</p>	<p>Acute uveitis: A rare adverse effect of miltefosine in the treatment of post-kala-azar dermal leishmaniasis</p>	<p>Acute anterior uveitis, a rare adverse effect, experienced by a patient treated with miltefosine for post-kala-azar dermal leishmaniasis. This adverse effect developed after 15 days of miltefosine consumption. The ophthalmic complication was completely resolved with antibiotics and steroid eye drops. After recovery from the ophthalmic complication, the patient was successfully treated with liposomal amphotericin B for the skin lesions.</p>

<p>Roberto Badaró, Larissa O Goncalves, Luana L Gois, Zuinara Pereira Gusmão Maia, Constance Benson, Maria Fernanda Rios Grassi</p>	<p>J Int Assoc Provid AIDS Care. 2015</p>	<p>Leishmaniasis as a Manifestation of Immune Reconstitution Inflammatory Syndrome (IRIS) in HIV-Infected Patients: A Literature Review</p>	<p>After the onset of highly active antiretroviral therapy (HAART), some HIV-infected patients present a severe inflammation in response to a latent or a previously treated opportunistic pathogen termed immune reconstitution inflammatory syndrome (IRIS). Commonly found characteristics included cutaneous involvement, regardless of Leishmania species; appearance of lesions unrelated to time of probable Leishmania infection; rapid recovery of CD4 count following HAART; and rapid progression.</p>
<p>A Perrin- Terrin, S Auriol, L Mahieu, A Debard, A Eden, M Cassagne, V Pagot-Mathis, F Malecaze, V Soler</p>	<p>J Fr Ophtalmol. 2014</p>	<p>Recurrent bilateral anterior uveitis due to Leishmania infantum in a patient with immune deficiency related to HIV infection: a case report and literature review</p>	<p>Efficacy of treatment can be monitored with weekly quantification of Leishmania DNA in the aqueous humor. Uveitis secondary to leishmaniasis is rare and serious. Physicians must be aware of this cause of uveitis, particularly in immunodeficient patients. The quantitation of Leishmania DNA in the aqueous humor is an indispensable tool for monitoring the disease.</p>

DUSN			
<p>Carlos Alexandre de Amorim Garcia Filho, Alexandre Henrique Bezerra Gomes,</p> <p>Ana Claudia M, De A. Garcia Soares and Carlos Alexandre de Amorim Garcia</p>	<p>Am J Ophthalmol 2012</p>	<p>Clinical Features of 121 Patients with Diffuse Unilateral Subacute Neuroretinitis</p>	<p>Subretinal tracks and focal alterations of the RPE was most common clinical feature found. Almost 70% of patients younger than 20 years.</p> <p>Most patients were diagnosed in the late stage (112/121 patients). This distribution reflects poor VA at the time of diagnosis, with 71% of patients with VA worse than 20/200 at presentation.</p> <p>In relation to patient age and worm identification, it was observed that the younger the patient, the higher the rate of larval location.</p> <p>Evaluation of all suspected patients, followed by a careful search for the worm and photocoagulation to destroy it, may prevent additional visual loss or may result in improved vision in early-stage patients.</p>
<p>Luiz H Lima, Gustavo B Melo, Claudio Zett, Fábio B Morais, Bruno C Leal, Michel E Farah, Rubens Belfort Jr</p>	<p>Ophthalmic Surg Lasers Imaging Retina. 2020</p>	<p>Optical Coherence Tomography Angiography Findings in Diffuse Unilateral Subacute Neuroretinitis</p>	<p>Cross-sectional OCT showed disruption of outer retinal layers in the foveal area and an irregular structure of the outer plexiform layer. En face OCT revealed hyperreflective spots and a large hyperreflective lesion in the foveal area correspondent to the outer retina disruption seen on cross-sectional OCT. OCTA demonstrated decreased vascular perfusion in both the superficial and deep retinal capillary plexuses along with choriocapillaris preservation.</p>

<p>Thiago José Muniz Machado Mazzeo , Mario Martins Dos Santos Motta , André Luiz Land Curi</p>	<p>J Ophthalmic Inflamm Infect. 2019</p>	<p>Diffuse unilateral subacute neuroretinitis: review article</p>	<p>Parasites of different sizes and species have been proposed as the etiologic agent of DUSN, including <i>Ancylostoma caninum</i>, <i>Toxocara canis</i>, and others. It may present as vitritis, multifocal gray-white lesions in the outer retina, and derangement of the retinal pigment epithelium, narrowing of the retinal vessels and optic atrophy. It is hypothesized that different infectious worms may be considered as the likely cause of both an autoimmune and toxic form of nematode retinopathy. Cases in which the worm can be identified, it is defined as confirmed DUSN, and eyes with the typical clinical features but without identification of the worm should be classified as presumed DUSN. In confirmed DUSN, the classic treatment is directly photocoagulation of the worm. Treatment of presumed DUSN cases with high-dose oral albendazole has shown encouraging results. Due to the possibility of this disease being, in part, autoimmune nematode retinopathy, corticosteroids associated with both albendazole or laser therapy, could be in any way beneficial.</p>
---	--	---	--

TREMATODE

<p>Sivakumar R. Rathinam, Lalan Kumar Arya, Kim R. Usha, <i>et al</i></p>	<p>Arch Ophthalmol. 2012</p>	<p>Novel Etiological Agent: Molecular Evidence for Trematode- Induced Anterior Uveitis in Children</p>	<p>Histopathological analysis has provided support that trematode infections can cause a characteristic granulomatous anterior uveitis in children from South India. Serologic testing is unreliable mainly because of cross-reactive antigens. Faecal egg identification likewise has limited utility because humans act as accidental hosts. Molecular diagnostic studies, in contrast, can identify individual species of trematodes involved in site-specific infections.</p>
<p>Rowayda Mohamed Amin, Alaa E Radwan, Mohamed B Goweida, Hesham F El Goweini, Ahmed M Bedda, Wael M Lotfy, Ahmed R H Ahmed</p>	<p>Jpn J Ophthalmol. 2019</p>	<p>Management of presumed trematode induced granulomatous uveitis in pediatric patients</p>	<p>The disease has been shown to affect children in rural villages after swimming in the fresh waters of the River Nile, causing anterior uveitis with white anterior chamber (AC) nodules that eventually evolve into retrocorneal scars. The lesions were only occasionally associated with subconjunctival nodules and dense vitritis. The uveitis in these cases is exceptional, and is presumably the result of incidental human infection under particular environmental circumstances. The inflammation is notably chronic and persistent, and vision is oftentimes lost to extensive AC scarring or phtthisis. A favorable response to steroid monotherapy is demonstrated in low grade disease, while surgical excision was found to be curative in patients with larger lesions or those showing suboptimal response to medical treatment.</p>

MISCELLANEOUS

<p>Anita Paul, Ashwin T Pammal</p>	<p>Indian J Ophthalmol. 2008</p>	<p>Ocular parasitosis: a rare cause of hypertensive uveitis</p>	<p>In cases of chronic, unilateral, granulomatous hypertensive uveitis causative factor can be parasitic worm. Therapeutic success in such patients depends upon early and complete surgical removal of the worm, which could be a real challenge as worms are highly mobile and only visible sporadically. Ocular parasitosis should be kept in mind as a differential diagnosis in treating non-responsive chronic hypertensive granulomatous inflammation, especially if the patient is of Southeast Asian origin or has recently visited the region.</p>
<p>Preeti Rawat, Manushree Gautam, C Nikhila, Rajdeep Jain</p>	<p>Indian Journal of Ophthalmology. 2016</p>	<p>Intraocular gnathostomiasis A rare case report from Central India</p>	<p>Humans become accidental host when they consume raw or undercooked meat of the definitive host or the second intermediate hosts such as brackish water fish, chicken, snails, and frogs or paratenic hosts such as birds. The most common manifestation of intraocular gnathostomiasis is anterior uveitis and intraocular parasite because it mostly localizes itself in the anterior segment of the eye. The other common manifestations are eyelid edema, conjunctival chemosis, hyphema, retinochoroidal, vitreous hemorrhage, and rarely, central retinal artery occlusion leading to blindness. The portal of entry into the eye may be posterior retina because intraocular gnathostomiasis has been associated with macular scarring, rupture of nasal branch of central retinal artery, or retinal tear with choroidal hemorrhage near the optic disc. High-dose albendazole, given for 3 weeks, has been used as specific chemotherapy for treatment of patients with gnathostomiasis.</p>

<p>G Kluxen, A Hörauf</p>	<p>Der Ophthalmologe. 2007</p>	<p>Ocular onchocerciasis: a key role for Wolbachia</p>	<p>Onchocerciasis is caused by the parasitic worm <i>Onchocerca volvulus</i>. When they die, either by natural attrition or after chemotherapy, the host response to degenerating worms can result in ocular inflammation (keratitis, uveitis, chorioretinitis, neuritis of the optic nerve) that causes progressive loss of vision and ultimately leads to blindness. Worms treated with the antibiotic doxycycline, which destroys <i>Wolbachia</i>, induced lower corneal stromal thickness and stromal haze (indicators of corneal oedema and opacity) and neutrophil infiltration compared with both untreated worms and worms that do not harbour <i>Wolbachia</i>. Worms recovered from patients treated for 6 weeks with doxycycline contained fewer <i>Wolbachia</i> bacteria and had abnormal embryogenesis, indicating a role for <i>Wolbachia</i> in the survival or fecundity of the worms. Antibiotic treatment may also reduce the severity of the inflammatory response.</p>
---------------------------	--------------------------------	--	--

<p>Dr. S. R. Rathinam, Dr. Radha Annamalai, Dr. Jyotirmay Biswas</p>	<p>Ocular Immunology & Inflammation. 2011</p>	<p>Intraocular parasitic infections</p>	<p>Parasitic diseases of human and animal populations are common in low-resource countries. Epidemiological investigations are not available from places where the disease is endemic. There is limited literature on the research-to-policy process from low- and middle-income countries. Policy makers have a variable understanding of economic analysis and burden of disease measures. Recommendations to facilitate the uptake of research into policy include improving the technical capacity of policy makers, implementing quality research, and dissemination of research results. Steps needed to be taken by the ophthalmologist include identification of the parasite, search for systemic involvement, treatment for elimination, and sequelae. It is also mandatory to increase the awareness among doctors toward the need for screening procedures, early diagnosis, and, finally, public health notification.</p>
--	---	---	--

Abbreviations used:

OT- Ocular Toxocara

DRI CT- Deep Range Imaging Optical Coherence Tomography

HAART- Highly active antiretroviral therapy



DIFFUSE UNILATERAL SUBACUTE NEURORETINITIS (DUSN)



Dr. Anamika Patel

Department of Vitreo-retina and Uveitis, LV Prasad Eye Institute, GMR Varalakshmi Campus, Vizag, Andhra Pradesh, India



Dr. Avinash Pathengay

L V Prasad Eye Institute, GMRV Campus, Vizag, Andhra Pradesh, India

Dr. Ketan Saoji

L V Prasad Eye Institute, GMRV Campus, Vizag, Andhra Pradesh, India

Dr. Jyothirmai Manasa

L V Prasad Eye Institute, GMRV Campus, Vizag, Andhra Pradesh, India

DIFFUSE UNILATERAL SUBACUTE NEURORETINITIS (DUSN)

Q. What are the stages in DUSN?

Answer: There are two stages in DUSN: Acute and Chronic¹

Acute or Early stage of DUSN: It's characterized by presence of multiple evanescent small round yellowish to greyish white lesions which appear in small cluster or crops seen at the level of outer retina affecting single quadrant at a time. Additionally, patient may present with anterior uveitis, relative afferent pupillary defect, vitritis, retinal vasculitis and papillitis in few, if not all cases. Commonly the macular and juxta macular locations are affected. The image here shows presence of multiple bright yellowish white well defined round lesions inferior to superotemporal arcade. At times, patients may present with subretinal yellowish white tracts or tunnels also called as Garcia's sign suggestive of larva migration in subretinal space.² It is interesting to note that larger worms are reported to leave tracts of clumped retinal pigment epithelium as their footprints and smaller worms are known to leave small chorioretinal atrophic scars.

Late or chronic stage of DUSN: It's characterized by presence of disc pallor, narrowed retinal arterioles, diffuse degeneration of retinal pigment epithelium and enhanced retinal inner limiting membrane reflex also called as orifice sign.³

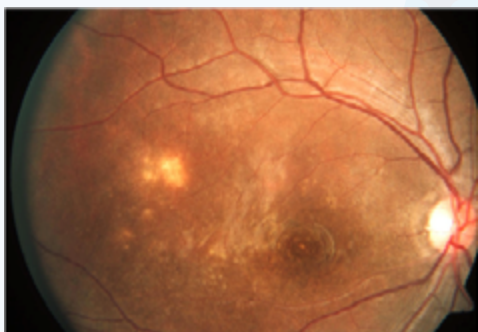


Image 1: Acute/Early stage DUSN showing presence of bright yellowish lesions (crops) in cluster

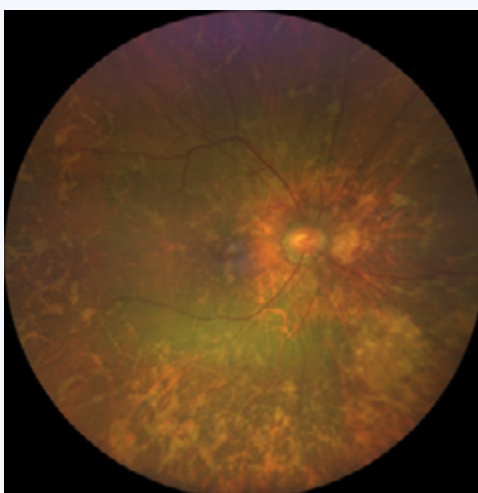


Image 2: Garcia's sign

Right eye image here is showing the presence of multiple zigzag shaped, subretinal tracts which appears to be yellowish with pigmented borders.

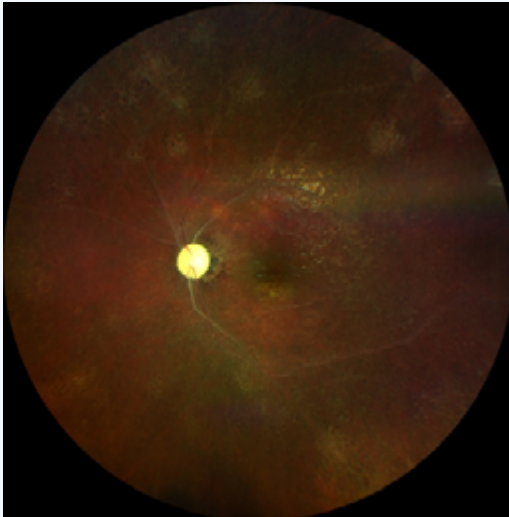


Image 3: Late/Chronic stage DUSN

The image here showcases the classic presentation of chronic stage of DUSN where, disc pallor, attenuated arterioles, RPE degeneration and enhanced ILM reflex are noted.

Q What are the causative organism names?

Answer: Several species of nematodes have been reported mainly *Toxocara canis*, *Baylisascaris procyonis*, *Ancylostoma caninum*, *B. procyonis* and *A. caninum*.⁴

Q. Why it is important to know stages of DUSN?

Answer: Two important reasons to identify which stage of DUSN patient is presenting with.

1. Identifying and treating the patient in acute stage of DUSN helps to avoid progression to chronic stage which usually leads to advanced irreversible visual loss.
2. It is important to differentiate active stage of DUSN from its very close mimicker MEWDS which is multiple evanescent white dots syndrome.⁴

Q. Why MEWDS, SLC and unilateral RP are considered as differential diagnosis in DUSN

Answer: Evanescence of white lesions are characteristically observed in both MEWDS and acute stage of DUSN. The distinctive feature of MEWDS is the evanescent white lesion when they fade away, they do not recur whereas in acute stage in DUSN they disappear in one quadrant and reappear in adjacent quadrant.

Dr Donald Gass went on to describe that any case previously diagnosed as MEWDS which fails to resolve within three weeks, has persistent fluorescein angiography pattern and shows presence of new lesions after 3 weeks, should be considered as DUSN.⁵

Early unilateral multifocal Serpiginoid choroiditis in TB endemic country could mimic DUSN but these lesions are never evanescent, activity is predominantly located in margins and signs of subtle scarring with or without hyperpigmentation is noted in the center of the scar.

5

Unilateral retinitis pigmentosa like appearance can be noted in the advanced/late stages/ chronic stages of DUSN. The following two observations like pallor of disc with attenuation of arterioles can be observed in both RP and chronic DUSN. However, the following three signs like presence of non-diffuse mottling of retinal pigment epithelium, lack of bony spicules pigmentation and prominent ILM reflex also called as orifice sign helps one to differentiate chronic stage of DUSN from unilateral RP clinically. Additional point that I would like to share is that unilateral Full Field ERG is never completely extinguished for scotopic and photopic responses in chronic DUSN. ⁵

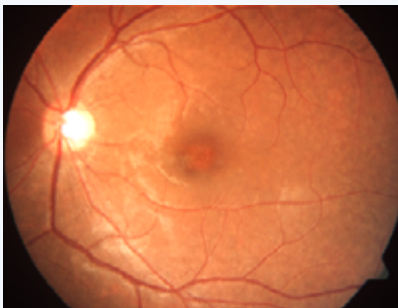


Image 4: MEWDS with foveal granularity

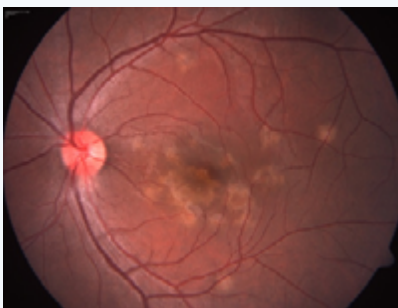


Image 5: Multifocal Serpiginoid choroiditis

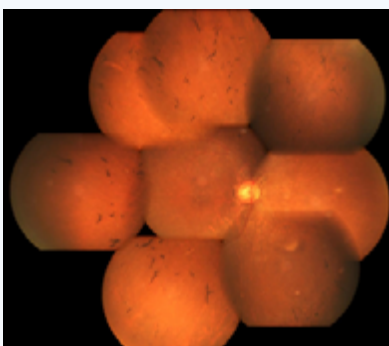


Image 6: Unilateral retinitis pigmentosa

Q. How does one identify DUSN is caused by a small worm or a large worm?

Answer: The causative nematodes are of two types based on their length – small and large.

Small worm(400-1000microns) appears as white or yellow outer retinal lesions , tends to move slowly and has slower rate of progression

Larger worm(1000-2000 microns)-appears grey brown in colour, moves faster and has rapid rate of progression¹

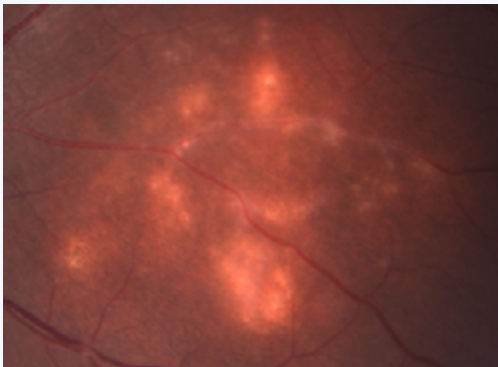


Image 7: Small worm

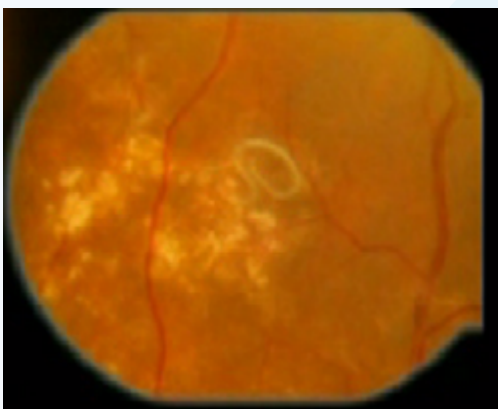


Image 8: Large worm

Q. How does one identify small worm?

Answer: Identification of worm/nematode is important because one may come across the same in both acute and chronic stage of DUSN. As we know there are two types of worms small and large.

Identifying larger worm is relatively easier and indirect ophthalmoscope helps in locating the same. For the small worm, one should actively search for it with fundus contact lens preferably with 78/90 diopter lens¹¹. Also, two pointers which would help in localizing the worm are:

1. Lookout for the worms near the vicinity of the outer retinal yellowish white lesions.
2. Look for number 6 or 9 (Image 9) like pattern to identify the worm. ⁵

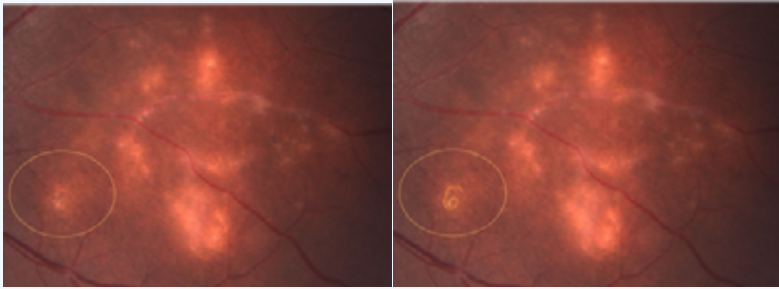


Image 9: Multiple yellowish crops showing number "6" and "9" in magnified view (yellow circle) The above image depicts the worm coiled near the multiple yellowish crops fashioned in numeric "6" pattern. One may see such morphological appearance of worm in pattern of numeric pattern "9" as well.

Q. What are the OCT characteristics of acute and chronic stage of DUSN?

Answer: DUSN is a clinical diagnosis and other investigative modalities could act as adjuvants.

Ocular Coherence Tomography (OCT) features are different both in acute and chronic stage. (Image 10a and 10b) As seen in this image here, one may notice presence of neurosensory detachment and shed photoreceptors in the early stage. Also, retinal nerve fiber layer appears thickened, which is reported to be a transitory edema. Late stage of DUSN as we now understand is a chronic of DUSN where one may observe neuroretinal atrophy and focal hyperreflectivity in the affected area.^{4,6}



Image 10a: OCT of Acute stage DUSN

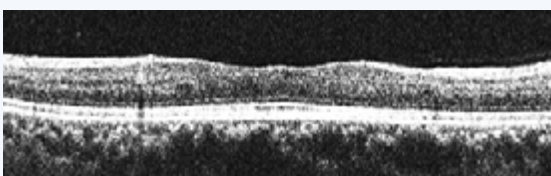


Image 10b: OCT of chronic stage DUSN

Image here shows following points in a case of chronic DUSN

1. Loss of foveal contour
2. Decreased CMT
3. Decreased RNFL thickness
4. Intact IS-OS

Q. What are the FFA characteristics of DUSN?

Answer: **Active stage** – The gray–white lesions appear hypofluorescent in early phase of angiogram and stain in the later phase of angiogram along with disc leakage due to leakage of dye from optic disc capillaries⁵. Perivenous leakage of dye is noted in cases of vasculitis.

It's interesting to know that despite the presence of neurosensory detachment, one may observe absence of leakage at fovea.⁵ (Image 11 a and b)

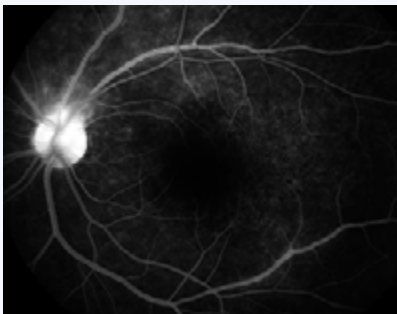


Image 11a: Disc Hyperfluorescence

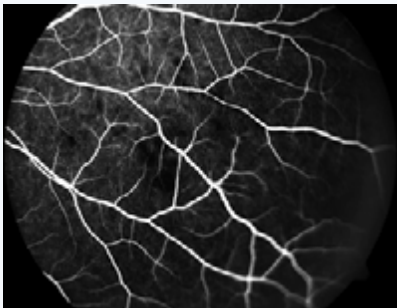


Image 11b: Active crops appearing hyperfluorescent

Late stage of DUSN: In chronic stage of DUSN one gets to observe irregular increase in background choroidal fluorescence, salt and pepper appearance with enlarged FAZ as seen in the left eye fluorescein angiography montage here.⁵(Image 11c)

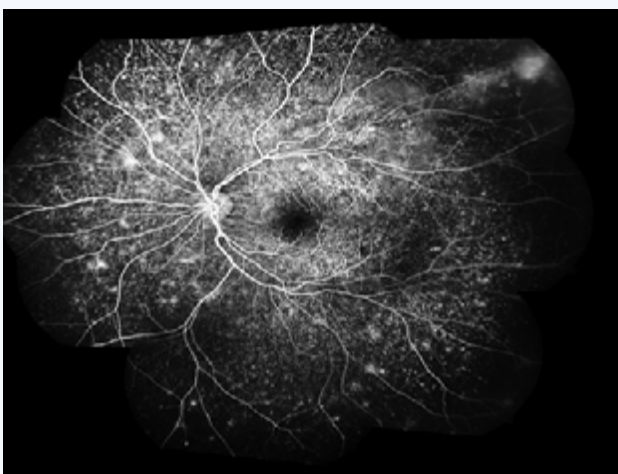


Image 11C: Late stage showing salt and pepper appearance with enlarged FAZ

Q. Once a diagnosis of DUSN is made how do we approach towards its management?

Answer: One may follow the algorithm depicted here. First question that we must ask ourselves is worm visible or not? If yes- opt for laser photocoagulation. If not visible, look for presence of vitritis. If vitritis is present-Start medical treatment (oral anti helminthics)⁷

If Vitritis is absent-opt for medical treatment along with scatter photocoagulation. (Image 12)

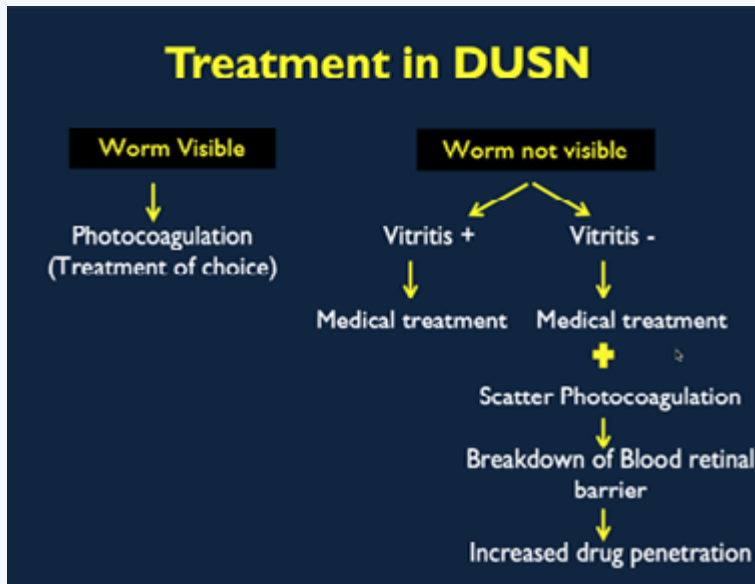


Image 12: Treatment algorithm

Q. What are the advantages of photocoagulation in DUSN?

Answer: It is prudent to identify the worm which is seen generally in the vicinity of the lesions. Direct laser photocoagulation is effective in destroying the worm since it makes worm immobile post laser application. (Image 13) Also, speed of laser is important to prevent migration to fovea. Direct laser photocoagulation in absence of vitritis helps in creating aseptic breakdown of blood retinal barrier and makes environment conducive for better penetration of anti-helminthics drugs.^{5,7-8}

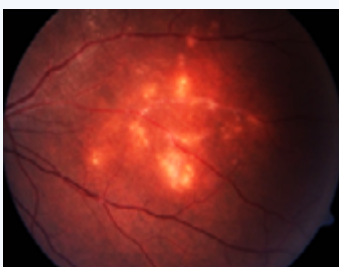


Image 13a: Pre- treatment Image

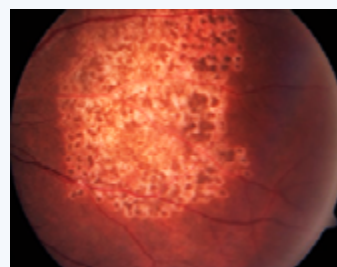


Image 13b: Post-treatment

In early stages of DUSN, prompt localization and destruction of the worm by photocoagulation may:

- 1.Improve the vision of patients
2. Halts disease progression⁵

Q. Role of oral anti helminthics in the management of DUSN

Answer: Ocular penetration of anti-helminthics is supposed to increase by disrupting blood-retina barrier by laser photocoagulation. We usually opt for 9/12 mg of ivermectin that is single dose after photocoagulation.

Albendazole is another broad-spectrum benzimidazole antihelminthic which could be used for 400 mg once every day for 3-5 days.⁹⁻¹¹

Q. Are corticosteroids mandatory in the management of DUSN?

Answer: No, it's not mandatory and we don't use it in our practice.

Use of corticosteroid is linked to the suppression of inflammation that occurs after the death of the nematode (secondary to treatment with photocoagulation or systemic medications such as anti-helminthic).

Summary / take home points

- DUSN is a clinical diagnosis
- There are two stages of DUSN: Early/active and Late/chronic
- Early/active stage of DUSN can mimic MEWDS, SLC and late/chronic stage of DUSN can mimic unilateral retinitis pigmentosa
- ERG is rarely completely extinguished in late/chronic stage of DUSN
- Treatment depends on whether worm is visualized. If visualized, consider direct photocoagulation of worm and if not visualized consider medical treatment +/- scatter laser photocoagulation.

References:

1. Gass JD, Scelfo R (1978) Diffuse unilateral subacute neuroretinitis. Journal of the Royal Society of Medicine. 71(2):95-111
2. Garcia Filho CA , Garcia CA , Arevalo JF . Imaging in the diagnosis and management of diffuse unilateral subacute neuroretinitis. Int Ophthalmol Clin 2012;52:283-9
3. Oréface F, Gonçalves ER, Siqueira RC et al (1994) Estudo de 21 casos de Neuroretinite subaguda unilateral difusa (DUSN)-dois casos de larva móvel sub-retiniana. Ver Bras Oftalmol. 53:467-489
4. Mazzeo, T.J.M.M., dos Santos Motta, M.M. & Curi, A.L.L. Diffuse unilateral subacute neuroretinitis: review article. J Ophthal Inflamm Infect 9, 23 (2019).
5. Gass JDM. Stereoscopic atlas of macular diseases: diagnosis and treatment. 5th ed. St Louis, MO: Mosby; 2012.
6. Berbel RF, Casella AM, de Souza EC, Farah ME (2014) Evaluation of patients with diffuse unilateral subacute neuroretinitis by spectral domain optical coherence tomography with enhanced depth imaging. Clinical ophthalmology. 8:1081-1087

7. Relhan N, Pathengay A, Raval V, Nayak S, Choudhury H, Flynn Jr HW. Clinical experience in treatment of diffuse unilateral subretinal neuroretinitis. *Clinical Ophthalmology (Auckland, NZ)*. 2015;9:1799.
8. Cherukuri N, Panchal B, Kaza H, Doshi S, Pathengay A. Role of PASCAL and optical coherence tomography angiography in the treatment of diffuse unilateral subacute neuroretinitis caused by large live motile worm. *Indian journal of ophthalmology*. 2019 Sep;67(9):1494
9. Maguire AM, Zarbin MA, Connor TB, Justin J. Ocular penetration of thiabendazole. *Arch Ophthalmol*. 1990;108(12):1675.
10. Gass JD, Callanan DG, Bowman CB. Oral therapy in diffuse unilateral subacute neuroretinitis. *Arch Ophthalmol*. 1992;110(5):675–680.
11. Gass JD, Callanan DG, Bowman CB. Successful oral therapy for diffuse unilateral subacute neuroretinitis. *Trans Am Ophthalmol Soc*. 1991;89: 97–112; discussion 113–116.



Recent Advances in Parasitic uveitis



Dr. Reema Bansal

Professor, Uveitis and Vitreo-Retina Services, Advanced Eye Centre
Post Graduate Institute of Medical Education and Research (PGIMER)
Chandigarh



Dr. Nikitha Ayyadurai

Uveitis and Vitreo-Retina Services, Advanced Eye Centre
Post Graduate Institute of Medical Education and Research (PGIMER)
Chandigarh

Recent Advances in Parasitic uveitis

Abstract

Parasitic causes of uveitis are more prevalent in the developing part of the world. *Toxoplasma gondii*, *Taenia canis*, *Onchocerca volvulus*, *Taenia solium*, *Ancylostoma caninum*, *Cysticercus celulosae*, and *Philophthalmus* are a few of the parasites responsible for morbid ocular infections. Herein we review the recent advances in parasitic uveitis that are prevalent in the Indian sub-continent.

Toxoplasmosis

Ocular toxoplasmosis is the most common cause of parasitic uveitis in both immunocompetent and immunocompromised individuals with variable prognosis. Epidemiology is yet to be studied in all the continents but recently certain strains are found to be more virulent than others due to their ability to evade host immune system like the South American variant than European strain. (1–3) Clinical manifestation can be variable and atypical especially in conditions causing systemic or local immunosuppression (intravitreal steroid). Multimodality imaging in punctate outer retinal toxoplasmosis (PORT) has shown outer retinal changes like disruption of the ellipsoid zone, interdigitation zones and retinal pigment epithelium, as well as inner retinal changes in the form of punctate, preretinal, hyperreflective lesions at the vitreoretinal interface, all of which regressed with treatment. (4)

Diagnosis is mainly clinical. In atypical cases, it is aided by serological tests. (5) To increase the sensitivity of polymerase chain reaction (PCR), Sugita et al. (6) suggested a 2-step PCR protocol for extremely small quantity ocular samples. A qualitative multiplex followed by quantitative RT-PCR was done to measure the *T. gondii* DNA titre. By this method sensitivity improved to 85%. Gomez CA et al. demonstrated that single-reaction, dual-target (B1, Rep529) RT-PCR for the detection of *T. gondii* DNA had similar sensitivity compared to the nested, conventional, single target (B1 gene) PCR. (7) Time of infection helps in deciding management in pregnant women to prevent vertical transmission. Recently, IgG avidity assay rather than identifying just IgG and IgM has been considered to be more reliable in differentiating newly acquired from remote infection causing recurrences. (8) IgG avidity index shifts from a low to a high by about 5 months from acquiring an acute infection. In pregnant females, pyrimethamine should be avoided in the first trimester due to its potential teratogenicity and sulfadiazine should be avoided in the last few weeks because of the risk of kernicterus in neonatal period. Spiramycin, azithromycin, intravitreal clindamycin are safe during pregnancy. When serum Ig M is positive or the IgG avidity is low, there is a risk of congenital toxoplasmosis in the foetus. (9)

Pyrimethamine and sulfadiazine (co-trimoxazole), plus corticosteroids, triple therapy is the frequently used treatment for ocular toxoplasmosis. Two Cochrane reviews have established the effectiveness of chemotherapy for ocular toxoplasmosis. The first one concluded that there is no evidence to support use of routine systemic antibiotic for acute ocular toxoplasmosis in immunocompetent individuals. Regarding treatment in chronic recurrent ocular toxoplasmosis, there was only weak evidence to reduce recurrence. (10)

The second Cochrane review did not identify any role of corticosteroids in the treatment of ocular toxoplasmosis from randomised control studies. (11) Yet, several questions remain unanswered regarding- duration, the dose of steroid and when to start steroid.

In vitro studies have shown that IL17 weakens the blood-retinal barrier playing a pivotal role in pathogenesis, thus making it a target for potential treatment. (12) Anti-IL17 monoclonal antibody like ixekizumab, brodalumab and secukinumab are already under trial for autoimmune diseases.(13–15)

Live-Attenuated RH:ΔNPT1 Strain of *T. gondii*, recombinant DNA vaccine encoding GRA14 and ROP13 genes, multi-epitope ROP8 DNA vaccine, DNA vaccine ROP29 and many others have shown good cellular and humoral immune response in animal trials. (16–19) Yet, Toxovax (live-attenuated tachyzoites of S48 strain) is the only vaccine available and that too approved for use in sheep. (20)

Toxocariasis

Ocular toxocariasis is caused by *Toxocara canis* or *catis*, a nematode. The ultra-wide-field (UWF) images compared to conventional fundus photography have better sensitivity as the periphery is better visualised in case of peripheral granuloma type. (21) In serologically confirmed unilateral toxocariasis, ultra-wide-field (UWF) imaging has detected granuloma in 91% of cases. Ultrasound bio microscopy (UBM) in *Toxocara* affected eyes with hazy medium has revealed granuloma, vitreous opacities, tractional retinal detachment and pseudocystic changes in the peripheral vitreous. (22) Other than characteristic clinical features, serological and immunological investigations like immunoelectrophoresis, ELISA, PCR aid in diagnosis. ELISA using various antigens like *Toxocara* excretory-secretory (TES), TES-58, TES-68, recombinant TES antigens (rTES), rTES-26, rTES-30, rTES-120, *Toxocara canis* larvae (TCLA) and antibodies IgG, IgG4 (subclass of IgG) have been studied. (23) The diagnostic efficacy of IgG antibody against *Toxocara* using ELISA has been recently studied in ocular fluid. (24,25) Patients with negative serology were found to have positive vitreous titre for toxocara antibody thus, a negative serology does not rule out ocular toxocariasis. Treatment strategy is inflammation control and surgical management of vitreo-retinal complications. The role of anthelmintics remains uncertain in ocular toxocariasis unlike systemic disease.

New formulations and delivery systems of anthelmintic drugs like microparticles produced by a spraying technique using sodium lauryl sulfate containing chitosan-encapsulated albendazole, polyethylene glycol (PEG)-conjugated ('pegylated') form of albendazole, liposome-encapsulated albendazole stabilized with PEG,

immunomodulator formulation of fenbendazole, phytochemical compounds extracted from *Picrasma quassioides*, *Ailanthus altissima* and *Ficus obtusifolia* has been studied in systemic toxocariasis.(23) Probiotics based on *Saccharomyces boulardii* which have already been found efficacious in prevention and treatment of some human gastrointestinal problems has been studied recently in mice model of systemic toxocara with promising results.(26)

Cysticercosis

Vitreous is the most common intra-ocular site for cysticercosis followed by subretinal space, optic nerve head and anterior chamber.

Investigations like ultrasound, CT, MRI, ocular coherence tomography(OCT) and UBM help in imaging the cyst and scolex. (27) Utility of serological tests like western blot and ELISA(enzyme-linked immunoassay), molecular PCR and mitochondrial DNA have been studied for systemic cysticercosis. (28,29) Serological studies have shown that IgG-ELISA has lower sensitivities in the diagnosis of Ocular cysticercosis than Neurocysticercosis. (30) In a recent study on ocular cysticercosis, ELISA using somatic and excretory-secretory(ES) antigens of metacestode were positive in 32.5% and 45% cases respectively. (31) ELISA was positive more frequently when the cyst was in extraocular location than intraocular. The poor sensitivity of the antibody tests in intraocular cases than extraocular cases was considered to be due to the immune privilege of the eye.

Management of ocular cysticercosis has always been a combination of antiparasitic medication, inflammation control and surgical removal. Albendazole is the anti-helminthic drug of choice. Etanercept-a tumour necrosis factor-alpha inhibitor has been tried in cases of neurocysticercosis to control inflammation due to anti-helminthic therapy and facilitate corticosteroid taper. (32) Many new benzimidazole derivatives and histone deacetylase inhibitors (entinostat, TH65, and TH92) are currently under clinical trial. (33-35)

Several glycoproteins of *Taenia solium* are studied as a target for the development of vaccines with some success in field trials, although, none is available for clinical use yet. (36,37)

Malaria

Malaria is endemic in our country. Its ocular manifestations have been studied and reported extensively. Pathogenesis of malarial retinopathy was considered similar to cerebral malaria, wherein sequestration of infected red blood cells in retinal microvasculature and endothelial dysfunction causes vascular obstruction resulting in ischemia and hypoxia. (38,39) Recently, a mice model has shown that the malaria parasites cross the blood-RPE (retinal pigment epithelium) barrier and infiltrate the neuro-retinal cells. (40)

Studies using Optical Coherence Tomography (OCT) has revealed hyper-reflective changes in walls of both large retinal vessels and capillaries which were seen even in clinically normal fundus. (41) The lumen of large vessels were found to be either hypo or hyper-reflective. Histologically, the hypo-reflective lumina correspond to vessels with parasitized erythrocytes sequestering around the vessel wall with normal blood in the lumen. The hyper-reflective lumina correspond to vessels filled with parasitized erythrocytes. All these changes resolved with treatment. Areas of retinal whitening have been demonstrated to have a paracentral acute middle maculopathy (PAMM) like findings on OCT as described in ischemic retinal diseases. (42)

In malaria-endemic areas, parasitaemia does not imply a diagnosis of severe malaria so, retinopathy could be a diagnostic tool in comatose patients, also increasing risk of mortality. (43) In third world countries where expertise is sparse Artificial-Intelligence can be of immense help. Automated software systems have been developed to analyze the retina to detect malaria retinopathy to aid in the diagnosis of cerebral malaria. (44,45) The software detects the Intravascular filling defects (IVFD) which are due to sequestration of parasitised erythrocytes in the microvasculature in fundus fluoresceine angiography of children with good precision compared to expert human observers.

Diffuse unilateral subacute neuroretinitis (DUSN)

DUSN is a form of retinopathy caused by *Toxocara canis* (most common), *Baylisascaris procyonis*, *Ancylostoma caninum*, *Gnathostoma spinigerum*, *Strongyloides stercoralis*, *Brugia malayi* and *Alaria*.(46)

Evidence of subretinal tunnels (Garcia's sign) and prominent internal limiting membrane (ILM) (Oréface's sign) has been demonstrated using OCT. (47) Optical coherence tomography angiography (OCTA) has been utilised to demonstrate a moving nematode in rare cases. (48) In definitive cases, treatment is laser ablation of the nematode and in presumptive cases, albendazole along with corticosteroids is the line of management. (49) Reports have demonstrated the worm becoming inactive and disappearing from OCTA post treatment as it does not have a vascular system by itself.(48,50)

Paediatric Presumed Trematode Infection

Trematode infections of human are rare. There are molecular (51) and histological evidences(52) for remanent of trematode present within acute nodular conjunctivitis and anterior chamber granuloma in paediatric cases. The epidemiology suggested the existence of an endemic waterborne trematode infection presumed to be caused by *Philophthalmus*, in the states of Tamil Nadu and Kerala.

A recent study using UBM has described the following features; retro corneal membrane, peripheral anterior synechiae, granulomatous anterior chamber reaction, subconjunctival nodule, iris granuloma, ciliary body granuloma, diffuse thickening of the ciliary body, cyclitic membranes, vitreous condensation over pars plana. (53)

Amin et al. have proposed a scoring system to aid in management wherein size of the granuloma, grade of inflammation and complications were taken into account. (54) According to the size of anterior chamber (AC) lesion - <3mm, 3-5 mm, >5mm was given a score of 1,2,3 respectively. Grade of AC inflammation (based on SUN grading of AC cells) - 0.5+ to 1+, 2+ to 3+, 4+ was given a score of 1,2,3 respectively. Complications at presentation - none, retrocorneal scar or corectopia, cataract, glaucomatous optic atrophy were scored as 0,1,2,3 respectively. Patients with total score <5 were managed medically with a topical and oral corticosteroid. Patients with total score ≥ 5 at presentation were managed surgically with excision of the granulomatous lesion.

Ocular Leishmaniasis

India is endemic for both cutaneous leishmaniasis and visceral leishmaniasis (Kala-Azar) and the recent co-infection with HIV has posed a diagnostic and management challenge. (55) Recently, its prevalence has increased among the organ transplant cohort.

Uveitis in leishmaniasis could be due to the infection per se or a part of immune reconstitution syndrome post-treatment with systemic anti-leishmanial or Highly Active Antiretroviral Therapy (HAART). (56) Ocular infection causes granulomatous uveitis and the parasite has been demonstrated in the ocular sample by PCR and Giemsa staining. They are to be treated with topical, systemic steroids along with anti-leishmanial drugs. Intracameral, intravitreal, intrastromal amphotericin B has been tried in anterior uveitis, panuveitis, kerato-uveitis respectively with good results. (57,58)

Onchocerciasis

Otherwise known as "African River Blindness," Onchocerciasis is a filarial infection caused by the nematode *Onchocerca volvulus*. Clinical manifestations include keratitis, live microfilariae in the anterior chamber, chorioretinitis, optic neuritis. There are a few cases reports of cutaneous and ocular onchocerciasis in India including a case where a live adult worm was extracted from the eye of a patient. (59)

Ophthalmomyiasis

Myiasis is the infestation of humans with maggots (larvae) of certain flies.

Larval tracks are often apparent in cases of ophthalmomyiasis interna in subretinal space or the vitreous. OCT imaging through the tracks shows subretinal hypo reflective areas, suggestive of subretinal tunnels. There are a few case reports of ophthalmomyiasis in India.(60,61)

Conclusion

Parasitic infections have morbidities due to their direct effect, toxins, and immune reaction. Given the increase in the load of immunocompromising conditions, there is a resurgence of parasitic diseases along with unusual presentations causing difficulty in diagnosis and delay in appropriate management. Proper and early diagnosis reduces morbidity and saves vision.

References

1. Pfaff AW, de-la-Torre A, Rochet E, Brunet J, Sabou M, Sauer A, et al. New clinical and experimental insights into Old World and neotropical ocular toxoplasmosis. *Int J Parasitol.* 2014 Feb;44(2):99–107.
2. de-la-Torre A, Sauer A, Pfaff AW, Bourcier T, Brunet J, Speeg-Schatz C, et al. Severe South American ocular toxoplasmosis is associated with decreased Ifn- γ /Il-17a and increased Il-6/Il-13 intraocular levels. *PLoS Negl Trop Dis.* 2013 Nov;7(11):e2541.
3. Hassan MA, Olijnik A-A, Frickel E-M, Saeij JP. Clonal and atypical *Toxoplasma* strain differences in virulence vary with mouse sub-species. *Int J Parasitol.* 2019 Jan;49(1):63–70.
4. Yannuzzi NA, Gal-Or O, Motulsky E, Swaminathan SS, Cunningham ET, Davis JL, et al. Multimodal Imaging of Punctate Outer Retinal Toxoplasmosis. *Ophthalmic Surg Lasers Imaging Retina.* 2019 May 1;50(5):281–7.
5. Park Y-H, Nam H-W. Clinical Features and Treatment of Ocular Toxoplasmosis. *Korean J Parasitol.* 2013 Aug;51(4):393–9.
6. Sugita S, Ogawa M, Inoue S, Shimizu N, Mochizuki M. Diagnosis of ocular toxoplasmosis by two polymerase chain reaction (PCR) examinations: qualitative multiplex and quantitative real-time. *Jpn J Ophthalmol* 2011;55:495-501.
7. Gomez CA, Sahoo MK, Kahn GY, Zhong L, Montoya JG, Pinsky BA, et al. Dual-target, real-time PCR for the diagnosis of intraocular *Toxoplasma gondii* infections. *Br J Ophthalmol.* 2019 Apr;103(4):569–72.

8. Moghaddas E, Hosseini SM, Sharifi K, Rezai A, Soleimanpour S, Miri Moghaddam MM, et al. IgG Avidity Test for Ocular Toxoplasmosis Diagnosis at a Tertiary Center, Northeast of Iran. *Iran J Immunol IJl*. 2019 Sep;16(3):258–64.
9. Paquet C, Yudin MH, Society of Obstetricians and Gynaecologists of Canada. Toxoplasmosis in pregnancy: prevention, screening, and treatment. *J Obstet Gynaecol Can JOGC J Obstet Gynecol Can JOGC*. 2013 Jan;35(1):78–81.
10. Gilbert RE, See SE, Jones LV, Stanford MS. Antibiotics versus control for toxoplasma retinochoroiditis. *Cochrane Database Syst Rev*. 2002;(1):CD002218.
11. Jasper S, Vedula SS, John SS, Horo S, Sepah YJ, Nguyen QD. Corticosteroids for ocular toxoplasmosis. *Cochrane Database Syst Rev*. 2013 Apr 30;4:CD007417.
12. Moroda M, Takamoto M, Iwakura Y, Nakayama J, Aosai F. Interleukin-17A-Deficient Mice Are Highly Susceptible to *Toxoplasma gondii* Infection Due to Excessively Induced T. *gondii* HSP70 and Interferon Gamma Production. *Infect Immun*. 2017 Nov 17;85(12):e00399-17.
13. Mease PJ, van der Heijde D, Ritchlin CT, Okada M, Cuchacovich RS, Shuler CL, et al. Ixekizumab, an interleukin-17A specific monoclonal antibody, for the treatment of biologic-naive patients with active psoriatic arthritis: results from the 24-week randomised, double-blind, placebo-controlled and active (adalimumab)-controlled period of the phase III trial SPIRIT-P1. *Ann Rheum Dis*. 2017 Jan;76(1):79–87.
14. Lebwohl M, Strober B, Menter A, Gordon K, Weglowska J, Puig L, et al. Phase 3 Studies Comparing Brodalumab with Ustekinumab in Psoriasis. *N Engl J Med*. 2015 Oct;373(14):1318–28.
15. Baeten D, Sieper J, Braun J, Baraliakos X, Dougados M, Emery P, et al. Secukinumab, an Interleukin-17A Inhibitor, in Ankylosing Spondylitis. *N Engl J Med*. 2015 Dec 24;373(26):2534–48.
16. Yang W-B, Wang J-L, Gui Q, Zou Y, Chen K, Liu Q, et al. Immunization With a Live-Attenuated RH:ΔNPT1 Strain of *Toxoplasma gondii* Induces Strong Protective Immunity Against Toxoplasmosis in Mice. *Front Microbiol*. 2019;10:1875.
17. Fatollahzadeh M, Eskandarian A, Darani HY, Pagheh AS, Ahmadpour E. Evaluation of Th17 immune responses of recombinant DNA vaccine encoding GRA14 and ROP13 genes against *Toxoplasma gondii* in BALB/c mice. *Infect Genet Evol J Mol Epidemiol Evol Genet Infect Dis*. 2021 Dec;96:105150.
18. Wang J-L, Zhang N-Z, Li T-T, He J-J, Elsheikha HM, Zhu X-Q. Advances in the Development of Anti-*Toxoplasma gondii* Vaccines: Challenges, Opportunities, and Perspectives. *Trends Parasitol*. 2019 Mar;35(3):239–53.

19. Chu K-B, Quan F-S. Advances in *Toxoplasma gondii* Vaccines: Current Strategies and Challenges for Vaccine Development. *Vaccines*. 2021 Apr 21;9(5):413.
20. Innes EA, Bartley PM, Buxton D, Katzer F. Ovine toxoplasmosis. *Parasitology*. 2009 Dec;136(14):1887-94.
21. Li S, Sun L, Liu C, Wang W, Huang S, Zhang T, et al. Clinical features of ocular toxocariasis: a comparison between ultra-wide-field and conventional camera imaging. *Eye Lond Engl*. 2021 Oct;35(10):2855-63.
22. Liu J, Li S, Deng G, Yang W, Chen W, Lu H. Ultrasound biomicroscopic imaging in paediatric ocular toxocariasis. *Br J Ophthalmol*. 2017 Nov;101(11):1514-7.
23. Moreira GMSG, Telmo P de L, Mendonça M, Moreira AN, McBride AJA, Scaini CJ, et al. Human toxocariasis: current advances in diagnostics, treatment, and interventions. *Trends Parasitol*. 2014 Sep;30(9):456-64.
24. Huang L, Sun L, Liu C, Li S, Zhang T, Luo X, et al. Diagnosis of Ocular Toxocariasis by Serum and Aqueous Humor IgG ELISA. *Transl Vis Sci Technol*. 2021 Jul 1;10(8):33.
25. Abd El-Aal NF, Basha M a. A, Eid AM. New insight into the diagnostic cut-off value of serum anti-Toxocara IgG for ocular toxocariasis in uveitis patients. *J Helminthol*. 2018 Nov 20;94:e12.
26. Cardoso PD, Walcher DL, da Silva Cadore P, Beheregaray AC, Cruz LAX, Klafke GB, et al. *Saccharomyces boulardii* reduces the mean intensity of infection in mice caused by the consumption of liver contaminated by *Toxocara canis*. *Parasitol Res*. 2020 Mar;119(3):1161-5.
27. Shi D, Li S, Guo Y, Guo X. A diagnostic analysis of imaging in ocular cysticercosis. *Zhonghua Yan Ke Za Zhi Chin J Ophthalmol*. 2000 Jan;36(1):56-8, 8.
28. Gómez-Morales MÁ, Pezzotti P, Ludovisi A, Boufana B, Dorny P, Kortbeek T, et al. Collaborative Studies for the Detection of *Taenia* spp. Infections in Humans within CYSTINET, the European Network on Taeniosis/Cysticercosis. *Microorganisms*. 2021 May 29;9(6):1173.
29. Yamasaki H, Nakao M, Sako Y, Nakaya K, Sato MO, Ito A. Mitochondrial DNA diagnosis for taeniasis and cysticercosis. *Parasitol Int*. 2006;55 Suppl:S81-85.
30. Sahu PS, Parija SC, Narayan SK, Kumar D. Evaluation of an IgG-ELISA strategy using *Taenia solium* metacestode somatic and excretory-secretory antigens for diagnosis of neurocysticercosis revealing biological stage of the larvae. *Acta Trop*. 2009 Apr;110(1):38-45.
31. Sahu PS, Sahu PK, Parija SC. Diagnosis of cysticercosis in eye and treatment follow-up of extra ocular forms by serum IgG ELISA. *Int J Ophthalmic Pathol*. 2015;4:1.

32. Nash TE, Ware JM, Coyle CM, Mahanty S. Etanercept to Control Inflammation in the Treatment of Complicated Neurocysticercosis. *Am J Trop Med Hyg.* 2019 Mar;100(3):609–16.
33. Palomares-Alonso F, Jung-Cook H, Pérez-Villanueva J, Piliado JC, Rodríguez-Morales S, Palencia-Hernández G, et al. Synthesis and in vitro cysticidal activity of new benzimidazole derivatives. *Eur J Med Chem.* 2009 Apr;44(4):1794–800.
34. González-Hernández I, Palomares-Alonso F, Becerril-Vega J, Melchor-Doncel de la Torre S, Hernández-Luis F, Rodríguez-Morales S, et al. Evaluation of New Benzimidazole Derivatives as Cysticidal Agents: In Vitro, in Vivo and Docking Studies. *Chem Pharm Bull (Tokyo).* 2019;67(12):1293–300.
35. Vaca HR, Celentano AM, Macchiaroli N, Kamenetzky L, Camicia F, Rosenzvit MC. Histone deacetylase enzymes as potential drug targets of Neglected Tropical Diseases caused by cestodes. *Int J Parasitol Drugs Drug Resist.* 2019 Apr;9:120-132.
36. Jimenez-Sandoval P, Castro-Torres E, González-González R, Díaz-Quezada C, Gurrola M, Camacho-Manriquez LD, et al. Crystal structures of Triosephosphate Isomerases from *Taenia solium* and *Schistosoma mansoni* provide insights for vaccine rationale and drug design against helminth parasites. *PLoS Negl Trop Dis.* 2020 Jan;14(1):e0007815.
37. da Costa GCV, Peralta RHS, Kalume DE, Alves ALGM, Peralta JM. A gel-free proteomic analysis of *Taenia solium* and *Taenia crassiceps* cysticerci vesicular extracts. *Parasitol Res.* 2018 Dec;117(12):3781–90.
38. Conroy AL, Glover SJ, Hawkes M, Erdman LK, Seydel KB, Taylor TE, et al. Angiopoietin-2 levels are associated with retinopathy and predict mortality in Malawian children with cerebral malaria: a retrospective case-control study. *Crit Care Med.* 2012 Mar;40(3):952–9.
39. Beare NA, Taylor TE, Harding SP, Lewallen S, Molyneux ME. Malarial retinopathy: a newly established diagnostic sign in severe malaria. *Am J Trop Med Hyg.* 2006 Nov;75(5):790–7.
40. Paquet-Durand F, Beck SC, Das S, Huber G, Le Chang, Schubert T, et al. A retinal model of cerebral malaria. *Sci Rep.* 2019 Mar 5;9(1):3470.
41. Tu Z, Gormley J, Sheth V, Proudlock FA, Seydel K, Taylor T, et al. Subclinical changes on hand-held optical coherence tomography in apparently malaria retinopathy-negative patients with cerebral malaria. *Invest Ophthalmol Vis Sci.* 2017 Jun 23;58(8):5135
42. Gormley J, Tu Z, Sheth V, Proudlock FA, Seydel K, Taylor T, et al. New insights into malaria retinopathy using optical coherence tomography. *Invest Ophthalmol Vis Sci.* 2017 Jun 23;58(8):5134.

43. Singh J, Verma R, Tiwari A, Mishra D, Singh HP. Retinopathy as a Prognostic Marker in Cerebral Malaria. *Indian Pediatr.* 2016 Apr;53(4):315–7.
44. Joshi V, Agurto C, Barriga S, Nemeth S, Soliz P, MacCormick IJ, et al. Automated Detection of Malarial Retinopathy in Digital Fundus Images for Improved Diagnosis in Malawian Children with Clinically Defined Cerebral Malaria. *Sci Rep.* 2017 Feb 15;7(1):42703.
45. Joshi VS, Wigdahl JC, Nemeth SC, Zamora G, Soliz P, Lewallen S, et al. Clinical implementation of malarial retinopathy screening system for improved diagnosis of cerebral malaria. *Invest Ophthalmol Vis Sci.* 2019 Jul 22;60(9):146.
46. Mazzeo TJMM, dos Santos Motta MM, Curi ALL. Diffuse unilateral subacute neuroretinitis: review article. *J Ophthalmic Inflamm Infect.* 2019 Dec 27;9:23.
47. Arevalo JF, Arevalo FA, Garcia RA, de Amorim Garcia Filho CA, de Amorim Garcia CA. Diffuse unilateral subacute neuroretinitis. *J Pediatr Ophthalmol Strabismus.* 2013 Aug;50(4):204–12.
48. Kalevar A, Jumper JM. Optical coherence tomography angiography of diffuse unilateral subacute neuroretinitis. *Am J Ophthalmol Case Rep.* 2017 Jun 22;7:91–4.
49. Myint K, Sahay R, Mon S, et al. Worm in the eye: the rationale for treatment of DUSN in south India *British Journal of Ophthalmology* 2006;90:1125-1127.
50. Cherukuri N, Panchal B, Kaza H, Doshi S, Pathengay A. Role of PASCAL and optical coherence tomography angiography in the treatment of diffuse unilateral subacute neuroretinitis caused by large live motile worm. *Indian J Ophthalmol.* 2019 Sep;67(9):1494–6.
51. Rathinam SR, Arya LK, Usha KR, Prajna L, Tandon V. Novel Etiological Agent: Molecular Evidence for Trematode-Induced Anterior Uveitis in Children. *Arch Ophthalmol.* 2012;130(11):1481–1484.
52. Rathinam SR, Usha KR, Rao NA. Presumed trematode-induced granulomatous anterior uveitis: a newly recognized cause of intraocular inflammation in children from south India. *Am J Ophthalmol.* 2002 Jun;133(6):773–9.
53. El Hefny E, Sabry D, Sewelam A, El Nokrashy A. Characteristics of Childhood Presumed Trematode-Induced Granulomatous Anterior Uveitis Using Ultrasound Biomicroscopy. *Ocul Immunol Inflamm.* 2021 May 20;1–5.
54. Amin RM, Radwan AE, Goweida MB, El Goweini HF, Bedda AM, Lotfy WM, et al. Management of presumed trematode induced granulomatous uveitis in pediatric patients. *Jpn J Ophthalmol.* 2019 Jan;63(1):119–25.

55. Burza S, Mahajan R, Sinha PK, van Griensven J, Pandey K, Lima MA, et al. Visceral Leishmaniasis and HIV Co-infection in Bihar, India: Long-term Effectiveness and Treatment Outcomes with Liposomal Amphotericin B (AmBisome). *PLoS Negl Trop Dis*. 2014 Aug 7;8(8):e3053.
56. Mignot G, Bhattacharya Y, Reddy A. Ocular Leishmaniasis - A systematic review. *Indian J Ophthalmol*. 2021 May;69(5):1052–60.
57. Gontijo CMF, Pacheco RS, Orefice F, Lasmar E, Silva ES, Melo MN. Concurrent cutaneous, visceral and ocular leishmaniasis caused by *Leishmania (Viannia) braziliensis* in a kidney transplant patient. *Mem Inst Oswaldo Cruz*. 2002 Jul;97(5):751–3.
58. Van Os L, Cnops L, Van Esbroeck M, Dhuhghaill SN, De Keizer RJW, Asselman V, et al. Slowly Progressive Keratouveitis in a Patient with Known Systemic Leishmaniasis and HIV. *Ocul Immunol Inflamm*. 2015 Jun;23(3):248–51.
59. Barua P, Sharma A, Hazarika NK, Barua N, Bhuyan S, Alam ST. A rare case of ocular onchocerciasis in India. *Southeast Asian J Trop Med Public Health*. 2011 Nov;42(6):1359–64.
60. Sardesai VR, Omchery AS, Trasi SS. Ocular myiasis with basal cell carcinoma. *Indian J Dermatol*. 2014 May 1;59(3):308.
61. Sachdev MS, Kumar H, Roop, Jain AK, Arora R, Dada VK. Destructive ocular myiasis in a noncompromised host. *Indian J Ophthalmol*. 1990 Oct 1;38(4):184.



CYSTICERCOSIS



Corresponding Author

Dr. Padmamalini Mahendradas

Head, Uveitis and Ocular Immunology, Narayana Nethralaya
Bangalore, INDIA

Dr. Sai Bhakti Mishra

Dr. Ankush Kawali

Dr. Namita Dave

Dr. Srinivasan Sanjay

Introduction:

Ocular parasitic infections, although rare can cause devastating manifestation unless identified timely and managed appropriately. Cysticercosis is one of such infection which may have a variety of presentations. It is caused by the *Taenia solium*, a parasite belonging to the class of Cestodes. Human are definitive hosts which harbour the adult parasite in the intestine whereas pigs are the intermediate hosts harbouring the larvae. Cysticercosis occurs in humans when they become intermediate host by ingesting the eggs via contaminated food and water (hetero-infection) or get reinfected by ingestion ova of the existing parasite (external auto-infection) or retrograde peristalsis (internal auto-infection).¹ The oncosphere penetrate the wall of the stomach and gets through the blood stream to various organs.

Cysticercosis is highly prevalent in northern states of Bihar, Orissa, Uttar Pradesh and Punjab.² A seroprevalence study from Chandigarh, reported anti-cysticercus antibodies in 17.3% while seroprevalence among the healthy blood donors from Pondicherry, was found to be 6.1%.^{3,4} In a World Health Organization (WHO) study conducted in rural pig-farming community of Uttar Pradesh, the prevalence was found to be 18.6%.⁵ Series of ocular cysticercosis has been reported from north as well as from south India.⁶⁻⁸ Cysticercosis is seen where sanitary conditions are poor and where raw or undercooked contaminated pork and beef are routinely consumed. Poor hygiene, unhealthy habits, and poverty could be the underlying causes for high prevalence of cysticercosis in certain regions.

Neurocysticercosis has been recognized as an international public health issue by WHO and a major cause of epilepsy.⁹ It can coexist with ocular cysticercosis in up to 10-24% of the cases.^{7,8} Intravitreal followed by subretinal and anterior chamber location are common for intraocular cysticercosis which is seen in the form of cyst with a scolex.^{10,11} Finding a live free-floating parasite *T. solium* in the anterior chamber is a rarity.^{12,13} Live parasite may not cause significant inflammation but the parasite's death brings on a marked release of toxic products, giving rise to sight threatening acute inflammatory reaction. Thus, it is important to identify the disease at early stage and undertake appropriate management. This article will discuss in detail about the intraocular manifestations of cysticercosis, diagnosis and its management.

Life cycle:

T. solium is a member of the phylum Platyhelminthes, class Cestoda, Order Cyclophyllidea and family Taeniidae.¹⁴ *T. solium* has a complex two-host life cycle. Human beings are the only definitive host and harbour the adult tapeworm (taeniasis), whereas both humans and pigs can act as intermediate hosts and harbour the larvae (cysticerci). Humans become infected when they ingest raw or undercooked pork that contain viable cysticerci. Upon reaching the small intestine, the scolex attaches to the intestinal wall and a proglottid chain grows. The scolex of the parasite contains four suckers and a double crown of prominent

hooks. It develops into adulthood in the small intestine by a process called strobilization. *T. solium* releases three to six proglottids per day, bearing 30,000 to 70,000 eggs per proglottid, into the intestine. In the small intestine, the adult worm may live for as long as 25 years without symptoms (taeniasis) and pass gravid proglottids intermittently with the faeces. The cysticercus larvae is semitransparent, opalescent, and oval in shape and may reach a length of 0.6 to 1.8 cm.¹⁵ Human cysticercosis occurs when *T. solium* eggs are ingested via fecal-oral transmission from a tapeworm infected host. The human then becomes an accidental intermediate host. These oncospheres (primary larvae) penetrate the intestinal mucosa and enter the circulatory system. (Figure 1)

Hematogenous spread occurs to neural, muscular, and ocular tissues. Here the oncospheres develop into secondary larvae (cysticerci). The incubation period varies from months to years.¹⁶ Host inflammatory response to cysticerci depends on the parasite's ability to evade host immunity. The inflammation is restricted to degenerating cysts whose ability to evade host defences is faltering. Lack of inflammation occurs with both healthy cysticerci and those that have involuted. Upon involution, cysts undergo granulomatous change and exhibit calcification.

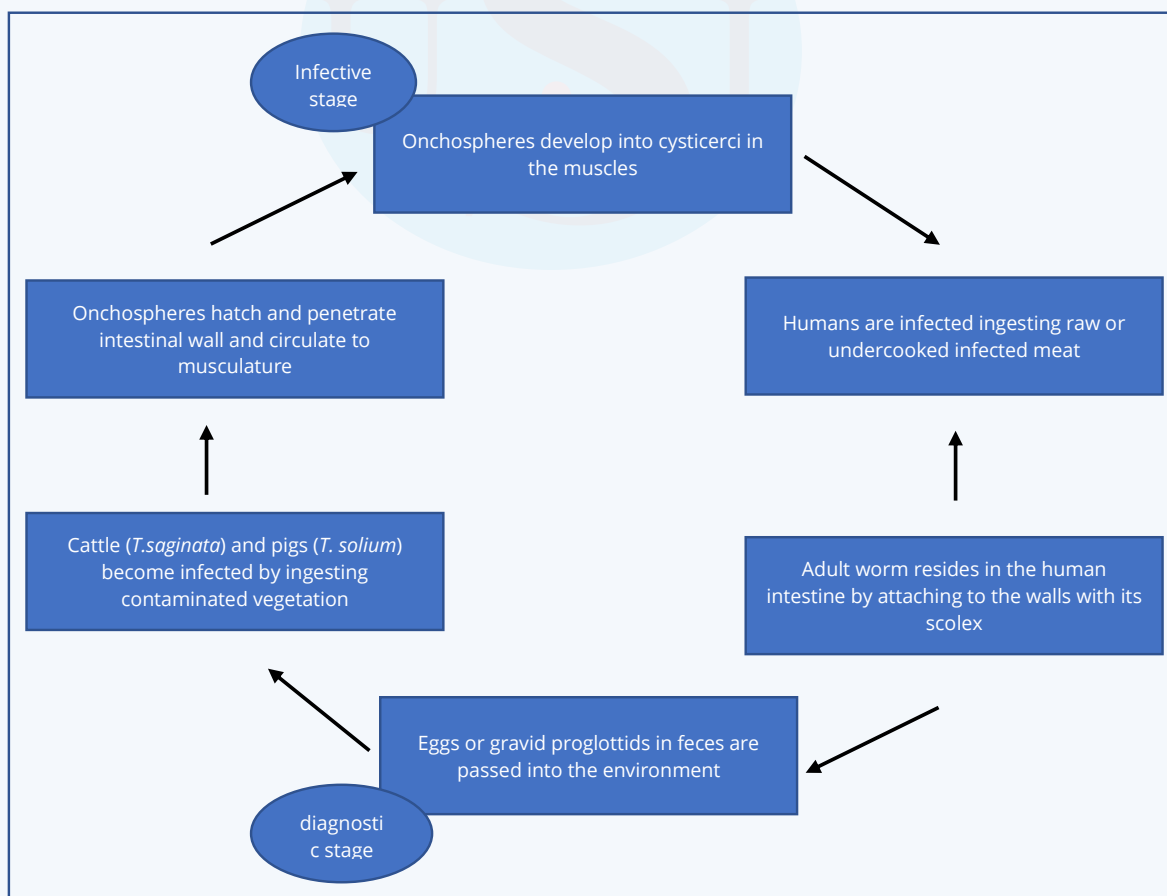


Figure 1: Life cycle of cysticercosis

STEP 1. Humans acquire the infection by eating the undercooked or raw flesh of an infected animal (pig or cattle)

STEP 2. Cystercerci migrate to the small intestine of the human host and develop into their adult tapeworm form. They attach to the intestinal wall with their scolices (hooked structures), and may persist in the intestines for long periods of time, even years.

STEP 3. The definitive host (infected humans) excretes the eggs or gravid proglottids in their feces into the environment that can remain viable from days to months. *T. solium* can be diagnosed at this point in the life cycle. Autoinfection can also occur at this point in the life-cycle via faeco-oral contamination. The eggs or gravid proglottids re-enter the body through the mouth and often travel to the central nervous system (CNS), the muscles or the eye, where they develop into cysticerci. The presence of cysticerci in these locations leads to the pathogenesis of cysticercosis (neurocysticercosis in the CNS).^{17,18}

STEP 4. The intermediate host (pigs and cattle) acquire infection by eating and digesting the eggs or gravid proglottids along with the parasitized or contaminated vegetation.

STEP 5. The gravid proglottids or eggs migrate to the animal intestine, break through the intestinal wall as oncospheres. Then via the circulatory system, they embed themselves in the pig or cattle musculature and develop into cysticerci (the infective form of *T. solium*). These cysticerci have the ability to persist in the muscle tissue for many years.

Systemic manifestations:

Clinical manifestations may depend on the affected organ; neuro-cysticercosis and ophthalmic cysticercosis are associated with substantial morbidity. The larval stage of the pork tapeworm (*T. solium*) infects the human nervous system, causing neuro-cysticercosis.¹⁹ Epileptic seizures are the commonest presentation of neuro-cysticercosis and generally represent the primary or sole manifestation of the disease.¹⁹

Seizures occur in 50–80% of patients with parenchymal brain cysts or calcifications but are less common in other forms of the disease.²⁰⁻²² In the endemic regions, recent onset of seizures in otherwise healthy teenage, young adult, or middle-aged individuals neuro-cysticercosis has to be considered.²³ Most of these patients are normal on neurological examination. Neuro-cysticercosis also presents with intracranial hypertension, hydrocephalus, or both in 20–30% of cases (the proportion varies according to the origin of the cases, higher in neurosurgical series). This syndrome is related to the location of parasites in the cerebral ventricles or basal cisterns, blocking the circulation of cerebrospinal fluid, and is caused by several different mechanisms—the presence of the parasite itself, ependymal inflammation, or residual fibrosis.^{24,25}

Outside of central nervous system, cysticercosis usually does not have major symptoms.¹⁹ Subcutaneous cysticercosis presents as small, movable, painless nodules that are most commonly noticed in the arms or chest. After a few months or even years, the nodules become swollen, tender, and inflamed, and then they gradually disappear.²⁶ Subcutaneous cysticercosis is very common in Asia and Africa. Biopsy or fine-needle cytology of a subcutaneous nodule helps to confirm the diagnosis of cysticercosis infection.²⁷ Muscular cysticercosis is a casual finding, appearing as dot-shaped or ellipsoidal calcifications following the muscle bundles in the thighs or arms, when radiography is done for an unrelated reason.

Ocular manifestations:

The clinical features of ocular cysticercosis depend on the ocular structure involved. Cysticerci can affect the eyelids and present as a subcutaneous, painless, mobile mass lesion with varying degrees of mechanical ptosis.²⁸ In the conjunctiva they form a painful or less commonly painless, yellowish, nodular subconjunctival mass with surrounding conjunctival congestion.¹ Intact larvae or cyst may be seen in the anterior chamber with or without anterior uveitis with fibrinous exudates.²⁹ When the parasite reaches the vitreous it can lead to intense vitritis, particularly if the worm dies in vitreous cavity. These patients present with floaters and blurring of vision.⁸ The parasite reaches the intraocular spaces commonly through the choroidal circulation and tends to settle in the subretinal space. (Figure 2) Preferred location is typically at the macula causing symptoms of metamorphopsia and loss of central vision.³⁰

Cysticercosis of extraocular muscle usually presents as recurrent pain, redness, proptosis, ocular motility restriction, diplopia and ptosis.^{28,31} Adnexal tissue involvement has also been reported. Patients may present with chronic dacryoadenitis and enlargement of the lacrimal gland. Lacrimal sac is rarely involved.³² Optic nerve compression by the cyst may cause decreased vision, disc edema and painful ocular motility.³³

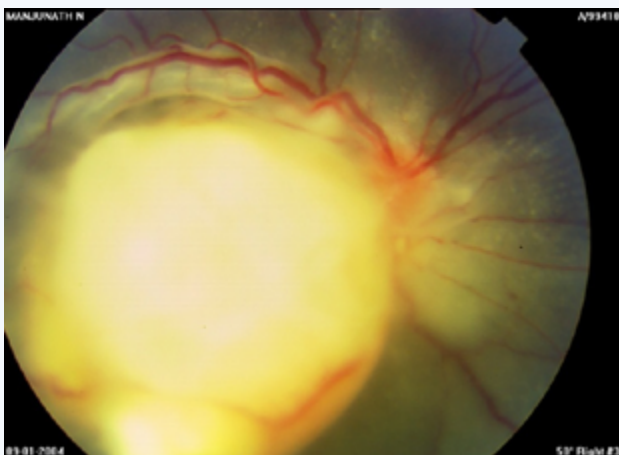


Figure 2: A 13 year old male presented with sudden decrease in the right eye vision since 3 days. On examination , the child diagnosed to have posterior uveitis in the right eye. Colour fundus photography of the right eye revealed vitritis ++ vitreous haze ++ hyperemia of

the disc, dilated tortuous retinal vessels, yellowish white circumscribed mass like lesion underneath the retinal vessel suggestive of subretinal cysticercus cyst.

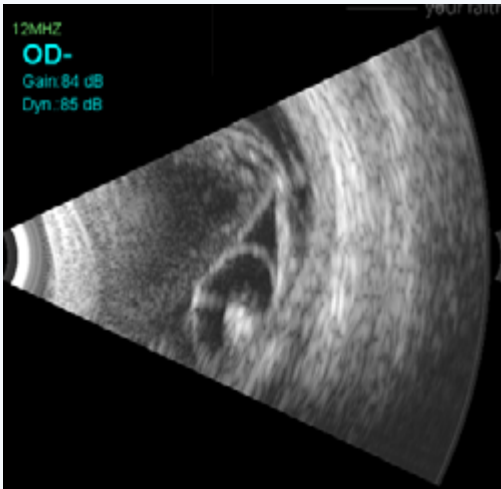


Figure 3

B scan ultrasonography showing multiple dot and membranous vitreous echoes with a subretinal well-defined cystic mass lesion with a hyperechoic central area suggestive of a scolex. This child underwent Pars plana vitrectomy followed by subretinal cyst removal and histopathological examination confirmed the presence of cysticercus cyst.

Diagnosis:

The diagnosis of ocular cysticercosis begins from history taking and comprehensive eye examination. Although, clinical history may be non-specific and symptoms may be rare and there might be no history of exposure to cattle and pigs or pork.³⁴ The presence of a cystic mass lesion in the eye may be the first clue for a final diagnosis of ocular cysticercosis. However, the definitive diagnosis is established following the identification of parasite by histopathological examination. Standard procedure requires parasitic specimen collection from suspected cystic lesion such as surgical removal or complete extraction using pars plana vitrectomy.³⁵ The parasitic cyst is typically round and a flat mount preparation is required to identify the parasite.

Visual acuity and visual fields may not be affected. Ophthalmoscopic examination may reveal a free-floating semi-translucent movable cyst with a pigmented surface. Imaging investigations such as ocular ultrasound can help in identifying parasitic cysts.³⁶⁻³⁹

The pathognomonic diagnostic feature of ocular cysticercosis on ultrasound is the presence of a well-defined cystic mass lesion with a hyperechoic central area suggestive of a scolex.^{36,37} (Figure 3) Ultrasound imaging has several advantages over other imaging techniques as such being more economical and easily repeatable by a single observer during follow up until complete resolution. For cases with intraocular cysticercosis, optical coherence tomography (OCT) is an alternate imaging technique. Spectral domain OCT may show a characteristic ring-like lesion with shadowing.³⁸

CT and MRI are helpful in diagnosing possible extraocular cysts and usually performed when there is an extraocular manifestation such as cranial nerve palsies. CT is more economical than MRI. By CT, a hypodense mass lesion with a central hyperdense scolex and MRI shows a hypointense cystic lesion with hyperintense scolex within an extraocular muscle as pathognomic findings of ocular cysticercosis. It is essential to investigate for possible cysticercosis at other parts of the body. At the minimum, it is necessary to perform brain investigation for neurocysticercosis.⁴⁰⁻⁴²



Figure 4 : Clinical photograph showing left eye proptosis, axial CT scan revealed the presence of cysticercus cysts in the left eye along with cyst in the brain due to neurocysticercosis. Patient identity needs to be hidden

However, an initial imaging may be equivocal or inconclusive. For such instances, additional laboratory investigations like serological tests including enzyme linked immunosorbent assay (ELISA) for serum antibodies against cysticercosis might be useful in active cases.⁴³ More complex tests like molecular PCR and mitochondrial DNA may support the clinical diagnosis in suspicious cases.⁴⁴

Treatment:

The two mainstay approaches for the management of ocular cysticercosis are antiparasitic drug therapy and surgical removal of parasitic lesion. The combined approach of drug therapy followed by surgical management is the recommended gold standard of treatment.⁴⁵

- Intraocular cysticercosis: Complete in-toto excision of the parasitic cyst is mandated in cases of intravitreal or subretinal lesions. Surgical removal can result in the definitive diagnosis and complete resolution of the disease.^{35,46} Nevertheless, surgical removal may either be difficult in case of a living mobile parasite or may result in increased ocular reaction to cysts. Hence, surgical removal must follow the administration of antiparasitic drug and corticosteroids for the control of inflammation following death of the parasite.⁴⁷
- Extraocular cysticercosis: Subconjunctival or intramuscular cysticercosis can be managed with antihelminthic drugs such as albendazole along with systemic steroids.
- Neurocysticercosis: Antiepileptic treatment must be given along with antihelminthic drugs and systemic steroids is important to prevent seizures as a host immune response to the dead parasite.

Albendazole is an effective antiparasitic drug that can kill larvae. It may be used as a single agent or be accompanied by praziquantel for favourable outcomes.⁴⁸⁻⁵¹ The recommended dose of albendazole is 15mg/kg/day for a month.⁴⁸ It is important to continue the medication postoperatively.⁵¹ Furthermore, classical cysticidal therapy may result in an untoward complication due to the induction of host inflammatory responses.⁵² Therefore, co-administration of corticosteroids is recommended and steroid therapy is continued until there are no signs of inflammation.

Many new benzimidazole derivatives have been developed and are undergoing clinical trials.^{53,54} Trichostatin A which is a pan histone deacetylase (HDAC) inhibitor is in clinical trials for cysticercosis treatment.⁵⁵ Etanercept is a new drug of interest when focusing on inflammation control in these patients. It has been found to be safe and highly effective in reducing inflammation and its use has been proposed to reduce steroid requirement.⁵⁶

Summary:

- Ocular cysticercosis although rare, can be a devastating manifestation of parasitic infestation.
- Humans are typically the definitive hosts harbouring the adult parasite while cattle and pigs are intermediate hosts harbouring the larva.
- Intravitreal followed by subretinal and anterior chamber cysts are the common presentations.
- B scan ultrasonography may reveal a well-defined cystic mass lesion with a hyperechoic centre suggestive of a scolex.
- High index of suspicion is important to rule out co-existing extraocular cysts and neurocysticercosis using CT and MRI.
- In-toto excision of the cyst along with its contents is recommended to prevent post-operative inflammation against parasitic contents.
- Combined surgical excision and medical treatment with antihelminthic drugs is the gold standard of treatment
- In cases with neurocysticercosis, it is important to add antiepileptic treatment along with standard care to prevent seizures from inflammation against the dead parasite.

References:

1. Dhiman R, Devi S, Duraipandi K, Chandra P, Vanathi M, Tandon R, Sen S. Cysticercosis of the eye. *Int J Ophthalmol* 2017;10(8):1319-1324.
2. National Health Portal, 2016. Taeniasis/cysticercosis. Available at: <https://www.nhp.gov.in/disease/communicable-disease/taeniasis-cysticercosis>. [Accessed 16 Nov 2021]
3. Khurana S, Aggarwal A, Malla N. Prevalence of anti-cysticercus antibodies in slum, rural and urban populations in and around Union territory, Chandigarh. *Indian J Pathol Microbiol.* 2006 Jan;49(1):51-3. PMID: 16625979.
4. Parija SC, Sahu PS. A serological study of human cysticercosis in Pondicherry, South India. *J Commun Dis.* 2003 Dec;35(4):283-9. PMID: 15909758.
5. Prasad KN, Prasad A, Gupta RK, Pandey CM, Singh U. Prevalence and associated risk factors of *Taenia solium* taeniasis in a rural pig farming community of north India. *Trans R Soc Trop Med Hyg.* 2007 Dec;101(12):1241-7.
6. Jain I S, Dhir S P, Chattopadhaya P R, Kumar P. Ocular cysticercosis in North India. *Indian J Ophthalmol* 1979;27:54-8.
7. Pushker N, Bajaj MS, Chandra M, Neena. Ocular and orbital cysticercosis. *Acta Ophthalmol Scand.* 2001 Aug;79(4):408-13. doi: 10.1034/j.1600-0420.2001.079004408.x. PMID: 11453864.
8. Sharma T, Sinha S, Shah N, Gopal L, Shanmugam MP, Bhende P, Bhende M, Shetty NS, Agrawal R, Deshpande D, Biswas J, Sukumar B. Intraocular cysticercosis: clinical characteristics and visual outcome after vitreoretinal surgery. *Ophthalmol.* 2003 May;110(5):996-1004.
9. World Health Organization. Control of neurocysticercosis, Report by Secretariat, 55th World Health Assembly Provisional agenda item 13.18 A55/23, 5 April 2002.
10. Wender JD, Rathinam SR, Shaw RE, Cunningham ET Jr. Intraocular cysticercosis: case series and comprehensive review of the literature. *Ocul Immunol Inflamm.* 2011 Aug;19(4):240-5.
11. Malik. S.R.K. Gupta A.K. and Chudhry, S. 1968. *Am. J. Ophthalmol.*, 66, 1168.
12. Sommering ST. Uber einen *Cysticercus cellulosae* im Menschlichen Auge. *Oken's Iseis* 1830;8:717-718.
13. Banstola A, Singh S, Maharjan S, Lamichhane G, Khatri A. *Taenia solium* in the Anterior Chamber: Surgical Management of a Rare Case of Live Free-Floating Parasite. *Case Rep Ophthalmol Med.* 2021;2021:4038691.
14. Odel JG, Moazami G. Diseases caused by helminths. In: Miller NR, Newman NJ (eds). *Walsh and Hoyt's Clinical Neuro-Ophthalmology*. Baltimore: Williams and Wilkins, 1997; 4439-44.

15. Markell EK, John DT, Krotoski WA. Medical Parasitology Eighth Edition. Pennsylvania: Saunders, 1999.
16. Davis, LE. "Neurocysticercosis" Emerging Neurological Infections edited by Power, C and Johnson RT. Taylor & Francis Group, 2005. 261-287
17. Garcia, Hector H., et al. "Diagnosis, treatment, and control of Taenia solium cysticercosis." Current Opinion in Infectious Diseases 16 (2003): 411-419. [4]
18. John, David T. and William A. Petri, Jr. Markell and Voge's Medical Parasitology. 9th ed. St. Louis, MO: Saunders Elsevier, 2006.
19. García HH, Gonzalez AE, Evans CA, Gilman RH; Cysticercosis Working Group in Peru. Taenia solium cysticercosis. Lancet. 2003;362(9383):547-556. doi:10.1016/S0140-6736(03)14117-7
20. Commission on Tropical Diseases of the International League Against Epilepsy. Relationship between epilepsy and tropical diseases. Epilepsia. 1994; 35:89–93. [PubMed: 8112262]
21. Chopra JS, Kaur U, Mahajan RC. Cysticerciasis and epilepsy: a clinical and serological study. Trans R Soc Trop Med Hyg. 1981; 75:518–20. [PubMed: 7324126]
22. Del Brutto OH, Santibanez R, Noboa CA, Aguirre R, Diaz E, Alarcon TA. Epilepsy due to neurocysticercosis: analysis of 203 patients. Neurology. 1992; 42:389–92. [PubMed: 1736171]
23. Garcia HH, Martinez M, Gilman R, et al. Diagnosis of cysticercosis in endemic regions. Lancet. 1991; 338:549–51. [PubMed: 1678809]
24. Lobato RD, Lamas E, Portillo JM, et al. Hydrocephalus in cerebral cysticercosis: pathogenic and therapeutic considerations. J Neurosurg. 1981; 55:786–93. [PubMed: 7310501]
25. Sotelo J, Guerrero V, Rubio F. Neurocysticercosis: a new classification based on active and inactive forms: a study of 753 cases. Arch Intern Med. 1985; 145:442–45. [PubMed: 3977513]
26. Dixon HBF, Smithers DW. Epilepsy in cysticercosis (Taenia solium): a study of seventy-one cases. QJM. 1934; 3:603–16.
27. Sahai K, Kapila K, Verma K. Parasites in fine needle breast aspirates—assessment of host tissue response. Postgrad Med J. 2002; 78:165–67. [PubMed: 11884700]
28. Rath S, Honavar SG, Naik M, Anand R, Agarwal B, Krishnaiah S, Sekhar GC. Orbital cysticercosis: clinical manifestations, diagnosis, management, and outcome. Ophthalmology 2010;117(3):600-605.
29. Mahendradas P, Biswas J, Khetan V. Fibrinous anterior uveitis due to cysticercus cellulosae. Ocul Immunol Inflamm 2007;15(6):451-454.

30. Lombardo J. Subretinal cysticercosis. *Optom Vis Sci* 2001;78(4):188-194.
31. Sundaram PM, Jayakumar N, Noronha V. Extraocular muscle cysticercosis-a clinical challenge to the ophthalmologists. *Orbit* 2004; 23(4):255-262.
32. Raoot A. Lacrimal sac cysticercosis: a rare site for manifestation. *Case Rep Ophthalmol Med* 2014;2014:961815)
33. Gurha N, Sood A, Dhar J, Gupta S. Optic nerve cysticercosis in the optic canal. *Acta Ophthalmol Scand* 1999;77(1):107-109)
34. Gonzales I, Rivera JT, Garcia HH, et al. Pathogenesis of *Taenia solium* taeniasis and cysticercosis. *Parasite Immunol.* 2016;38:136-46.
35. Zou Y, Wang F, Wang HB, et al. Disseminated cysticercosis in China with complex and variable clinical manifestations: a case series. *BMC Infect Dis.* 2019;19:543.
36. Pujari A, Chawla R, Singh R, Mehta A. Ultrasound-B scan: an indispensable tool for diagnosing ocular cysticercosis. *BMJ Case Rep.* 2017;2017. pii: bcr-2017-219346
37. Shi D, Li S, Guo Y, Guo X. A diagnostic analysis of imaging in ocular cysticercosis. *Zhonghua Yan Ke Za Zhi.* 2000;36:56-8, 8.
38. Kennelly KP, Morgan JP, Keegan DJ, et al. Objective assessment of symptomatic vitreous floaters using optical coherence tomography: a case report. *BMC Ophthalmol.* 2015 Mar 8;15:22.
39. Tripathy SK, Sen RK, Akkina N, Hampannavar A, Tahasildar N, Limaye R. Role of ultrasonography and magnetic resonance imaging in the diagnosis of intramuscular cysticercosis. *Skeletal Radiol.* 2012 Sep;41: 1061-6.
40. Wiwanitkit S, Wiwanitkit V. Intraocular cysticercosis. *Ocul Immunol Inflamm.* 2012;20:61
41. Sharma AK. Ocular cysticercosis: diagnosis and treatment. *Nepal J Ophthalmol.* 2014;6:240-1.
42. Joob B, Wiwanitkit V. Clinical Profile of Ocular Cysticercosis. *Ocul Immunol Inflamm.* 2018;26:558.
43. Mukhija R, Agarwal D, Pujari A, et al. Orbital cysticercosis. *BMJ Case Rep.* 2018; 2018. pii: bcr-2017-224028.
44. Yamasaki H, Nakao M, Sako Y, et al. Mitochondrial DNA diagnosis for taeniasis and cysticercosis. *Parasitol Int.* 2006;55 Suppl:S81-5.
45. Wiwanitkit, Viroj. (2020). Diagnostic and management strategies of ocular cysticercosis: Current perspectives. *Expert Review of Ophthalmology.* 15. 10.1080/17469899.2020.1806714.

46. Madigubba S, Vishwanath K, Reddy G, et al. Changing trends in ocular cysticercosis over two decades: an analysis of 118 surgically excised cysts. *Indian J Med Microbiol.* 2007;25:214-9.
47. Berche M, Hayot B, Mokrane M, et al. Ocular cysticercosis, typical forms and treatment. *Ophtalmologie.* 1990 Jul-Aug;4(4):377-9.
48. Pandey S, Malhotra HS, Garg RK, et al. Quantitative assessment of lesion load and efficacy of 3 cycles of albendazole in disseminated cysticercosis: a prospective evaluation. *BMC Infect Dis.* 2020 Mar 14;20(1):220.
49. Gnanamoorthy K, Suthakaran PK. Disseminated cysticercosis in an immunocompetent individual. *Ann Afr Med.* 2019;18:51-53.
50. Chopra R, Kapoor H, Chopra A. Ocular myocysticercosis: favorable outcomes with early diagnosis and appropriate therapy. *Nepal J Ophthalmol.* 2012 Jul-Dec;4(2):315- 8.
51. Wani VB, Kumar N, Uboweja AK, et al. A case of submacular cysticercosis treated by pars plana vitrectomy in Kuwait. *Oman J Ophthalmol.* 2014;7:144-6.
52. Al-Saeed WM, Oleiwi Al-Kuraishi AH, et al. Neurocysticercosis: A new concept and insight into basic and future pharmacotherapy. *J Pak Med Assoc.* 2019;69:S113-S118.
53. Palomares-Alonso F1, Jung-Cook H, Pérez-Villanueva J, et al. Synthesis and in vitro cysticidal activity of new benzimidazole derivatives. *Eur J Med Chem.* 2009 Apr;44(4):1794-800
54. González-Hernández I, Palomares-Alonso F, Becerril-Vega J, et al. Evaluation of New Benzimidazole Derivatives as Cysticidal Agents: In Vitro, in Vivo and Docking Studies. *Chem Pharm Bull (Tokyo).* 2019;67:1293-1300.
55. Vaca HR, Celentano AM, Macchiaroli N, et al. Histone deacetylase enzymes as potential drug targets of Neglected Tropical Diseases caused by cestodes. *Int J Parasitol Drugs Drug Resist.* 2019 Apr;9:120-132.
56. Nash TE, Ware JM, Coyle CM, et al. Etanercept to Control Inflammation in the Treatment of Complicated Neurocysticercosis. *Am J Trop Med Hyg.* 2019;100:609- 616.

Rewards and Recognitions



Uveitis Society (India)

(((VIRTUAL)))

USICON 2021

20th Annual Conference of Uveitis Society (India)

8th | 9th | 10th October

We had a great conference with excellent academic deliberations and interesting discussions by national and international faculty . The highlight of the conference was the participation by many of our budding young uveitis specialists who presented challenging cases, e-posters, interesting photos and a record number of freepapers.

- Free papers presentation - 18
- Challenging case presentations - 09
- e -posters - 71
- Photo exhibits - 29

We have no words to thank enough our esteemed faculty who gave their valuable time to make this conference a grand success.

A hearty congratulations to all the prize winners.

We also wish to thank all the industry friends for their support.



Bayer Zydus Pharma



USI - Executive Committee



Dr. G Venkataswamy Endowment Award 2021 Uveitis Society (India) Citation

Dr. Virender Sangwan, Director Innovations at Dr. Shroff's Charity Eye Hospital, New Delhi (India), is internationally acknowledged for his innovative simple limbal endothelial transplantation (SLET) to repair eye damage. He is the founder member of Uveitis society of India. He is the former head of ocular immunology and uveitis service from 2003 to 2012 and Director cornea and anterior segment and uveitis service from 2004 to 2012 at L.V Prasad Eye Institute, Hyderabad, India.

Known for his academic and innovative skills. He served as the Director for Innovations center at L.V Prasad Eye Institute, Hyderabad, India.

He has been at the fore front of clinical research in Ophthalmology and was ranked 4th in India in medical, dentistry and animal husbandry for being the most productive scientist in the year 2002-2014.

Dr. Sangwan provided a major breakthrough in providing an innovative technique of affordable and simple limbal endothelial transplantation (SLET) for which he was awarded the Dr. Shanti Swarup Bhatnagar Award by the Prime Minister of India in the year 2006.

Dr. Sangwan has been a recipient of several awards since his post-graduation. To name a few of his numerous awards and orations, he received the Best of Show award at the 2013 American Academy of Ophthalmology Annual Meeting, first Dr. Paul Dubord Chair of Cornea in 2012 by L.V Prasad Eye Institute, Hyderabad, Dr. R M Siboo oration on "stem cell therapy -the promise and the potential" in 2012 by Indian Medical Association, Hyderabad and Dr. Patnaik Oration on cell based therapy for ocular surface reconstruction in 2011 at Dr. Shroff's Charity Eye Hospital and award lecture at the annual meeting of international ocular surface society in 2011, Hollywood, FL, USA. Rangachari Award for best free paper in AIOS-2005.

Dr. Sangwan has published more than 262 original research papers in peer reviewed international and national journals which are extensively cited and 30 book chapters. He has been on the editorial board of various international and national journals.

The Uveitis Society (India) is privileged and honored to confer
Dr. G Venkataswamy Endowment Award 2021 on

Dr. Virender S Sangwan

in appreciation of his outstanding contributions to the field of Uveitis and Ophthalmology



Uveitis Society (India)

USICON 2021 (Virtual)

20th Annual Conference of Uveitis Society (India)

8th | 9th | 10th October

Dr. Vishali Gupta
President

Dr. Kalpana Babu
Vice President

Dr. Manisha Agarwal
Secretary

Dr. Sudharshan S
Treasurer





★ **AWARD WINNERS 2021** ★

Free Paper

Dr. Carl Herbort Travel Award for Best Free Paper



Dr. Mamta Agarwal

Topic : Infectious scleritis -clinical characteristics, causative factors and treatment outcomes in an Indian population

Institute : Sankara Nethralaya, Chennai

Dr. Narsing A Rao Award for Best Free Paper



Dr. Atul Arora

Topic : Subretinal Hyper-reflective Material in Posterior Uveitis

Institute : Advanced Eye Centre, PGIMER, Chandigarh



★ **AWARD WINNERS 2021** ★

Free Paper

Appreciation Award-1

Dr. Reema Bansal

Topic : Cytokine expression profile in tears of COVID-19 patients

Institute : Advanced Eye Centre, PGIMER, Chandigarh

Appreciation Award-2

Dr. Nitin Kumar

Topic : Clinical profile of uveitis patients with Blau syndrome:
An experience from a tertiary center in North India

Institute : Advanced Eye Centre, PGIMER, Chandigarh

Appreciation Award-3

Dr. Rupesh Agrawal

Topic : Choroidal Microvascular Alterations in COVID-19 Patients

Institute : Tan Tock Seng Hospital, Singapore



★ **AWARD WINNERS 2021** ★

Prof. Amod Gupta Young researcher award 2021



Dr. Aniruddha Agarwal

Topic : Clinical and Multimodal Imaging Clues in Differentiating between Tuberculomas and sarcoid choroidal Granulomas

Institute : Cleveland Clinic, Abu Dhabi.

Challenging Case

1st Prize

Dr. Raji Kurumkattil

Topic : Disseminated Histoplasmosis:How an environmental fungi cause havoc in immunocompromised Host

Institute : Command Hospital (Central Command) Lucknow

2nd Prize

Dr. Nikitha Ayyadurai

Topic : Relentlessly progressive hereditary vascular dystrophy

Institute : Advanced Eye Centre, PGIMER, Chandigarh

3rd Prize

Dr. Sabia Handa

Topic : Cytomegalovirus retinitis in a patient with Goods syndrome

Institute : Advanced Eye Centre, PGIMER, Chandigarh



★ AWARD WINNERS 2021 ★

Challenging Case

Appreciation 1

Dr. Srishti Agarwal

Topic: Culture positive rifampicin-resistant Mycobacterium Tuberculosis related choroidal granuloma in a monocular patient

Institute: Advanced Eye Centre, PGIMER, Chandigarh

Appreciation 2

Dr. Saurabh Luthra

Topic: A Leopard Can't change its spots!

Institute: Drishti Eye Institute, Dehradun

Appreciation 3

Dr. Shalin Shah

Topic: Retinal vasculitis and immune cytopenia as presenting signs of Sjogren syndrome

Institute: Dr Shroff's charity eye hospital, New Delhi, India



★ AWARD WINNERS 2021 ★

E Poster Presentation

1st Prize

Dr. Satabdi Nanda

Topic: Unilateral Central Retinal Vein Occlusion With Frosted Branch Angitis in a young male post recovery from Institute COVID-19 and administration of Covaxin

Institute: Prabha Eye Clinic And Research Centre
Vittala International Institute Of Ophthalmology , Bangalore

2nd Prize

Dr. Bhavana Vasudev

Topic: Post pyrexial neuro retinitis, a type three hypersensitivity
immune complex mediated ocular manifestation

Institute: Narayana Nethralaya, Bangalore

3rd Prize

Dr. Vivek Dave

Topic: Application and validation of a novel inflammatory score in the clinical grading
of infectious endophthalmitis

Institute: L V Prasad Eye Institute



★ AWARD WINNERS 2021 ★

E Poster Presentation

Appreciation 1

Dr. Kanwaljeet H. Madan

Topic: Study of different ocular manifestations in post COVID patients

Institute: Thind Eye Hospital

Appreciation 2

Dr. Amit Nene

Topic: Unilateral Optic Neuropathy with Choroiditis after COVID-19 Vaccination

Institute: Isha Netralaya, Kalyan, Maharashtra, India

Appreciation 3

Dr. Kajree Gupta

Topic: MultiColor Confocal Scanning Laser Ophthalmoscope Imaging (MCI) in
Posterior Uveitis

Institute: Postgraduate Institute of Medical Education and Research, Chandigarh



★ AWARD WINNERS 2021 ★

Photo Contest

1st Prize

Dr. Gazal Patnaik

Topic: Ocular PearlSS...

Institute: Sankara Nethralaya, Kolkata

2nd Prize

Dr. Mayur R. Moreker

Topic: Progressive Outer Retinal Necrosis

Institute: Bombay Hospital Institute of Medical Sciences, Mumbai

3rd Prize

Dr. Subina Narang

Topic: SLE presenting as bilateral combined vessel occlusion

Institute: Government Medical College Hospital, panchkula

★ **AWARD WINNERS 2021** ★

Photo Contest

Appreciation 1

Dr. Sameera Nayak

Topic: Central frosted branch angitis(green arrow), peripheral granular and haemorrhagic cytomegalovirus retinitis(black arrow)

Institute: L V Prasad Eye Institute

Appreciation 2

Dr. Anamika Patel

Topic: Vogt-Koyanagi-Harada disease showing sunset glow and dalen fuch nodules in inferior, nasal and temporal peripheral retina

Institute: LV PRASAD EYE INSTITUTE, Vizag

Appreciation 3

Dr. Lekha T

Topic: Havoc due to a Drug! Sudden onset of bilateral anterior uveitis with choroidal detachment in an otherwise healthy patient, remember to take treatment history

(eg., Topiramate)

Institute: Giridhar Eye Institute , Kochi



Uveitis Society (India) congratulates



Dr. Jyotirmay Biswas

Head of Uveitis and Ocular Pathology

Sankara Nethralaya

for having been conferred the prestigious

B. K Narayan Rao award

by AIOS-2021

“A very well deserved award for the
best teacher and a great researcher”

Dr. Jyotirmay Biswas

has won the second Best Poster Presentation Award
for Best Original Study/ Case Series
for the Poster titled

“Cytopathology Correlation of Biopsy Specimens in Uveitis
from a Tertiary Eye Care Center in South India”
at CYTOCON 2020-21

Dr. Manisha Agarwal

has been awarded

Dr. A C Agarwal Trophy

by Delhi Ophthalmological Society

Dr. Aniruddha Agarwal

has won the

Rhett Buckler Award

by American Society of Retina Specialists



Uveitis Society (India)

G Venkataswamy Endowment Lecture Awardees

2021: Dr. Virender S Sangwan

"Ocular inflammation and immune modulation "

2019: Dr. Rajeev Buddi

The 1990s: Genesis of USI and 'Hyperlocal' Uveitic entities

2018: Prof. Rathinam S

"Let there be joy of doing something beautiful"

2017: Prof. Amod Gupta

"Serpiginous like choroiditis-Present, Past and future"

2016: Dr. Jyotirmay Biswas

"My journey in uveitis"

2015: Prof. Narsing A Rao

"Pathology & Molecular advances in Uveitis"

Uveitis Society (India)



Directory
2021

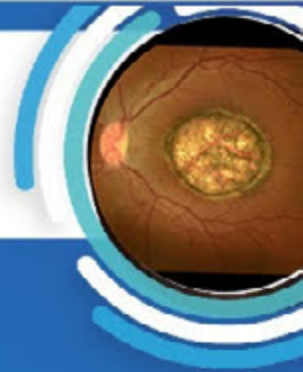
A membership directory with the contact details of all the members of the society has been made. The printed version of it will be handed over to you personally by Sun Pharma representatives or sent by courier . We thank Sun Pharma for their support in bringing out the directory

USI Academic Programme in 2021



Uveitis Society (India)

UVEITIS UP-TO-DATE



27th June 2020, Saturday

5:00 – 6:30 PM IST

Zoom Webinar ID: 934 3418 4860 Password : FUNDUS

Toxoplasma Retinochoroiditis



Introduction

Vishali Gupta
President US (I)



Moderator

Manisha Agarwal
Secretary US (I)



SPEAKER & PANELIST

Carlos Pavesio
London

5.05 – 5.15 pm

Toxoplasma Retinochoroiditis :
Clinical manifestations & diagnosis



SPEAKER & PANELIST

Andre Curi
Brazil

5.15 – 5.25 pm

Local versus systemic therapy for
Toxoplasma Retinochoroiditis & Prophylaxis

5.25 – 5.45 pm

Q & A

CHALLENGING CASES

PANELISTS



Jyotirmay Biswas



Parthopratham Majumdar



Alay S. Banker

5.45 – 5.50 pm

Case 1

Anup Kelgaonkar

5.50 – 5.55 pm

Case 2

Nivedita Nair

5.55 – 6.00 pm

Case 3

Sabia Handa

6.00 – 6.30 pm

Panel Discussion

Watch live stream on our channel : "Uveitis Society"



YouTube

<https://youtu.be/1Uhlm0tV4cU>

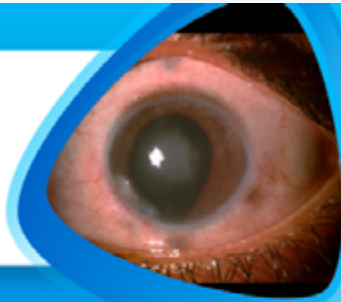


Uveitis Society (India)



Uveitis Society (India)

UVEITIS UP-TO-DATE 2



Viral Anterior Uveitis : All you need to know



25th July
2020, Saturday



5:00 – 6:30 pm
IST



Introduction

Dr. Vishali Gupta
President US(I)



Expert

Dr. Manfred Zierhut
Germany



Moderator

Dr. Manisha Agarwal
Secretary US(I)



Dr. Chee Soon Phaik
Singapore

5:00 – 5:15 pm

Clinical features to differentiate between HSV, VZV and CMV viral anterior Uveitis



Dr. Somasheila Murthy
Hyderabad

5:15 – 5:30 pm

Treating viral anterior Uveitis: When and How?



Moderator

Dr. Kalpana Babu
Bengaluru

5:30 – 5:40 pm

Case Presentation 1



Moderator

Dr. Reema Bansal
Chandigarh

5:40 – 5:50 pm

Case Presentation 2

Dr. Gazal Patnaik
Sankara Nethralaya, Chennai

5:50 – 6:30 pm

Panel Discussion with Audience participation

Watch live stream on our channel "Uveitis Society"



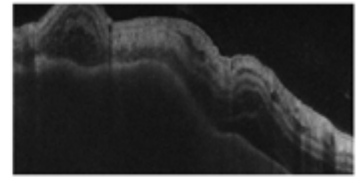
YouTube <https://youtu.be/aktzJqWhpoE>



Uveitis Society (India)



UVEITIS 3 UP-TO-DATE



OCT & OCTA in Uveitis



29th August
2020, Saturday



5:00 – 6:30 pm
IST



Introduction

Dr. Vishali Gupta
President US(I)



Moderator

Dr. Manisha Agarwal
Secretary US(I)



Speaker

Dr. Alessandro Invernizzi
Milan, Italy

5:00 – 5:30 pm

OCT in Uveitis



Speaker

Dr. Piergiorgio Neri
Abu Dhabi, UAE

5:30 – 6:00 pm

OCTA in Uveitis



Panelist

Dr. Aniruddha Agarwal
Chandigarh



Panelist

Dr. Padmamalini Mahendradas
Bengaluru

6:00 – 6:30 pm

Panel Discussion with Audience participation

Also Watch on : YouTube **LIVE**

Watch live stream on our channel "Uveitis Society"
www.youtube.com/uveitissociety-india



UVEITIS 4 UP-TO-DATE



FFA & ICG in Uveitis



26th September
2020, Saturday



5:00 – 6:30 pm
IST



Introduction

Dr. Vishali Gupta
President US(I)



Moderator

Dr. Manisha Agarwal
Secretary US(I)



Speaker

Dr. Marion R. Munk
Bern, Switzerland

5:05 – 5:25 pm

**Wide field Angiography
in Uveitis**



Speaker

Dr. Francesco Pichi
Abu Dhabi, UAE

5:30 – 5:50 pm

**Indocyanine green
angiography in Uveitis**



Panelist

Dr. Shishir Narain
*Shroff Eye Centre
New Delhi*



Panelist

Dr. Mohit Dogra
*PGIMER
Chandigarh*

5:50 – 6:30 pm

Panel discussion with audience participation

Also Watch on : YouTube **LIVE**

Watch live stream on our channel "Uveitis Society"

www.youtube.com/uveitissociety-india



WEBINAR 1



BASIC UVEITIS COURSE

January 16, 2021

01:00 PM (CET)

IUSG Goes Virtual

IUSG is pleased to announce a series of webcasts covering a vast majority of Uveitis topics. We are pleased to announce the first webcast on 16th Jan 2021 at 1 PM CET that shall cover the basics of Uveitis including epidemiology, history, anatomy and basic workup in a Uveitis patient.

Manfred Zierhut

Vishali Gupta

Marc de Smet

Speakers:



John Kempen



Daniel Vasconcellos-Santos



Peter McClusky



Reema Bansal



Manfred Zierhut

Basic Uveitis Course: January 16, 2021 (Day 1)

Basic Uveitis

(John Kempen, Daniel Vasconcellos-Santos, Peter McClusky, Reema Bansal)

• Anatomy related to uveitis	Reema Bansal	10 min
• Epidemiology of the various types of uveitis	John Kempen	20 min
• History of uveitis	Manfred Zierhut	15 min
• Differentiation of the anatomical types of uveitis	Daniel Vasconcellos-Santos	15 min
• How history of patients may help to identify the type of uveitis	Peter McClusky	15 min
• Investigations for uveitis, minimal diagnostics	Daniel Vasconcellos-Santos	20 min
• Controversy: unclear findings, contrary opinions, hot topics	all speakers	15 min
• Discussion	all speakers	10 min



BASIC UVEITIS COURSE SERIES WEBINAR 2

Supported by
Asia-Pacific Intraocular Inflammation Study Group

Sponsored by
Uveitis Society of India



**February 06, 2021
01:00 PM (CET)**

Dear colleagues,

We thank you for an overwhelming response to our first webinar. Continuing with the series, we are pleased to announce the second webcast on 6th Feb 2021 at 1 PM CET that shall cover the basics of diagnostics in Uveitis including anatomical types, serology, imaging, radiologic imaging and systemic evaluation in various forms of Uveitis.

Manfred Zierhut

Vishali Gupta

Marc de Smet

Speakers:



Basic Uveitis Course: February 06, 2021

Diagnostics

(Vishali Gupta, Moncef Khairallah, Justus Garweg, Rafael Grajewski)

- | | | |
|---|--------------------|--------|
| • Anatomical types of uveitis and the associated disorders | Rafael Grajewski, | 10 min |
| • Diagnostics for anterior uveitis: clinical diagnosis, serological testing, role of AC-tap, radiological methods | Justus Garweg, | 20 min |
| • Diagnostics for intermediate uveitis: clinical findings, difference to posterior and anterior uveitis, associated disorders and how to detect them, role of VR-biopsy, role of FLA, OCT | Moncef Khairallah, | 25 min |
| • Diagnostics for posterior uveitis: serology, radiological findings, diagnostic ppV, choroidal biopsy, MRI, OCT, autofluorescence, FLA, ICG | Vishali Gupta, | 30 min |
| • Minimal diagnostics, necessary for each type | Rafael Grajewski, | 05 min |
| • Controversy round table | all speakers, | 10 min |
| • Discussion | all speakers, | 20 min |



BASIC UVEITIS COURSE SERIES WEBINAR 3

Supported by
Asia-Pacific Intraocular Inflammation Study Group



Sponsored by
Uveitis Society of India



March 06, 2021
01:00 PM (CET)

Dear colleagues,

Thanks to all participants of the second webinar. We are now announcing the 3rd Basic Uveitis webinar for March 6th, 2021. Here we will discuss more in detail anterior and intermediate uveitis regarding their clinical, diagnostic procedures and therapeutical approaches.

Manfred Zierhut

Vishali Gupta

Marc de Smet

Speakers:



Basic Uveitis Course: March 06, 2021

Anterior and intermediate Uveitis (Soon Phaik Chee, Debra Goldstein, Manfred Zierhut)

Manfred Zierhut	Various anatomical types of uveitis and the typically associated disorders	10 min
Manfred Zierhut	Clinic of anterior uveitis	30 min
Debra Goldstein	Clinic of intermediate uveitis	20 min
Soon Phaik Chee	Diagnostic methods for anterior and intermediate uveitis	10 min
Debra Goldstein	Treatment of anterior uveitis	15 min
Soon Phaik Chee	Treatment of intermediate uveitis	10 min
All speakers	Controversy	15 min
All speakers	Discussion	10 min



BASIC UVEITIS COURSE SERIES WEBINAR 4

Supported by
Asia-Pacific Intraocular Inflammation Study Group



Sponsored by
Uveitis Society of India



Sponsored by



April 03, 2021 01:00 PM (CET)

Dear colleagues,

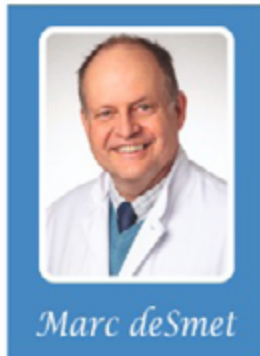
After the last webinar covering anterior and intermediate Uveitis, we would like to invite you to the 4.th IUSG webinar which will cover the huge group of posterior uveitis. We will show you first how various clinical findings can help to differentiate this group. But in the last few years tremendous progress in imaging has helped not only to differentiate posterior uveitis but also to understand the pathophysiology.

Manfred Zierhut

Vishali Gupta

Marc de Smet

Speakers:



Marc deSmet



Fernando Arevalo



Annabelle Okada

Basic Uveitis Course: April 03, 2021

Posterior uveitis (Marc deSmet, Fernando Arevalo, Annabelle Okada)

Marc deSmet	Anatomical types of uveitis and associated disorders, pathophysiology of posterior uveitis	20 min
Annabelle Okada	Differentiating posterior uveitis I - vasculitis, White Dot Syndromes, retinal detachment	20 min
Fernando Arevalo	Differentiating posterior uveitis II - location of inflammation, vitreous involvement	20 min
Marc de Smet	Diagnostics for posterior uveitis	15 min
Arevalo, deSmet, Okada	Imaging for posterior uveitis	30 min
All Speakers	Controversy	10 min
All Speakers	Discussion	05 min

REGISTER NOW



BASIC UVEITIS COURSE SERIES WEBINAR 5

Supported by
Asia-Pacific Intraocular Inflammation Study Group



Sponsored by
Uveitis Society of India



Sponsored by



May 08, 2021

Tokyo 9 pm | India 5:30 pm | Brussels 2 pm | Philadelphia 8 am

Dear Colleagues,

In our basic uveitis course we now had introduced you to the different locations of uveitis. In the consequent next step we want to invite you to the 5.th IUSG webinar which will cover the principles of uveitis treatment. Besides some basic information we will introduce you to the various groups of drugs, informing you about mechanisms, indications and what else you should know when you order such drugs. Also the use of surgery will be presented here. Finally typical faults in uveitis treatment will get presented.

Manfred Zierhut

Vishali Gupta

Marc de Smet

Speakers:



Sofia Androudi



Nida Sen



Manfred Zierhut



Vishali Gupta

Basic Uveitis Course: May 08, 2021

Treatment (Sofia Androudi, Nida Sen, Manfred Zierhut)

Manfred Zierhut	Therapy concepts for uveitis	10 min
Sofia Androudi	Therapy with corticosteroids	15 min
Nida Sen	Therapy with immunosuppressive drugs	15 min
Vishali Gupta	Therapy of infectious forms of uveitis	15 min
Nida Sen	Therapy with biologics	15 min
Sofia Androudi	How to integrate surgery in uveitis treatment	10 min
Manfred Zierhut	Top 10 of faults in the treatment of uveitis	10 min
All speakers	Controversies	20 min
All speakers	Discussion	10 min



UVEITIS - WEBINAR 6

Supported by
Asia-Pacific Intraocular Inflammation Study Group



Sponsored by
Uveitis Society of India



Sponsored by



June 05, 2021

Tokyo 8 pm | India 4:30 pm | Brussels 1 pm | Philadelphia 7 am

Dear Colleagues,

After finishing the basic uveitis course in May we now will offer you distinctive uveitis topics. With the 6th IUSG webinar we will present topics regarding one of the most often detectable anterior uveitis types, the HLA-B27 associated form. Our speakers from Australia and US will update you regarding the genetics, the clinics and the treatment of this disease. You may see: the diagnosis is easy and so is the treatment for most of the patients.

Manfred Zierhut

Vishali Gupta

Marc de Smet

Speakers:



Uveitis Course: June 05, 2021

HLA-B27 and uveitis (Peter McClusky, Dennis Wakefield, Jim Rosenbaum, Manfred Zierhut)

Dennis Wakefield	Anterior Uveitis: genetics and epidemiology	15 min
Peter McClusky	Clinical features and grading	15 min
Dennis Wakefield	Systemic disease and anterior uveitis	15 min
Jim Rosenbaum	HLA-B27, Microbiome and anterior uveitis (prerecorded)	15 min
Manfred Zierhut	New concept: "HLA-B27 Typical acute anterior Uveitis"	15 min
Peter McClusky	Approach to treatment of anterior uveitis	15 min
Dennis Wakefield	New therapy for anterior uveitis	15 min
All speakers	Controversies for "HLA-B27 associated uveitis"	10 min
All speakers	Discussion	05 min

REGISTER NOW



UVEITIS - WEBINAR

7

Supported by
Asia-Pacific Intraocular Inflammation Study Group



Sponsored by
Uveitis Society of India



Sponsored by



July 03, 2021

Tokyo 9 pm | India 5:30 pm | Brussels 2 pm | Philadelphia 8 am

Dear Colleagues,

In June we started to step deep into anterior uveitis, the most often diagnosed form of uveitis. From the HLA-B27 associated (or "typical") forms (covered in June and still available on our videos) we often see viral induced anterior uveitis. These forms, presented at the 7.th IUSG webinar, are clinically different and need, of course, another treatment than immune mediated forms of anterior uveitis.

Manfred Zierhut

Vishali Gupta

Marc de Smet

Speakers:



Uveitis Course: July 03, 2021

Viral anterior uveitis (Soon-Phaik Chee, Jolanda de Groot-Mijnes, Uwe Pleyer, Debra Goldstein)

Jolanda de Groot	Immune response vs.infection in viral disorders	15 min
Soon-Phaik Chee/Uwe Pleyer	Clinic of viral anterior uveitis	25 min
Soon-Phaik Chee	Clinically differentiating among viruses	10 min
Jolanda de Groot	Diagnostics in viral anterior uveitis	15 min
Uwe Pleyer	Treatment of viral anterior uveitis	20 min
Debra Goldstein / whole panel	Cases	20 min
All speakers	Controversies and discussion	15 min

REGISTER NOW



UVEITIS - WEBINAR

8

Supported by
Asia-Pacific Intraocular Inflammation Study Group



Sponsored by
Uveitis Society of India



Sponsored by



August 07, 2021

Tokyo 9 pm | India 5:30 pm | Brussels 2 pm | Philadelphia 8 am

Dear Colleagues,

In August our webinar is dedicated to one of the classical ocular disorders, tuberculosis (TB) induced uveitis. Over the centuries the spectrum of intraocular entities induced by tuberculosis became more and more clear. We now know that ocular TB can be a combination of infection and immune response, both effects may need treatment.

Manfred Zierhut

Vishali Gupta

Marc de Smet

Speakers:



Uveitis Course: August 07, 2021

TB (Vishali Gupta, Mamta Agarwal, Rupesh Agarwal, Soumyava Basu, John Kempen, Salil Mehta)

Rupesh Agarwal	Nomenclature of ocular tuberculosis	10 min
Soumyava Basu	Pathophysiology and immunology of TB	15 min
John Kempen	Epidemiology of TB	12 min
Vishali Gupta	Clinic of ocular TB	20 min
Salil Mehta	Diagnostics of TB	15 min
Vishali Gupta	Treatment of ocular TB	10 min
Mamta Agarwal	Problem of multidrug resistant TB	08 min
Soumyava Basu	Pulmonary vs. extrapulmonary TB	10 min
All speakers	Controversies (Round Table)	10 min
All speakers	Discussion	10 min

REGISTER NOW



UVEITIS - WEBINAR 9

Supported by
Asia-Pacific Intraocular Inflammation Study Group



Sponsored by
Uveitis Society of India



Sponsored by



September 04, 2021

CEST 3 PM | Japan 10 pm | Australia 11 pm | USA West Coast time 6 am | India 6:30 pm

Dear Colleagues,

Tuberculosis, the topic of our August webinar, has a lot of similarities to sarcoidosis, and therefore our next webinar will update you about sarcoidosis and uveitis. This includes the most recent diagnostic criteria, the clinic of extraocular and ocular sarcoidosis, differential diagnostic aspects to TB and other uveitis topics. Finally the treatment strategies will be presented.

Manfred Zierhut

Vishali Gupta

Marc de Smet

Speakers:



Uveitis Course: September 4, 2021

Current Concepts in the Diagnosis and Management of Ocular Sarcoidosis

Moderators: Manabu Mochizuki, Quan Dong Nguyen, Justine Smith

Justine Smith	Introduction	05 min
Onn Min Kon	Sarcoidosis as a systemic disease	15 min
Manabu Mochizuki	GOIW criteria for the diagnosis of ocular sarcoidosis	15 min
Jennifer Thorne	SUN perspective on ocular sarcoidosis	10 min
Debra Goldstein	Differential diagnosis of ocular sarcoidosis	10 min
Manfred Zierhut	Drug-induced sarcoidosis	10 min
Rupesh Agrawal	Distinguishing sarcoidosis from tuberculosis	10 min
Hiroshi Takase	Management of ocular sarcoidosis	15 min
Quan Dong Nguyen,	Case presentations and group discussion:	15 min
All Speakers	Controversy and discussion	15 min



UVEITIS - WEBINAR 10

Supported by
Asia-Pacific Intraocular Inflammation Study Group



Sponsored by
Uveitis Society of India



Sponsored by



October 02, 2021

CEST 2 PM | Japan 9 pm | Australia 10 pm | USA West Coast time 5 am | India 5:30 pm

Dear Colleagues,

The next webinar shall deal with one of the most important areas of Uveitis i.e., Imaging. In the current era of Multimodal imaging, we are really spoiled for choice with so many newer imaging modalities available. However, the translational value of these modalities is still evolving. This webinar shall have experts dealing with the interpretation of these imaging modalities followed by a panel discussion that shall help the participants choosing the right one for their patients.

Manfred Zierhut

Vishali Gupta

Marc de Smet

Speakers:



Francesco Pichi



Ester Carreño Salas



Marion Munk



Alessandro Invernizzi



Aniruddha Agarwal

Uveitis Course: October 2, 2021

Differential Diagnosis of Uveitis and role of Multimodal imaging

Francesco Pichi, Ester Carreño Salas, Marion Munk, Alessandro Invernizzi, Aniruddha Agarwal

IMAGING FOR ANTERIOR UVEITIS

Alessandro Invernizzi	The Cell: Automated assessment of AC inflammation	12 min
Marion Munk	Reservoir Dog: Automated flare assessment for AC inflammation	12 min

IMAGING OF THE RETINA VASCULATURE IN POSTERIOR UVEITIS

Marion Munk	How To Lose a Dye In 10 Days: will swept source-OCTA replace conventional angiography?	12 min
Ester Carreño	The Incredible Hulk: Indocyanine green angiography and other modalities to assess the choroid	12 min

OLD BUT GOLD

Alessandro Invernizzi	The Last Boy Scout: OCT, the Swiss Army knife of the uveitis specialist	12 min
Francesco Pichi	Face Off: En face OCT to assess the outer retinal changes in uveitis	12 min

CUTTING EDGE IMAGING IN UVEITIS

Francesco Pichi	Grease: imaging of the vitreous gel to get insight into uveitis	12 min
Aniruddha Agarwal	Six Feet Deep: advanced imaging of the choroid in uveitis	12 min

All speakers	Panel Discussion on 'choosing the right Imaging Modality for a given patient'	24 Min
---------------------	---	--------

REGISTER NOW



UVEITIS - WEBINAR 11

Supported by
Asia-Pacific Intraocular Inflammation Study Group



Sponsored by
Uveitis Society of India



Sponsored by



November 06, 2021

CEST 2 PM | Japan 9 pm | Australia 10 pm | USA West Coast time 5 am | India 5:30 pm

Dear Colleagues,

Thanks a lot for the overwhelming response and continued enthusiasm towards our monthly webinars. Following the special webinar on COVID, we now have a webinar on HIV; a disease that can perplex any uveitis experts by causing a myriad of opportunistic infections. In the forthcoming webinars our experts shall discuss several issues related to intraocular inflammations related to HIV including opportunistic infections, the diagnostic and management challenges. As usual we will have discussion at the end where experts would be discussing some of the controversies related to this topic. Looking forward to seeing you all virtually.

Manfred Zierhut

Vishali Gupta

Marc de Smet

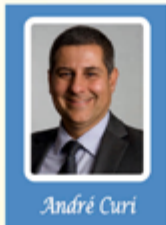
Speakers:



Alejandra Torre



Jolanda de Groot



André Curi



Remco Peters



Derrick Smit



Daniel Vasconcellos-Santos



Debra Goldstein

Uveitis Course: November 6, 2021

Special webinar on HIV

Alejandra de-la Torre, Jolanda de Groot, André Curi, Remco Peters, Derrick Smit, Daniel Vasconcellos-Santos, Debra Goldstein

Special webinar on HIV

Remco Peters	Epidemiology of HIV-infection	15 min
Jolanda de Groot	Pathogenesis of HIV	15 min
Daniel Vasconcellos-Santos	Viral ocular infections in HIV-infected individuals	20 min
Derrick Smit	Ocular Tuberculosis in HIV-infected individuals	10 min
Debra Goldstein	Syphilis in HIV patients	10 min
Alejandra de-la Torre	Ocular toxoplasmosis in HIV Patients	10 min
André Curi	Unusual intraocular infections	15 min
All speakers	Controversies	10 min
All speakers	Discussion	15 Min

REGISTER NOW



UVEITIS - WEBINAR 12

Supported by
Asia-Pacific Intraocular Inflammation Study Group



Sponsored by
Uveitis Society of India



Sponsored by



December 11, 2021

CET 1 PM | Japan 9 pm | Australia 11 pm | USA West Coast time 5 am | India 5:30 pm

Dear Colleagues,

We would like to thank you all for your continued interest and support in making IUSG webinars a great success. Continuing with our series on specific diseases, we bring you one of the most common infections causing posterior uveitis i.e., Toxoplasmosis. This webinar aims to discuss the epidemiology, pathogenesis, genetics, clinical aspects, diagnosis, management and future perspectives on Toxoplasmosis with panel discussion on controversies in Toxoplasmosis. We look forward to see you all once again.

Manfred Zierhut

Vishali Gupta

Marc de Smet

Speakers:



Alejandra de-la-Torre



Rubens Belfort



Justus Garweg



Pikka Jokelainen



José G. Montoya



Jorge Gómez-Marín

Uveitis Course: December 11, 2021

Toxoplasmosis

Alejandra de-la-Torre, Rubens Belfort, Justus Garweg, Pikka Jokelainen, José G. Montoya, Jorge Gómez-Marín

Webinar on Toxoplasmosis

Pikka Jokelainen	Epidemiology of <i>Toxoplasma gondii</i> infections	10 min
Alejandra de-la-Torre	Pathophysiological aspects of ocular toxoplasmosis: Host-parasite interaction	15 min
Jose G. Montoya	Genetics of <i>Toxoplasma gondii</i>	15 min
Justus Garweg	Diagnostics for ocular toxoplasmosis: Clinical diagnosis, serological testing, the role of AC-tap, images	20 min
Rubens Belfort	Therapy for ocular toxoplasmosis: systemic therapy, intraocular therapy, prophylaxis	20 min
Jorge Gómez-Marín	Perspectives: Is it possible to create a vaccine for <i>Toxoplasma gondii</i> ?	15 min
All speakers	Controversies	15 min
All speakers	Discussion	10 Min

REGISTER NOW



UVEITIS - WEBINAR 13

Supported by
Asia-Pacific Intraocular Inflammation Study Group



Sponsored by
Uveitis Society of India



Sponsored by



January 08, 2022

CET 1 PM | Japan 9 pm | Australia 11 pm | USA West Coast time 5 am | India 5:30 pm

Dear Colleagues,

Once again we would like to thank you all for your continued support to our IUSG monthly webinar series that have completed one successful year. Entering the new year, we shall begin with a topic that has been introduced on demand and this is about starting a Uveitis Clinic. Keeping in mind the diverse settings of all our attendees, we have invited our speakers representing several settings and they will share with you their personal experiences and how they overcame the challenges one normally would meet in setting up the uveitis clinic.

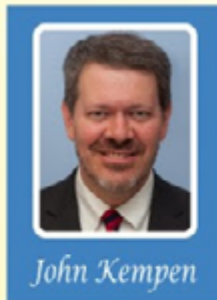
We wish you all and your families a very healthy and happy 2022 and hope to see you all virtually in the first webinar of 2022.

Manfred Zierhut

Vishali Gupta

Marc de Smet

Speakers:



John Kempen



Soumyava Basu



Marc deSmet



Manfred Zierhut



Steven Yeh

Uveitis Course: January 08, 2022

How to start a Uveitis Clinic

John Kempen, Soumyava Basu, Steven Yeh, Marc deSmet, Manfred Zierhut

How to start a Uveitis Clinic

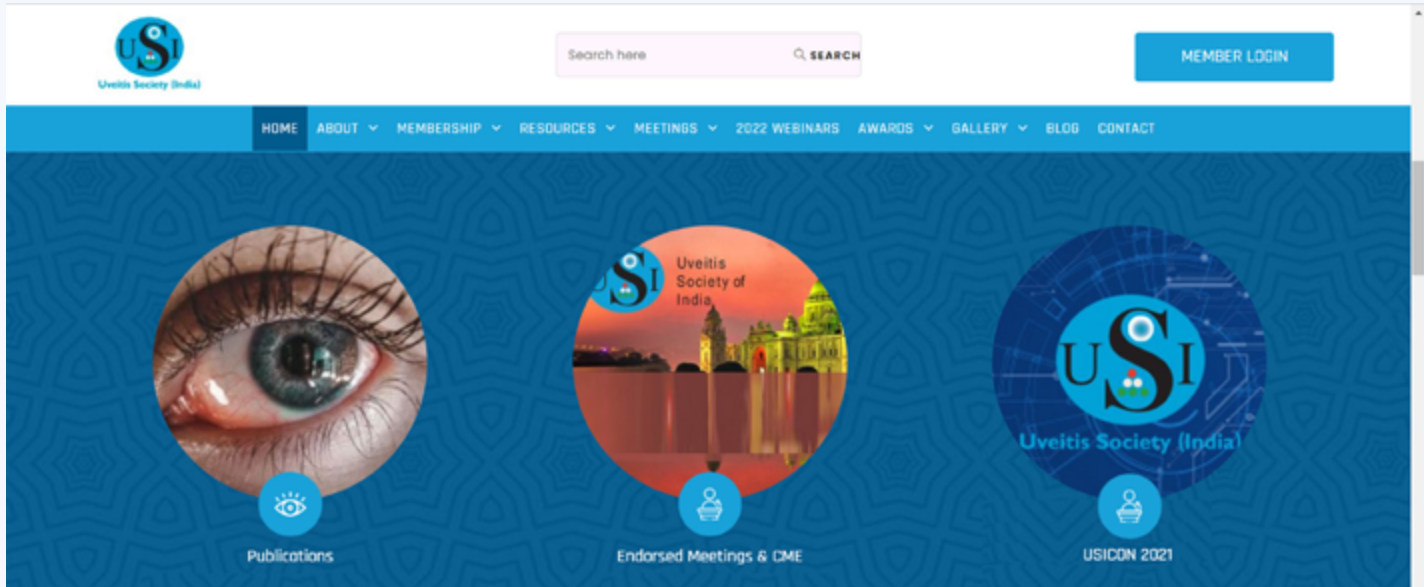
Marc de Smet	From University to private practice (Switzerland)	20 min
Soumyava Basu	From the Academic uveitis in a high-volume setting (India)	20 min
John Kempen	From University to Ethiopia	20 min
Steven Yeh	From University to Emergency (Ebola epidemic)	20 min
Manfred Zierhut	Education for emerging countries	20 min
all speakers	Panel – How to start a uveitis clinic	20 min

REGISTER NOW



Revamped USI website

www.indianuveitis.org



- Easy to use and navigate
-
- Compatible in all the internet browsers.
-
- Compatible with all the devices / mobiles / Tablets.
-
- Secured with SSL Certification
-
- Blog integration
-
- Online membership enrolment with secured payment gateway link.
-
- Exclusive members login area.
-
- USICON 2021 conference proceedings
-
- Videos of the "Webinars"

© 2021 Uveitis Society of India



THANK
YOU

With due perseverance and hard work of the editorial and scientific committee members, we have been able to publish this edition of the USI Newsletter for the readers. I sincerely wish to convey my heartfelt special thanks to Dr. Vishali Gupta Dr. Kalpana Babu, Dr. Manisha Agarwal Dr. Sudharshan S, Dr. Jyothimay Biswas, Dr. S R Rathinam, Dr. Padmamalini Mahendradas Dr. Reema Bansal, Dr. Parthoprati Dutta Majumder, Dr. Soumyava Basu, Dr. Dipankar Das Mrs. Veidhehi J and Design Team of Hallmark Events for sparing their precious time to co-edit the contents of this issue. Thanks to all the fraternity members who have contributed their manuscript.

The encouragement from all my friends and seniors is highly appreciated.

With high regards,
Dr. S Bala Murugan
Editor-in-chief
Mob: 9080518580
Email: drbalamuruganms@gmail.com

Disclaimer: *Facts and opinions in articles published in the Newsletter are solely the personal statements of respective authors. Authors are responsible for all contents in their article(s) including accuracy of the facts, statements, citing resources, and so on. Please excuse any possibility of human error by the authors, editors.*

The scientific committee wishes to thank Dr. Badrinath Talwar and Dr. S Bala Murugan for the cover page images of a 7 year child with worm in anterior chamber associated with posterior segment changes.

USI TREASURER



I consider it an honour to thank the team behind this Newsletter.

One person behind this excellent masterpiece which is academically enriching and a visual treat in equal measure is Dr. S Bala Murugan.

He has been persistent and its almost solely because of his efforts ably supported by the scientific committee.

Authors/Discussant are most important without whom this is not possible. Have to be always thankful for their valuable time and knowledge sharing.

Its always a great pleasure and a wonderful experience to be part of the team lead by Dr. Vishali Gupta

Who seems to always have novel ideas which gets implemented perfectly by Dr. Manisha Agarwal.

Hope this edition helps all ophthalmologists to become well versed and independent so that they no longer are "parasites" on anyone else during management of such patients.

Am sure you all will enjoy it because we did

Stay safe and be happy!

Dr. Sudharshan S



Uveitis Society (India)



LV Prasad Eye Institute



Uveitis Society (India)

USICON 2022

21st Annual Conference of Uveitis Society (India)

BLOCK YOUR DATES

14th
OCTOBER

15th
OCTOBER

16th
OCTOBER

H Y D E R A B A D

Organizing Secretary - Dr. Mudit Tyagi



Dr. Vishali Gupta
President

Dr. Kalpana Babu
Vice President

Dr. Manisha Agarwal
Hon. Secretary

Dr. Reema Bansal
Jt. Secretary

Dr. Sudharshan S
Treasurer

Await more details at www.indianuveitis.org

Address for Communication



Uveitis Society (India)

C/O Hallmark Events, Maruthi ' 688, 1st Floor, 6th Main
3rd Block, BEL layout, Vidyananyapura, Bangalore - 560097

Ph: 080 - 23646880 | **Mob:** 9591732274 | 9845671462

Email: uveitissociety@gmail.com

Designed by



Bangalore / Coimbatore / Chennai / Hyderabad / Kochi

www.indianuveitis.org