STATE BOARD OF OPTOMETRY
2450 DEL PASO ROAD, SUITE 105, SACRAMENTO, CA 95834
P (916) 575-7170 F (916) 575-7292 www.optometry .ca.gov

Continuing Education Course Approval Checklist

Title: White-Dot Syndromes and Phacomatoses

Provider Name: Dr. Jeong-Ah Jennifer Kim

☑Completed Application
Open to all Optometrists? ☑Yes ☐No
Maintain Record Agreement? ☑Yes ☐No

☑Correct Application Fee

☑Detailed Course Summary

☑Detailed Course Outline

☑PowerPoint and/or other Presentation Materials

☐Advertising (optional)

☑CV for EACH Course Instructor

☑License Verification for Each Course Instructor
Disciplinary History? ☐Yes ☑No

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CONTINUING EDUCATION COURSE APPROVAL APPLICATION

$50 Mandatory Fee

Board Use Only

Receipt #	Payor ID	Beneficiary ID	Amount
1-2769	385688	629891	50

Pursuant to California Code of Regulations (CCR) § 1536, the Board will approve continuing education (CE) courses after receiving the applicable fee, the requested information below and it has been determined that the course meets criteria specified in CCR § 1536(g).

In addition to the information requested below, please attach a copy of the course schedule, a detailed course outline and presentation materials (e.g., PowerPoint presentation). Applications must be submitted 45 days prior to the course presentation date.

Please type or print clearly.

Course Title	**Course Presentation Date**
WHITE DOT SYNDROMES AND PHACOMATOSES	03/14/2017

Course Provider Contact Information

Provider Name

JEONG-AH (First) KIM (Last) JENNIFER (Middle)

Provider Mailing Address

27107 TOURNEY RD
Street **City** SANTA CLARITA **State** CA **Zip** 91355

Provider Email Address jenniferkim1000@hotmail.com

Will the proposed course be open to all California licensed optometrists?	☒ YES ☐ NO
Do you agree to maintain and furnish to the Board and/or attending licensee such records of course content and attendance as the Board requires, for a period of at least three years from the date of course presentation?	☒ YES ☐ NO

Course Instructor Information

Please provide the information below and attach the curriculum vitae for each instructor or lecturer involved in the course. If there are more instructors in the course, please provide the requested information on a separate sheet of paper.

Instructor Name Dr. Mohammani will be providing the lecture for this course "White Dot Syndromes..."

AMIR (First) ISFAHANI (Last) (Middle)

License Number G078449	**License Type** MD
Phone Number (___) ___	**Email Address** amir.h.isfahani.org

I declare under penalty of perjury under the laws of the State of California that all the information submitted on this form and on any accompanying attachments submitted is true and correct.

Signature of Course Provider *Date* 2-1-17

27107 Tourney Road

Santa Clarita, CA 91355

February 9, 2017

CALIFORNIA BOARD OF OPTOMETRY
2450 Del Paso Road, Suite 105

Sacramento, CA 95834

To whom it may concern:

I am submitting a request for continuing education approval for the Kaiser Permanente Mammoth Ocular Symposium (3/12/17-3/14/17) less than the required 45 days because we have had a last minute cancellation from one of our speakers. Thus, Drs. Howard Cohen and Gary Groesbeck have volunteered to give lectures to replace the speaker who had to cancel.

Thank you so much for your understanding and my apologies for this unforeseeable change in our speakers.

If you need to contact me, please email me at jenniferkim100@hotmail.com or call me at 323-574-8957.

Sincerely,

Jeong-Ah Jennifer Kim, OD
CA Lic 11674TLG

27107 Tourney Road
Santa Clarita, CA 91355
March 4, 2017

State Board of Optometry
2450 Del Paso Road, Suite 105
Sacramento, CA 95834

To whom it may concern:

Thank you for your attention to the Kaiser Permanente Mammoth Ocular Symposium 2017 continuing education approval submission. In anticipation of receiving deficiency notifications for the other lectures, I have included a summary of each of the lectures and the respective powerpoint presentations.

There will be 7 lectures from 3/12/17-3/14/17:

The Retinal and Choroidal Dystrophies lecture is relevant to diagnosing and providing proper care as optometrists perform retinal exams on a regular basis. As optometrists continue to go toward medical aspects of eye care, this lecture will keep us well informed regarding various retinal conditions.

The Update on Cataract Surgery is relevant to optometrists because this is one of the most common referrals we make. It is important for optometrists to remain informed about advancements and changes to cataract surgeries so that we can properly educate our patients.

The Retinal White Dot Syndromes lecture is relevant in providing proper optometric care with respect to retinal diseases. Such retinal conditions may lead to discovering the underlying systemic condition giving rise to the specific white dot syndrome.

The Corneal Ectasias and Cross-Linking lecture provides information for conditions such as keratoconus and its treatment with cross-linking. Optometrists are often the first to diagnose keratoconus thus it's important that we know about various medical treatments, in addition to contact lenses and glasses.

The IOL Materials and Design lecture provides information regarding the details of lens implants for cataract patients. IOL materials and designs are topics that are commonly discussed between optometrists and their patients.

The Sports Injuries lecture is relevant as patients come into our clinics with various sports injuries sustained at school, sporting teams/clubs, and times of recreation. It is

important to anticipate and know what injuries can be sustained as optometrists provide a wide range of eye care.

The Benign Eyelid Lesions lecture provides information and visuals regarding eyelid lesions that optometrists observe daily. This will help to properly diagnose benign lesions and contrast those with lesions that need further work ups and/or referrals.

I apologize for submitting the lectures less than the 45 day request. I was waiting for all the presentations so that the lectures can be submitted together. The Benign Eyelid Lesions and Sports Injuries lectures were submitted less than the 45 request because there was a last minute cancellation of one of the original speakers, thus Drs. Groesbeck and Cohen prepared the presentations thereafter. In the future, an earlier deadline will be proposed so that the submissions will be on time.

I am attaching 2 checks that have already been deposited, one for $250 and the other for $100. All the files could not be sent in one email because the files were too large so there are 3 emails total which contain the required documents.

Thank you very much for your attention.

Sincerely,

Jeong-Ah Jennifer Kim, OD
CA Lic 11674TLG

Mammoth Ocular Symposium 2017

White Dot Syndromes: (Kourosh Mohammadi, MD)
Retina White Dot Syndromes and Phacomatoses

Acute posterior multifocal placoid pigment epithliopathy
- Case presentation
- bilateral inflammatory disease affecting the retinal pigment epithelium and outer retina
- presentation: healthy adult viral prodrome possible meningeal symptoms followed by suddn bilateral blurred vision.
- EXAM:
    -- Few Vitreous cells
    -- multiple yellow placoid lesions diagnosis is usually made by clinical appearance and characteristic fluorescein angiographic findings. Fluorescein angiogram demonstrates early hypofluorescence with late hyperfluorescent staining of the placoid lesions (See Figures 2a & b).2 Indocyanine green angiography also shows hypofluorescent spots corresponding to the placoid lesions.

- Differential diagnosis: metastatic tumors, viral retinitis, toxoplasma retinochoroiditis and pneumocystis choroiditis.

- Adenovirus type 5 infection has been serologically documented in patients with APMPPE.
- Histopathology of patients with APMPPE has not been studied. lesions thought to occur at the level of the retinal pigment epithelium or the choriocapillaris.
- Other systemic associations: meningoencephalitis, nephritis, and hearing loss

- Treatment:
    resolves spontaneously in two to 12 weeks and does not usually require treatment
    sometimes systemic corticosteroids may speed resolution of the placoid lesions and/or serous detachment.
- Prognosis:
    -- usually full recovery within 2-6 weeks

Multiple Evanescent White Dot Syndrome

- case presentation
- unilateral inflammatory chorioretinopathy that
- Typical patient: young, healthy 20-40y/o female, 1/2 have half have viral prodrome prior to disease onset.
- Symptoms: unilateral acute painless vision loss, temporal paracentral scotoma (enlaged physiologic blind spot) possible shimmering lights in the temporal visual field

- Signs Ocular findings: vitreous cell, optic disc edema and characteristic multiple white spots at the level of the retinal pigment epithelium or deep retina in the posterior pole. These spots are generally between \

10 and 100 m. The fovea has a characteristic granular appearance in the acute phase of the disease which may remain after resolution of disease.

A small percentage of patients may have chronic MEWDS and may develop choroidal scarring.

- Dx: typical ocular findings. Visual field testing may reveal an enlarged blind spot.

- Imaging: Fluorescein angiography often reveals punctate staining of the pigment epithelium in the posterior pole and disc leakage.

The punctate staining is sometimes in the shape of a wreath ICG angiography shows multiple hypofluorescent areas in the posterior pole.

- ERG: Reduction of a-wave amplitude on the electroretinogram in acute phase of disesae which normalizes after resolution of disease.

- Differential diagnoses includes APMPEE, acute macular neuroretinopathy, multifocal choroiditis panuveitis, birdshot retinochoroidopathy, diffuse unilateral subacute neuroretinitis, primary intraocular lymphoma and sarcoidosis. It is felt that idiopathic blind spot enlargement, MEWDS and MCP may represent a spectrum of the same disease process.19-21

-- Prognosis: MEWDS has a self-limited course, and no specific treatment is required. The white dots and disc edema fade within two to six weeks; however, visual symptoms such as the enlargement of blind spot, temporal scotoma, and photopsias may take several months to resolve. Recurrence is uncommon but has been reported in 10 to 15 percent of patients.15,22 Prognosis for patients with recurrences is also excellent.22 Choroidal neovascularization is a rare late complication of MEWDS.23

Serpiginous Choroiditis (geographic helicoid choroidopathy)

- case presentation
- choroidal inflammatory disease that typically affects healthy patients in the 20-70 y/o.
-- Symptoms: painless onset of paracentral scotoma and vision loss. asymmetric but usually becomes bilateral over time.
-- signs: some inflammatory response in the anterior chamber or the vitreous, but this is variable and minimal. Chorioretinal lesions are grey-white and associated with retinal and RPE edema when active .

Lesions typically start in the peripapillary region and move in a serpiginous or snake-like fashion around the posterior pole and progress to involve the macula gradually over time
-- course: The disease process has a step-wise, progressive course. Typical new lesions of serpiginous choroiditis appear contiguous to old atrophic ones.

A macular variant of serpiginous choroiditis has also been described with active lesions initially occurring in the macula without peripapillary involvement.

Subretinal hemorrhage and subretinal fluid is characteristic of choroidal neovascularization that complicates up to one-third of patients with serpiginous choroiditis.

-- Diagnosis is based on typical clinical appearance and fluorescein angiography.

-- imaging: Fluorescein angiography demonstrates early hypofluorescence and late hyperfluorescence of active lesions. Laboratory evaluation is unrevealing. In the original descriptions of serpiginous choroiditis,
most patients had a history of pulmonary tuberculosis or were purified protein derivative skin test positive;however, treatment with antituberculous agents does not impact the course of serpiginous choroiditis.
Tuberculous choroiditis can indeed look identical to serpiginous choroiditis.30

-- Differential diagnosis: tuberculous choroiditis, APMPPE, relentless placoid chorioretinitis, multifocal choroiditis and panuveitis, birdshot retinochoroidopathy and ocular histoplasmosis syndrome.

Multifocal Choroiditis and Panuveitis

-- case presentation
-- multifocal chorioretinal lesions with significant anterior chamber and vitreous inflammation.
-- Demographics: myopic females 20-60y/o life, with a mean age of onset of 33 years.
-- Symptoms: Acute onset of bilateral (75%) blurred vision, photopsias, and scotomata.
-- Ocular findings: multiple yellow or gray lesions at the level of the choroid and retinal pigment epithelium 50-1000 m and can be numerous (as many as several hundred at a time).

The lesions usually are concentrated in the midperiphery. chronic lesions are punched-out atrophic scars that develop pigmentation over time. occasional optic disc edema.

Peripapillary scarring and prominent linear chorioretinal streaks also may be present. Choroidal neovascularization can be present, in addition to peripapillary fibrosis.

Almost all patients have vitreous inflammation, and many have anterior chamber inflammation. The patient also may present with cystoid macular edema.

-- Diagnosis: based on history exam and imaging.

-- Fluorescein angiography demonstrates that active lesions show early hypofluorescence and late hyperfluorescence. However, if patients present at a later stage, the active lesions usually have scarred or

are in the process of scarring, thereby giving early hyperfluorescence and late staining. If choroidal neovascularization is present, it usually is observed as early hyperfluorescence with a lacy appearance

and a late leakage of dye. ICG angiography shows both active and chronic lesions as hypofluorescent. ICG angiography of choroidal neovascularization reveals hyperfluorescence.

-- Differential diagnosis: POHS. [35] POHS The lesions in POHS are less numerous and smaller.But POHS does not present with anterior segment and vitreous inflammation. sarcoidosis and birdshot retinochoroidopathy.

-- Pathogenesis: unknown, ? association with EBV

-- Prognosis is variable, with final visual acuity of 20/40 or better in 66% of eyes.

-- Treatment: Oral steroids are helpful in patients with active posterior segment inflammation or with cystoid macular edema. Topical corticosteroids are helpful if there is severe anterior segment inflammation.
However, cases in which corticosteroids have not improved the inflammation have been described.

-- Complications: choroidal neovascularization, which develops in 30% of patients. Laser photocoagulation or intravitreal bevacizumab may be indicated in these patients. Oral steroids also may be used for
choroidal neovascularization since they have been shown to decrease the neurosensory detachments associated with choroidal neovascular membranes.

Related conditions: PIC (acute bilateral loss of vision and photopsias and scotoma in young myopic female) but lesions in PIC are smaller and PIC has no AC or Vitreous inflammation and rarely have recurrences.

DSF (diffuse subretinal fibrosis): In addition to the presence of multifocal choroiditis, a prominent fibrosis exists. The fibrosis is predominantly at the area of previous inflammatory lesions,

and a turbid, subretinal fluid that overlies the lesions also is present. Rare with poor prognosis.

PIC and DSF represent a spectrum of disease as it relates to MCP. PIC = milder form of disease, DSF = more severe form.

Birdshot Retinochoroidopathy (vitiliginous choroiditis),

Case presentation
Presentation: Female with painless graduall blurred vision, floaters, and achromatopsia.
> 90% of patients with birdshot chorioretinopathy are HLA-A29 positive.

Ocular findings: multiple depigmented yellow-white patches scattered throughout the fundus radiate from the optic nerve and follow the larger choroidal vessels.
+/- Vitritis, optic disc edema, and cystoid macular edema

Diagnosis" is by clinical examination, fluorescein angiography, and HLA-A29 status.
Fluorescein angiography: demonstrates mild hyperfluorescence and staining in the late phase.
The pathogenesis is unknown at this time; however, speculation exists regarding an autoimmune etiology.
Treatment: Ocular and systemic corticosteroids are generally the treatment of choice.
Prognosis: Birdshot retinochoroidopathy is a chronic disease with multiple recurrences, and, guarded visual prognosis

Presumed Ocular Histoplasmosis Syndrome

Presentation: Male or Female Pt in 40's from Ohio and Mississippi River Valley. May be asymptomatic or may have central or paracentral vision loss.

Exam: No vitritis or anterior uveitis. + PPA, + atrophic choreoretinal lesions, and sometimes cnv. Sometimes linear streaks in midperiphery.

Pathophysiology: H capsulatum; however, the organism has never been isolated from the choroid.

Imaging: Fluorescein angiography shows the typical features of choroidal neovascularization, ie, early lacy hyperfluorescence with late leakage.

Management: intravitreal bevacizumab.

Punctate inner choroidopathy (PIC)

is an idiopathic inflammatory disorder of the choroid which was first described by Watzke et al in 19841.

Etiology

The etiology has remained unclear with a wide spectrum of theories proposed. PIC was proposed to be a variant of multifocal choroiditis and panuveitis (MFCPU), a form of limited myopic degeneration or a variant of Multifocal Choroiditis (MFC). Other theories have proposed an inflammatory or infectious thrombosis of the choriocapillary layer by as of yet an unidentified organism. A previous study suggested an association betwee

Presentation: (90%) female 18 - 50 y/o presents with bilateral (80%) blurred vision floaters, photopsias, possible metamorphopsia (vision 20/50 to 20/400)

Ocular findings: Bilateral white-yellow chorioretinal lesions usually 100-200 microns diameter develop at the level of the inner choroid and retinal pigment epithelium (RPE) which rarely extend to the midperiphery

NO vitritis. Lesions 80% bilateral and progress to atrophic scars that appear punched-out.

Choroidal neovascular membrane 40 to 75% of cases

Pathophysiology: Unknown but thought to be inflammatory (Light and electron microscopy of the CNVM showed lymphocytes at the level of the inner choroid with sparing of the choriocapillaris.)

Diagnosis: Hx exam and FA

Fluorescein angiography: Early hyperfluorescence, variable late leakage/staining of acute lesions, leakage in presence of cystoid macular edema (CME) and choroidal neovascular membrane (CNVM).

PIC lesions are hyperfluorescent in the early arterial phase, with staining observed in the arteriovenous phase. In some cases the lesions blocked fluorescence in the early arterial phase and stained thereafter.

More lesions were seen on FA than on clinical examination. As the disease progresses damage to the RPE occurs and FA demonstrates punctate RPE window defects. Leakage of fluorescein into the subretinal space was

observed in patients with a serous neurosensory retinal detachment1. Descriptions of both the pathology and clinical features of CNVMs in PIC have also been reported. Olsen et al described the FA characteristics

in 6 eyes. PIC CNVMs appeared as focal areas with an irregular, lacy network of neovascularization, with hyperfluorescence in the early phase and leakage in the late phase. Over time the newer vessels linked to

form a larger neovascular complex with multiple feeder vessels originating from individual neovascular buds. The subsequent fibrotic response lead to a dumbbell-shaped pattern of subretinal fibrosis10.

Early transit of the fluorescein angiogram shows early hyperfluor- escence of the CNV lesion.

Indocyanine green (ICG):

Multiple midphase hypofluorescent lesions in the peripapillary posterior pole, corresponding to those seen on exams. ICG is a useful tool in the diagnosis of PIC.

It has been reported to show subclinical hypofluorescent spots in 32% of affected eyes, thereby increasing the diagnostic potential in patients who have evaded clinical diagnosis11.

Tiffin et al described unusual abnormalities of the choroidal vasculature in PIC12. Several areas of obvious hypofluorescence corresponded to the site of the visible subretinal lesions;

larger choroidal vessels were noted to cross these areas. In addition, several choroidal vessels demonstrated localized points of hyperfluorescence situated close to the vessel wall/ border.

The authors suggested that the hypofluorescent areas corresponded to localized choroidal hypoperfusion, whereas the localized points of hyperfluorescence on the vessel walls might indicate an associated vasculitis. The presence of larger choroidal vessels running through the hypofluorescent areas could imply that the vasculitic process is confined to smaller choroidal vessels and the choriocapillaris12.

Early phase of the fluorescein angiogram reveals hyperfluorescence corresponding to the CNV lesion with hypofluorescent borders corresponding to the pigmented borders of the lesion.

Electrophysiology : normal ERG usually but abnormal EOG (mild abnormality of arden ratio due to involvement of rpe)

Visual fields: enlarged blind spot.

OCT: homogenous thickening of retina above lesions that improves with disease improvement.

Laboratory test: none except histo is negative.

Differential diagnosis includes Acute Posterior Multifocal Plaquoid Pigment Epitheliolopathy, Behcets disease, Harada disease, Leukemia, Myopic degeneration, Multiple evanescent white dot syndrome (MWEDS),

Pars planitis, Presumed ocular histoplasmosis, Sarcoidosis, Sympathetic ophthalmia, Serpiginous choroiditis, Vogt-Koyanagi-Harada disease or Whipples disease.

treatment: usually none unless there are lesions close to fixation or there is cnv.

Systemic corticosteroids have been used alone or indeed combined as part of a multimodal approach. The usual starting dose is 1 mg/kg (60- 80 mg oral daily) for 3-5 days and subsequently tapered16.

Lesions may show a marked improvement however this may be without an improvement in visual acuity due to CNVM formation and subsequent subfoveal fibrosis17.

One case report showed the value of oral steroids in a 28 year old pregnant female with PIC after intravitreal lucentis and PDT have failed to arrest disease progression18. Interestingly one would expect that inflammatory activity of PIC or other autoimmune inflammatory diseases would be suppressed during pregnancy and exacerbated in the postpartum period19, 20. A case report by Rao et al demonstrated a flare up of choroiditis in the first trimester18.

The multimodal approach to treatment has also been used in the management of PIC. One such study examined 5 patients treated with PDT combined with oral prednisolone (1 mg/kg body weight/day) which was started 5 days before PDT over a 12 month followup period and found a mean improvement in vision of 15 letter following a mean of 2 PDT treatments21.

Intraocular corticosteroid implants and injections

Intravitreal triamcinolone

One of the more commonly used methods of administration has been the intravitreal injection of 4 mg of triamcinolone. One recent retrospective study studied fourteen patients (14 eyes) over 12 month follow-up who had PIC and idiopathic CNVM. Patients were treated with combined intravitreal triamcinolone (4 mg) and PDT. The mean logMAR BCVA improved significantly from 0.52 at baseline to 0.20 at 1 year (Wilcoxon signed-ranks test, P = 0.003)22.

Intravitreal dexamethasone implant

More recently an intravitreal implant containing 0.7 mg or 0.35 mg of dexamethasone for posterior uveitis releases the medication over a 6 month period. The implant is biodegradable (containing poly D, L- lactide-co- glycolide polymer (PLGA) matrix) and is administered via a 22-gauge applicator. A recent multicenter trial examined the use of the dexamethasone implant (both 0.35 and 0.70 mg) in posterior and intermediate uveitis and found a significant improvement in degree of inflammation and visual acuity over a 6 month followup compared to sham treatment with a slightly higher incidence of raised IOP in both implants23.

Intravitreal fluocinolone acetonide implants

Injectable, non-biodegradable, intravitreal implants containing 0.59 mg of fluocinolone acetonide releases its contents over 36 months. The medication is released at a nominal initial rate of 0.6 g/day, decreasing over the first month to a steady state between 0.3-0.4 g/day over approximately 30 months. The cylindrical device is 3.5 mm in length and 0.37 mm in diameter and injected into the vitreous cavity using a 25-gauge needle. Recently the results of the the MUST trial (Multicenter Uveitis Steroid Treatment trial) were published. This was a multicenter trial across the United States examining the effectiveness of standardized systemic therapy versus the fluocinolone acetonide implant therapy for the treatment of severe non-infectious intermediate, posterior uveitis or panuveitis. Although this in theory includes PIC cases, the specific diagnoses were not discussed in the results. It reported that neither treatment were superior to the other with a detectable degree of power in terms of visual acuity, quality of life or degree of inflammation24. Conversely another study in Europe examined the effectiveness of an intravitreal fluocinolone acetonide implant versus standard systemic therapy in noninfectious uveitis and found intravitreal injections were superior with no treatment-related side-effects compared with standard of care25.

Mycophenolate mofetil

Mycophenolate mofetil suppresses the immune system by selectively inhibiting the purine biosynthesis enzyme inosine monophosphate dehydrogenase (IMPDH), thereby resulting in depletion of guanosine nucleotides that are essential for purine synthesis used in the proliferation of B and T lymphocytes26. Mycophenolate mofetil has has been shown to decrease the frequency of attacks in recurrent PIC. This was used in conjunction with fundus autofluorescence to monitor and predict the response to treatment27. Other multicenter studies have examined its role in uveitis and found it was effective in approximately 50 % of all patients it was used in 28. This study did not specifically divide its participants into diagnostic categories, it examined patients with anterior uveitis (20.3%), intermediate uveitis (11.9%) and posterior uveitis or panuveitis (39.8%).

Thalidomide

Thalidomide has little role in the treatment of CNVM due to PIC although one case report by Ip et al showed that it failed to prevent a recurrence of a choroidal neovascular membrane in a 38 year old patient with bilateral CNVM secondary to PIC29.

Sirolimus (rapamycin)

Sirolimus is a macrolide antibiotic and potent immunosuppressive agent and was first discovered as a product of the bacterium Streptomyces hygroscopicus in a soil sample sample from Easter Island- an island also known as Rapa Nui. Its mode of action involves inhibiting the

binding of the cytosolic protein FK-binding protein 12 (FKBP12) and thereby inhibiting the secretion of IL-2. It has been reported to be used successfully in a patient with juxtafoveal PIC-associated CNVM30.

Interferon B-1A

One study reported the resolution of disease activity following the treatment of chronic recurrent PIC with interferon B-1A31. There has been scant reports on this specific modality of treatment for PIC in the literature.

Intravitreal bevacizumab and ranibizumab

Several case series have reported the successful treatment of CNVM with anti-VEGF treatments32, 33. Although anti-VEGF agents have not been examined in pregnant patients with PIC, it has been successfully used in the treatment of CNVM with good results34. Rouvas et al followed a cohort of 16 patients including 5 with PIC over a period of 70 weeks following intravitreal injection of ranibizumab35. They found an improvement in mean foveal thickness and visual acuity as well as significant regression in CNVM over the course of the study35. It remains to be seen whether the advent of VEGF-TRAP holds the key to widening the anti-VEGF spectrum for the white dot syndromes, including PIC. Without treatment CNV is inevitably progressive36, 37.

Photodynamic therapy

Several reports have substantiated PDT as an effective treatment option in extrafoveal or juxtafoveal CNV due to PIC. PDT has been advocated as a viable option if outcome without treatment is likely to be poor, and preliminary success in ocular histoplasmosis syndrome, angioid streaks, idiopathic, and other conditions has been reported38, 39, 40, 41, 42, 43, 44, 45. With the widespread use of anti-VEGF treatment its role continues to decline. Studies of subfoveal CNVs which had failed to improve with a single dose of immunosuppressive therapy showed an improvement in visual acuity after they were treated with PDT46. A multimodal approach using a combination of PDT and intravitreal triamcinolone have also been used for the treatment of CNV47. This was described in a cohort of 15 patients who showed a significant improvement in visual acuity at 3 and 6 months but a worsening at 12 months47. Although PDT can be useful in selective circumstances, its role remains limited in CNV secondary to PIC.

Medical follow up[edit source]

Patients are followed at periodic intervals by a uveitis/retinal specialist depending on level of inflammation/pathology.

Surgery[edit source]

Submacular translocation surgery Although currently submacular translocation surgery is no longer advocated for ARMD related CNVM, recent studies have examined its use in a cohort of patients with progressive use from non-ARMD submacular diseases including PIC. They primarily examined final visual acuity and found a large percentage of subjects gained >3 lines of visual acuity (38%) and achieved a final visual acuity of = 20/50 (31%) over a mean followup of 28 months48. The submacular surgery trial examined a cohort of patients following submacular surgery and recurrent CNV developed in 58 % of patients. One recent publication examined the ultrastructural and pathological features of CNVMs in PIC in a patient with PIC who initially had intravitreal bevacizumab followed by submacular surgery when this failed50. This study noted recurred in on eye of a PIC patient with bilateral CNVMs who had submacular surgery in both eyes. This was consistent with the study by Olsen et al in which four out of six eyes developed a recurrence of CNV following surgical excision10.

Surgical follow up[edit source]

Close follow up after surgical intervention is necessary. Patients should be monitored for recurrence of disease.

Complications[edit source]

CNVM as well as subretinal fibrosis can develop leading to poorer visual outcomes.

Prognosis[edit source]

Visual prognosis is good in the absence of CNVM with 50-75% of eyes having visual acuity better than 20/25. The course is usually self-limited with recurrences common, usually in the first 3 months 49. The two major causes of visual loss are CNVM and subretinal fibrosis. One study of 136 patients noted CNVM in 74 (66%) of cases. In eyes with choroidal neovascularization, the mean logMAR visual acuity was 0.63 at study entry, 0.63 at 12 months, 0.61 at 2 years, and 0.71 at final review (mean, 6.1 years). Brown et al reported a cohort with a mean length of followup of 51 months. The final average VA was 20/40 or better in 77% of eyes (23 eyes) and 20/50 or worse in 23% (7 eyes). In 20% of eyes (6 eyes) it was 20/200 or worse8.

White Dott Syndromes

Kourosh Mohammadi M.D.

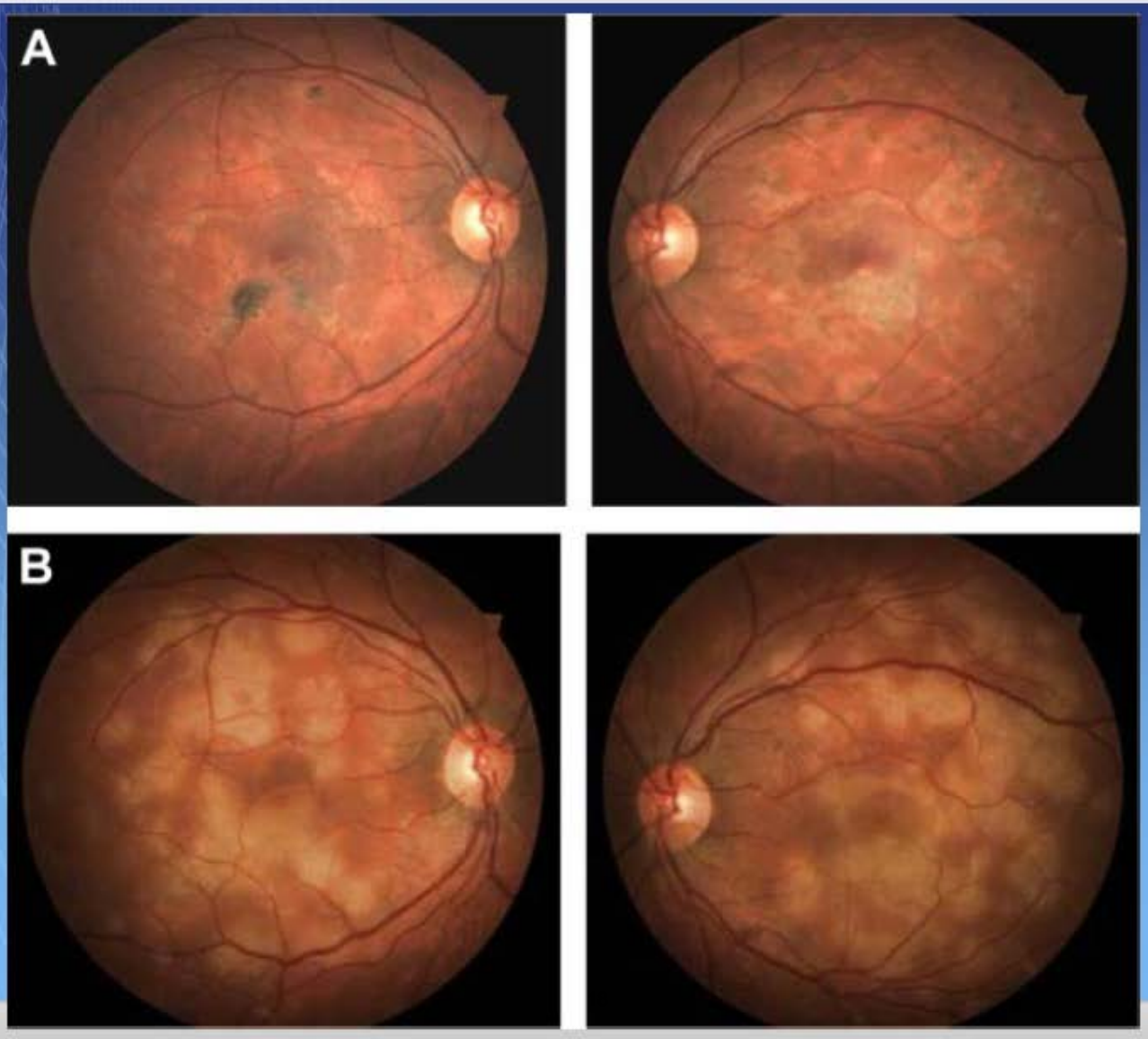
SCPMG Mammoth Symposium 2017

Overview of White Dott Syndromes

- Inflammatory Choroidopathy Of Unknown Origin
- White inflammatory lesions in Choroid, RPE, and/or retina
- Variable Prognosis
- Some require no treatment
- Some require steroid and immunomodulators
- Same may be variable phenotypic manifestations of same disease

Case Number 1

- 29y/o patient presents with 2 days of cold symptoms and sudden bilateral decreased vision.
- On exam:
- 20/200 ou
- Few Vitreous Cells ou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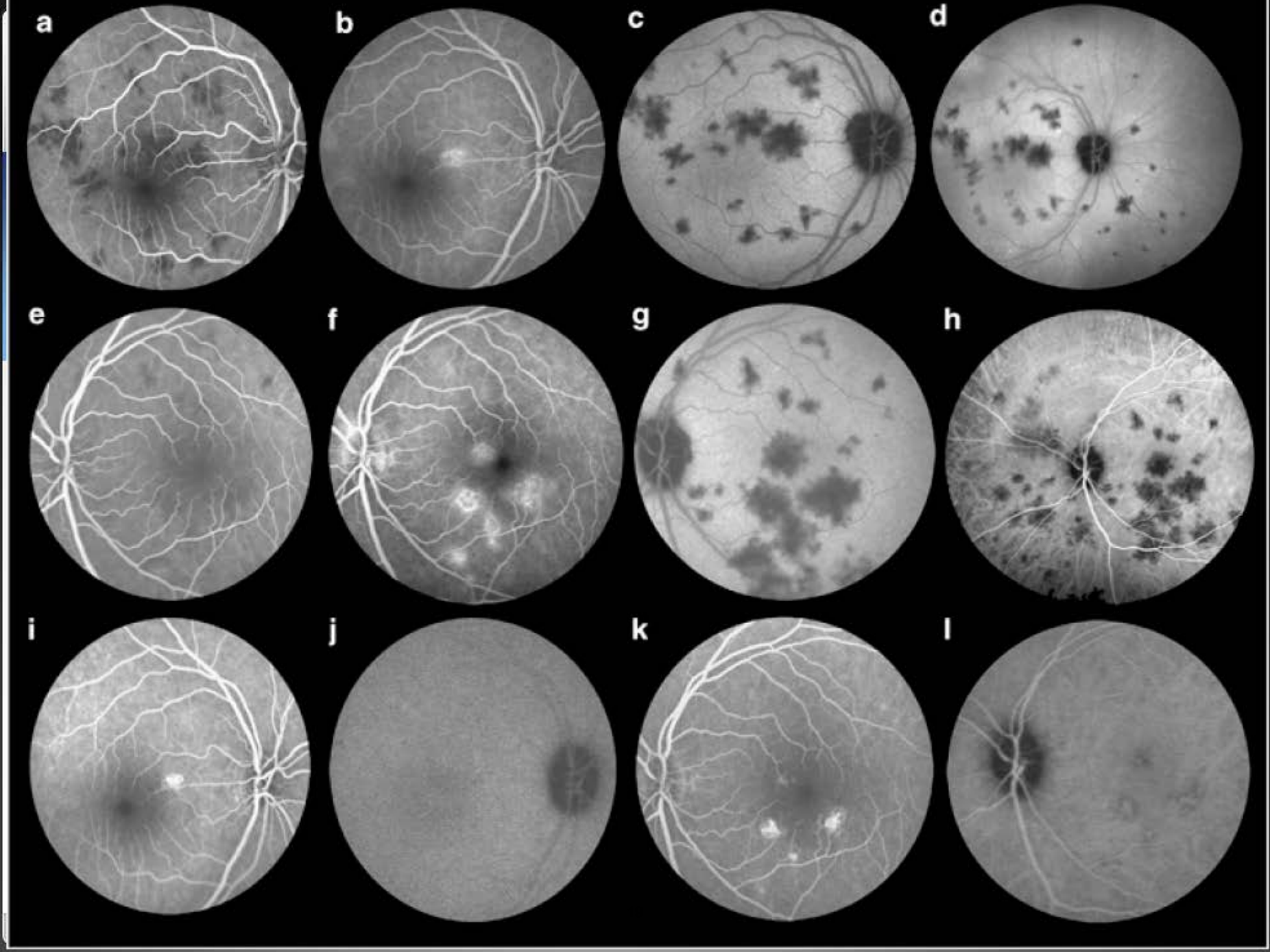
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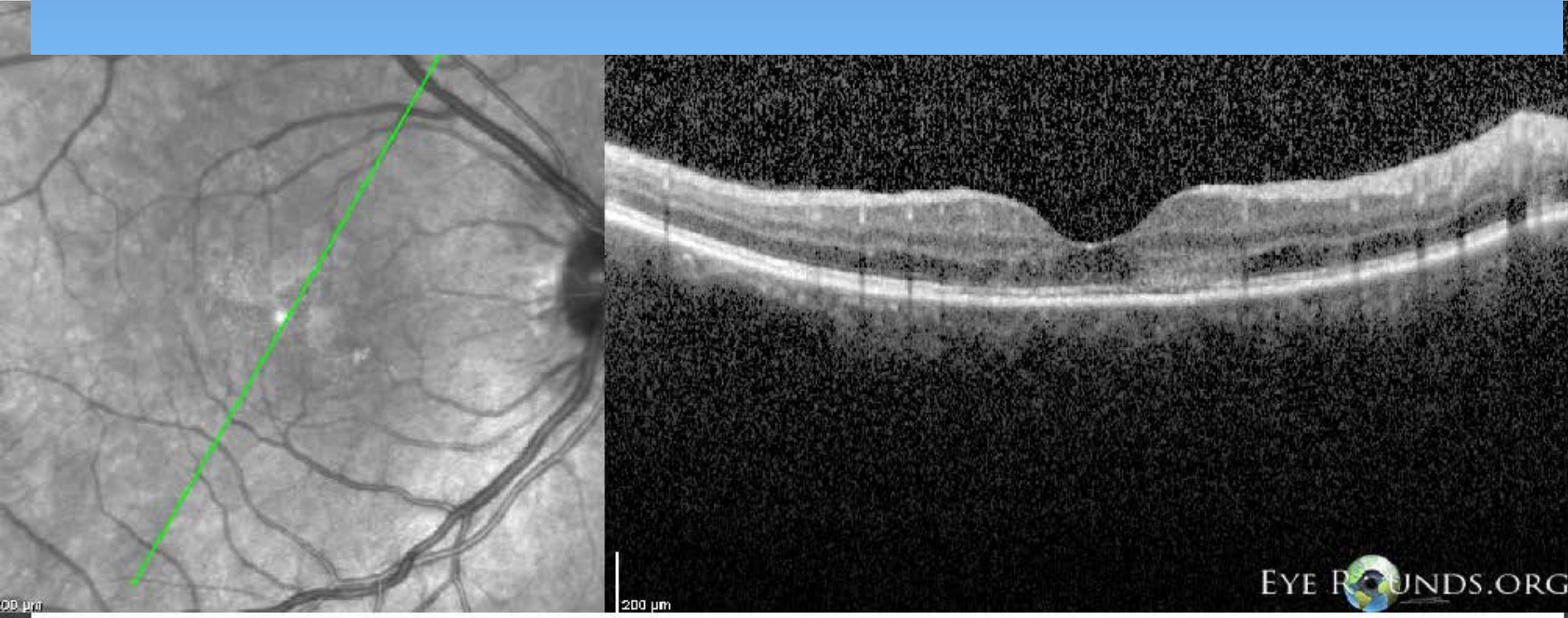
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Acute posterior multifocal placoid pigment epithliopathy (APMPPE)

PRESENTATION:

Healthy adult with viral or meningeal prodrome

Sudden vision loss 20/40 to CF

Exam:

+ Anterior Cell

50% Vit cell

Large Placoid Lesions

APMPPE Diagnostic Studies

- fluorescein angiographic findings:
  - early hypofluorescence with late hyperfluorescent staining of the placoid lesions
  - 3 months later shows hyperfluorescent scars
- Indocyanine green angiography
  - Hypofluorescent spots corresponding to the placoid lesions.
  - 3 months later decreased hypofliuorescent spots
- OCT
  - Disruption of outer retina layers
- EOG: abnormal
- EEG: abnormal

APMPPE Treatment

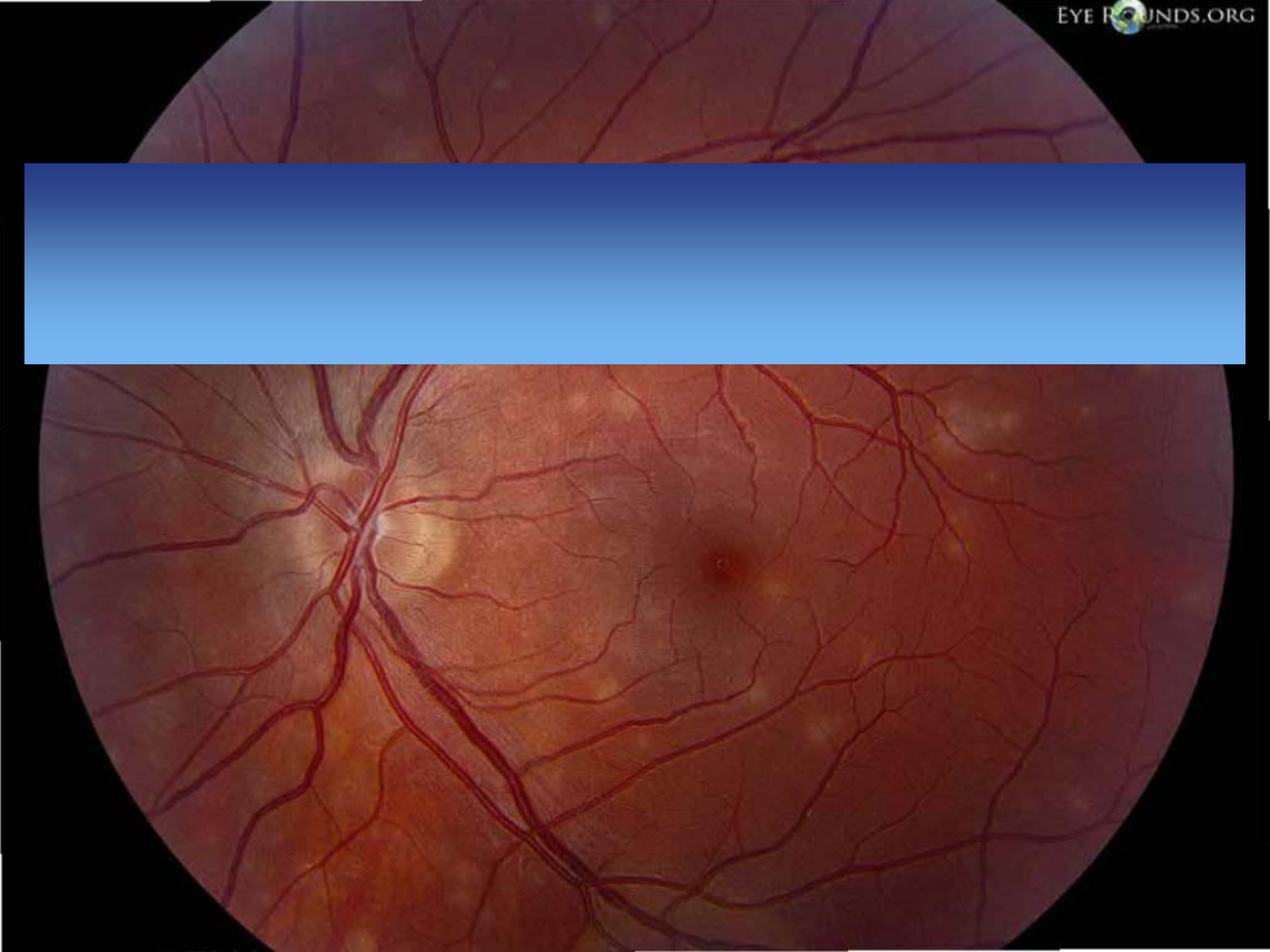
- resolves spontaneously in two to 12 weeks and does not usually require treatment
- sometimes systemic corticosteroids may speed resolution of the placoid lesions and/or serous detachment.

APMPPE Continued

- Differential diagnosis: metastatic tumors, viral retinitis, toxoplasma retinochoroiditis and pneumocystis choroiditis, Serpigenous Choroiditis, other white dot syndromes
- Adenovirus type 5 infection has been serologically documented in patients with APMPPE.
- Histopathology not been studied
- lesions thought to occur at the level of the retinal pigment epithelium or the choriocapillaris.
- Other systemic associations: meningoencephalitis, nephritis, and hearing loss
- -Prognosis:
  - usually full recovery within 2-12 weeks
  - 80% Better than 20/40
  - Can be about 20/200 in patients with Foveal lesions

Case number 2

- 26 y/o Female noted painless central scotoma in left eye noticed while reading last week which has not improved
- VA: pd 20/20 os 20/50 phni
- Anterior chamber DQ
- 1+ Vitreous Cell
- 100-200 micron Retinal White Lesions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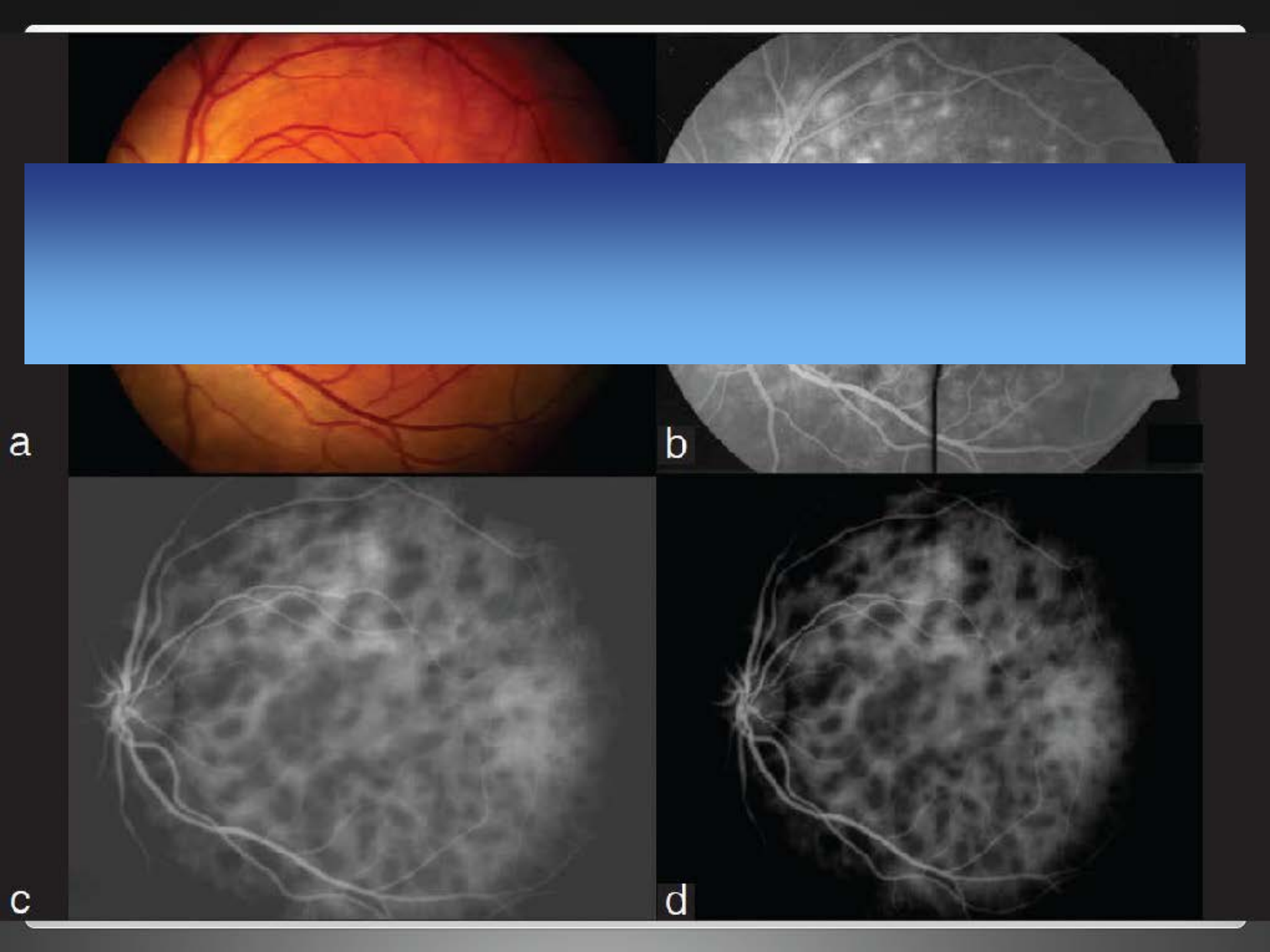
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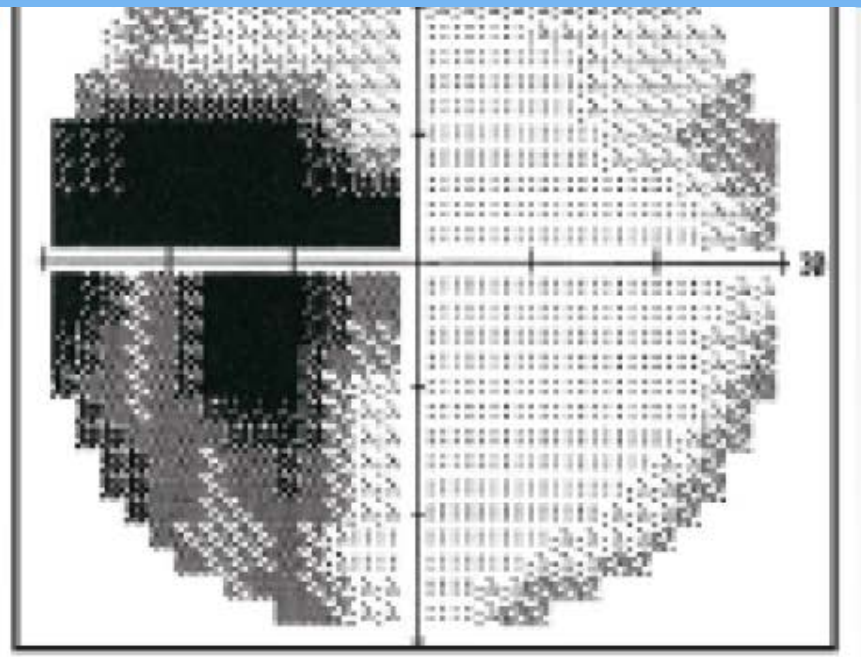
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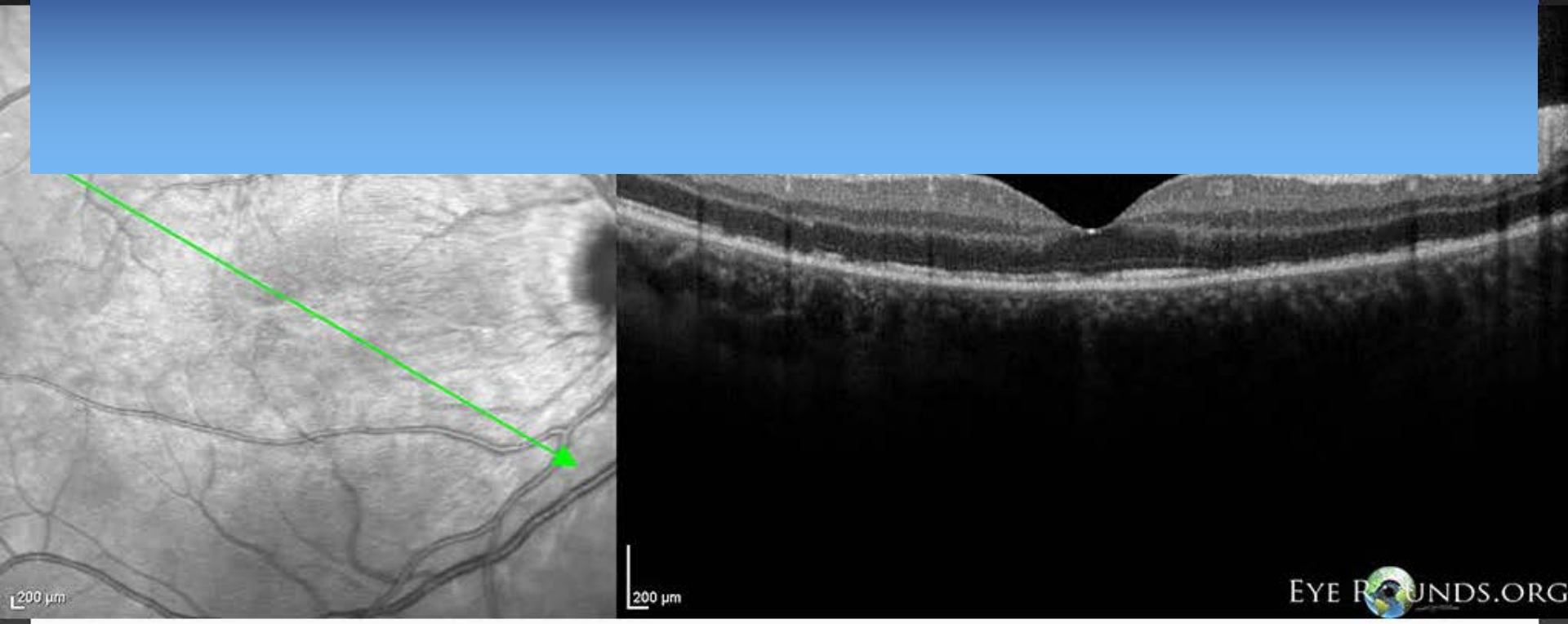
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Multiple Evanescent White Dot Syndrome

- Presentation:
- 20-40 y/o healthy female (F>M 4to1)
- Unilateral painless vision loss, temporal paracentral scotoma, possible shimmering lights in periphery
- unilateral inflammatory chorioretinopathy that
- half have viral prodrome prior to disease onset.

+ vitreous cell, optic disc edema and characteristic multiple white spots (10-100 microns) at the level of the retinal pigment epithelium or deep retina in the posterior pole. The fovea has a characteristic granular appearance in the acute phase of the disease which may remain after resolution of disease.

Diagnostics

- Visual field testing: enlarged blind spot.
- Fluorescein angiography often reveals punctate staining of the pigment epithelium in the posterior pole and disc leakage. The punctate staining is sometimes in the shape of a wreath.
- ICG angiography shows multiple hypofluorescent areas in the posterior pole.
- ERG: Reduction of a-wave amplitude on the electroretinogram in acute phase of disesae which normalizes after resolution of disease.

Differential Diagnosis

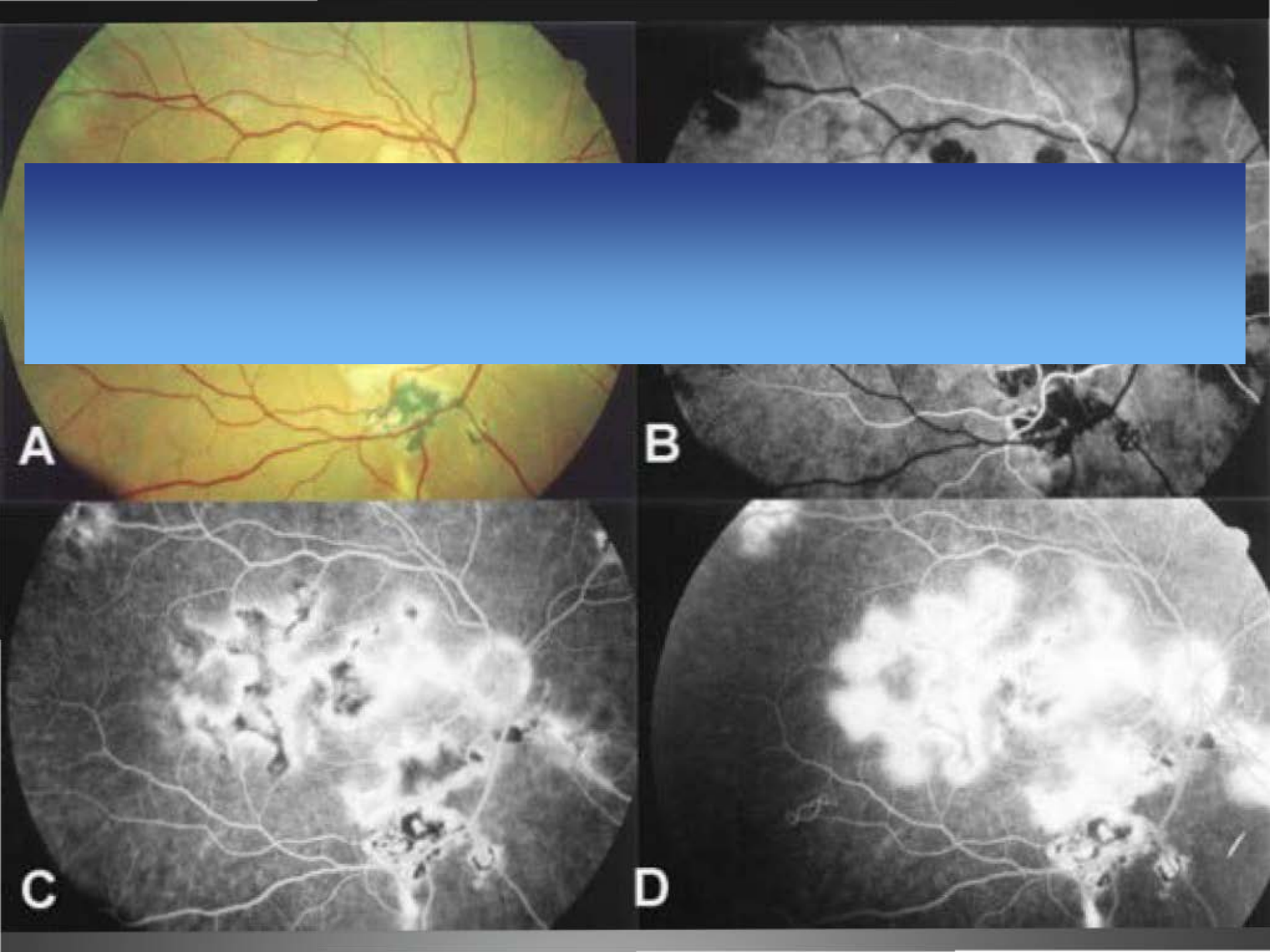
- Differential diagnoses includes APMPEE, acute macular neuroretinopathy, multifocal choroiditis panuveitis, birdshot retinochoroidopathy, diffuse unilateral subacute neuroretinitis, primary intraocular lymphoma and sarcoidosis. It is felt that idiopathic blind spot enlargement, MEWDS and MCP may represent a spectrum of the same disease process.

Prognosis / Treatment of MEWDS

- self-limited course
- no specific treatment is required.
- The white dots and disc edema fade within two to six weeks,
- visual symptoms such as the enlargement of blind spot, temporal scotoma, and photopsias may take several months to resolve.
- Recurrence 10 to 15 percent of patients.15,22
- Prognosis for patients with recurrences is also excellent.22
- Choroidal neovascularization is a rare late complication of MEWDS.23

Serpiginous Choroiditis (geographic helicoid choroidopathy)

- Presentation:
- 45 y/o patient (male or female) presents with scotoma noticed in central or paracentral visual axis over last few weeks and it is not improving.
- No other symptoms.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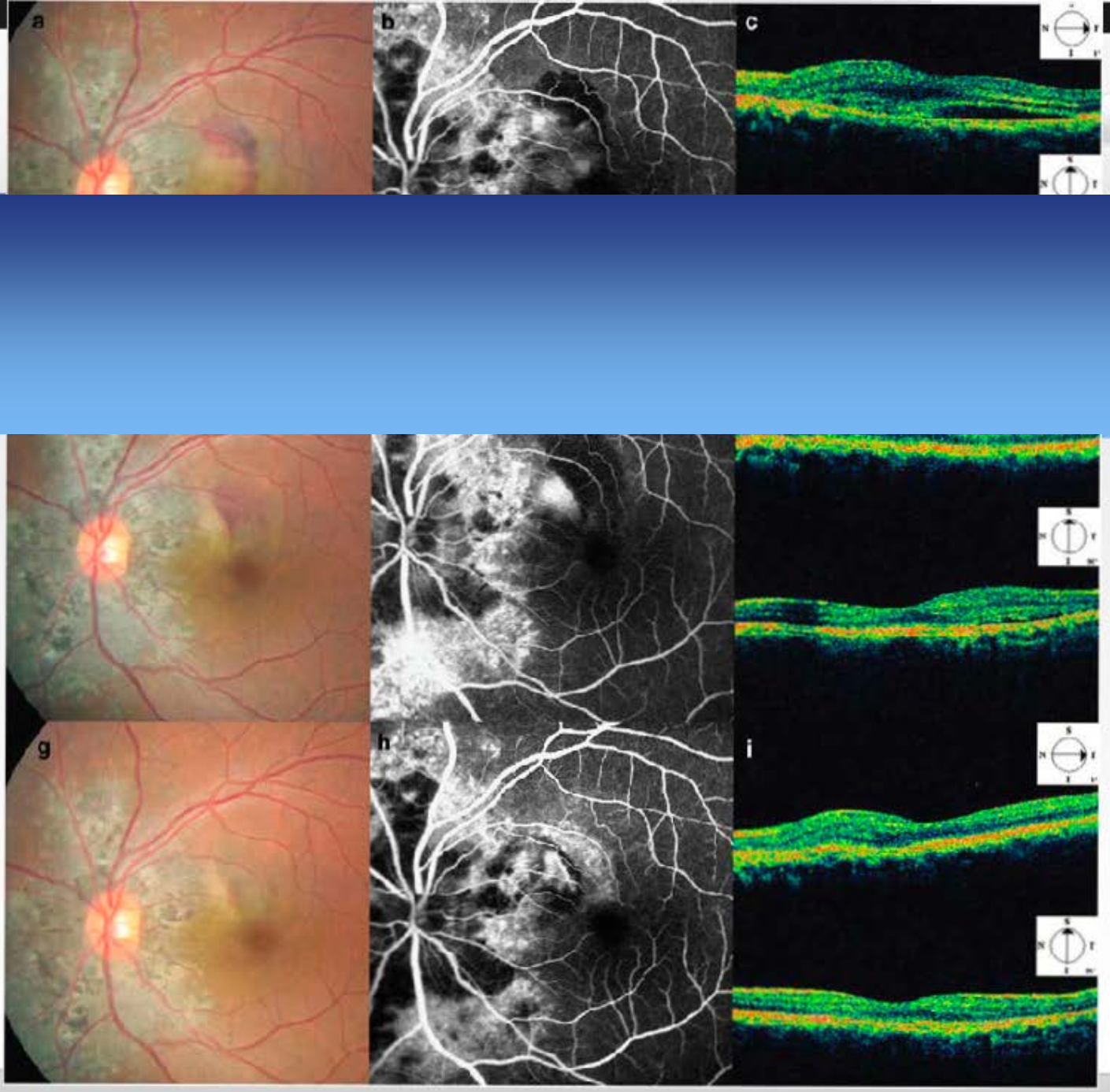
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Serpiginous Choroiditis (geographic helicoid choroidopathy)

- Healthy 20-70 y/o male or female
- painless onset of paracentral scotoma and vision loss
- asymmetric but usually becomes bilateral over time.
- No AC reaction, 30% have vit cell
- Chorioretinal lesions are grey-white and associated with retinal and RPE edema when active .
- Lesions typically start in the peripapillary region and move in a serpiginous or snake-like fashion around the posterior pole and progress to involve the macula gradually over time

Serpiginous Choroiditis (geographic helicoid choroidopathy) Cont

- course: The disease process has a step-wise, progressive course. Typical new lesions of serpiginous choroiditis appear contiguous to old atrophic ones.
- A macular variant of serpiginous choroiditis has also been described with active lesions initially occurring in the macula without peripapillary involvement.
- Subretinal hemorrhage and subretinal fluid is characteristic of choroidal neovascularization that complicates up to one-third of patients with serpiginous choroiditis.

Diagnosis of Serpiginous Choroiditis

- Diagnosis is based on typical clinical appearance and fluorescein angiography.
- Fluorescein angiography demonstrates early hypofluorescence and late hyperfluorescence of active lesions.
- some patients have had a history of pulmonary tuberculosis or were purified protein derivative skin test positive. But treatment with antituberculous agents does not impact the course of serpiginous choroiditis.
- Tuberculous choroiditis can indeed look identical to serpiginous choroiditis.30

Differential Diagnosis

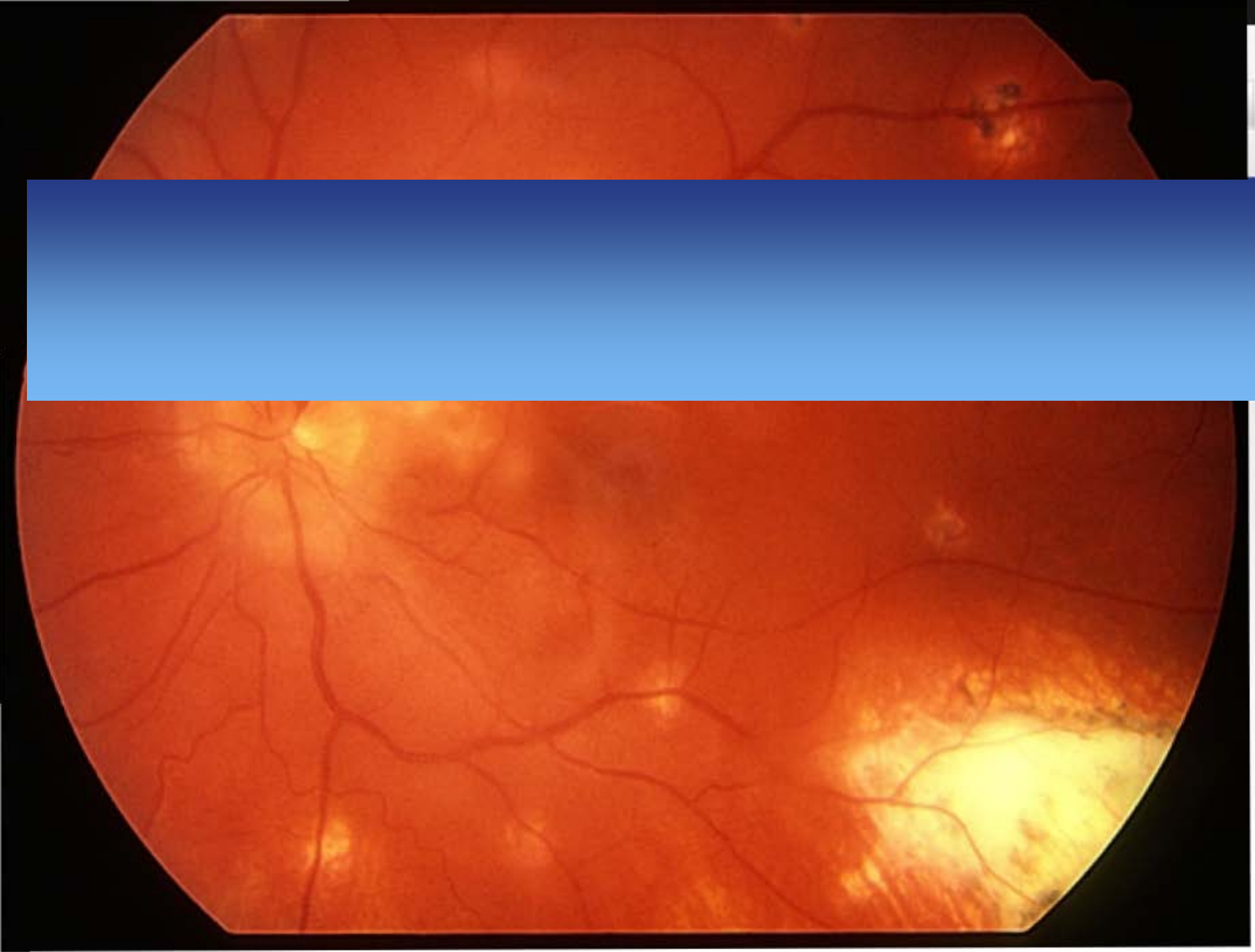
- tuberculous choroiditis, APMPPE, relentless placoid chorioretinitis, multifocal choroiditis and panuveitis, birdshot retinochoroidopathy and ocular histoplasmosis syndrome.

Treatment / Prognosis of Serpigenous Choroiditis

- Treatment:
  - Anti-VEGF can be considered if there is CNVM
  - PDT can be considered for extrafoveal lesions
  - Systemic and local steroid as well as immunosuppressives have been tried but no convincing data.
- Prognosis:
  - Poor
  - Progressive
  - Bad if there is cnvm or fovea involved

Case #3

- 33 y/o female presents with bilateral blurred vision 20/50 ou for few weeks
- + floaters, + photopsias, + scotomas
- + inflammation in the AC and Vitreous
- multiple yellow or gray lesions at the level of the choroid and retinal pigment epithelium 50-1000 µm and can be numerous (as many as several hundred at a time).



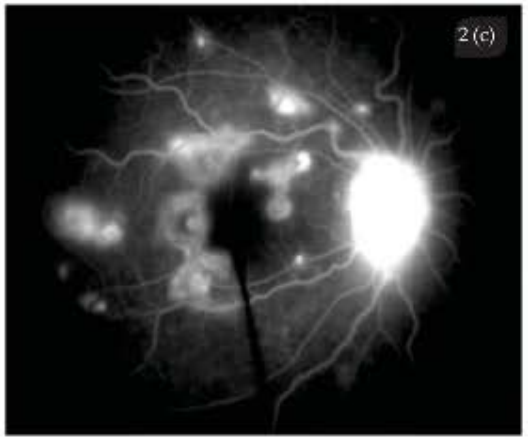


Figure 2 a: showing fundus photo withcreamy placoid lesions of APMPPE
2 b: FFA of APMPPE early staged showing hypofluorescence the lesions
2 c: FFA of APMPPE lakstage showing hypofluorescence the lesions

Multifocal Choroiditis and Panuveitis (MCP)

- Demographics: myopic females 20-60y/o life, with a mean age of onset of 33 years.
- Symptoms: Acute onset of bilateral (75%) blurred vision, photopsias, and scotomata.
- Ocular findings:
  - 52% AC reaction, and 98% Vitreous Cell
  - multiple yellow or gray lesions at the level of the choroid and retinal pigment epithelium 50-1000 µm and can be numerous (as many as several hundred at a time).

Diagnostics

- FA: early hypofluorescent lesions (which correspond to the fundus lesions but are typically more numerous) with late hyperfluorescent staining. Healed lesions reveal transmission window defects.
- If patients present at a later stage, the active lesions usually have scarred or are in the process of scarring, thereby giving early hyperfluorescence and late staining. If choroidal neovascularization is present, it usually is observed as early hyperfluorescence with a lacy appearance
- ICG angiography shows both active and chronic lesions as hypofluorescent. ICG angiography of choroidal neovascularization reveals hyperfluorescence.
- Imaging findings suggest choroidal choriocapillary perfusion abnormalities with secondary involvement of the RPE & photoreceptors

Differential diagnosis MCP

- POHS. The lesions in POHS are less numerous and smaller. But POHS does not present with anterior segment and vitreous inflammation. sarcoidosis and birdshot retinochoroidopathy

MCP Treatment

- -- Treatment: Oral steroids are helpful in patients with active posterior segment inflammation or with cystoid macular edema. Topical corticosteroids are helpful if there is severe anterior segment inflammation.
- However, cases in which corticosteroids have not improved the inflammation have been described.

MCP Complications

- -- Complications: choroidal neovascularization, which develops in 30% of patients. Laser photocoagulation or intravitreal bevacizumab may be indicated in these patients. Oral steroids also may be used for
- choroidal neovascularization since they have been shown to decrease the neurosensory detachments associated with choroidal neovascular membranes.

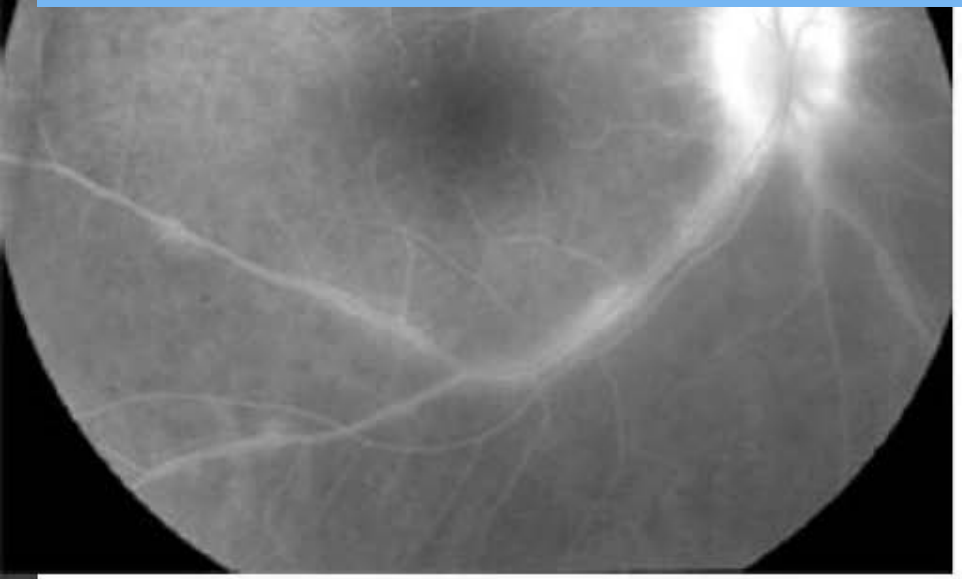
MCP Facts

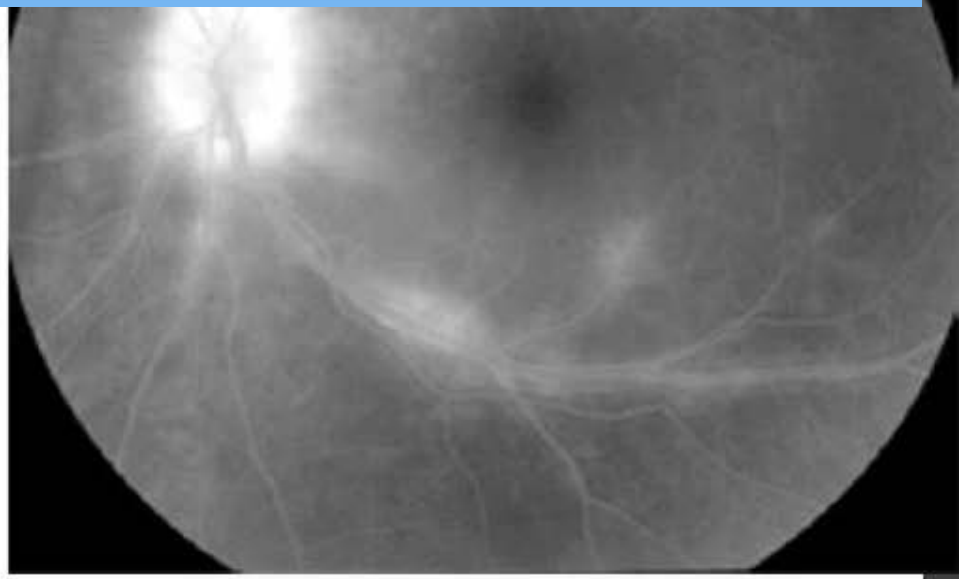
- Pathogenesis: unknown, ? association with EBV
- Prognosis is variable, with final visual acuity of 20/40 or better in 66% of eyes.
- Related conditions:
  - PIC (acute bilateral loss of vision and photopsias and scotoma in young myopic female) but lesions in PIC are smaller and PIC has no AC or Vitreous inflammation and rarely have recurrences.
  - DSF (diffuse subretinal fibrosis): In addition to the presence of multifocal choroiditis, a prominent fibrosis exists. The fibrosis is predominantly at the area of previous inflammatory lesions, and a turbid, subretinal fluid that overlies the lesions also is present. Rare with poor prognosis.
  - PIC and DSF represent a spectrum of disease as it relates to MCP. PIC = milder form of disease, DSF = more severe form.

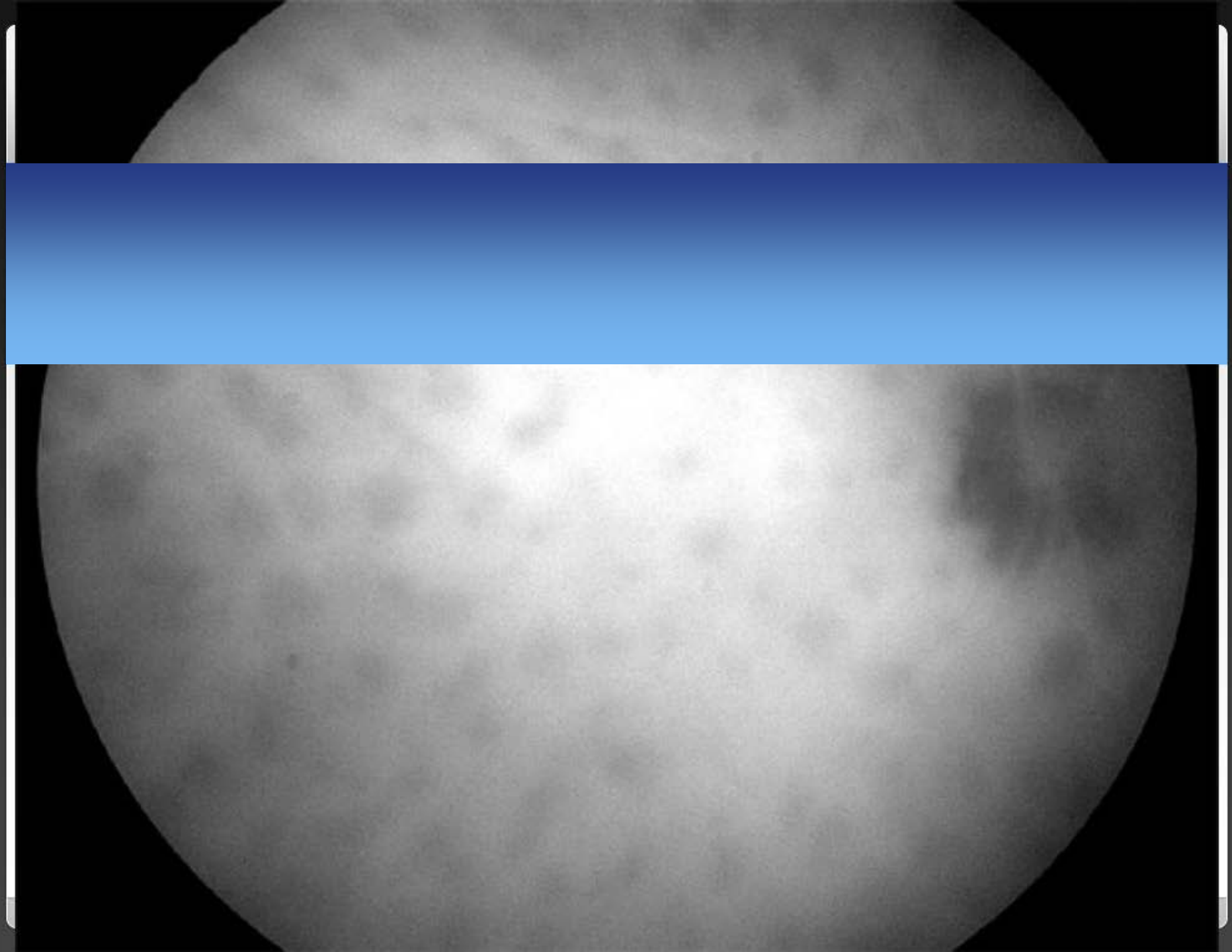
Case Presentation

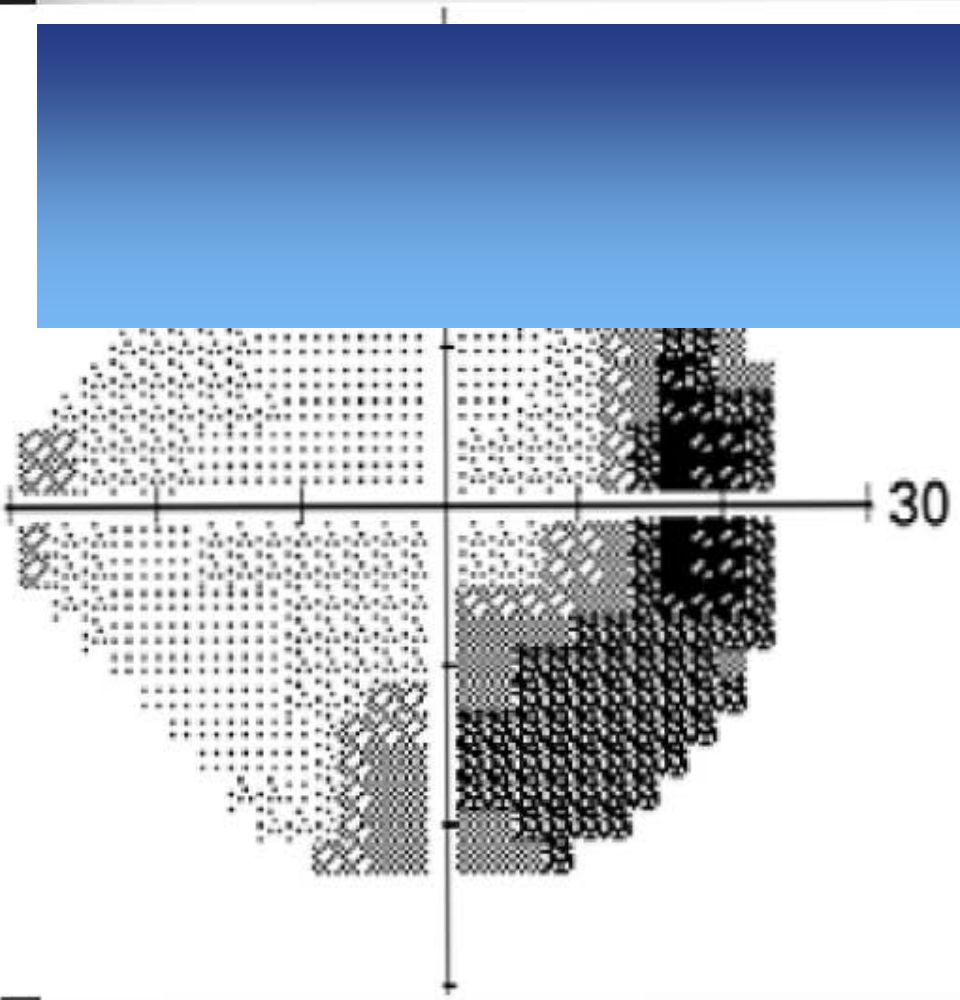
- Presentation:
  - 50 y/o female p/w 1 week of blurred vision, floaters, nyctalopia, achromatopsia, glare and photopsia
- Exam:
  - (30%) ac reaction
  - (100%) vitritis
  - multiple depigmented yellow-white patches scattered throughout the fundus radiate from the optic nerve and follow the larger choroidal vessels, +/- optic disc edema, and +/- cystoid macular edema

Fig. 5. Birdshot chorioretinopathy. *A:* Older birdshot chorioretinopathy lesions show widespread depigmented scarring, optic atrophy, and vascular attenuation. *B:* Fluorescein angiogram shows hypofluorescent lesions with polycystic macular edema. *C:* Indocyanine green angiography shows hypofluorescent lesions.





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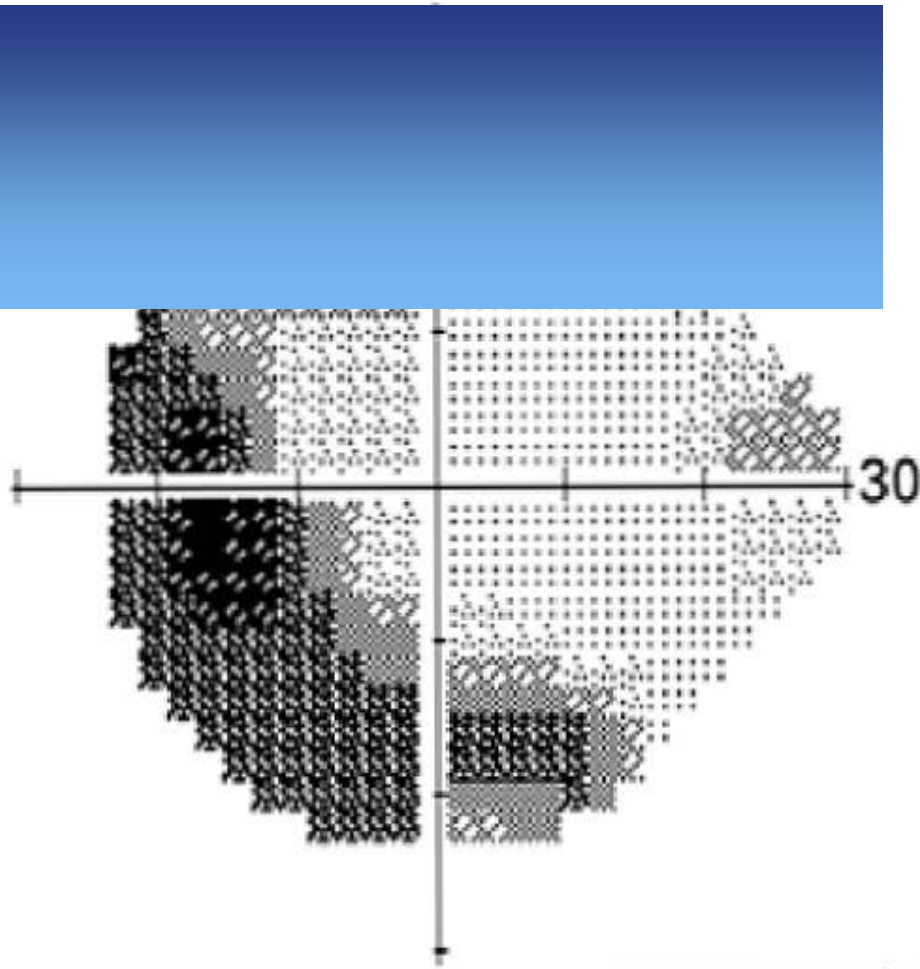
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Birdshot Retinochoroidopathy (vitiliginous choroiditis)

- > 90% of patients with birdshot chorioretinopathy are HLA-A29 positive.
- Age 35 to 70 (avg 50)
- Ocular findings: multiple depigmented yellow-white patches scattered throughout the fundus radiate from the optic nerve and follow the larger choroidal vessels.
- + Vitritis, +/- AC reaction, optic disc edema, and cystoid macular edema
- Diagnosis" is by clinical examination, fluorescein angiography, and HLA-A29 status.
- Fluorescein angiography: demonstrates mild hyperfluorescence and staining in the late phase.
- The pathogenesis is unknown at this time; however, speculation exists regarding an autoimmune etiology.

Treatment / Prognosis

- Treatment: Ocular and systemic corticosteroids are generally the treatment of choice.
- Prognosis: Birdshot retinochoroidopathy is a chronic disease with multiple recurrences, and, guarded visual prognosis

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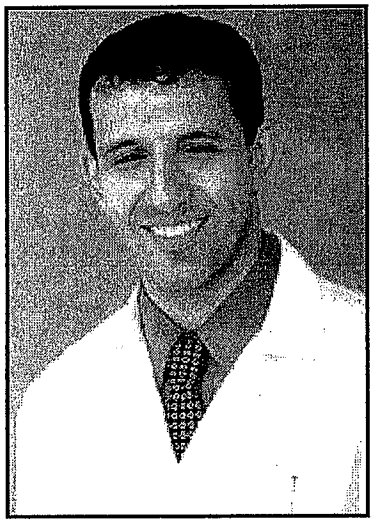
Dr. Mohammadi



San Diego LASIK Surgeon Education and Work History:

Dr. Mohammadi is a graduate of Duke University and the University of California San Diego School of Medicine. He attended Duke University from 1995 to 1999 during which time he continuously remained on the dean's list with distinction. While studying Public Policy at Duke University in 1998, his interest in public health drove him to draft a policy recommendation that earned a grant to support his hometown San Pedro California's Harbor Free Clinic. He graduated Magna Cum Laude in 1999 from Duke University and entered medical school at the UCSD School of Medicine. His strong fascination with eyesight drove him to start research in ophthalmology during his first year in medical school. His ophthalmology research earned two separate prestigious NIH research grants and a number of publication in major medical journals and scientific meetings. He graduated from the UCSD School of Medicine with honors in 2004; finished his medical internship at Boston University; and entered sub-specialty ophthalmology and ocular surgery training at the UCSD Department of Ophthalmology in 2005. He completed his surgical training with world-renowned ocular surgeons at UCSD and entered private practice in 2008. After three years of private practice, he decided to accept a position as a clinical instructor at the UCSD Veterans Hospital where he serves veterans today and helps train young ophthalmic surgeons.

Philosophy of San Diego LASIK eye surgery center:

Dr. Mohammadi believes in providing all his patients with the best possible care at a reasonable price. As an author for some of the most prestigious ophthalmology medical journals himself, he constantly and critically reads recent scientific research and applies it to his day-to-day practice of medicine. Dr. Mohammadi believes that a doctor's office should be a caring environment where healthcare professionals provide care in the way they would want for themselves and their own family: with compassion and precision. He has a personal distaste for sales-focused medical practices. He takes pride in a conservative approach where safety of patients is valued above all else and no pressure or sales tactics are used to influence patient choices.

Interests:

Dr. Mohammadi lives a very active lifestyle similar to many of his San Diego LASIK patients. In his free time, he enjoys kiteboarding, tennis, bicycling, running, paddle-boarding, and basketball. He also meditates regularly which helps him maintain a strong focus and calm disposition during his life, practice of medicine, as well as during ocular surgery. He enjoys spending time with his family and also doing community service.

Curriculum Vitae:

Comprehensive Ophthalmologist and San Diego LASIK Surgeon

CITIZENSHIP: U.S.A.

LANGUAGES SPOKEN: English, Farsi, and Spanish.

SURGICAL EXPERIENCE:

- San Diego Laser Eye Surgery (PRK, LASIK, EPI-LASIK)
- Cataract Surgery (Including Restor, Tecnis, Toric, and other premium intraocular lenses)
- Blepharoplasty and Eyelid Surgery
- Pterygium
- Glaucoma Lasers and Photocoagulation
- Retina Lasers (Focal, Grid, Barrier, and PRP)
- BOTOX and Facial Fillers

EDUCATION:

- B.A. in Public Policy Studies, Duke University Durham, NC
- M.D. University of California San Diego, San Diego, CA
- Glaucoma Research Fellowship University of California San Diego, San Diego, CA
- Medical Internship Boston University Medical Center, Boston, MA
- Ophthalmology Residency University of California San Diego, La Jolla, CA

LICENSURE:

- Board Certified Ophthalmologist by the American Board of Ophthalmology
- California Medical License

SOCIETIES:

- Association for Research in Vision and Ophthalmology