PRIMARY eye care
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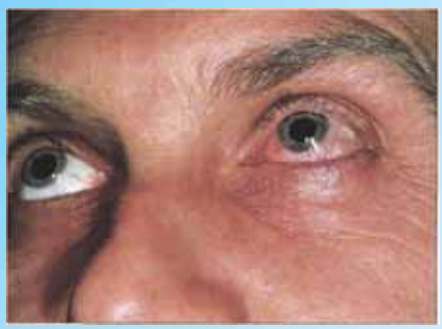
A common infection - could this be affecting the eyes of your patient?

Mr. LD is a 56 year old male from South Africa. He has lived in Wellington for a number of years now. For a week, the patient's left eye had been quite red and he was aware of a visual disturbance that he describes as "a band of blurriness" superior to central fixation in "a horizontal arc." The left eye vision seemed better when he looked off fixation.



There was no pain, photophobia, discharge or itching. He is an occasional soft contact lens wearer, and his spectacles are only six months old. His distance refraction has been stable since about 2004, and his presbyopic progression has been typical. He keeps good health, with a history of occasional migraines, hay fever, and a recent URTI. He is on a statin for hypercholesterolemia and an antihistamine nasal spray.

So from the history and presenting symptoms, I am already considering my differential diagnoses:

- FB/Trauma
- Dry eye with RCE
- Blepharitis
- Allergic response
- Infective
- ACG
- Uveitis
- Vascular
- Vitreo-retinal
- Optic neuritis
- Other…?

His refraction from six months earlier showed mild myopia and good visual acuities (VA) of 6/5 for each eye. However, at this appointment, his VA was R: 6/5 and L: 6/19. There was no improvement with pinhole. The pupils were a regular size; all reflexes were normal and equal. I could not elicit a RAPD.

The patient was aware of a visual disturbance that he describes as "a band of blurriness"

On external examination, the right eye appeared quiet and normal. The left eye appeared red, with generalized G2+ conjunctival congestion. Circum-limbal congestion was G3+.

The lids appeared quiet, there was no marginal inflammation or swelling, and there was no sign of problems seen on lid eversion. The tears were clean, stable (maybe a little copious), but with no discharge or mucous stranding. Both corneas were clear, with no hazing, scars, infiltrates, or epithelial loss. There was no Sodium Fluorescein or Lissamine Green staining. However, the left cornea did show G2 fine generalized endothelial precipitates. The anterior chamber (A/C) was quiet in the right eye, but the left exhibited G3 cells in the aqueous. Intra-ocular pressures were R: 10mmHg L: 36mmHg. Gonioscopy showed angles open past TM in all quadrants.

Revised DDx:

- Acute anterior uveitis (AAU)
- Angle closure glaucoma (ACG)
- Posner-Schlossman Syndrome (PSS)
- Trabeculitis
- Intermediate Uveitis
- Posterior Uveitis
- Vascular
- Optic neuritis

ACG: This is unlikely, as the symptoms had been present for about a week, and the pain and pupils do not fit the classic presentation; nor did the slit lamp findings. Perhaps the angle closure is intermittent. He had been in a darkened theatre for a couple of hours and this may have set it off.

Posner-Schlossman Syndrome (PSS): is another cause of intermittent IOP increases. PSS also known as glaucomatocyclitic crisis, is a disease typified by acute, unilateral, recurrent attacks of elevated intraocular pressure (IOP) accompanied by mild anterior chamber inflammation. Mr. LD is in the right age group (20-50yo), and the vision is commonly blurred during an attack.

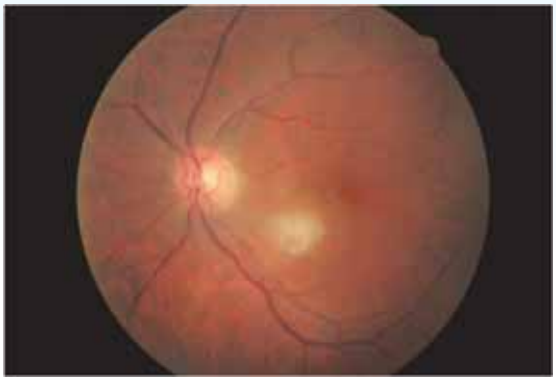
Trabeculitis: Inflammation of the trabecular endothelium does affect aqueous outflow and can have various causes. However this diagnosis seems to be one of exclusion.

AAU: This classically presents with miosis, photophobia, pain, circum limbal-flush and the presence of A/C cells. But in AAU the IOP is usually lowered due to ciliary body shutdown from inflammation and increased uveal outflow. However, on occasion, IOPs can be elevated due to inflammatory cells blocking the trabecular meshwork (TM). Also, the pupils were regular and equal, with no miosis. There was no photophobia or pain.

Intermediate uveitis/posterior uveitis: These can lead to "spillover" into the A/C. It would also account for the reduced symptoms of discomfort and photophobia. The IOP would be more likely to be raised, as the ciliary body/aqueous function would not be impaired and inflammatory cells from the spillover could be affecting outflow.

Optic neuritis: The symptoms for optic neuritis can be a bit vague. Pain was not reported to be worse on eye movement and there was no RAPD noted. I must confess that I really did not consider this a likely cause and omitted colour vision and Red-Cap test out of my assessment.

Dilated fundus examination revealed the following picture:



What we appear to have here is an acute chorio-retinitis with some vitritis and the A/C cells are a "spill-over" anterior uveitis. The location of the lesion certainly accounts for the visual symptoms and findings.

The DDx is now looking like:

- CMV necrotizing retinitis
- HZV
- HSV
- Fungal retinitis (candidiasis)
- Ocular toxoplasmosis
- Ocular toxocariasis
- TB
- Sarcoidosis
- Syphilis

Whatever the diagnosis, there will need to be specific medical tests now to establish/confirm the diagnosis. The most likely diagnosis is **Toxoplasmosis.** Mr. LD was referred urgently to the Wellington Hospital Eye Department where tests were performed to screen for TB, Syphilis, Toxoplasmosis, Toxocariasis, Sarcoidosis and whatever shows up on full blood counts.

In humans, Toxoplasmosis is one of the most common parasites; serological studies estimate that up to a third of the global population has been exposed to and may be chronically infected with it, although infection rates differ significantly from country to country. Toxoplasmosis is one of the most frequently identifiable causes of uveitis worldwide. In fact, Toxoplasma gondii infection is the most common cause of

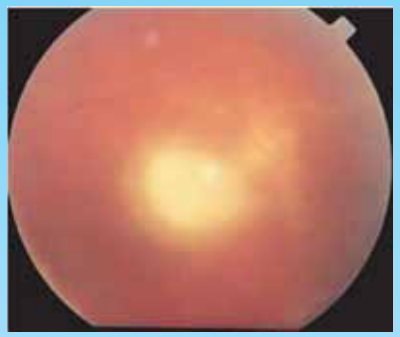
infectious posterior uveitis in non immuno-compromised individuals, and second only to cytomegalovirus retinitis in patients with HIV/AIDS.

In a study performed in Germany, toxoplasmosis accounted for 4.2% of all cases of uveitis at a referral centre. Around 5000 people develop symptomatic OT each year in the United States. OT is a complication of both acute acquired and reactivated congenital in immuno-competent but particularly in immuno-compromised individuals

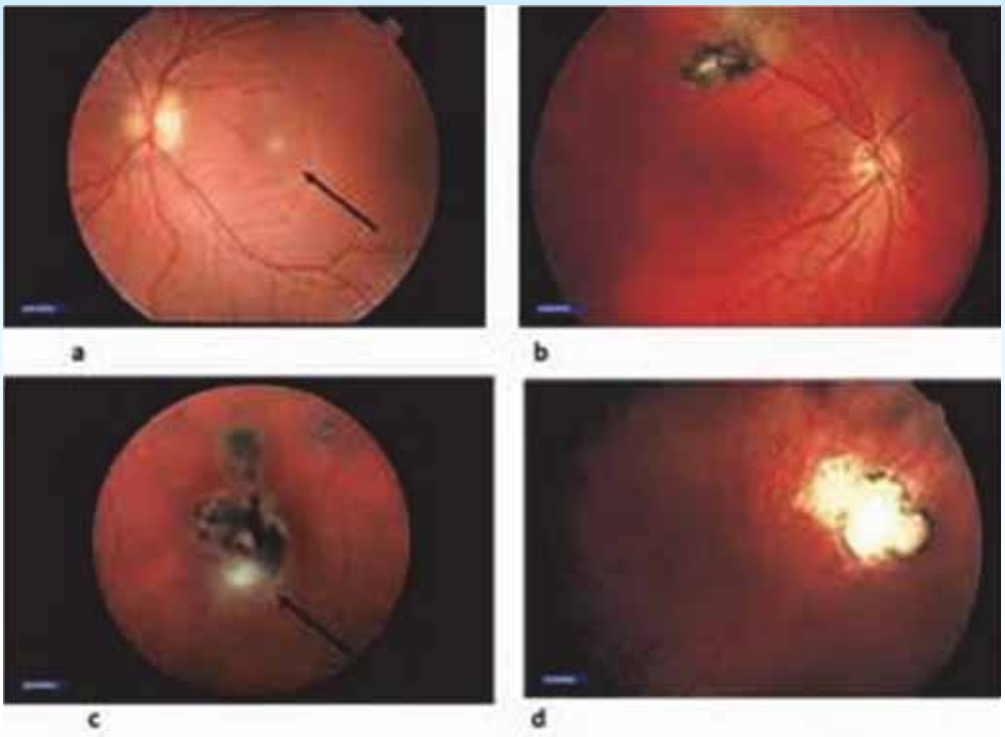
The hallmark of ocular toxoplasmosis is a necrotizing retinochoroiditis, which may be primary or recurrent. In primary ocular toxoplasmosis, a unilateral focus of necrotizing retinitis is present at the posterior pole in more than 50% of cases. The area of necrosis usually involves the inner layers of the retina and is described as a whitish, fluffy lesion surrounded by retinal edema. The retina is the primary site for the multiplying parasites, while the choroid and the sclera may be the sites of contiguous inflammation.

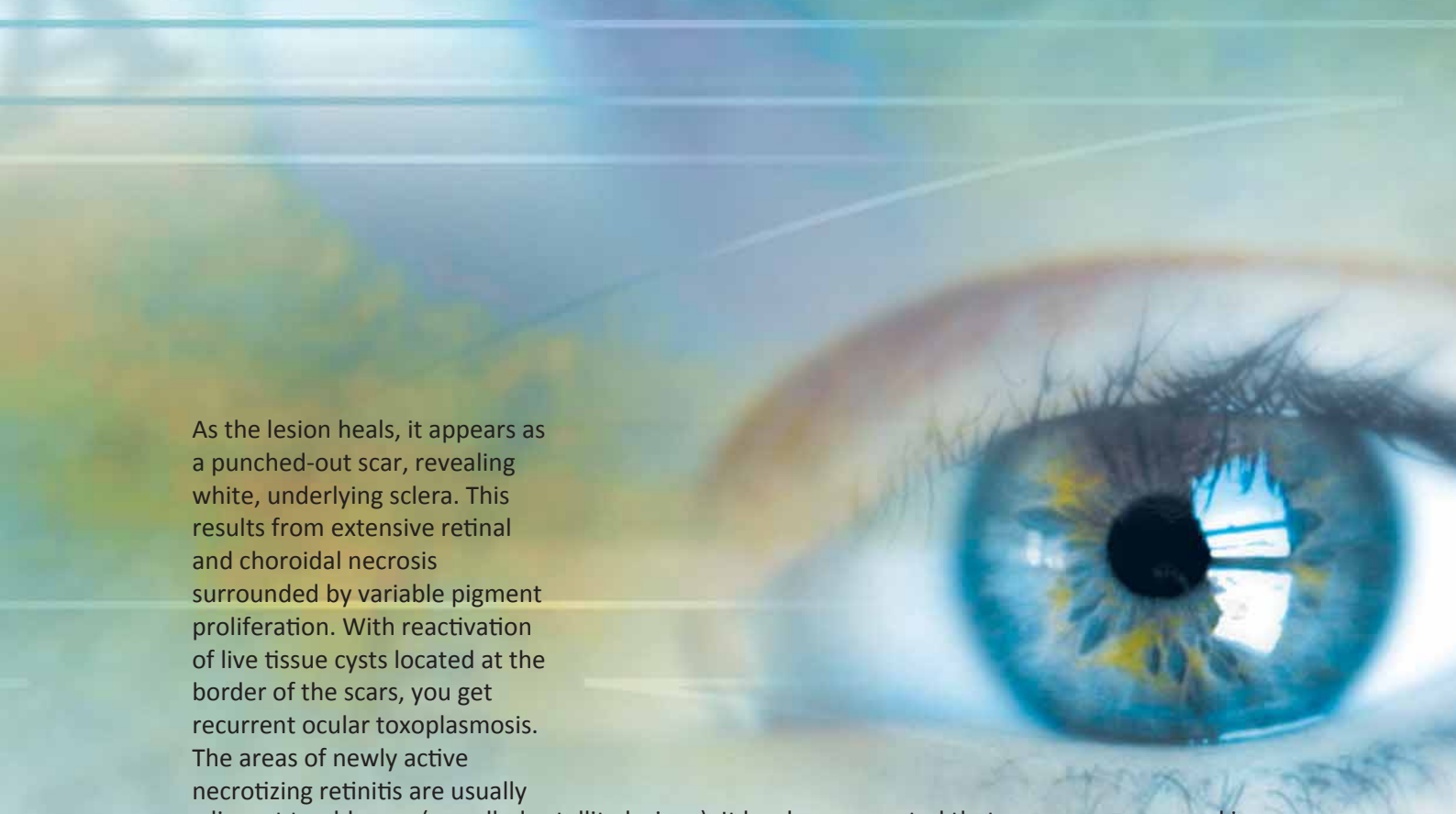
Toxoplasma antigens are responsible for a hypersensitivity reaction that may result in retinal vasculitis and granulomatous or non-granulomatous anterior uveitis. In many cases, the inflammatory reaction is severe, and the details of the fundus are not visible.

This appearance has been termed a "headlight in the fog."



Posterior vitreous detachment is commonly seen, and patients may develop precipitates of inflammatory cells on the posterior vitreous face, referred to as vitreous precipitates. Thick, vitreous strands and membranes may be present and may require vitrectomy.





As the lesion heals, it appears as a punched-out scar, revealing white, underlying sclera. This results from extensive retinal and choroidal necrosis surrounded by variable pigment proliferation. With reactivation of live tissue cysts located at the border of the scars, you get recurrent ocular toxoplasmosis. The areas of newly active necrotizing retinitis are usually adjacent to old scars (so-called satellite lesions). It has been reported that recurrence occurred in approximately in 4 out of 5 patients and that the risk was higher two years after the first episode. The risk of recurrence is the highest immediately after an episode of active disease and recurrence have a tendency to occur in clusters. The tentative diagnosis was made for Toxoplasmosis and treatment was commenced to reduce the inflammatory response, reduce the IOP and to treat the presumed toxoplasmosis.

Toxoplasmosis was confirmed as the diagnosis from the investigations. Here was the course of management:

- **Azopt drops** 1gtt bd left eye
- **Pred forte drops** 1gtt qds left eye.
- **Co-trimoxazole oral** 960mg bd.

The toxoplasmosis diagnosis was confirmed, but the Co-Trim was ceased due to the appearance of a skin rash. The retinitis was completely settled by three months and Mr. LD was discharged, armed with an Amsler Grid to monitor for further changes. His vision has remained at 6/12 in his left eye.

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