

## Case Record 16

# Acute Ideopathic Non-Granulomatous Iritis



July 2009

Dr Peter Frampton  
DOptom MSc FCOptom  
BAppSc(Optom)(AUS) DipTp(AS)  
DipTp(SP) DipTp(IP)



## INTRODUCTION

A personal observation is that many optometrists, while keen to be involved in community 'red eye' shared care schemes, articulate fear in the direct prescribing of drugs. As a profession, our primary hurdle is not at the point of prescribing, but is developing diagnostic confidence; without which the prescribing of therapeutics is extremely risky.

While symptoms and case history should give strong clues (Scott 2008, Ostler 1993, Farina and Mazarin 2006), it is easy to overlook a possibility, particularly if more obvious co-existing observations dominate the clinical picture; particularly relevant with contact lens wearers. A systematic approach is therefore essential and must include visual acuity, IOP, examination of the iris and anterior chamber, palpebral and bulbar conjunctiva and all levels of the cornea and epithelium (with and without stains) (George 2007). The possibility of posterior eye involvement must not be discounted, particularly if iritis is found. Consideration as to whether the condition being investigated is bilateral or unilateral is vital as is examination of the fellow eye for less obvious signs of binocular involvement.

Uveitis can be either monocular or binocular with a variety of possible aetiologies (Knott 2009). While independent prescriber optometrists may prescribe any licensed medicine for ocular conditions, this must only be done within the area of expertise of the individual optometrist (GOC 2008, College of Optometrists 2009). Iritis must be accurately assessed; steroid use in a misdiagnosed infective condition could aggravate the problem (BNF 2009). The decision as to whether the particular presentation falls within the individual's abilities to manage must also be made, suspicion of underlying systemic disease may necessitate referral.

# Case 1 Idiopathic Acute Non-granulomatous Iritis

## Male, CL Wearer DOB 11/5/79

### 18/7/05 Presenting complaint

LE red and painful over 3 days. Removed C/L 2/7 ago after day out. Woke yesterday in extreme pain, slightly better today. Very light sensitive and eye quite red.

### Past Ocular History

Contact lens wearer for 6 years with no problems. Daily wear schedule, comfort usually good with wear time up to 16 hours.

No other problems previously. No recent trauma

### Family Ocular History

Myopia.

### General Health

No medications. No allergies. Non-smoker

Feeling well. Did not have any problems before Saturday

No history of trauma or surgery.

VA with Specs R 6/6, L 6/7.6

Goldmann R 16, L 16

Pupils E&A D,C & N

### Slit Lamp

#### RIGHT

Cornea Clear-No Nafl staining  
Stroma and Endothelium Clear

Conjunctiva Clear

Evert lid – normal

AC Clear

#### LEFT

Cornea Clear-No Nafl staining  
Stroma and Endothelium Clear. NoKP

No Seidel Sign

360° Perilimbal Injection CCLRU 4+

Evert lid – normal

A/C flare 1+ and cells 1+

### Fundsocopy 1% Trop

No synechiaie. Vitreous clear. Vasculature normal. No retinal or vascular inflammation.

### Differential diagnosis

Idiopathic Acute Anterior Iritis.

1% Cyclopentolate bid  
Predforte (Prednisolone Acetate 1%) q1h during the day  
Review 24/24

19/7/05 5.30pm

Much improved. Eye white and comfortable. Not keen on cycloplegic – focus difficult.

VA with Specs R 6/6, L 6/6  
IOP R 16, L 16

Slit Lamp

AC clear

No synechiae

Conjunctival hyperaemia CCLR 2

Plan

Continue for a further 4/7 at:

Predforte q1h

Cyclopentolate 1% bid

Review in 4/7

24/7/05 9.00am

Eye quiet and fine. Comfort excellent.

VA with specs R 6/6, L 6/6

Goldmann R 15, I 16

Slit Lamp

Fully resolved. Eye quiet. AC clear and no conjunctival injection.

Plan

Stop cycloplegia

Taper steroids

Predforte q3h 1/52

Predforte bid 3/7

Predforte qd 3/7

Review in 2/52

7/8/05 10.00am

Coped with drops well. Eye feels fine, no photophobia or blurriness. Last drops yesterday

VA with Specs R6/6, L 6/6

Goldmann R 16, L 17

### Slit Lamp

AC clear, No conjunctival injection.

### Advice

Advice on recurrences given. Return immediately if symptoms. Otherwise review in 1/12

## DISCUSSION

Gordon (2007) suggests that the diagnosis of uveitis should be one of exclusion. This would not seem an appropriate policy; the possibility of iritis should be proactively considered for all 'red' eyes, particularly monocular red eyes. The diagnosis of Idiopathic Acute Iritis however should be after the exclusion of other aetiologies. Iritis can be secondary to infection, trauma, systemic disease, surgery, neoplastic processes and ischaemia (Knott 2009). The decision to treat, as an IP optometrist, could well depend on the need to involve other medical specialties.

In this case the patient was young and in excellent general health. No previous history of iritis could be elicited and the monocular, non-granulomatous presentation and lack of signs of trauma or more posterior inflammatory processes made the diagnosis of Idiopathic Acute Iritis most probable.

A single episode of non-granulomatous iritis in an otherwise healthy individual does not warrant investigation (George 2007, Gordon 2007, Knott 2009, Ray-Chaudhuri 2005). The same authors recommend cycloplegia for comfort and to prevent synechiaie formation, coupled with topical steroids to reduce the inflammatory processes. Regardless of level of inflammation, aggressive treatment is recommended; Ray-Chaudhuri (2005) stresses the tendency to

underestimate the disease process and therefore under treat it. The dosing of Rimexolone for uveitis is q1h for week one, q2h week two, qid week three reducing to bid for four days and qd for the remaining three days (BNF 2009). The same document simply states every 1 or 2 hours until inflammation is controlled and then taper for Prednisolone Acetate 1%. Ray-Chaudhuri (2005) is also less prescriptive and suggests the steroid should be tapered when there is definite improvement. In this case the patient showed no adverse reactions to the Predforte and, while significant improvement was noted in 24 hours the maximum dosing was maintained for a further four days, ensuring effective treatment.

An adverse effect of both cycloplegics and steroids is elevated IOP. The use of cyclopentolate was not of risk. Angle depth was estimated as Grade 4+ for each eye (van-Herrick's); only angles less than III have been found to be closable (Palmborg 1996). Corticosteroids however seem to affect IOP by increasing resistance to outflow within the trabecular meshwork (Skuta & Morgan 1996, Boyd & McLeod 1964) and therefore steroid responders cannot be predicted and all patients must be monitored closely for elevation in IOP.

As an IP optometrist would elevated IOP during steroid treatment necessitate onward referral? IP optometrists must only use drugs specifically within their licensed guidelines (GOC 2008, College of Optometrists 2009). The only hypotensive drugs licensed for treatment of secondary glaucomas are the  $\beta$ -blockers (electronic medicines compendium 2009). In this case the patient had no general health contra-indications and temporary use of a  $\beta$ -blocker could be considered if the patient had been a steroid responder. Regardless of licensing limitations a  $\beta$ -blocker would be the drug of first choice as the prostaglandins are pro-inflammatory mediators (Camras 1996) and intuitively would not be best suited to an inflammatory condition.

Choice of steroid could also influence IOP control in a sensitive patient.

Prednisolone and Dexamethasone show higher instances of IOP elevation than fluorometholone (Onofrey, skorin and Holdemen 1998). These authors also suggest that Rimexolone, while still a very potent steroid, shows lower hypertensive effects.

Regular reviews, even in uncomplicated cases, are therefore vital. Next day review confirmed that improvement was evident and a misdiagnosis was unlikely. A second check before commencing steroid tapering, followed by a third at the end of treatment was considered adequate in this case. A review several weeks after cessation of treatment is also advocated by George (2007) to ensure absence of residual inflammation.

## REFERENCES

1. BNF 57 (March 2009). British National Formulary. Accessed [www.bnf.org](http://www.bnf.org)
2. Boyd TAS and McLeod LE. (1964). Circadian Rhythms of Plasma Corticoid Levels, Intraocular Pressure and Aqueous Outflow Facility in normal and Glaucomatous Eyes. *Ann NY Academy of Science*, 117, 597-613.
3. Camras CB. (1996). Prostaglandins. In Ritch R, Shields MB and Krupin T (eds). *The Glaucomas (second edition) Vol III Glaucoma Therapy*. Mosby. USA
4. College of Optometrists. (2009). Guidance for Optometrist Prescribers. [www.college-optometrists.org](http://www.college-optometrists.org)
5. Electronic Medicines Compendium. (2009). Accessed [www.emc.medicines.org](http://www.emc.medicines.org)
6. European Glaucoma Society. (2003). Terminology and Guidelines for Glaucoma Edition II. Dogma. Accessed [www.eugs.org](http://www.eugs.org)
7. Farina G and Mazarin G. (2006). Red Eye Evaluation. [emedicine.medscape.com/article/1216540](http://emedicine.medscape.com/article/1216540). Accessed [www.trapdatabase.com](http://www.trapdatabase.com)
8. George R K. (2007). Uveitis, Anterior, Nongranulomatous. [emedicine.medscape.com/article/1209595](http://emedicine.medscape.com/article/1209595). Accessed [www.tripdatabase.com](http://www.tripdatabase.com)
9. GOC. (2008). A Handbook for Optometry Specialist Registration in Therapeutic Prescribing. General Optical Council. [www.optical.org](http://www.optical.org)
10. Gordon K. (2007). Iritis and Uveitis. [emedicine.medscape.com/article/798323](http://emedicine.medscape.com/article/798323). Accessed [www.tripdatabase.com](http://www.tripdatabase.com)
11. Knott L. (2009). Uveitis. [www.patient.co.uk](http://www.patient.co.uk). Document ID 1599. Accessed [www.tripdatabase.com](http://www.tripdatabase.com)
12. Onofrey B E, Skorin L and Holdeman N R. (1998). Ocular Therapeutics Handbook, A clinical manual. Lippincott-Raven. USA.

13. Ostler H B. (1993). Diseases of the External Eye and Adnexa: A Text and Atlas. Williams and Wilkins. USA.
14. Palmberg P. (1996). Gonioscopy. In Ritch R., Shields M.B. and Krupin T. *The Glaucomas (Second Edition) Vol I Basic Sciences* . Mosley. USA.
15. Ray-Chaudhuri (Ed). (2005). Royal Victoria Infirmary Eye Casualty Guidelines. The Newcastle upon Tyne Hospitals NHS Trust. Access [www.newcastle-hospitals.org.uk](http://www.newcastle-hospitals.org.uk)
16. Scott O. (2008). Conjunctivitis. Mentor. Patient UK [www.patient.co.uk/showdoc/40025912](http://www.patient.co.uk/showdoc/40025912). Accessed [www.tripdatabase.com](http://www.tripdatabase.com)
17. Skuta GL and Morgan RK. (1996). Corticosteroid-induced Glaucoma. In Ritch R, Shields MB and Krupin T (eds). *The Glaucomas (second edition) Vol II Glaucoma Therapy*. Mosby. USA.