Key Clinical Topics in Ophthalmology

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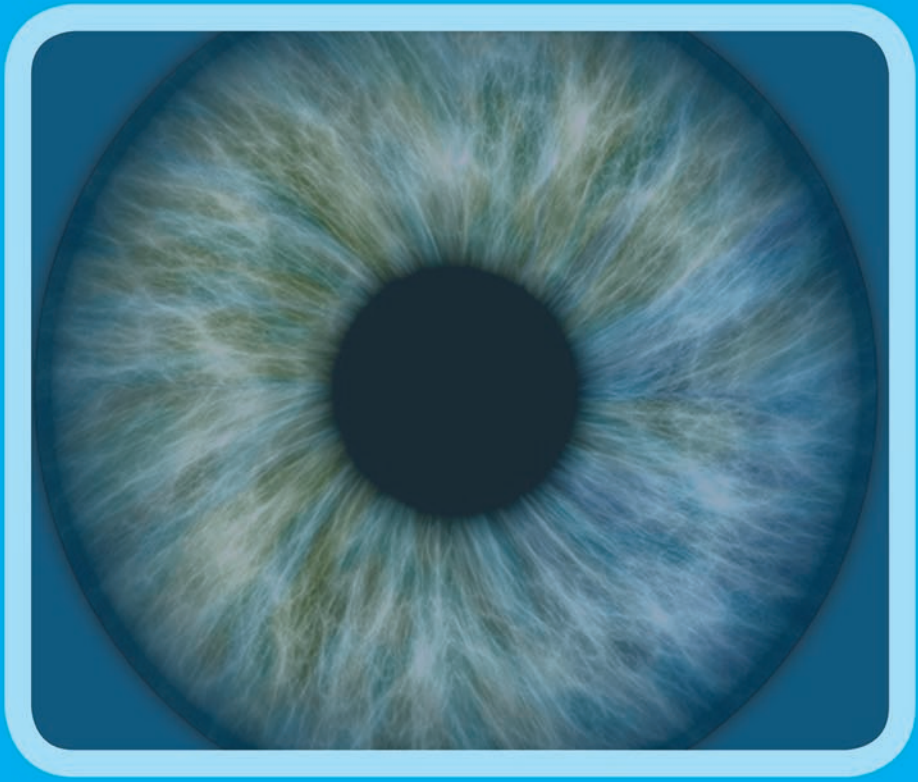

Contents

4 Anterior uveitis

Key points

- Occurs due to inflammation of the ciliary body and iris
- Characterised by recurrent attacks of pain, photophobia and a red eye
- Multiple different possible causes but 80% will remain idiopathic after investigation
- Must be differentiated from more serious infectious uveitis of the posterior segment with a dilated fundal examination

Epidemiology

Uveitis is an uncommon condition with an incidence of around 12 in 1000,000. Men and women are affected equally although the disease has been reported to be more severe in males. Uveitis can develop at any age but is most common in those aged over 20 years.

Risk factors

Most cases remain idiopathic with no cause found, however the human leucocyte antigen (HLA) B27 variant, which is present in up to 8% of the population, is found in up to 20–50% of uveitis cases so is a known genetic risk factor. The most common identifiable causation is an underlying disease which affects the immune system and predisposes to the dysregulation seen as part of the pathophysiology.

Pathophysiology

It is believed that an external antigen sets off the immune system which then reacts to the ocular tissues due to genetic and environmental risk factors which have sensitised it. While the initiating event is not fully understood we have evidence that the activated immune system, through T-lymphocytes and macrophages, leads to localised inflammation in the uveal tissue that breaks down the blood-ocular barrier. White blood cells and protein pass into the anterior chamber and lead to the clinical signs described below.

Although most remain idiopathic there are many cases where a source of antigen can be discovered, and these are divided into infectious and non-infectious causes.

- Non-infectious:
  - Seronegative spondyloarthropathies associated with HLA-B27 (ankylosing spondyloarthritis, psoriatic arthritis, reactive arthritis and inflammatory bowel disease associated)
  - Juvenile idiopathic arthritis (JIA)
  - Sarcoidosis
  - Behçet's disease
  - Localised ocular causes including trauma and hypermature cataracts
  - Specific syndromes – Fuchs' heterochromic cyclitis
- Infectious:
  - Bacterial – Syphilis, Lyme disease, tuberculosis
  - Viral – Herpesvirus family, Ebola virus
  - Fungal – Microsporidia

Clinical features

Symptoms

Inflammation in any organ is associated with pain and when the principle affected tissue is the muscles in ciliary body and iris, this produces aching pain on pupillary constriction and accommodation. This manifests as visual blurring, photophobia and pain on near focusing such as reading.

Examination

The affected eye will be red with a characteristic vasodilation around the limbus called ciliary flush. The characteristic signs of anterior uveitis are keratic precipitates (KP) and cells in the anterior chamber (AC) – the presence and amount of which are often used to grade severity and assess response to treatment. Keratic precipitates are deposits of inflammatory material and cells on the corneal endothelium. These can be small (dusting) or large and greasy (mutton-fat) KP. The same inflammatory material congregates and floats in the AC as cells and flare which is the

proteinaceous element. These can be counted and graded using an angled 1 by 1 mm bright slit lamp beam (see **Tables 4.1** and **4.2**).

The pupil may be miosed due to a spasming painful pupillary constrictor muscle and may be adherent to the anterior lens capsule called posterior synechiae. If these posterior synechiae seal off the pupil in 360° then iris bombe may develop and cause angle closure and acute glaucoma. The iris may also show granulomatous nodules at the pupillary margin called Koeppe's nodules or present in the ciliary zone called Busacca's nodules. Intraocular pressure may be high due to bombe or inflammatory adhesions between the iris and trabecular meshwork, but conversely may be low due to reduced aqueous production. The fundus must be examined with a dilated examination to exclude posterior uveitis presenting with anterior signs.

Investigations

Given most cases are idiopathic, the majority of investigations performed for anterior uveitis will be normal or negative. They should therefore be targeted and restricted to cases which are recurrent, bilateral, granulomatous or have associated systemic signs or symptoms.

A good history is essential to tailor your investigations according to systemic symptoms, e.g. of rheumatic back pain for HLA-B27 or chronic cough for sarcoid. A history of tuberculosis (TB) exposure will lead you to TB testing and intravenous drug use to HIV testing.

Table 4.1 SUN classification for grading anterior chamber cells – count the cells

0	–
1–5	+/–
6–15	+
16–25	++
26–50	+++
> 50	++++

Table 4.2 SUN classification for grading anterior chamber flare – degree of blur when examining the iris

None	–
Hardly any	+
Definite blur but still clear iris detail	++
Obvious blur and iris details hazy	+++
Fibrinous flare	++++

Initial investigations

- Blood testing – full blood count (FBC), urea and electrolytes, treponemal (syphilis) serology
- Chest X-ray – sarcoid and tuberculosis
- Urinalysis – looking for blood and protein
- Optical coherence tomography (OCT) – may be normal or show cystoid macula oedema

Targeted investigations

Based on results of the initial investigations/ suspicions:

- Tuberculosis testing – Mantoux skin testing or interferon-gamma (IFN-γ)release assay on blood
- Serology for HIV, Lyme disease
- HLA-B27 testing for seronegative spondyloarthropathies
- Further testing under medical physicians including sarcoid specific imaging, etc.

Diagnosis

Anterior uveitis has characteristic symptoms and signs which make diagnosis relatively easy. The skill comes in ensuring there is no other secondary cause or deeper inflammation and then deciding which cases to investigate to rule out causes as described above. Anterior uveitis can be classified in many different ways; non-granulomatous or granulomatous (mutton-fat KPs, iris nodules), acute or chronic (lasting > 3 months) and infectious or non-infectious based on the causes.

Differential diagnosis

- Conjunctivitis
- Keratitis
- Episcleritis
- Scleritis

- Posterior, panuveitis or endophthalmitis (examine the whole eye and beware severe postoperative inflammation)
- Pigment dispersion syndrome (referred from optometry with iris transillumination, Krukenberg's spindle looking like KP)

Treatment

Treat the inflammation and pain

- Topical corticosteroids – start with intensive treatment and taper down over several weeks. Hourly dexamethasone 0.1% drops applied day and night should be reduced to 2 hourly then 6 times a day, 5 times a day etcetera. Steroid ointments can be used at night as an alternative but are not as effective as waking to put in the drop in the initial stage
- Cycloplegic agents – paralyse the spasming muscles relieving pain but blurring vision, e.g. cyclopentolate 1% given 3 times a day
- Very severe or resistant cases may require steroid injected either sub-conjunctivally or periocular

Prevent complications

- Cycloplegia also dilates the pupil and can prevent posterior synechiae formation
- Drops to lower intraocular pressure
- Taper the drops over time to prevent secondary iatrogenic complications of steroid use such as ocular hypertension or cataract formation

Complications

With the correct use of treatment these complications are less likely but include posterior synechiae and iris bombe, cataract, cystoid macula oedema and band keratopathy.

Glaucoma and uveitis are common partners due to multiple causes including iatrogenic steroid response, synechial angle closure, physical blockage of the trabecular meshwork with cells and iris bombe.

Prognosis for vision is good when chronic anterior uveitis doesn't develop but certain categories of disease such as JIA associated uveitis have a worse prognosis with a quarter developing severe visual loss.

Further reading

Williams GS, Westcott M (Eds). Practical Uveitis: Understanding the Grape. Taylor and Francis; 2017.

Related topics of interest

- Intermediate uveitis (p. 185)
- Posterior uveitis (p. 259)
- Retinal vasculitis (p. 286)

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