Appraisal, work-up and diagnosis of anterior uveitis
A practical approach

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Introduction

The diagnostic yield in uveitis has significantly improved in the last 15 to 20 years, in part because a clear classification is available to the clinician but even more so because diagnostic tests have greatly improved lately.

With a systematic approach, based on history and clinical examination and guided by laboratory tests and other investigational tests, a diagnosis can be expected in about 70% of cases.

A compilatory approach such as it is exposed in most uveitis textbooks has to be avoided. (Figure 1a & 1b) Endless lists of diseases the exposure to which the patient is supposed to tick on a checklist are not contributing to a diligent work-up of uveitis and are even counterproductive as this may push the clinician into the wrong direction.

![Fig. 1a & 1b Example of the traditional lists that are featured in most uveitis books](image)

It is much more productive to use a comprehensive approach that avoids a detailed anamnesis (history) at the beginning of the consultation but limits the initial anamnesis (history) to the immediate complaints of the patient followed immediately with a thorough clinical examination. Once the clinical signs have been established and have allowed to characterize the type of uveitis (granulomatous versus non-granulomatous) the anamnesis (history) can be resumed by asking oriented questions. The clinician should then try to classify the uveitis more precisely, refer to the local epidemiological data and come up with a working diagnosis, which he should always be prepared to change. At this point targeted investigations and laboratory tests should be performed. The result is a diagnosis which has higher degree of probability that can be followed by appropriate therapy. In case no diagnosis can be suspected non-specific therapy may have to be started or therapy can be withheld. In a proportion of cases the evolution of the disease will lead to the diagnosis. (Flow-chart 1)
Flow-chart 1: general principles in the appraisal of a uveitis case: proceed with guidance of the clinical signs

1. Short initial history
2. Clinical examination
3. Oriented history based on clinical examination
4. Epidemiological data of the area
5. Classification
6. Laboratory tests based on the clinical examination
7. Special investigations based on the clinical signs
8. Clinical evolution
9. Diagnosis
10. Treatment
The work-up of anterior uveitis

Definition

The anatomical classification of uveitis into anterior, intermediate and posterior forms is very useful to conduct the work-up and eventually to reach a diagnosis, even though inflammation does not always respect these anatomical boundaries. Anterior uveitis is the term used for the group of inflammatory disorders for which the preponderant part of the inflammation is situated at the level of the pars plicata of the ciliary body, the retroiridal space, the iris and the anterior chamber.

Symptoms and signs of anterior uveitis

The severity of symptoms in anterior uveitis ranges from no symptoms in chronic disease such as anterior uveitis related to juvenile idiopathic arthritis (JIA) to very severe symptoms in acute uveitis such as HLA-B27 related uveitis. Symptoms of acute anterior uveitis include photophobia, redness, pain, decreased vision and tearing in the absence of discharge.

The signs of anterior uveitis are listed in table 1

| 1.1. Conjunctival injection |
| 1.2. Keratic precipitates |
| 1.3. Aqueous flare / fibrinous clots (Figure 2) |
| 1.4. Posterior synechiae between the iris and the capsule of the lens (Figure 3) |
| 1.5. Aqueous cells / hypopyon (Figure 4) |
| 1.6. Iris rubeosis (usually reversible) |
| 1.7. Iris nodules (Koepppe / Busacca) (Figures 5a & 5b) |
| 1.8. Iris atrophy (herpes uveitis and Fuchs’ uveitis) |
| 1.9. Intraocular pressure changes (hypotony in severe acute anterior non-granulomatous uveitis; hypertony in granulomatous uveitis) |

Figure 2. Fibrinous clot in anterior chamber in typical case of HLA-B27 related uveitis.
Figure 3. Posterior irido-lenticular synechiae. The ring of pigment deposits on the crystalline lens show where the iris was attached (synechiae). There is one remaining synechia at six o'clock on the verge of detaching itself following the administration of massive dilating drops.

Figure 4. Hypopyon. Sedimented white cells form a level at the bottom of the anterior chamber, associated with a ring of fibrine on the surface of the crystalline lens (top) indicating broken synechiae. One remaining synechia on the meridian of 7 o'clock.

Figure 5a. Koepppe nodules. Two Koepppe nodules at the border of the iris associated with synechiae.
1. The **conjunctival injection** in anterior uveitis can be diffuse or localized circumferentially at the limbus (perikeratic injection) or mixed (diffuse and perikeratic injection).

2. The morphology of **keratic precipitates (KPs)** is very useful to help distinguish non-granulomatous from granulomatous uveitis. Small diffuse KPs causing dusting of the endothelium are characteristic for non-granulomatous uveitis such as HLA-B27 related acute anterior uveitis. When KPs become larger than endothelial dust they can be individualised and correspond to granulomatous KPs. The morphology and distribution of granulomatous KPs is often useful in orienting towards a more specific diagnosis within granulomatous causes (table 2). Medium and large size KPs are called "mutton fat" KPs. There is a lot of confusion regarding the characterisation of KPs. In many textbooks only mutton-fat KPs are termed granulomatous and small granulomatous KPs as those found in Fuchs’ uveitis are erroneously termed as non granulomatous. Therefore Fuchs’ uveitis is mistakenly classified as non-granulomatous in many textbooks. It is useful to distinguish between fresh and chronic (old) muton-fat KPs. Old mutton-fat KPs tend to be less white, pigmented and less dense in the centre.

**Figure 5b. Busacca Nodules.** UBM picture showing nodule within the stroma in a patient with tuberculous uveitis.
Table 2. Granulomatous keratic precipitates (KPs)

<table>
<thead>
<tr>
<th>Type of KP</th>
<th>Clinical entity to suspect</th>
</tr>
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<tbody>
<tr>
<td>Small stellate, even distribution no gravitation</td>
<td>Fuchs' (heterochromic) uveitis (&lt;Figure 6&gt;)</td>
</tr>
<tr>
<td>Small gravitational or inferior random distribution</td>
<td>CMV uveitis (&lt;Figure 7&gt;)</td>
</tr>
<tr>
<td>Small/medium/ size, focal spherical distribution under stromal keratitis</td>
<td>h.simplex/zoster keratouveitis (&lt;Figure 8&gt;)</td>
</tr>
<tr>
<td>Medium/large gravitational distribution (mutton-fat)</td>
<td>sarcoidosis, tuberculosis, toxoplasmosis (&lt;Figure 9&gt;)</td>
</tr>
<tr>
<td>Very few (2-5)small/medium/large size, inferior peripheral (iridocorneal angle)</td>
<td>Posner-Schlossmann syndrome</td>
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</tbody>
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**Figure 6. Fuchs uveitis.** Small stellate non-gravitationally distributed keratic precipitates (KPs) typical of Fuchs' uveitis.

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**Figure 7. KPs in CMV anterior uveitis.** Scarce unilateral KPs of different sizes (small to medium) in a case of cytomegalovirus (CMV) anterior uveitis.

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**Figure 8a. Herpes KPs.** Usually herpes/zoster KPs are arranged in a round disciform pattern and are small to medium sized.
Figure 8b. Herpes KPs. Usually arranged in a round disciform pattern very well visible against retroillumination. Note also sectorial iris atrophy at the top of the picture at the level of the superior iris.

Figure 9. Mutton-fat KPs. This type of gravitational large KPs can be seen in tuberculosis and sarcoidosis. In toxoplasmosis the anterior inflammation sometimes associated with the retinitis is also made of large mutton-fat KPs, although the KPs are usually less numerous.

3. Anterior chamber flare is caused by exudation of proteins into the normally clear aqueous humor from iris vessels or across the ciliary body epithelium following the breakdown of the blood-aqueous barrier. The intensity of flare is measured in a standard fashion following the grading system proposed by the Proctor Group in San Francisco in 1959 (table 3),\(^1\) that is however only qualitative. A beam 1mm wide and 3mm long with the highest light intensity and 16X magnification on the BQ Haag-Streit slitlamp is used. When the concentration of proteins in the aqueous is very high, they agglomerate and form fibrinous clots, a finding more common in acute non-granulomatous uveitis. (Figure 2)

4. Depending on the amount and composition of aqueous inflammatory proteins adherences between the iris and anterior capsule of the crystalline lens can form (posterior synechiae). (Figures 3 & 4)

Table 3. Slit-lamp grading of aqueous flare (1 mm X 3mm beam)

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>No flare</td>
<td>0</td>
</tr>
<tr>
<td>faint, just detectable</td>
<td>+</td>
</tr>
<tr>
<td>moderate, iris details clear</td>
<td>++</td>
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Since a few years it is now possible to measure flare in a quantitative and objective fashion, using **laser flare photometry (LFP)**. (Figure 10) This new technology makes flare the only quantitative parameter to measure intraocular inflammation. So far cells were judged more accurate to measure inflammatory activity in uveitis. At best this measurement is however only semi-quantitative. LFP was shown to be more sensitive than slit-lamp assessment of cells to measure the evolution of inflammatory activity, making flare the new gold standard to assess intraocular inflammation. LFP allows to detect subclinical flare intensities and changes, that can be predictive of clinical recurrence. It allows to detect resistance of inflammation to treatment and closer follow-up of therapy, often leading to corticosteroid sparing in the treatment. When available, laser flare photometry certainly allows improved management of uveitis.²

**5. Aqueous cells** used to be the reference parameter for inflammatory activity because their evaluation was quantifiable by slit-lamp examination. Nowadays this is no more true when LFP is available. LFP is the quantifiable gold standard to measure inflammatory activity even in chronic inflammation with chronic breakdown of the hemato-ocular barriers, as it has been shown that even when there are no cells, LFP can detect active inflammation that responds to therapy. Grading of cells in the anterior chamber has been standardized by Hogan et al. at the Proctor Foundation in 1959.¹ (table 4)

<table>
<thead>
<tr>
<th>No cells</th>
<th>0</th>
</tr>
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<tbody>
<tr>
<td>1-5 cells</td>
<td>±</td>
</tr>
<tr>
<td>6-10 cells</td>
<td>+</td>
</tr>
<tr>
<td>11-20 cells</td>
<td>++</td>
</tr>
<tr>
<td>21-50 cells</td>
<td>+++</td>
</tr>
<tr>
<td>&gt; 50 cells</td>
<td>++++</td>
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</table>

It is important to make the difference between pigment clumps and inflammatory cells and to examine the anterior chamber prior to mydriasis as cells and especially pigment dispersion can sometimes be seen after pupillary dilatation.

When the quantity of cells is very dense they sediment and cause a **hypopyon**, a sign more often seen in HLA-B27 related uveitis, Behçet's uveitis and uveitis related to juvenile idiopathic arthritis (JIA). (Figure 4)

6. In severe and longstanding uveitis **iris rubeosis** can develop. It is in fact most often a pseudo-rubeosis that is reversible after introduction of anti-inflammatory treatment. Even when a real rubeosis has
developed, it is usually situated at the pupillary border of the iris and is much less aggressive and proliferative than ischemic rubeosis iridis. In Fuchs’ uveitis with extensive iris atrophy iridial vessels can be seen and correspond to a pseudo-rubeosis.

7. Iris nodules (Figures 5a & 5b)
In granulomatous uveitis two types of iris nodules can develop. When situated at the pupillary margin (and on the surface of the iris) they are called Koeppe nodules, have a fluffy appearance and a size going from very small barely visible excrescences to frank nodules. When nodules are situated in the body of the iris stroma they are called Bussaca nodules.

8. Sectorial or widespread iris atrophy is a sign rather specific for herpes simplex or herpes zoster uveitis and is a useful diagnostic help. Diffuse atrophy is very often seen in Fuchs’ uveitis.

9. Intraocular pressure changes due to uveitis can present either as hypotension or hypertension. Hypotony is usually measured in severe uveitis involving the ciliary body such as acute anterior non-granulomatous HLA-B-27 related uveitis. Hypertony is usually associated with granulomatous uveitis, especially herpes simplex or herpes zoster uveitis but also sarcoidosis or spill-over anterior uveitis associated with toxoplasmic retinochoroiditis. In recent years, CMV anterior uveitis, a newly recognised usually unilateral entity very often causing hypertony has been described. 3

The work-up of anterior uveitis

The anatomical diagnosis of anterior uveitis has first to be verified by excluding spill-over inflammation associated with uveitis of the posterior segment (intermediate or posterior uveitis). To exclude posterior involvement pupil dilatation is mandatory in all cases. Secondly, the type of clinical presentation has to be characterized as non-granulomatous or granulomatous in order to correctly orient work-up and differential diagnosis.

Non-granulomatous uveitis is characterised mainly by the type of keratic precipitates that presents as fine KPs producing endothelial dusting. In severe cases fibrinous clotting or hypopyon can occur depending on whether protein influx or cellular infiltration is predominant. In case of severe inflammation it is also common to find posterior synechiae and pressure tends to be more often decreased than increased.

Table 5. Common causes of non-granulomatous anterior inflammation in anterior uveitis or panuveitis

<table>
<thead>
<tr>
<th>Cause</th>
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<tbody>
<tr>
<td>HLA-B27 related uveitis</td>
</tr>
<tr>
<td>Behçet’s uveitis</td>
</tr>
<tr>
<td>Juvenile rhumatoid arthritis related uveitis</td>
</tr>
<tr>
<td>Uveitis associated with scleritis</td>
</tr>
<tr>
<td>Uveitis associated with streptococcal infection</td>
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</tbody>
</table>
**Granulomatous uveitis** is characterized by KPs that are larger than the dusty KPs of non-granulomatous uveitis. They are better individualized but their size varies depending on the inflammatory process. The medium and large size granulomatous KPs are called mutton-fat KPs. (Figure 9) Other characteristic features of granulomatous uveitis are Koepppe and Bussaca nodules. (Figures 5a & 5b) Synechiae are common in more pronounced inflammation. Pressure changes when present are usually characterized by increased intraocular pressure.

Although this distinction is a very useful working classification, the subdivision is not an absolute one. A granulomatous uveitis may initially present as non-granulomatous when dusty KPs are very thick so that before taking its granulomatous aspect. Conversely in rare cases a non-granulomatous uveitis can take an aspect that might be qualified as granulomatous. In most cases the distinction is however rather well delineated.

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**Table 6. Common causes of granulomatous anterior inflammation**

1. **in anterior uveitis**
   - Fuchs' uveitis
   - Herpetic (herpes simplex, varicella-zoster, Epstein-Barr) uveitis
   - CMV anterior uveitis
   - Posner-Schlossmann syndrome
   - Sarcoidosis
   - Tuberculosis
   - Syphilis

2. **in panuveitis**
   - Sarcoidosis
   - Vogt-Koyanagi -Harada syndrome, sympathetic uveitis
   - Tuberculosis
   - Syphilis
   - Toxoplastic retinochoroiditis
   - Necrotizing herpetic retinopathies (acute retinal necrosis)
   - Post-surgical inflammation (propionibacterium acnes)

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**Work-up of non-granulomatous anterior uveitis**

In case of simple, fibrinous or hypopyon nongranulomatous uveitis, the only first-line work-up test we presently perform is the detection of the HLA-B27 antigen. HLA-B27 testing is performed even if the inflammation is only moderate. If the test is positive it will avoid further unnecessary testing during a subsequent episode and it is reassuring for the patient and the doctor to know the specific diagnosis, especially when it is a benign disease. In case of a positive result no further investigation is performed at the ophthalmological level. It is however recommended to take an oriented history that will allow, with the help of the internist or rhumatologist when necessary, to subclassify the affection into ankylosing spondylarthritis, Reiter's syndrome, Crohn's disease, ulcerative colitis or simply into HLA-B27 uveitis without systemic associated disease. About 50-55% of acute anterior non-granulomatous uveitis are HLA-B27.
positive in Europe, but this varies from one geographical area to another, being for instance quite low in Japan. In the remaining 45-50% of cases, a specific diagnosis is more difficult to establish. If the episode of HLA-B27 negative non-granulomatous uveitis is of limited severity and/or responds readily to classical topical corticosteroid therapy, no further investigation is performed.

Flow-chart 2: diagnostic steps in non-granulomatous uveitis

**Diagnostic approach: non-granulomatous anterior uveitis**

- Non-granulomatous anterior uveitis
  - HLA-B27 antigen
    - Positive
      - Hypopyon Aphtae, genital ulceration (⇒ HLA-B51)
    - Negative
      - Resolution
      - Recurrence
        - No further work-up
  - In children
  - Bilaterality
  - ANA
    - Juvenile idiopathic arthritis (JIA)
    - Renal pathology B-2-microglobulin
  - Examine posterior segment !!!

**TINU-syndrome**

**TINU = Tubulo-interstitial nephritis and uveitis**

**ANA = antinuclear antibodies**

**HURC = haemorhagic ulcerative recto-colitis**

**HLA-B51 = HLA-B27 antigen**
In case of an anterior uveitis with hypopyon signs and symptoms found in Behçet's syndrome should be searched for, in particular oral and/or genital ulcerations, cutaneous signs such as erythema nodosum and pustules, arthralgies, thrombophlebitis or central nervous system involvement. If Behçet's uveitis is suspected we find it useful to look for the HLA-B51 antigen that, when present, represents an additional argument for the diagnosis of Behçet's uveitis, especially in the milder forms of Behçet's uveitis seen in the European caucasian population. Isolated anterior Behçet's uveitis can occur but posterior involvement should be searched for by funduscopy and is best investigated by performing a fluorescein angiography looking for retinal vasculitis.

In case of non-granulomatous uveitis in children (with or without band keratopathy) history should be directed towards juvenile idiopathic arthritis (JIA). Inflammatory symptoms can be completely absent, contrasting with the severe signs of uveitis such as hypopyon and extensive synechiae that can characterise JIA related uveitis. Uveitis is usually associated with the pauciarticular form of JIA and testing should include anti-nuclear antibodies (ANA) that are present in up to 70% of JRA patients with uveitis. In elderly children it is also useful to test for the presence of HLA-B27 antigen. A bilateral non granulomatous uveitis in children, but also in adults, should prompt to search or exclude tubulointerstitial nephritis and uveitis syndrome (TINU), an often neglected diagnosis. Renal function should be tested, starting with the dosage of creatininemia requiring sometimes renal biopsy and urinalysis should be performed looking for glucosuria and dosage of beta-2-microglobulin which is found to be elevated in TINU.5

In children pars planitis can initially present with a pronounced anterior participation and can be mistaken for an anterior uveitis if the posterior segment is not carefully analyzed.

In case of non-responding HLA-B27 negative anterior uveitis or in case of recurrence we pursue the work-up in the same fashion as for a granulomatous uveitis

**Work-up of granulomatous uveitis**

Before starting the work-up of granulomatous uveitis, it is important to exclude Fuchs' heterochromic uveitis because this condition, when sufficiently typical, does not need any work-up. Furthermore corticosteroid treatment should be withheld in Fuchs' uveitis to avoid the side-effects of a treatment that usually has no impact on the inflammatory process. Characteristic findings of Fuchs' uveitis include fine stellate granulomatous keratic precipitates that usually do not accumulate inferiorly by gravitation but are more uniformly distributed over the whole surface of the endothelium,( Figure 6) fine Koepppe nodules at the pupillary edge of the the iris, prominent vessels in the irido-corneal angle seen by gonioscopy and absence of posterior synechiae. Heterochromia is only present in fair-coloured irises but not in dark irises. Therefore the name of this entity should not include any more the term of heterochromic and should simply be called Fuchs' uveitis. It should be kept in mind that involvement can be bilateral. Laser flare photometry shows a
very moderate breakdown of blood-aqueous barrier of 10.2±3.5 ph/ms (normal 3.5-4.0 ph/ms) that remains relatively stable over time and usually does not respond significantly to anti-inflammatory treatment.

Spill-over anterior granulomatous uveitis as it can occur in very inflammatory toxoplasmic retinochoroiditis has to be excluded by performing a detailed examination of the posterior segment with funduscopy.

**Flow-chart 3 : diagnostic steps in granulomatous uveitis.**

**Granulomatous uveitis : systematic diagnostic approach 1**

**Granulomatous uveitis**

**Step 1**
exclude Fuchs’ uveitis

- small stellate KPs, non gravitational, regularly distributed over the cornea
- vitreous infiltration
- usually unilateral
- Koepple nodules possible
- polar posterior cataract
- heterochromia

**Step 2 : first-line lab. tests**

- Lysozyme (L)
- Angiotensin converting enzyme (ACE)
- Tuberculin skin test
- Multiple herpetic serologies (polyclonal activation)

**Serum L & ACE elevated**

- DTH anergy
- Herpetic serologies show polyclonal activation

**Sarcoidosis**

most probable

**Tuberculosis**

probable

**Chest X-ray &/or CT-scan, BAL, Biopsy, Ga-scan**

**Chest X-ray, TB cultures**

BAL = bronchio-alveolar lavage ; DTH = delayed type hypersensitivity ; Ga-scan = galium scan ; IFN = interferon; TB = tuberculosis
The first-line laboratory tests performed to investigate granulomatous uveitis are serum angiotensine converting enzyme (ACE) and lysozyme, products indicating the presence of granulomatous lesions. ACE can be normally elevated in children and serum lysozyme levels tend to be progressively more elevated in elderly persons. It is therefore important to perform both tests.

The second step is to differentiate between sarcoid, tuberculous or other granulomatous causes. In order to differentiate between tuberculosis and sarcoidosis, multiple skin tests, measuring delayed type hypersensitivity reaction to several antigens to which the adult patient should be normally reacting (MultiMérieux® containing tuberculin, streptococcus, diphteria, tetanos, tricophyton and candida antigens) are performed to search for anergy, a strong argument for sarcoidosis. This test is however rarely performed nowadays as the Merieux® multi-antigen applicator is difficult to find. In a patient vaccinated for tuberculosis or that is known to have been exposed to a tuberculous infection a PPD skin test that has become negative has the same diagnostic value. We find it also useful to look for polyclonal antibody activation that is present in up to 85% of the patients with sarcoidosis and was also found in patients with ocular sarcoidosis. For this purpose, serologies to four herpes viruses to which most of the adult population has been exposed (herpes simplex, herpes zoster, cytomegalovirus and Epstein-Barr) are performed. ELISA serology detects exposure to these viruses and complement fixation serology is done to establish whether the antibody titers are elevated. An isolated elevated titer to one virus only might be indicative of a viral etiology. Polyclonal activation however is an additional element for sarcoidosis. This non-specific antibody elevation is the cause of some of the false-positive diagnosis of presumed infectious uveitis, relying only on a serology such as Lyme borreliosis. A positive serology is not a confirmation of ocular Lyme disease. We followed five cases with uveitis and a positive Lyme serology that had negative anterior chamber antibody ratios (Goldmann-Witmer coefficient) and for whom the diagnosis was finally sarcoidosis. Patients with a compatible clinical picture and positive ACE & lysozyme tests in presence of cutaneous anergy have a probability of over 95% to have ocular sarcoidosis.

On the other hand when the PPD tuberculin skin test is hyperpositive this should raise the suspicion of a tuberculous granulomatous uveitis. Nowadays the next test to be performed is one of the gamma-interferon releasing assay that are testing blood lymphocytes of patients in order to detect whether there are lymphocytes reacting in vitro when put in presence with specific proteins coming from Mycobacterium Tuberculosis. When the patient’s lymphocytes release gamma-Interferon, it means that the patient has been exposed to the bacteria and tuberculosis should be actively researched.

Syphils serology is performed either routinely or in case of a positive history. In case of undefined diagnosis, serology for Lyme borreliosis is performed with the known limitations of the value of a positive serology.

Toxoplasmic retinochoroiditis can sometimes present as a granulomatous (hypertensive) anterior spill-over uveitis. The presence of a retinal focus orients clearly into this direction. In order to make the diagnosis possible toxoplasmic serology should be performed shown simply the presence IgG antibodies indicating that the patient has been in contact once in his life with Toxoplasma Gondii.
In case of negative ACE / lysozyme test and a non-contributary skin test a herpetic uveitis should be suspected. Clinical signs that are very suggestive of herpes simplex / zoster uveitis are ocular hypertension and iris atrophy (found both in herpes simplex and varicella-zoster uveitis). Laboratory confirmation of herpes simplex / zoster anterior uveitis can be obtained by the detection of intraocular production of antibodies in the aqueous humor (Goldmann-Witmer coefficient). Aqueous paracentesis is however not performed routinely in these cases but reserved for sight-threatening diseases such as necrotic herpetic retinopathies (NHR) that include acute retinal necrosis. It is also performed in uveitis suspected to be herpetic but that does not reapond to classical combined systemic antiviral and topical antiviral therapy to detect CMV DNA in the aqueous.

A condition that can be associated with anterior granulomatous uveitis is multiple sclerosis (MS). In most cases posterior segment findings such as pithlebitis and vitritis are usually present. In patients with a history compatible with MS investigations should be directed towards MS beginning with a cerebral MRI.11

**Figure 10. Laser flare photometry.** This machine is measuring the content of proteins in the anterior chamber and hence the level of inflammation in an objective and quantitative fashion. The instrument shines a laser into the anterior chamber and the electronic detector is counting the number of photons that are back-scattered. Back-scattered photons are proportional to the amount of particles (proteins) and hence proportional to the intraocular inflammation.

Granulomatous uveitis: systematic diagnostic approach 2

**Granulomatous uveitis**

Exclude Fuchs Uveitis !!!

**First-line laboratory tests**
- Lysozyme (L)
- Angiotensin converting enzyme (ACE)
- PPD TB skin test
- Multiple herpetic serologies (polyclonal activation)

**Sarcoidosis**

ACE & L positive + anergy

**Tuberculous uveitis** (chorioretinitis)

PPD ++++ and +tive G-IFN releasing test

**Syphilis serology**

ACE, L negative

**Syphilitic uveitis** (chorioretinitis)

**Lyme serologies**

**R-choroidal focus**
Toxopl. Gondii Abs +

**Lyme uveitis**

**Toxoplasmatic retinochoroiditis**

**HSV/VZV uveitis probable**

L & ACE normal
DTH non-contributive
+tive herpes serology
Intraocular HT
Iris atrophy
Unilateral

Resists usual ttt: CMV uveitis

CMV = cytomegalovirus; DTH = delayed type hypersensitivity; G-IFN = gamma-interferon; HT = hypertension; TB = tuberculosis; ttt = treatment
References