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# VITRITIS IN INDIAN POPULATION

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## INTRODUCTION

Vitritis, or more accurately known as vitreous humor inflammation, is an important clinical entity. It is the most consistent sign of intermediate uveitis (cyclitis) and can also occur in association with certain non-uveitis entities including intraocular lymphoma, trauma, etc. The inflammation could be induced by an infectious agent or trauma, but in majority the underlying mechanisms appear to be autoimmune in nature. The term 'Intermediate uveitis' encompasses the inflammation of pars plana that could be of various etiologies including sarcoidosis, tuberculosis, etc. The term 'pars planitis' is used when the intermediate uveitis is idiopathic in nature.

## CLINICAL PRESENTATION

Patients complain of floaters or blurry vision. Pain, photophobia and redness are unusual. Children may be more prone to develop anterior segment inflammation including posterior synechiae<sup>1,2</sup> and band keratopathy<sup>3,4</sup>. Vitritis is the most consistent sign of intermediate uveitis. The vitreous humor cellular reaction has the appearance of "dust" at the slit lamp biomicroscope<sup>5</sup>. Vitritis may become so dense so as to obscure the retina entirely<sup>6,7</sup> and cause profound loss of vision. These white cells are present in both the anterior and posterior vitreous (diffuse clouding). Examination of posterior vitreous humor is necessary to evaluate fully the degree of vitritis, as it is usually most prominent posteriorly. Characteristic mobile, round, white, focal vitreous humor opacities, dubbed "oeufs de formi" (ant's eggs) in French<sup>8</sup>, can be seen in the inferior peripheral vitreous. They lie close to the retina but are not in contact with it. They are not specific for pars planitis and may occur with any kind of inflammation of the peripheral fundus or with an extensive and diffuse uveitis<sup>9</sup>. Later, the vitreous shows degenerative changes with fibre-like cylindrical condensations of coarse vitreous strands<sup>10,11</sup>. Posterior vitreous detachment is common.

The hallmark of pars planitis are the white or yellowish-white exudates (posterior hypopyon) and collagen bands (snow banks) over the pars plana, with extension into the ora serrata and peripheral retina.

They are typically located inferiorly, but may also be found superiorly or divided into multiple foci.

Chronicity produces gliosis and collagen deposition similar to that seen in preretinal membranes<sup>12,13</sup>.

## DIFFERENTIAL DIAGNOSIS OF VITRITIS

Vitritis could be due to infectious or non-infectious etiologies. Certain non-uveitis conditions might present as vitritis and mimic chronic uveitis. These are termed as 'masquerade syndromes'.

### Infectious causes

1. Tuberculosis
2. Syphilis
3. Toxoplasmosis
4. Toxocariasis
5. Lyme disease
6. Endogenous endophthalmitis
7. Parasitic infestations e.g., cysticercosis

### Non-infectious causes

1. Sarcoidosis
2. Behcet's disease
3. Multiple sclerosis
4. Inflammatory bowel disease
5. Fuch's heterochromic uveitis
6. Collagen vascular disease
7. Eales' disease

### Masquerade syndromes

1. CNS lymphoma
2. Other tumors e.g., Retinoblastoma, malignant melanoma, medulloepithelioma
3. Metastasis from breast, lung or renal carcinomas into the eye
4. Paraneoplastic syndromes

Following are some of the common entities:

### Intermediate Uveitis

Intermediate uveitis is a common type of uveitis in children and young adults. It is one of the four major categories of uveitis in the classification scheme proposed by the International Uveitis Study Group<sup>14</sup>. This classification scheme subdivides uveitis into anterior, intermediate, posterior and panuveitis based on the principal anatomic site of inflammation. The diagnosis of intermediate uveitis is made when intraocular inflammation primarily involves the vitreous, peripheral retina and pars plana ciliaris.

The term intermediate uveitis has been designated by the International Uveitis Study Group for the entities that have been previously called cyclitis, peripheral uveitis, chronic cyclitis, vitritis and pars planitis<sup>15-21</sup>.

### Epidemiology

In a large series from uveitis referral practices, intermediate uveitis represented 4-25% of patients<sup>22, 23-26</sup>. Pars planitis occurs in patients between the ages of 5-40 years and has a bimodal distribution with a young group in the age range 5-15 years and an older group in the age range 20-40 years. There has been no striking sex predilection or familial tendency.

In our population from North India, it constituted 198 of 1233(16.06%) patients of uveitis seen between January 1996 and June 2001. Out of these, 181(91.4%) were idiopathic in nature, tuberculosis was the etiology in 8(4%) cases, sarcoidosis in 8(4%) and toxoplasmosis in 1(0.5%) case<sup>27</sup>.

### Pathogenesis

The pathogenesis of intermediate uveitis largely is unknown. Autoimmune reactions against vitreous, peripheral retina and ciliary body have been proposed. Several studies have looked for human leukocyte antigen (HLA) associations in Intermediate Uveitis. Davis et al found associations with HLA – DR 2 and HLA – DQ1<sup>28</sup>.

### Clinical features

Patients complain of floaters or blurred vision.

Vitritis is the most consistent sign of intermediate uveitis. Presence of the characteristic pars plana exudates (snow balls) and snowbanking facilitates diagnosis. Eyes with exudates have been considered to be more severely involved<sup>29</sup>.

Cuffing of retinal vessels with inflammatory cells is a common feature and there is occasionally obliterative vasculitis. These changes are most common in the terminal branches of retinal veins, and arterioles are rarely involved<sup>30</sup>. Neovascularisation of the disc (NVD), of the retinal periphery (NVE), or of the iris (NVI) can occur in most severe disease. NVD or NVE may lead to vitreous hemorrhage and NVI to neovascular glaucoma.

Papillitis and optic nerve edema are uncommon. Cystoid macular edema (CME) is common in pars planitis, being the most common cause of decreased visual acuity<sup>31-34</sup>. Vitreous traction may be involved in the generation of CME. Cataract is common in protracted pars planitis. Retinal traction, retinal detachment, subretinal exudation with serous retinal detachment are rare.

### Aetiology

As in any case of uveitis, it is important to rule out following causes:

#### Tuberculosis

Tuberculosis may affect any ocular structure. Tuberculous uveitis may appear as a chronic granulomatous iridocyclitis, peripheral uveitis, disseminated choroiditis or panuveitis. The associated iridocyclitis is usually granulomatous, characterized by mutton-fat KPs and extensive posterior synechiae. The presence of choroidal tubercles classically suggests hematogenous dissemination. Retinal periphlebitis in TB is usually a result of extension from choroidal disease. An immune mechanism has been proposed in the pathogenesis of retinal periphlebitis in patients with tuberculin hypersensitivity. The diagnosis is based on clinical features with a positive tuberculin skin test, pulmonary radiographic evidence of TB, PCR positivity from intraocular fluids and a response to empiric anti-tuberculous therapy.

#### Toxoplasma Retinochoroiditis

Active toxoplasma lesions are round or oval, yellow-white, adjacent to a pigmented old scar or satellite lesions and are usually in the posterior retina. Peripheral lesions have been described, including a wide ring-like lesion near the extreme periphery, resembling the snowbanking seen in pars planitis<sup>35,36</sup>. Active toxoplasmosis may cause such a heavy vitreal

reaction that the retinal lesion itself cannot be directly visualized ("headlight in the fog"). There may be "spillover" anterior segment inflammation with small to medium-sized, round white or large, mutton-fat KPs in the cornea. It is usually acute in onset in contrast to the insidious onset in intermediate uveitis. Diffuse or segmental vasculitis may be seen.

### **Toxocariasis**

In children, toxocariasis is a major diagnostic consideration when dealing with unilateral intermediate uveitis<sup>37</sup>. Patients with ocular toxocariasis are usually free of systemic findings. The characteristic unilateral chronic endophthalmitis with peripheral granuloma and tractional bands extending from the disc to the posterior pole is distinct from intermediate uveitis. History of infected puppies or pica and serological testing (ELISA) for toxocara antigen are extremely helpful.

### **Sarcoidosis**

Sarcoidosis should be suspected in all patients with intermediate uveitis. Unlike pars planitis, sarcoid uveitis is slightly more common in females, with an older age of presentation<sup>38</sup>. Chest radiograph, serum ACE levels, serum lysozyme and gallium scans support the diagnosis, but only tissue biopsy (transbronchial lung biopsy) confirms it.

### **Behcet's Disease**

Characterized mainly by its retinal vascular involvement, Behcet's disease has usually bilateral ocular involvement with recurrent episodes of uveitis<sup>39</sup>. An anterior uveitis is frequent with an associated hypopyon. An isolated vitritis is not characteristic but is significant. HLA typing (HLA-B 51), a compatible history and systemic evaluation are helpful.

### **Syphilis**

Anterior uveitis is the most common finding of secondary syphilis<sup>40</sup>. The pathogenesis of uveitis appears to be related to both infectious and immunogenic components. Posterior uveitis can occur as diffuse or localized choroiditis or chorioretinitis<sup>41</sup>, most often affecting the posterior pole<sup>42</sup> and juxtapapillary area<sup>43</sup>. Progressive posterior uveitis can lead to disc edema, vitritis, retinal vasculitis, uveal effusion and exudative retinal detachment<sup>44</sup>. Late syphilis may present with anterior or posterior uveitis.

Vitritis as the primary manifestation of ocular syphilis in patients with HIV infection:

HIV positive patients with syphilis may present with atypically dense vitritis. In these patients, vitritis may be the first manifestation of syphilis. The regimen for neurosyphilis provides effective therapy. Moreover, in some patients, syphilitic vitritis may be the initial manifestation of HIV disease.

### **Lyme Disease**

It may present with unilateral or bilateral uveitis which usually resolves rapidly on treatment with antibiotics. Rarely, it can cause intermediate uveitis. Serology for *Borrelia burgdorferi* should be performed if suspicion is high based on exposure or review of systems which include erythema migrans like skin rash, cardiac conduction defects, and cranial nerve palsies.

### **Endogenous endophthalmitis**

Endogenous infections, bacterial or fungal, reach the eye via the bloodstream, usually as part of a disseminated infection. The infective element initiates a focal choroiditis, retinitis or cyclitis. When this initial focus of infection extends into the vitreous, it produces inflammation and involves all structures of the eye, producing endogenous endophthalmitis. Vitreous inflammation may be so severe that it obscures view of the fundus and makes clinical diagnoses difficult. Sudden onset and a compatible history are helpful in making a correct diagnosis<sup>45</sup>.

### **Cysticercosis**

Although any structure within or around the eye can be involved in ocular cysticercosis, the vitreous and retina are most likely affected<sup>46</sup>. The parasite may be present within the vitreous or in the subretinal space<sup>47</sup>. Vitritis is usually severe in ocular cysticercosis.

### **Multiple Sclerosis**

Patients with multiple sclerosis may develop ocular disease resembling other forms of intermediate uveitis. Retinal periphlebitis and vitritis have been reported to occur in 5-20% of patients of multiple sclerosis<sup>48</sup>. Breger suggested an autoimmune disorder directed against an unknown antigen, common to both eye and myelin nerve sheath<sup>49</sup>. Multiple sclerosis does not have to be active for intermediate uveitis to be present. Intermediate uveitis may precede multiple sclerosis by more than 7 years<sup>50</sup>. A subset of intermediate

uveitis patients may be at a risk for the subsequent development of multiple sclerosis<sup>51</sup>.

#### **Fuch's Heterochromic cyclitis**

It can produce a unilateral, chronic, low grade anterior segment inflammation with spillover of the cells into the vitreous cavity. There is no snowbank. Multiple stellate keratic precipitates and heterochromia iridis constitute this diagnosis. Posterior synechiae preclude the diagnosis. Vitritis may be significant to cause CME.

Transient Vitreous Inflammatory Reactions associated with combination antiretroviral therapy in patients with AIDS and Cytomegalovirus Retinitis

Patients with AIDS and CMV Retinitis may develop transient intraocular inflammation associated with combination antiretroviral therapy. This inflammation reflects an improved immune response against cytomegalovirus.

#### **Masquerade Syndromes**

These comprise a group of disorders that occur with intraocular inflammation and are often misdiagnosed as chronic idiopathic uveitis. Apart from certain non-malignant conditions masquerading as uveitis, many of the masquerade syndromes are malignant processes.

#### **Intraocular Lymphoma**

Vitritis without fundus lesions has been reported with intraocular lymphoma<sup>52,53</sup>. Elderly patients presenting with vitreous cells may be indicative of intraocular lymphoma. Diagnostic vitrectomy, cytological evaluation of CSF and neuroimaging are necessary for establishing diagnosis.

#### **Other malignant conditions**

Tumors such as retinoblastoma, malignant melanoma or medulloepithelioma can disseminate into the vitreous and simulate intermediate uveitis. Atypical vitreous cells and a mass, detectable by funduscopy or ultrasonography constitute these diagnosis. In children, apart from retinoblastoma and medulloepithelioma, acute myelogenous leukemia and juvenile xanthogranuloma can masquerade as uveitis.

Breast, lung and renal carcinomas can metastasize to the choroid and produce signs and symptoms of uveitis. Such patients need a complete medical evaluation to

detect the primary cancer. Vitreous biopsy may reveal the presence of malignant cells.

#### **Paraneoplastic Syndromes:**

Cancer-associated retinopathy (CAR) may manifest as uveitis. Histologically, there is destruction of photoreceptors<sup>54</sup> which is thought to be immune-mediated. Visual loss may be explained by optic disc pallor, vascular sheathing and RPE disturbances.

Bilateral Diffuse Uveal Melanocytic Proliferation (BDUMP) is another paraneoplastic disorder simulating as uveitis, found in association with a systemic malignant process. Multiple, round, raised, subretinal red patches with or without exudative retinal detachment and vitreous cells can be seen.

#### **LABORATORY INVESTIGATIONS**

The diagnosis of intermediate uveitis is clinical. Routine tests should include a complete blood count to look for WBC abnormalities (as in a malignant masquerade syndrome), erythrocyte sedimentation rate, a tuberculin skin test, chest radiograph to screen for tuberculosis and sarcoidosis, and TPHA for syphilis. Fluorecein angiography is useful for retinal or choroidal pathologies, subtle CME and capillary non-perfusion.

Serological tests (ELISA) may be indicated for toxoplasmosis, toxocariasis, HIV and Lyme disease. Aqueous tap and Vitreous biopsy may be required for cytological or microbiological evaluation and Polymerase chain reaction. In suspected cases of endogenous endophthalmitis, blood culture and urine culture may prove contributory. MRI may be required to look for multiple sclerosis. Cranial imaging with lumbar puncture are indicated to rule out large cell lymphoma.

#### **TREATMENT**

The goal of the treatment is to ameliorate vision threatening complications secondary to intermediate uveitis like CME, cataract, glaucoma and exudative retinal detachment.

If a specific cause of intermediate uveitis is diagnosed (such as tuberculosis, sarcoidosis, toxoplasmosis, syphilis), then the treatment is directed against the particular disorder and a relevant medical consultation is sought for systemic involvement.

The mainstay of the treatment of intermediate uveitis is periocular corticosteroid injections or oral steroids. Posterior sub-tenon Triamcinolone Acetonide injection may be given every 6-8 weeks until resolution of CME or return of 20/20 visual acuity. Giving the injection superotemporally in the sub-Tenon space as far posteriorly and close to the globe as possible, results in a deposit of the drug closer to the macula. Posterior injection also reduces the risk of intraocular pressure rise.

When a series of periocular injections have failed, oral Prednisolone in the dose of 1.0 mg/kg daily are given and tapered over 6-12 weeks, depending upon the response. All possible complications of corticosteroids should be looked for on follow-ups. Topical betamethasone and cycloplegics are given for associated anterior segment inflammation.

Severe, bilateral, uncontrolled cases of vitritis and patients who are intolerant to corticosteroids may require the use of immunosuppressive agents. Cyclophosphamides, Methotrexate, Azathioprine, Chlorambucil and Cyclosporin A have proven to be effective. But these agents have serious systemic side effects which should be monitored by an experienced physician. Very severe cases may need Combination therapy (Prednisolone plus immunosuppressives, or two or three different immunosuppressives).

### **Surgical management**

Pars plana vitrectomy may be indicated for diagnostic as well as therapeutic purpose to remove vitreous antigens and inflammatory cells and mediators, playing an important role in CME.

Other indications of vitrectomy, although rare, include vitreous opacification, cataract, tractional retinal detachment, epiretinal membranes and vitreous hemorrhage.

### **Specific causes**

Cases diagnosed as intraocular tuberculosis are treated with a course of anti-tubercular therapy (4 drug regimen) and oral steroids. ATT comprises of Isoniazid (5-10mg/kg/day), Rifampicin (10mg/kg/day), Ethambutol (10-15mg/kg/day) and Pyrazinamide (15-20mg/kg/day) with Pyridoxine (10mg/day) supplementation. Ethambutol and Pyrazinamide are discontinued after 4 months and patient is maintained on 2 drug-regimen (INH and Rifampicin) for upto a

total of 18 months. Oral steroids are tapered over a period of 6-8 weeks. ATT, in our experience, reduces the recurrence rate.

The "classic" antimicrobial therapy for active ocular toxoplasmosis consists of a combination of pyrimethamine, sulphadiazine and corticosteroids with Folic acid supplementation. Initiation of oral corticosteroid therapy is delayed for 24-48 hours after starting antimicrobial agents to allow adequate blood levels of antimicrobials<sup>55</sup>. The other approach is to use Clindamycin in a dose of 300 mg QID as the sole antimicrobial agent in combination with corticosteroids<sup>56</sup>, started a day before oral corticosteroids and continued for a few days after tapering off steroids for a total period of 6 weeks. Clindamycin appears to be concentrated in ocular tissue and can penetrate tissue cyst walls.

Penicillin remains the treatment of choice for syphilis<sup>57</sup>. Patients with active syphilitic uveitis are treated as for neurosyphilis-Penicillin G Sodium 2-4 million units every 4 hours intravenous for 2 weeks after a negative skin test.

Core Vitrectomy with intravitreal antibiotics (antibacterial or anti fungal) along with administration of intravenous antibiotics is recommended for endogenous endophthalmitis.

Other specific indications for vitrectomy include intravitreal or subretinal cysticercosis, ocular toxocariasis for granuloma excision and tractional retinal detachment.

### **PROGNOSIS**

The visual prognosis depends upon status of the macula and vitreous opacification in the early course of the disease. Cataract, secondary glaucoma and tractional retinal detachment affect the visual outcome in later stages.

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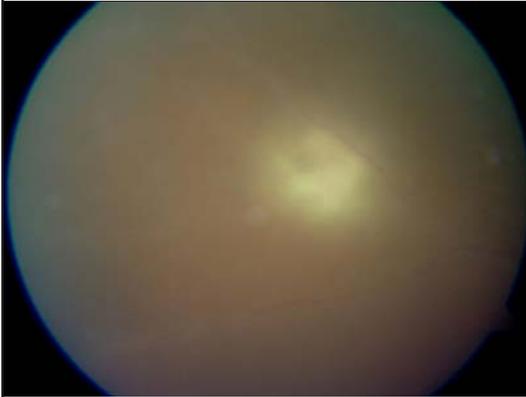


Fig.1. A 26-years old male with a "headlight in the fog" appearance OD.ELISA was positive for IgM in the vitreous and serum.Treated with Clindamycin and oral corticosteroids.vision improved from 6/60 to 6/12.

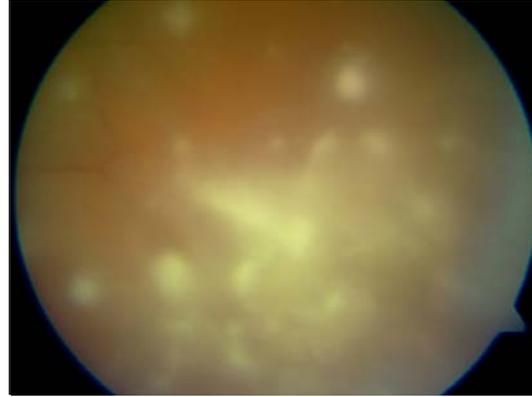


Fig.2. An 18-years old male with endogenous endophthalmitis OS.Pars plana vitrectomy was done and intravitreal antibiotics given.Vision improved from 6/36 to 6/6.

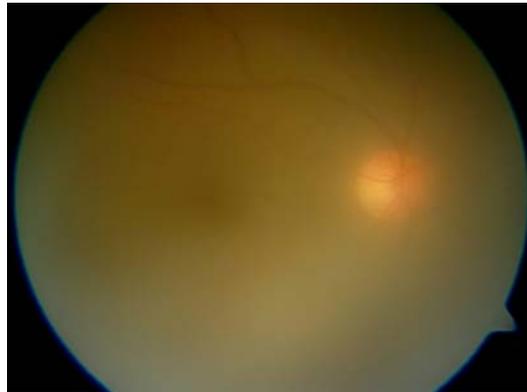


Fig.3 . A 50-years old male with severe vitritis OD.Vision was 4/60.Routine investigations were normal . Vitreous biopsy revealed B- cell Lymphoma.MRI ruled out CNS lymphoma.Pars plana vitrectomy was done and intravitreal methotrexate given.Vision improved to 6/12.