Uveitis in children is an important cause of ocular morbidity and severe vision loss. The diagnosis and the management are challenging in such population due to some unique presentations. Majority of the cases are idiopathic with infectious causes contributing to about one fifth of the cases. Various causes of infectious uveitis include tuberculosis, toxoplasmosis, toxocariasis. Anterior uveitis is the most common anatomical type of uveitis in children followed by almost equal incidence of posterior, intermediate and panuveitis. The examination is often difficult in children and diagnostic evaluation is challenging. The management requires multidisciplinary approach involving the rheumatologist and uveitis specialist. Exclusion of infectious and masquerade causes is of utmost importance before starting any immunomodulatory treatment. The primary aim of the treatment is to reduce the inflammation as early as possible to prevent complications. Corticosteroids are the main pillar stone for the management. The dosage and the route of the steroids should be optimized according to the growth and development of the child as it may have to be given for a longer duration. Steroid sparing agents such as methotrexate, cyclosporine, azathioprine and biologic agents are reserved for children refractory to treatment with steroids. Children tend to develop complications of uveitis more often than in adults, which include band keratopathy, cataract, glaucoma, cystoid macular edema, amblyopia and strabismus.

Uveitis in children constitutes of 4-10% of all uveitis population. Uveitis in such population is often challenging in terms of diagnosis and management as well as the spectrum of complications associated with it.

The diagnosis of uveitis in children may be delayed due to the varied presentation of uveitis. The anterior uveitis often is asymptomatic without the classical symptoms of pain, redness or photophobia (white iris). Strabismus and leukocoria may be the presenting symptoms which can cause misdiagnosis and delay in treatment. Also, the examination in children is often difficult. Examination under anaesthesia may be required for complete examination of the child. Some of the masquerade syndromes such as retinoblastoma, leukemia, juvenile xanthogranuloma may present as uveitis as a primary presentation. Therefore, the approach to uveitis in children is very different as compared with that in adults.

Epidemiology
Based on the anatomical site of involvement, anterior uveitis is the most common type of uveitis in children. Posterior, intermediate and panuveitis constitute almost equal percentage of pediatric uveitis. Majority of the pediatric uveitis cases are idiopathic. Juvenile idiopathic arthritis is the most common known etiology overall. Infectious uveitis constitute nearly one-fifth of the cases. Among the infectious cases, tuberculosis and toxoplasmosis are the most common etiologies. Intermediate uveitis is commonly idiopathic in children. Bilateral involvement is the norm in pediatric uveitis with males being affected slightly more than the females. The etiologies of pediatric uveitis are mentioned in (Table 1) and the important causes are being considered in the following section.

Juvenile Idiopathic Arthritis
It is a group of arthritis, which often presents below the age of 16 years and persists for at least 6 weeks. Unlike the adult onset rheumatoid arthritis which has symmetric joint involvement, characteristic rheumatoid nodules and a chronic inflammatory course, JIA has asymmetric joint involvement and the children outgrow the disease with time. It is one of the common causes of anterior uveitis in children with prevalence of uveitis in patients of JIA varying between 14 to 24%. The oligoarticular subtype accounts of 50% of the JIA cases and is diagnosed when fewer than 5 joints are involved during the first 6 months of the disease. It is more common in females and the onset is usually before 6 years of age. Chronic anterior uveitis (CAU) is most commonly associated with oligoarticular JIA. Approximately 30% to 50% of oligoarticular JIA patients develop CAU. The polyarticular subtype is diagnosed when more than 4 joints are involved in the first 6 months of disease. It is more common in females and can be further subdivided based on rheumatoid factor (RF) results. RF positive disease mimic adult onset rheumatoid arthritis and is not associated with uveitis. RF negative polyarticular JIA is associated with uveitis in 5% to 10% of cases. Systemic disease or Still disease is seen in children less than 5 years of age and affect both sexes equally. Uveitis develops in less than 5% of the cases.

Psoriatic arthritis and enthesitis-related arthritis are less common subtypes of JIA which may also be associated with CAU.

Major risk factors for developing uveitis in patients with JIA are female sex, antinuclear antibody (ANA) seropositivity, oligoarticular arthritis, rheumatoid factor sero-negativity and early (less than 6 years) age of onset of arthritis.

Clinical features: The most common extra articular manifestation of JIA is uveitis. Uveitis in JIA is usually bilateral, anterior, non-granulomatous uveitis with a chronic relapsing course. Children with CAU associated with JIA are typically asymptomatic and therefore routine ophthalmologic screening is essential for early diagnosis and
timely treatment. Otherwise, affected subjects may present after development of serious ocular complications and severe visual impairment. These may include complicated cataract, band shaped keratopathy, hypotony, and phthisis bulbi.

Screening guidelines have been developed; patients with oligo- or polyarticular JIA with onset of arthritis at 6 years of age or younger, duration of arthritis 4 years or less, and ANA-seropositivity should be screened for uveitis within 6 weeks of diagnosis and then at 3-month intervals.8

**Seronegative spondyloarthopathies (SpA)**

These include ankylosing spondylitis, reactive arthritis, psoriatic arthritis, and enteropathic arthritis. The common systemic findings are axial arthritis and enthesitis. Adolescent males are more commonly involved.

Clinical features: Ocular manifestation is with unilateral acute anterior uveitis, occurring in one-fourth of the patients. The diagnosis is made based on presence of systemic findings supportive of SpA and negative rheumatoid factor.9

**Tubulointerstitial nephritis and uveitis**

Tubulointerstitial nephritis and uveitis (TINU) is a rare immune mediated entity occurring in children with female preponderance.10 The median age of onset is 15 years.

Clinical features: The child may have fever, flank tenderness, anorexia, and weight loss. Ocular presentation may be bilateral non-granulomatous anterior uveitis or posterior uveitis in form of chorioretinitis and multifocal choroiditis.11 Suspected cases may be advised urinalysis which shows presence of glycosuria, proteinuria, aminoaciduria, and microscopic hematuria. Renal biopsy is confirmatory. Although the disease usually resolves spontaneously, systemic steroids are often used to prevent structural renal damage.

**Herpetic Anterior Uveitis**

Herpes simplex virus (HSV) and varicella zoster virus (VZV) can cause unilateral acute granulomatous or non-granulomatous anterior uveitis in children.12 Patchy or sectoral iris atrophy may occur. The intraocular pressure may be high in herpetic infections contrary to low intraocular pressure seen in non-infectious anterior uveitis.

VZV anterior uveitis is more severe and results in segmental iris atrophy (from occlusive vasculitis) and pupillary distortion as compared to relatively mild uveitis with round iris atrophy in HSV infection.

Definitive diagnosis requires polymerase chain reaction (PCR) of the aqueous humor sample to detect the viral DNA.

**Idiopathic Intermediate Uveitis (Pars Planitis)**

According to the anatomic classification of uveitis by the Standardization of Uveitis Nomenclature (SUN) Working Group, the term “intermediate uveitis” defines a subset of uveitis where the vitreous is the primary site of inflammation. Pars planitis is a diagnostic term that defines a subset of idiopathic intermediate uveitis where there is snowbank or snowball formation.13

It usually affects children and adolescents. Various associations with HLA-DR2 and HLA-DR15 have been reported.14

Clinical features: Children with pars planitis may be asymptomatic and are usually diagnosed during a routine examination. Some children are diagnosed only after significant visual impairment or the development of complications that cause leukocoria or strabismus. Typical clinical findings include mild to moderate anterior segment inflammation, diffuse vitreous cells and haze, snowballs and snowbanks located inferiorly. Peripheral retinal vasculitis may also be present. Band keratopathy, peripheral corneal endotheliopathy, and posterior synechiae may be seen in childhood pars planitis but are very rare in adults. Optic disc edema and cystoid macular edema are the most frequent complications.15

Dense vitreous condensation may sometimes cause leukocoria. Vitreous haze and cataract may cause amblyopia in a young child with pars planitis. Cystoid macular edema is the leading cause of visual morbidity in intermediate uveitis.15

**Behçet Uveitis**

Although Behçet disease is more common in third and fourth decade of life, it may occur in childhood as well. There are no internationally accepted diagnostic criteria for childhood-onset Behçet disease. Recurrent oral ulcers are the most common presentation of Behçet disease in children. Uveitis is less common (34%) in this group than in adults.16 The age of presentation in pediatric Behçet disease is in adolescence (10-15 years).17 There is a male predominance in the pediatric age group, similar to adult-onset Behçet uveitis.17 A positive family history has been reported in 20–47% of pediatric cases from endemic areas, implying the role of genetic factors in the early onset of the Behçet disease.18 HLA-B51 haplotype is seen in 60% of the cases. B*5101 is the most common allele in this serotype.

Clinical features: The majority of patients have bilateral involvement and recurrent panuveitis with retinal vasculitis. Cataract, intraocular pressure elevation, macular edema, and optic atrophy are the most common complications.18

**Sarcoidosis**

Childhood sarcoidosis is a multisystemic granulomatous inflammatory disorder. While older children may present with pulmonary involvement, young children with early-onset non-hereditary sarcoidosis (onset before age 5, familial autosomal dominant Blau syndrome) typically present with a triad of arthritis (involving knee and wrist), skin lesions and anterior uveitis.19
Clinical features: Anterior uveitis is the most common presentation in children with sarcoidosis. Granulomatous KPs, iris nodules, and peripheral and broad-based posterior synechiae are typical findings. Other than iris nodules at the pupillary margin (Koeppe nodule) and in the iris stroma (Busacca nodule), nodules may also be found in the anterior chamber angle (Berlin nodule). Uveitis in sarcoidosis has to be differentiated from JIA associated uveitis. Inflammation of the posterior segment in the form of retinal vasculitis or multifocal choroiditis can be seen in sarcoidosis; however, uveitis is typically non-granulomatous and confined to the anterior segment in JIA.

Serum angiotensin-converting enzyme levels may be misleading because children tend to have higher levels than adults. The diagnosis of sarcoidosis is usually clinical based on typical signs of ocular disease and laboratory abnormalities, although definitive diagnosis requires biopsy specimen showing non-caseating granulomatous inflammation.

Toxoplasmosis

Congenital toxoplasmosis occurs via transplacental transmission of *Toxoplasma gondii* especially when the mother acquires the disease later during the pregnancy. Congenital toxoplasmosis scars are an incidental finding and are usually bilateral (Figure 1). Acquired infection can occur in childhood but is unilateral. Reactivation of the congenital disease with active satellite lesions adjacent to the healed lesion is the primary presentation in childhood.

Clinical features: Ocular toxoplasmosis presents as focal necrotizing retinochoroiditis with vitritis (“headlight in fog” appearance) in children, similar to that seen in adults. It may also present as panuveitis. Complication such as choroidal neovascularization in the affected healed area is relatively more common in pediatric cases than adults.

The diagnosis is clinical with laboratory tests assisting in the diagnosis. Polymerase chain reaction and determination of Goldmann–Witmer coefficient using aqueous humour or serum are the important laboratory tools.

Toxocara

Ocular toxocariasis manifests in children and early adolescents. Infection is by ingestion of toxocara eggs. The egg matures into larvae and migrates in various tissue including the eye.

Clinical features: Ocular presentation is unilateral in 90% of the cases. Toxocara retinochoroiditis appears as a well demarcated, elevated mass lesion in the posterior pole with overlying vitritis. It may also present as peripheral granuloma or chronic dense vitritis mimicking endophthalmitis. Immunologic studies are generally not reliable for toxocariasis.

Tuberculosis

Clinical features: Ocular tuberculosis can have a variety of presentations in children including granulomatous anterior uveitis, intermediate uveitis, posterior uveitis in the form of choroidal tuberculosis, tuberculoma, subretinal abscess, serpigenoid choroiditis (Figure 2) or panuveitis. Broad based posterior synechiae, retinal vasculitis with perivascular choroiditis/scar and multifocal serpiginochoroiditis are suggestive of tubercular infection.
Laboratory diagnosis of intraocular TB is challenging in the children. Based on the clinical findings, microbiological results, immunologic tests and radiology features, the diagnosis of confirm TB, probable TB or possible TB is made. Mantoux test has limited use since there may be cross reactivity with antigens used in the test with previously administered BCG vaccines. Interferon gamma release assay (IGRA) is more specific. High resolution CT chest is more useful than X-ray in cases with suspected systemic focus of infection. Aqueous or vitreous PCR may help in confirming ocular infection in suspect cases but is less sensitive and not feasible in real world scenario.

**Masquerade Syndromes In Children**

Masquerade syndromes are one of the rare cause of uveitis in children. A high index of suspicion is required for the early diagnosis. These should be suspected in cases with incomplete or poor response with anti-inflammatory therapy. The causes include tumors such as retinoblastoma, leukemia and juvenile xanthogranuloma, congenital abnormalities such as Coats disease, retained intraocular foreign bodies following trauma and endogenous endophthalmitis.

**Diagnosis**

Early and correct diagnosis is a challenge for uveitis in children. The diagnosis may be difficult in preverbal and asymptomatic children. The examination in children is often difficult. Also, uveitis in children may have unique presentations which may cause misdiagnosis. Diagnostic approach vary for pediatric uveitis as compared to uveitis in adults.

**Management**

The primary aim of management is to reduce the active inflammation and hence decrease the complications associated with it. The management principles for uveitis are different in children as compared with adults. Exclusion of an infectious cause of uveitis and masquerade syndromes is of utmost importance before the administration of nonspecific anti-inflammatory and immunomodulatory treatment.

**Infectious uveitis**

The management of infectious uveitis requires tailored treatment approach. Treatment of herpetic anterior uveitis is with systemic antivirals (oral acyclovir 400 mg five times daily for 2-4 weeks) along with topical-corticosteroid and cycloplegic agents. In recurrent cases, oral prophylaxis (acyclovir 400mg twice a day) may be given to prevent relapse. Herpetic eye disease study–1 (HEDS-1), a randomized trial performed in 1990s, studied the efficacy of oral acyclovir (400 mg five times daily for 10 weeks) in addition to topical corticosteroids for treating HSV iridocyclitis. Although the originally planned recruitment could not be completed and the trial was stopped, the treatment failure was significantly less with additional oral acyclovir treatment. HEDS-2 study demonstrated the efficacy of low dose acyclovir prophylaxis (400mg twice a day for 1 year) in preventing recurrence of any form of HSV eye disease including iritis.

Toxoplasma retinochoroiditis is aimed at preventing multiplication of the protozoa. Treatment regimen includes pyrimethamine (2 mg/kg first day then 1 mg/kg each day) combined with sulfa-diazine (50-100mg/day in two divided doses) for 1 month. Folic acid supplementation (7.5 mg per day) is required along with anti-protozoal treatment. Trimethoprim and sulfamethoxazole combination in double strength may also be used for treatment. Intermittent therapy with trimethoprim-sulfamethoxazole combination is useful in preventing recurrences.

Steroids are the mainstay treatment for toxocara which is aimed at reducing the inflammation and preventing secondary complications such as development of tractional membranes and retinal detachment. Oral steroids are used in the dose of 0.5-1mg/kg/day. Anti-helminthic therapy is of unproven value in management; although few studies support the use of albendazole (15mg/kg in two divided dose) or thiabendazole to eradicate the organism.

The management of ocular tuberculosis is with 4 drug regimen (Rifampicin 15 mg/kg, maximum dose 600mg/day; Isoniazid 10 mg/kg, maximum dose 300 mg/day; Pyrazinamide 35 mg/kg; and ethambutol 20 mg/kg) for 6-9 months. Once diagnosis is made and treatment is started, it should not be stopped before 6 months because of lack of response, unless an alternative diagnosis is reached. This is because poor or suboptimal response can be due to immune reaction from death of the microorganism, which in addition needs anti-inflammatory therapy. Steroid are to be used judiciously under cover of anti-tubercular treatment, as it might lead to flare up or reactivation of disease if used alone. Treatment response is assessed at 2 months and at the end of treatment (6-9months) based on resolution of TB lesions and compatible uveitis.

**Non-Infectious Uveitis**

Corticosteroids remain first-line treatment for non-infectious uveitis in children. Topical corticosteroids are initially used for treatment of anterior segment inflammation. Periocular or subtenon corticosteroid injections may be used for treatment of intermediate or posterior uveitis, especially in unilateral cases or for the treatment cystoid macular edema. Systemic corticosteroids are used only for short-term treatment in children because of significant systemic side effects associated with their prolonged use, including Cushing syndrome, growth retardation, weight gain, hypertension, osteoporosis, gastrointestinal disturbance, psychosis and electrolyte imbalance. There is increased risk of corticosteroid induced cataract and glaucoma. Steroid-induced glaucoma occur more rapidly and may be refractory to treatment in children. It is difficult to detect and monitor intraocular pressure elevation in young children.

Due to such complications, early use of corticosteroid sparing agents or immunosuppressants is advisable. Immunosuppressive therapy is also advised if there is active systemic disease. Non-biologic agents such as antimetabolites (methotrexate) and signal transduction
inhibitors (cyclosporine) are commonly in management of chronic non-infectious uveitis in children. Methotrexate is the most widely used first-line immunomodulatory agent in children with uveitis because of its long-term safety profile in this age group. However, it takes 1-2 months to achieve the desired plasma levels. It is a folic acid analogue given at 0.15mg/kg orally once a week for at least 3 months and/or until stable quiescence, entirely without corticosteroids, is observed with concurrent folic acid supplementation. Folic acid supplementation of 1mg per day for 5 days per week is required to decrease the bone-marrow toxicity. The dosage of methotrexate should be reassessed as the child grows according to the weight. Methotrexate can be given in subcutaneous form if the dose exceeds 17.5mg or there is no clinical remission even with increased oral dosage. Cytopenia and transaminitis can occur with treatment, therefore liver function test and hemogram is advised every 4-6 weekly.

Second-line immunosuppressive agents include azathioprine, cyclosporin and mycophenolate. Cyclosporin A is a calcineurin inhibitor that prevents T-cell activation. It can be used as a combination therapy with other antimetabolites. It is effective as a primary treatment in Behçet’s disease-associated uveitis. Initial dosing is recommended at 3-5mg/kg/day divided into two doses, followed by a maintenance dose of 2-3 mg/kg/day. Alkylating agents, such as cyclophosphamide and chlorambucil, are generally avoided in children because of serious potential side effects.

Biologic agents that have been used for pediatric uveitis include tumor necrosis factor (TNF) alpha inhibitors (adalimumab, infliximab), IL-2 inhibitor (dalizumab), IL-6 inhibitor (tocilizumab), T-cell activation inhibitor (abatacept), and CD-20 inhibitor (rituximab). These agents are used in patients who fail to respond to conventional immunosuppressive therapy and are at high risk of visual loss. Both infliximab and adalimumab have been successfully used for treatment of resistant pediatric uveitis. Infliximab, a mouse/human chimeric antibody, is given by intravenous infusion at doses of 5-20 mg/kg every 4 weeks, after an induction period. Adalimumab, a fully humanized monoclonal antibody, is given in subcutaneous form at a dose of 20-40 mg every 7 to 14 days. Some data suggest that adalimumab may be slightly more effective than infliximab in achieving remission in JIA associated uveitis. A close co-ordination is required between ophthalmologist and pediatric rheumatologist for starting the biologic therapy and to monitor the systemic side effects.

Complications
Children tend to develop complications of uveitis more often than in adults, which include band keratopathy, peripheral anterior synechiae, posterior synechiae, cataract, glaucoma, cystoid macular edema, epiretinal membrane, retinal detachment and phthisis bulbi. Surgical intervention for the complication of uveitis in children also tend to be complicated, with higher failure rates.

Conclusions
To conclude, multiple challenges are faced related to the diagnosis and treatment of pediatric uveitis. Clinical manifestations of uveitis are different in children as compared to the adults. Active inflammation need to be aggressively controlled with immunosuppressants to prevent ocular complications. Drug-related side effects should be monitored and therapy should be modified accordingly. Immunomodulatory therapy requires a close coordination between the ophthalmologist and pediatric rheumatologist to maximize the efficacy of treatment and minimize the ocular and systemic side effects.

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