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Pars Planitis Epidemiology, Diagnosis, Follow-Up and Prognosis

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ABSTRACT

Pars planitis (PP) is an idiopathic intermediate uveitis accompanied by snowbanks and snowballs that often affects the pediatric and adolescent age groups. PP accounts for 5-26.7% of pediatric uveitis in different series. Histopathological and clinical findings indicate autoimmune etiology. It shows bilateral and asymmetrical involvement. While patients often complain of blurred vision and floaters, sometimes PP can be asymptomatic. Complications develop as a result of chronic involvement. Diagnosis is made by clinical examination and imaging methods. Treatment aims to suppress inflammation in the acute period and to reduce the frequency, severity and complications of exacerbations in the long term. The ultimate goal is to prevent ocular morbidity by providing complete remission. Conventional treatments include corticosteroids and immunomodulatory (IMT) agents such as methotrexate (MTX), azathioprine (AZA), cyclosporine A (CSA), mycophenolate mofetil (MFM). In recent years, new treatment options including biological agents such as anti-TNF-α therapy have become widespread and are used effectively in treatment. The most important point regarding the necessity of surgical treatment is that surgical success depends on the complete suppression of ocular inflammation with medical treatment. Therefore, it must be ensured that full inflammation control is achieved before surgery.

Keywords: Pars planitis, pediatric uveitis, biological agent

Pars Planitis Epidemiyolojisi, Tanısı, Takibi ve Prognozu

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ÖZ

Pars planit (PP) sıklıkla pediatrik ve adölesan yaş grubunu etkileyen snowbank ve snowball'ların eşlik ettiği idiyopatik intermediate üveittir. PP farklı serilerde pediatrik üveitlerin %5-26,7'sini oluşturmaktadır. Histopatolojik ve klinik bulgular otoimmün etiyolojiye işaret eder. Bilateral, asimetrik tutulum gösterir. Hastalar sıklıkla bulanık görme ve uçuşma şikayetiyle başvurur. Bazen de asemptomatik seyreder. Kronik tutulum sonucu komplikasyon gelişimine rastlanabilir. Tanı klinik muayene ve görüntüleme yöntemleriyle konur. Tedavinin amacı akut dönemde enflamasyonu baskılamak, uzun dönemde ise atakların sıklığını, şiddetini ve komplikasyonları azaltmaktır. Nihai amaç, tam bir remisyon sağlanarak oküler morbiditenin önlenmesidir. Geleneksel tedaviler arasında steroitler ve metotreksat (MTX), azatioprin (AZA), siklosporin A (CSA), mikofenolat mofetil (MFM) gibi immunomodülatuar (İMT) ajanlar bulunmaktadır. Son yıllarda; anti-TNF-α tedavisi gibi biyolojik ajanları kapsayan yeni tedavi seçenekleri yaygınlaşmış olup tedavide etkin şekilde kullanılmaktadır. Cerrahi tedavi gerektiğinde ise dikkat edilmesi gereken en önemli nokta; cerrahi başarının oküler enflamasyonun medikal tedavi ile tamamen baskılanmasına bağlı olduğudur. Bu nedenle cerrahi öncesi tam enflamasyon kontrolü sağlandığından emin olunmalıdır.

Anahtar sözcükler: **P**ars planitis, pediatric üveit, biolojik ajan

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Introduction

Intermediate uveitis is primarily an inflammation of the anterior vitreous, ciliary body and peripheral retina. It can be idiopathic or associated with infectious or systemic diseases. Pars planitis (PP) is described as idiopathic intermediate uveitis accompanied by snowbanks and snowballs, which predominantly affects the pediatric and adolescent age group[1]. In children, intermediate uveitis often presents as PP.

1.Epidemiology

The prevalence and incidence of the disease vary according to geographical regions and genetic characteristics. PP accounts for 5-26.7% of pediatric uveitis in different series[2, 3-5]. In a retrospective study conducted by Özdal et al. from Türkiye, the main cause of pediatric uveitis was found to be PP (24%)[5]. Soylu et al. found it to be the 3rd most common (9%) cause of pediatric uveitis after Behçet's and toxoplasma uveitis[4]. The typical onset of the disease occurs between the ages of 6 and 10[3, 4]. Symptoms begin on average around age 6, and most patients are diagnosed before age 14[6]. Although both genders can be affected, male gender predominates at young ages and female gender comes to the fore in adolescence[7, 8]. PP is bilateral in 75-80% and may show asymmetric involvement[9].

2.Etiopathogenesis

Histopathological and clinical findings suggest autoimmune etiology. It is thought to be an autoimmune response to ocular antigens that have not yet been identified playing a role in the etiology of the disease. It is thought that the retinal blood vessels, the ciliary body, or the pars plane itself may be the source of these antigens. T-helper cells have been found in retinal vascular infiltrates and snowbank structures. Due to the intense detection of T cells in the vitreous, the role of an autoimmune mechanism in which T cells are dominant is accepted.

3.Genetic factors

A genetic predisposition is believed to exist as a familial relationship is found in approximately 15% of patients and HLA DR2 is present in 50–70% of patients[10]. Associations between PP and HLA-DR2, DR15, B51 and DRB1*0802 haplotypes suggest immunogenetic predisposition. Patients who are HLA-DR15 positive have been reported to have systemic manifestations of other HLA-DR15-related disorders such as multiple sclerosis (MS), optic neuritis, and narcolepsy, indicating a common genetic alteration[11].

4.Clinical characteristics

Symptoms usually begin in one eye and have an insidious onset. It typically starts with mild blurring of vision and floaters. Donaldson and colleagues reported blurred vision in 74% of cases and floaters in 61% at diagnosis[12]. Although more rare, patients may present with red eyes, pain, light sensitivity, vision loss, strabismus and leukocoria. Sometimes the disease has an asymptomatic course and uveitis can be detected during routine eye examination. Especially in young patients; due to the difficulty in expressing complaints, delays in diagnosis and high complication rates at the time of diagnosis are encountered[13]. Especially in younger patients, diagnosis is delayed and the risk of developing complications that may lead to permanent vision loss increases.

PP is a disease that mostly has a bilateral course, but the severity of inflammation may be asymmetrical. Various studies have reported bilateral involvement at rates as high as 92%[3,9]. Typical clinical findings include mild to moderate anterior segment inflammation, diffuse vitreous cells and haze, and snowbanks and snowballs located in the retinal periphery[14]. PP extends from the anterior segment to the posterior segment. Inflammatory cells in the anterior chamber are the most common in anterior segment involvement. Small, round, white keratic precipitates are found on the corneal endothelium in approximately 50% of eyes. Peripheral corneal endotheliopathy which indicates the autoimmune etiology is characterized by peripheral corneal edema and large sheep fat keratic precipitates (KP) at the border of the edematous and normal cornea[15]. As the inflammation becomes chronic anterior segment involvement such as band keratopathy, anterior and posterior synechiae and cataract may be encountered and is more common in childhood than in adults[14].

The posterior segment and vitreous involvement pattern are important clinical features of PP. Characteristic findings include snowbank, snowball, peripheral vasculitis, diffuse cells and haze in the vitreous. Snowballs are yellow-white inflammatory deposits usually found in the middle and lower periphery. Snowbank is defined as exudates on the inferior pars plana. In approximately 60-65% of cases, the snowbank begins inferiorly and can spread and accumulate 360° in front of the peripheral retina. Donaldson et al. found the presence of snowballs in 67.4% and snowbanks in

97.8% of eyes with PP[12]. Although peripheral retinal vasculitis and vein sheathing is seen in PP, its occurrence varies between 17-90% in different clinical studies[31]. Optic disk inflammation is common in PP, its frequency goes up to 70% especially when the screening is done with fundus fluorescein angiography (FFA)[6, 16].

Patients with suspected PP should undergo a careful fundus examination with scleral depression, and the presence of snowball opacity and pars plana exudate should be investigated. In PP, the prevalence of exudate and the presence of more serious vitreous inflammation are often associated with cystoid macular edema (CME). In some cases, intravitreous hemorrhage may occur due to neovascularization of the vitreous base[17].

5.Diagnosis

There is no specific diagnostic test. Diagnosis is made by clinical ophthalmological examination. In a patient with suspected intermediate uveitis, the diagnosis is confirmed after excluding accompanying conditions like infectious (toxocariasis, peripheral toxoplasmosis, Lyme uveitis, tuberculosis, syphilis) and autoimmune (Behcet's disease, sarcoidosis, multiple sclerosis) systemic diseases. Optic coherence tomography (OCT) is widely used because it is fast and easily reproducible. OCT is effective in the follow-up of patients with epiretinal membrane (ERM), vitreomacular traction, and foveal atrophy, as well as macular edema. It is important in visual prognosis prediction as it can provide a detailed evaluation of the retinal layers[18]. In FFA; widespread fluorescein leakage from retinal vessels, optic disc inflammation and hyperfluorescence due to (CME) are observed. While there is no neovascularization, peripheral retinal traction and vasculitic changes on clinical examination, snowbank may show early hyperfluorescence and leakage, and this has been thought to be related to occult neovascularization[19]. Snowbanks located in the peripheral retina may appear as a fibrovascular mass. It is possible to demonstrate this with ultrasonography. Ultrasound biomicroscopy (UBM) shows that the pars plana is thickened and the exudates settled in the peripheral retina and pars plana are homogeneous, medium-density reflective opacities[20].

6.Complication

Pars planitis can lead to permanent damage to ocular structures and blindness due to complications, especially if diagnosis is delayed[21]. Delays in diagnosis and treatment may occur due to its chronic and asymptomatic course. It has been reported that children with uveitis onset at a young age (≤7 years) are more prone to the development of cataracts, glaucoma and vitreous hemorrhage and have a worse visual prognosis compared to older children (>7 years)[22]. Common complications are cataracts, CME, vitreous opacities and optic disc edema. Band keratopathy, amblyopia, ERM formation, vitreous condensation, neovascularizations, retinal detachment (RD) and cyclitic membranes are also seen as a result of long-term PP.

The most common complications are optic disc edema and CME. CME, the most common cause of low vision, is associated with poor visual prognosis. DeBoer et al. reported that it was observed in 44% of children with PP[16]. Ellipsoid zone (EZ) loss on OCT is associated with poor visual acuity in eyes with uveitic macular edema. Other complications include corneal endotheliopathy (corneal graft rejection-like appearance), posterior synechiae, cyclitic membrane, vasculitis, vitreous opacities and inferior peripheral retinoschisis which occurs almost only in children[23]. Optic disc neovascularization has been associated with severe inflammation[11].

Dense vitreous condensations are a cause of leukocoria that may be misdiagnosed as cataracts. Posterior subcapsular cataract is common in children with PP and poses a serious risk for amblyopia[23].

ERM formation was found to be directly related to disease chronicity and the mean time between disease onset and ERM formation was 7-8 years[12]. It has also been reported that the presence of ERM associated with uveitic macular edema is associated with worse visual acuity after treatment[24].

Retinal detachment (tractional, rhegmatogenous or exudative) is rare and has been reported in about 10% of cases in different studies[7,12,25]. The development of retinoschisis and tractional RD in the periphery of the retina is thought to be the result of traction of gliosis caused by the previous snowbank. Peripheral retinoschisis is stable and self-limiting in most cases[26]. Another view focuses on vascular etiology, suggesting that chronic inflammation causes peripheral angiogenesis, which in turn leads to exudative RD, retinoschisis, intraretinal edema and cyst formation[27].

Glaucoma is due to decreased aqueous outflow and blockage, which can develop secondary to many causes such as peripheral anterior synechiae, increased protein concentration in the aqueous humor, trabecular inflammation and damage. Surgical treatment may be required in the presence of high intraocular pressure that cannot be

controlled with topical treatment. The success rate and long-term efficacy of surgery may be limited in uveitic patients. It is a complication seen in approximately 6-8% of patients and requires surgery in half of the cases[11].

Other rare complications include macular hole and macular ectopia.

Amblyopia may occur due to dense band keratopathy, vitreous opacities, vitreous haze, cataracts occluding the visual axis or persistent macular edema.

7.Prognosis

The natural course of PP is variable. According to studies, part of the patients have self-limiting disease while other part of the patients have a prolonged active disease with frequent exacerbations and the rest of the patients have chronic disease after a few exacerbations. PP's chronic and insidious nature and the anterior segment's symptoms' usual quiet characteristics may cause a lot of pediatric patients to have permanent visual loss. In children, PP prognosis is strongly associated with vitreous inflammation's severity. While more severely inflamed eyes are more prone to CME and other macular complications, eyes that developed vitreous bands may result in retinal traction and RD[28]. To improve patients' general prognosis, the main goal is sufficient control of inflammation and rapid detection of disease-associated complications[29]. One of the most important factors that affect visual prognosis is the age at the onset of the disease. In pediatric PP patients, visual acuity at the diagnosis and follow-up is poorer compared to adult patients. It is shown that children who were diagnosed at the age of 7 and younger are more prone to complications and poorer visual prognosis compared to older children[22]. Another study has shown that the onset of disease at 10 years old and before, male gender, apparent vitreous blurriness and macular edema existence are markers for poor prognosis[9].

8.Treatment

Uveitis in pediatric patients is a chronic disease that may have relapses and poor prognosis. It is important to use a multidisciplinary approach with a team consisting of ophthalmologists and pediatricians while managing the treatment[30].

Before starting the treatment, uveitis' relation to systemic diseases and infections should be researched and the mechanism of action and adverse reactions of the therapeutic agents should be known well. Patients must be well informed about the test and the treatment plan and should be followed closely in terms of the side effects.

The main goal of the treatment is to suppress the inflammations acutely and to reduce the frequency and severity of attacks and complications in the long term. The ultimate goal is to provide full remission and to prevent ocular morbidity.

For pediatric patients, conventional treatment for non-infectious uveitis includes topical, periocular, intravitreal or systemic CS and immunomodulator agents (IMT) such as methotrexate (MX), azathioprine (AZA), cyclosporin A (CSA), mycophenolate mofetil (MFM). In recent years, new treatment options which contains biologic agents like anti-TNF-α have become prevalent and used efficiently.

In our PP practice, treatment's first step is the usage of CS which forms the basis of the treatment. When a patient needs long-term therapy with steroids, IMT must be considered. If the patient has refractory uveitis, poor prognostic factors and developed complications at the time of diagnosis, IMTs must be started along with CS without delay as the first-line therapy. IMT agents can be used as monotherapy or combined with other agents. The choice of IMT agent can change according to ophthalmologists' preference and experience, also patient's clinical findings and age.

In conventional immunosuppressive treatment-refractory and uncontrolled uveitis or situations when adverse effects cause treatment discontinuation, biological agents are considered the treatment of choice. Adalimumab (ADA) is the first choice when switching the biological agent treatment. In some cases, when uveitis is very severe and cannot be controlled with anti-TNF-α, tocilizumab (anti-IL 6) treatment can be started.

8.1 Corticosteroids

Corticosteroids are the first-line treatment in PP. Topical CS is mainly used in the treatment of anterior segment inflammation, although its effect is insufficient in the treatment of intermediate and posterior uveitis, especially in phakic cases[31]. Topical CSs are ineffective in posterior segment inflammation because they cannot penetrate the vitreous. In these cases; subconjunctival, peribulbar, intravitreal or systemic CS treatment can be applied. Periocular or subtenon CS injections might be a treatment choice for intermediate and posterior uveitis, specially in unilateral cases and for CME. The most common complications of periocular CS applications are; increased intraocular

pressure, cataracts and aponeurotic ptosis[9]. Others; herpes virus reactivation, delayed healing of corneal wounds, corneascleral thinning, subconjunctival hemorrhages, myopia, central serous chorioretinopathy (CSCR), microcyst formation in the iris/ciliary body.

CS side effects are related to the average dose and duration of treatment. However, serious side effects may occur even with low doses. Systemic CSs are used only for short-term treatment in children due to significant systemic side effects associated with long-term use, such as cushingoid changes, growth retardation, increased appetite, weight gain, restlessness, hypertension, osteoporosis, gastrointestinal upset, psychosis, electrolyte imbalance and pseudotumor cerebri.

The induction dose of oral prednisolone is 1-2 mg/kg. When a faster and stronger effect is required, intravenous methylprednisolone 30 mg/kg may be preferred.

In patients who do not respond adequately to high-dose CS or are dependent on high doses, additional IMT agents should be started. Moreover, in patients who present with serious ocular complications and have risk factors for the development of new complications, IMT agents combined with CS can be started at the first visit.

8.2.Conventional immunomodulatory Treatment Agents

8.2.1Antimetabolites

Methotrexate

Methotrexate is the most commonly used and first-choice IMT agent in children with uveitis. It is a folate analog that inhibits the enzyme dihydrofolate reductase.

MTX is administered to children once a week orally or subcutaneously at a dose of 10-15 mg/m2. At the end of 6-8 weeks, the dose can be safely increased up to 30 mg/m2, depending on the response and tolerance to the drug. The therapeutic effect is usually seen after 6 to 10 weeks[28]. The subcutaneous route is better tolerated in children with nausea or in patients with poor oral bioavailability.

Side effects of treatment depend on the dose and duration of treatment. Since MTX is a folic acid antagonist, it should be used in conjunction with folic acid. Aversion; It is an undesirable side effect that may occur during treatment. Before an oral or subcutaneous dose, children often experience abdominal pain, nausea, and may vomit. if these symptoms significantly affect the child's quality of life; It is important not to insist on treatment and to use alternative agents instead of MTX. However, the most common side effect of MTX is that it affects liver function and increases transaminase levels. Side effects such as gastrointestinal toxicity, liver cirrhosis, hematological toxicity, pneumonia, lung fibrosis and teratogenicity may occur during the use of MTX. In case of inadequate response with MTX, other IMT agents or combined treatment can be started.

Azathioprine

It is a purine nucleotide analog and is given 1-2 mg/kg/day (30-60 mg/m2) orally. The therapeutic effect mainly occurs within 1-3 months of use. Most side effects were in the form of gastrointestinal tract complaints, usually at higher doses, while malignancies were only rarely reported with long-term treatment[32]. The most common side effects are bone marrow suppression with leukopenia, thrombocytopenia and hepatotoxicity. Monthly complete blood count and liver function test (LFT) control should be performed during drug monitoring.

Mycophenolate Mofetil

It is a prodrug. It inhibits the proliferation of human T and B lymphocytes and suppresses the antibody production of B cells[33]. It is better tolerated than AZA. MFM has high oral bioavailability and should be taken on an empty stomach. Antacids reduce the bioavailability of the drug by 15%. The recommended drug dose for uveitis is 2 g/day.

Up to 30% of patients experience nausea, gastrointestinal upset and diarrhea. Less commonly reported side effects of AZA are leukopenia, hair loss and fatigue. Patients should be monitored with a complete blood count once a week for 4 weeks, then twice a month for 2 months, then once a month. LFT should be performed every 3 months.

8.2.2.Calcineurin Inhibitors

Cyclosporine

CsA, a calcineurin inhibitor that suppresses T cell activation, has limited efficacy in pediatric uveitis when used alone. It is usually applied as a combined treatment.

Important side effects associated with cyclosporine use include nephrotoxicity, hypertension, hepatotoxicity, anemia, gingival hyperplasia, hypertrichosis, nausea, vomiting and tremor. It is less nephrotoxic in children than in adults due to higher renal clearance. Patients should be monitored for side effects with kidney and liver

function tests and blood pressure measurements. The recommended dose of CsA is 2.5-5 mg/kg per day[34].

8.3.Biological agents

They provide effective treatment in the immune system by affecting specific molecules (proteins) in the inflammatory process. The majority of these agents are monoclonal antibodies[35]. These drugs are used as next-line therapy in the treatment of uveitis when CS and conventional immunosuppressive therapy fail to suppress ocular inflammation or when steroids must be avoided. They can be used alone or in combination with conventional agents. The most commonly used biologics in the treatment of uveitis are TNF-α inhibitors; Especially ADA and infliximab (IFX) are preferred. Different biological agents such as tocilizumab (anti-IL 6) can be tried in patients resistant to TNF inhibitors.

Inhibition of TNF-alpha has been shown to reduce leukocyte activity, function (rolling, adhesion), and vascular leakage. This mechanism may explain its effectiveness in inflammatory ocular diseases[36]. TNF-α has been found to increase vascular endothelial growth factor (VEGF) production in choroidal endothelial cells and VEGF is responsible for macular edema in patients with uveitis. Anti-TNFs reduce VEGF-α levels in plasma by inhibiting TNF-α production in the treatment of uveitic macular edema[37].

TNF inhibitors can occur in 2 ways; in the form of soluble receptor fusion protein (etanercept) or monoclonal antibodies (IFX, ADA, golimumab, certolizumab). IFX consists of partly human and partly mouse antibodies and is chimeric, whereas ADA is a completely human antibody. Their activities are similar but there are some differences. ADA binds to TNF-α with higher affinity than etanercept or IFX, and therefore its use in treatment has proven to be advantageous[38].

Although TNF-α inhibitors are used in the treatment of sarcoidosis and psoriasis, the reason is not fully explained and they may paradoxically cause sarcoidosis-like involvement in the lungs and psoriatic skin lesions. TNF-α inhibitors are contraindicated in multiple sclerosis. Infections such as tuberculosis, human immunodeficiency virus (HIV), syphilis, HBV, HCV and toxoplasma should be excluded before starting treatment. Common side effects of TNF-α inhibitors include hypersensitivity, more serious side effects include infections, hematological reactions, malignancies and myocardial infarctions.

Patients using TNF-α inhibitors require regular blood evaluations, including complete blood count, LFT, blood urea nitrogen, and serum creatinine levels every 6 weeks.

İnfliximab

Standard practice is intravenous treatment at weeks 0, 2 and 6. Thereafter, it can be given at 4 or 8 week intervals. The half-life of IFX is 10 days; however, its effects may persist for up to 2 months. Due to its chimeric nature, IFX is often given together with MTX or another IMT agent to reduce anti-chimeric antibody formation and increase the duration of drug effectiveness[36]. It is a good option in pediatric uveitis when rapid effectiveness is required and CSs are avoided due to side effects. Pediatric patients may require higher doses or more frequent infusions than adults.

Adalimumab

Adalimumab is a fully humanized monoclonal antibody against TNF-α. Therefore, chimeric antibody formation is not observed. However, some patients may develop antibodies against ADA, which may reduce the effectiveness of the drug over time[36]. Several prospective studies, including the VISUAL I clinical trial, have demonstrated the efficacy and safety of anti-TNF drugs in treating chronic and refractory uveitis and reducing the use of CS[39].

The standard ADA protocol is subcutaneous administration of 80 mg if the patient weighs 30 kilograms or more in the first application, followed by a 40 mg dose 1 week later, and then 40 mg doses every 2 weeks. For patients weighing less than 30 kilograms, the dose is halved.

The most common side effects in adalimumab treatment are injection site reactions and allergic reactions[39]. Cases of cellulitis, pneumonia, appendicitis, herpes zoster, urinary tract infection, gastrointestinal tract abscess and gastroenteritis, and more rarely tuberculosis and opportunistic infections, have been reported with ADA. Other serious side effects such as demyelinating disorders, lupus-like syndrome, and congestive heart failure are rare. The advantage of ADA over other anti-TNF agents is that it can be applied without requiring admission to any hospital or healthcare institution.

In a prospective multicenter case series including 131 patients from different age groups, it was shown that ADA therapy could significantly improve anterior chamber and vitreous inflammation with the ability to reduce CS. Complete resolution of CME was demonstrated in 70% of eyes at 6 months[40]. Studies have shown that ADA and IFX are effective in providing inflammation control; their success and effectiveness are similar[41]. The ease of application of ADA (it can be applied subcutaneously) and the fact that it does not require application to a

healthcare institution are important advantages over IFX. In another study; It has been suggested that ADA will provide a superior response because it binds to TNF-α, which is present not only in the circulation but also on the cell surface and weekly or biweekly administration of ADA will provide less variable serum levels than periodic infusions of IFX[42]. In another study comparing ADA and IFX for the treatment of pediatric chronic non-infectious uveitis, remission rates were similar. However, ADA was found to be more effective than IFX in terms of maintenance of remission. Recently, it has been reported that the use of ADA as the first anti-TNF-α agent in treatment is more effective than its use in cases of IFX failure[43,44].

8.4. Surgical treatment

In complications that develop as a result of a chronic course, surgical interventions may be required in addition to medical treatment. The most important point is that surgical success depends on the complete suppression of ocular inflammation with medical treatment. Therefore, it should be ensured that complete inflammation control is achieved before surgery[45].

Chelation therapy is effective in band keratopathy, but the recurrence rate is high in uveitic eyes. Therefore, chelation therapy is recommended for eyes at risk of amblyopia or serious vision loss[46].

Cataract surgery in pediatric patients may be difficult due to a lack of scleral rigidity and existing ocular complications such as band keratopathy and posterior synechiae. In recent years, in addition to good preoperative inflammation control, modern surgical techniques such as phacoemulsification and the development of foldable hydrophobic acrylic intraocular lenses (IOLs) have resulted in successful visual results after cataract surgery with lens implantation in the capsular bag. Postoperative inflammation control plays a major role in this success. IOL implantation during cataract surgery has been a subject of debate for many years. It was widely believed that IOL implantation after cataract extraction was contraindicated due to the high rate of fibrotic membrane formation around the postoperative IOL[47]. Current studies show the opposite. It has been shown that postoperative complication rates are similar in aphakic and pseudophakic eyes, and long-term postoperative visual outcomes in pseudophakic eyes are better than in aphakic patients[48].

The effectiveness of trabeculectomy in glaucoma may be limited and temporary, especially in the uveitic patient population due to severe postoperative inflammation and fibrosis[49]. Hypotonia or hypertonia may be more common in uveitic patients in the early postoperative period. Glaucoma drainage implant surgery and goniotomy are other surgical methods that can be applied.

Laser photocoagulation can be performed in cases with peripheral neovascularization, retinal traction, and retinoschisis. Pars Plana vitrectomy (PPV) is especially performed in patients who develop vitreous condensation, intravitreous hemorrhage, retinal detachment and ERM. PPV also provides mechanical clearance of inflammatory mediators in patients with active inflammation and CME resistant to medical therapy[31].

Conclusion

PP is one of the most common causes of childhood uveitis. Early diagnosis is made in symptomatic patients with a careful examination. With the introduction of new generation drugs, especially biological agents, disease activity is effectively suppressed and the development of complications is prevented. At the same time, patients should be monitored for ocular and systemic side effects of the drugs during treatment. If diagnosis is delayed and inflammation control is not achieved, complications that may result in blindness may develop.

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