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Rheumatologic Disorders and Their Ocular Manifestations – Clinical Insights and Therapeutic Approaches

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Abstract

Introduction and purpose: Rheumatic disorders, a broad group of conditions primarily affecting the joints, can have significant systemic manifestations that extend beyond musculoskeletal involvement. One such often-overlooked aspect of these diseases is the impact they can have on the eyes. Ocular symptoms are common in many rheumatic diseases and can range from mild irritation to severe sight-threatening complications. This article explores the range of ocular symptoms associated with rheumatic diseases, emphasizing their clinical presentations and the importance of early recognition and treatment.

Materials and methods: A review was conducted in PubMed including publications regarding ocular manifestations of rheumatic diseases. Literature was searched using the following terms: "uveitis in rheumatic disorders", "spondyloarthropathies", "**systemic lupus erythematosus**", "HLA B27 uveitis" and "treatment of rheumatic diseases".

Brief description of the state of knowledge: The eyes may be affected either directly through inflammation of various ocular structures or indirectly as part of the systemic manifestations of autoimmune processes. Conditions such as rheumatoid arthritis, systemic lupus erythematosus, Sjögren's syndrome and ankylosing spondylitis often present with ocular involvement including dry eye syndrome, uveitis, scleritis, episcleritis and retinal vasculitis. While some ocular findings may be asymptomatic others can lead to significant visual impairment if not properly managed.

Conclusions: Rheumatic diseases are often associated with a range of ophthalmic complications which can sometimes develop before any other systemic signs of the rheumatic condition appear, making them crucial indicators of potential underlying disease. In many cases, the visual system may be the first to show signs of involvement and recognizing these symptoms early is essential for timely diagnosis and management.

Introduction

The uveal tract is the middle layer of the eye wall, consisting of the iris, ciliary body and choroid. The anatomical classification distinguishes between: anterior uveitis (involving the iris and anterior part of the ciliary body) – uveitis anterior (UA), intermediate uveitis (involving the flat part of the ciliary body and the peripheral choroid, including the adjacent retina) – uveitis intermedialis (UI), posterior uveitis (involving the choroid behind the posterior border of the ciliary body) – uveitis posterior (UP) and panuveitis (involving the entire uveal

tract) [1-3]. Clinically, inflammation is classified based on its duration: acute (lasting up to 3 months), chronic (lasting more than 3 months) and recurrent (characterized by periods of remission and relapse) [1]. Anterior uveitis is the most common form of uveitis. Among all cases of UA, 25-60% are associated with autoimmune diseases [2]. The symptoms of UA include eye pain and redness, strong photophobia and a tendency for eyelid spasm (blepharospasm). Symptoms typically have a sudden onset and are associated with a decrease in visual acuity and pain. Approximately 20% of UA cases lead to serious complications including vision loss, disability, and permanent incapacity to work. Causes of reduced visual acuity include cataracts, glaucoma (inflammatory or steroid-induced), cystoid macular edema (CME), band keratopathy, development of epiretinal membranes, optic neuropathy and damage resulting from choroid and retina inflammation (immunosuppression, opportunistic infections). Therefore, patients with UA should undergo thorough diagnostic evaluation (if necessary, by an interdisciplinary team of ophthalmologists and rheumatologists) to implement effective treatment as soon as possible, optimally addressing both the eye and joint involvement.

Table 1. Classification of spondyloarthropathies based on uveal tract involvement and symptoms

Eye segment	**Anterior uveitis**	**Intermediate uveitis**	**Posterior uveitis**
Anatomical part of the eye	Iris Ciliary body	Vitreous body	Choroid Retina
Symptoms reported by the patient	red eye, pain, tearing, photophobia, blurred vision	visual floaters, blurred vision	visual floaters, scotomas, decreased visual acuity, loss of vision
Findings on physical examination	eyelid spasm, decreased visual acuity, ciliary injection, deposits on the corneal endothelium, Tyndall phenomenon in the aqueous humor of the anterior chamber	**inflammatory exudate in the vitreous body,** peripheral perivascular inflammation	inflammatory cells, **haze** (or **fogging**), condensations in the vitreous body, choroiditis, retinitis
Disease	ankylosing spondylitis, juvenile, idiopathic arthritis, inflammatory bowel disease - associated arthritis, psoriatic arthritis, reactive arthritis, sarcoidosis, Behcet disease, herpes simplex virus or varicella zoster virus infection	involvement of only this segment is atypical for SpA - may occur in multiple sclerosis associated intermediate uveitis, tubulointerstitial nephritis, Lyme disease, sarcoidosis, syphilis or vasculitis	reactive arthritis, Behcet syndrome, eye injury, Lyme or Bartonella disease, cytomegalovirus infection, syphilis or toxoplasmic retinitis, acute retinal necrosis (herpes simplex virus or varicella zoster virus retinitis)

Rheumatoid arthritis

Rheumatoid arthritis (RA) is an autoimmune disorder characterized by chronic inflammation of the synovial membrane of the joints, leading to its proliferation and, as a result of pro-inflammatory cytokines, cartilage damage, the formation of bone erosions and gradual destruction of bone tissue. Ocular involvement is one of the extra-articular manifestations of RA, more commonly seen in individuals with a long disease history, rarely being the first symptom. Although it can affect any part of the eye, the anterior segment is most frequently involved [4,5]. Among the various ocular symptoms in RA, dry eye syndrome (DES) is the most common one with the reported prevalence ranging from 18% to as high as 90% [4]. Episcleritis, occurring in around 5% of RA patients, is the second most common ocular symptom and is characterized by redness, discomfort and photophobia [4,5]. Compared to the general population, episcleritis in RA is more frequently bilateral. Scleritis, another ocular manifestation in RA, is an inflammatory process that is associated with severe pain and potential vision loss due to complications such as cataracts, glaucoma or exudative retinal detachment. This type of inflammation typically occurs in patients with a long history of RA and is found in 1-6% of them [4]. The sclera is anatomically divided into anterior and posterior segments. Anterior scleritis includes non-necrotizing diffuse and nodular forms, as well as necrotizing scleritis, which may present with or without signs of inflammation. A distinctive feature of RA is necrotizing anterior scleritis without signs of inflammation, also known as scleromalacia perforans. This form is painless, does not affect vision, and mainly affects older women with chronic RA [4,5]. It progresses with sudden vascular closure, leading to extensive areas of necrosis, which heals with scleral thinning and exposure of the choroid [5]. Peripheral ulcerative keratitis, which is associated with corneal epithelial defects, may lead to corneal perforation and vision loss [4]. Approximately 35% of cases are associated with underlying rheumatic diseases, particularly long-standing RA, but also vasculitis, relapsing chondritis, and systemic lupus erythematosus (SLE). Anterior uveitis, which affects the iris or both the iris and ciliary body, accounts for 14-42% of all inflammatory eye diseases in RA patients [4].

Spondyloarthropathies

Spondyloarthropathies (SpA) are a group of inflammatory diseases characterized by symptoms affecting both the musculoskeletal system and other organs. The joint-related symptoms include inflammation of the spine and sacroiliac joints (known as axial involvement),

along with peripheral joint inflammation, enthesitis, tendon sheath inflammation, and dactylitis (known as peripheral involvement) [6,7]. Extra-articular manifestations include uveitis (25-30%), skin psoriasis (10-25%), inflammatory bowel diseases (5-10%) and cardiovascular symptoms, including aortic valve involvement (1-10%) [7]. SpA is often associated with the HLA-B27 antigen, though its frequency varies between different diseases within the group. While about 10% of the general population carries the HLA-B27 antigen, only 1-5% of these individuals will develop an inflammatory joint disease [8]. The strongest association is seen with ankylosing spondylitis (AS), where approximately 90% of patients test positive for HLA-B27 [8]. Uveitis is the most common extra-articular manifestation of SpA, affecting 21-33% of patients. It may even be the first symptom, preceding joint changes [6,9]. It affects about 37% of patients with inflammatory bowel disease - associated arthritis, about 33% of patients with AS (30-50%), 26% with reactive arthritis (ReA), 25% with psoriatic arthritis (PsA) and approximately 13% with undifferentiated spondyloarthropathy [6,9]. The presence of HLA-B27 is linked to more frequent relapses and a more severe course of uveitis [8]. Some forms of SpA have a predilection for affecting specific parts of the uveal tract. The anterior uveal tract is by far the most commonly affected segment in SpA, while involvement of the posterior uveal tract or generalized inflammation of the uveal tract (panuveitis) is extremely rare. When it does occur, it is more commonly associated with infectious conditions (such as tuberculosis or syphilis), sarcoidosis, Behçet's disease or Vogt-Koyanagi-Harada syndrome (a rare autoimmune disorder that involves chronic choroiditis, premature graying of hair, patchy hair loss and potential meningitis). A key concern for SpA patients with uveitis is the secondary development of permanent complications due to the inflammation in the uveal tract. The most common complications include the formation of posterior synechiae (13-91%), cataracts (7-28%), glaucoma and cystoid macular edema, which is the leading cause of vision disturbances in uveitis [8].

Ankylosing spondylitis

Ankylosing spondylitis is linked to the highest rate of acute anterior uveitis among all spondyloarthropathies. This form of uveitis is typically acute, unilateral and recurrent, with both eyes rarely being affected at the same time [8]. Some studies suggest that eye involvement is correlated with the clinical activity of peripheral arthritis [10].

Reactive arthritis

Reactive arthritis is triggered by a previous infection of the urinary tract, respiratory or gastrointestinal system. As a result of an autoimmune process in genetically predisposed individuals, arthritis develops. The most common pathogens associated with the development of reactive arthritis include *Salmonella, Campylobacter, Yersinia, Shigella, Chlamydia* spp., and *Mycoplasma* spp. [11] There is a particularly strong association between reactive arthritis and the presence of HLA-B27. In the context of spondyloarthritis (SpA) linked to infections from pathogens like *Shigella,* this correlation is found in 80-90% of patients; for *Yersinia,* in 70-80% and for *Chlamydia,* in 40-50%. This confirms that the presence of HLA B27 plays a role in the pathogenesis of both anterior uveitis and SpA [11,14]. Furthermore, a strong association has been found between the occurrence of anterior uveitis and the presence of HLA A-9 and B40 antigens [11]. A common early symptom of reactive arthritis, occurring in 2% of cases during the early stages of the disease, is conjunctivitis. It is most common in patients with reactive arthritis caused by *Chlamydia* spp., particularly in sexually acquired reactive arthritis (SARA), where the inflammation is caused by a pathogen transmitted sexually [11,12]. In these patients, identifying those with uveitis who are at risk of further complications is crucial. Reactive arthritis is self-limiting in about half of the cases; however, relapses occur in 50% of patients and 17% experience chronic joint inflammation [12]. In chronic reactive arthritis, in addition to the previously mentioned conjunctivitis, the most common ocular symptoms are anterior uveitis (92%) and posterior uveitis (64%) [11]. Other observed conditions include corneal inflammation, cataracts, intermediate uveitis, scleritis, cystoid macular edema, and glaucoma [13].

Psoriatic arthritis

Ocular symptoms affect up to one-third of patients with psoriatic arthritis, with the most common being conjunctivitis (19.6%). Other eye manifestations include acute uveitis (15%), iritis (7.1%), dry eye syndrome (2.7%) and episcleritis (1.8%) [6,12]. Unlike ankylosing spondylitis, uveitis in psoriatic arthritis tends to be chronic and is more frequently bilateral. Ocular symptoms are more common in patients with psoriatic arthritis compared to those with psoriasis alone [13].

Spondyloarthropathies associated with inflammatory bowel diseases

In SpA associated with inflammatory bowel diseases, uveitis is found in around 25% of patients [9], with half of these cases being preceded by a complete clinical picture of both gastrointestinal and joint symptoms [15]. The course of uveitis in these patients differs from that seen in individuals with ankylosing spondylitis (AS) or undifferentiated SpA. Most affected patients are women with an insidious onset of symptoms. The condition is typically bilateral and tends to be chronic, showing little to no tendency for spontaneous resolution [16]. The most commonly observed conditions are scleritis and acute uveitis, which are more frequent in individuals with Crohn's disease than in those with ulcerative colitis (UC) [12].

Juvenile Idiopathic Arthritis

Juvenile idiopathic arthritis (JIA) is the most common rheumatic disease in children, defined as arthritis with onset before the age of 16, lasting at least 6 weeks, and diagnosed after ruling out all other causes [17]. In addition to joint inflammation, the autoimmune process associated with JIA can lead to systemic complications, with uveitis being the most common. Uveitis affects 16-57% of JIA patients (with an average of 22.1%), though this varies by JIA subtype [18]. Approximately 75% of uveitis cases are bilateral [18,19] and it typically develops within 4 years of the first joint symptoms, although in 3-10% of cases, it precedes joint symptoms [18,20]. The risk of developing uveitis is highest in the oligoarticular form (21-62%) and lowest in the systemic form of JIA [21-23]. Risk factors include sex, JIA subtype, age at diagnosis, disease duration and the presence of antinuclear antibodies (ANA) and rheumatoid factor (RF). The highest risk group includes girls diagnosed with oligoarticular JIA before age 6 and with positive ANA [18,22]. In JIA, uveitis predominantly manifests as chronic, recurrent, non-granulomatous inflammation of the anterior uveal tract [13]. However, in juvenile idiopathic arthritis associated with enthesitis (JIA-ERA), acute uveitis with typical symptoms - such as eye and head pain, photophobia, redness, tearing and vision problems - is more common [18,19]. In the chronic form, the initial course remains asymptomatic. According to some authors, uveitis in oligoarticular JIA is asymptomatic in up to 87% of cases [23]. Children and parents may not notice the gradual decline in vision, which can persist for years and lead to irreversible, vision-threatening damage. The most common complications include: posterior synechiae (8-75%), cataract (8-38%), secondary glaucoma (8-38%) with subsequent optic neuropathy and band keratopathy (7-70%) [18,23]. Some children may develop ocular hypotony (4%), which can lead to ocular atrophy [23]. In severe cases of chronic inflammation,

cystoid macular edema and retinal detachment may occur [23]. Around 17% of JIA patients experience dry eye syndrome, and 23.9-33.6% of patients show visual impairment during the initial eye examination. Therefore, regular eye examinations are crucial for children with JIA [19]. In 2018, a group of European experts in pediatric rheumatology and ophthalmology developed recommendations for screening, diagnosis, and treatment of uveitis associated with JIA as part of the SHARE program. An eye exam should be performed within 6 weeks for all patients suspected of having JIA. The frequency of further eye exams depends on the severity of the disease and the recommendations of collaborating specialists in rheumatology and ophthalmology [24].

Systemic Lupus Erythematosus

Systemic lupus erythematosus (SLE) is an autoimmune disorder with a complex underlying mechanism that causes chronic inflammation of tissues and organs, including the eyes [25]. The inflammatory process can affect almost all structures of the eye and the surrounding orbital tissues [26,27]. Ocular symptoms often reflect the systemic activity of the disease and may sometimes be the first indication of SLE [25]. Approximately one-third of SLE patients experience eye-related issues, with dry eye syndrome being the most common [25]. Inflammation of the optic nerve and retinal vessel occlusion - resulting from immune complex deposition, inflammation and subsequent emboli formation - are associated with a poor prognosis and irreversible vision loss [26]. Secondary to dry eye syndrome, damage to the cornea can occur, manifesting as scarring, ulceration or fibrous keratitis. Although posterior uveitis is rare, it is typically associated with vasculitis [28]. Scleral inflammation affects approximately 2.4% of SLE patients, usually young women without signs of other organ involvement, in contrast to the pediatric population, where scleral inflammation (though rarer) is associated with systemic involvement [25]. Scleritis occurs in approximately 2% of SLE patients and is a potentially sight-threatening condition, sometimes leading to exudative retinal detachment or cystoid macular edema [25,26]. Retinopathy affects 29-50% of SLE patients and is linked to disease activity, indicating insufficient disease control [25,26,28]. Retinal changes occur directly due to vasculitis and indirectly due to hypertension associated with lupus nephritis [26]. The fundus appearance in lupus retinopathy resembles changes seen in hypertension and diabetes. In mild cases, small intraretinal hemorrhages, cotton wool spots and blood vessel tortuosity are common. In more advanced cases, focal and generalized narrowing of the arterial vessels is observed and in severe cases retinal arterioles become occluded, leading

to proliferative retinopathy, vitreous hemorrhages and retinal detachment [26]. Lupus choroidopathy with exudative retinal detachment is a rare ocular manifestation of SLE, usually observed in patients with high clinical disease activity, concomitant vasculitis, nephropathy, high blood pressure and the presence of antiphospholipid antibodies [25]. Cases of secondary closed-angle glaucoma have also been reported [26]. Optic neuropathy affects 1.6-3.6% of SLE patients and includes optic neuritis and ischemic neuropathy, which presents as painless vision loss [25,26]. There is poor recovery prognosis. Additionally, if optic neuritis is the only symptom of SLE, it may pose diagnostic challenges when differentiating from demyelinating causes.

Systemic Sclerosis

Eye involvement in systemic sclerosis (SSc) affects most patients and results not only from the fibrotic process involving the structures of the orbit, which is considered an advanced stage of the disease, but also from immunological disturbances and vascular pathologies [27]. In systemic sclerosis, there is hardening of the eyelid skin, which leads to difficulty closing the eyelids (57-77% of patients), subsequently resulting in dry eye syndrome (48.9-64.7%) [28,29]. Factors contributing to the development of dry eye syndrome include disorders of tear film secretion (both quantitative and qualitative), as well as accelerated evaporation from the surface of the eyeball. In 14-23% of patients with systemic sclerosis, secondary Sjögren's syndrome is present [29]. Vasculopathy in systemic sclerosis plays a dominant role in the pathology of the choroid. Small vessel obliteration and areas of avascularity and ischemia in the choroidal membrane have been observed in up to 50% of patients [27,29]. Vascular changes such as telangiectasia, neovascularization, vascular tortuosity and varicose dilation also significantly impact the conjunctiva. The aforementioned vascular alterations resemble those seen in nailfold capillaroscopy [29]. The link between systemic sclerosis and retinopathy is not fully understood, but some studies indicate that it may affect up to one-third of SSc patients. However, factors such as concurrent hypertension or diabetes should also be considered. Glaucoma and cataracts are more frequently observed in SSc patients compared to the general population, though further research is needed to clarify their connection to the disease [29].

Sjögren’s Syndrome

Primary Sjögren's syndrome (pSS) is a chronic, inflammatory autoimmune disease characterized by the presence of antinuclear antibodies SS-A (Ro-52 and Ro-60) and SS-B (La), leading to lymphocytic infiltration of exocrine glands, especially the lacrimal and salivary glands, resulting in their destruction and symptoms of dry eyes, dry mouth, dry throat and vaginal dryness [30,31]. About 30% of patients with pSS experience systemic symptoms due to involvement of the lungs, kidneys, nervous system, muscles, joints and skin [31]. Patients with pSS are at a 16-fold higher risk than the general population for developing MALT lymphoma, which affects 5-10% of patients and is most often localized in the salivary glands [30,32]. The prevalence of pSS ranges from 0.5-4.8% and is more common in women [31]. Sjögren's syndrome can be classified as either primary (without associated diseases) or secondary (with other autoimmune diseases such as rheumatoid arthritis, lupus, systemic sclerosis or mixed connective tissue disease). The secondary form often presents with symptoms of dryness and may include typical autoantibodies, though it can also occur without them, with symptoms of the primary autoimmune disease being more prominent. The most common ocular symptom of pSS is dry eye syndrome, which affects about 90% of patients and is more severe than idiopathic dry eye syndrome [36]. This is caused by insufficient tear production (water phase deficiency) or excessive evaporation, leading to damage to the surface of the eyeball [33]. In pSS, hyposecretion of tears is observed due to the altered, inflamed lacrimal glands. What is more, meibomian gland insufficiency may coexist, worsening dryness due to excessive evaporation (lipid phase deficiency), resulting in a sandy feeling in the eyes, burning, itching and photophobia. In addition to dry eye syndrome, pSS can lead to other ocular complications, often due to factors like vasculitis rather than direct lacrimal gland involvement. These complications include corneal ulceration (6.3%), anterior uveitis (3.2%), scleritis or episcleritis (3.2%), retinal vasculitis (0.8%) and optic neuropathy (1.6%) [4]. Patients with pSS and ocular symptoms have been found to have higher disease activity and an increased risk of mortality, with the risk being twice as high compared to other individuals in a referenced study [34]. The current classification criteria for primary Sjögren's syndrome (pSS) set by ACR/EULAR in 2016 (Table 2) include two ophthalmic assessments for objective dry eye symptoms: the Schirmer test and ocular surface staining. The latter can be evaluated using the Ocular Staining Score (OSS), which involves scoring changes observed under a slit lamp after staining with lissamine green and fluorescein, or through the van Bijsterveld method, such as Bengal rose staining. [35]. Every patient suspected of having pSS, as well as those with an

established diagnosis, requires an ophthalmic evaluation to identify potential complications of dry eye syndrome.

Table 2. 2016 ACR/EULAR Classification Criteria for Primary Sjögren's Syndrome

Criteria	**Description**	**Points**
Ocular Symptoms	Presence of symptoms of dry eyes (e.g., feeling of dryness, sandy sensation, or foreign body in the eye).	1 point
Oral Symptoms	Presence of symptoms of dry mouth (e.g., feeling of dry mouth or frequent need to drink liquids to swallow food).	1 point
Schirmer's Test (Eye Dryness Test)	A test to measure tear production (Schirmer's test < 5 mm in 5 minutes indicates dryness).	1 point (if positive)
Ocular Staining (Fluorescein)	Staining of the ocular surface to assess damage (score ≥ 4 in the cornea and conjunctiva under a slit-lamp examination).	1 point (if positive)
Salivary Gland Biopsy	Presence of focal lymphocytic sialadenitis in minor salivary glands, assessed by biopsy (focus score ≥ 1).	3 points (if positive)
Anti-SSA (Ro) or Anti-SSB (La) Antibodies	Presence of anti-SSA (Ro) or anti-SSB (La) antibodies.	3 points (if positive)
Total Score	The points are added together, and the total score determines whether the patient meets the criteria for pSS. **Diagnosis of Primary Sjögren's Syndrome (pSS)** is made if the patient scores **4 points or more** based on the above criteria.	**≥ 4 points**
Exclusion criteria: prior radiation of the head and neck, active HCV infection (confirmed by polymerase chain reaction), AIDS, sarcoidosis, amyloidosis, graft-versus-host disease, IgG4-related disease.		

Idiopathic Inflammatory Myopathies

Dermatomyositis (DM) and polymyositis (PM) are idiopathic inflammatory myopathies characterized by progressive, symmetrical muscle weakness, primarily affecting the proximal muscles. Besides muscles, other organs, including the heart, gastrointestinal system, skin, lungs and eyes, can also be affected [37]. Ocular symptoms are uncommon in DM and even rarer in PM. When they do occur, they can affect any part of the eye, with the most frequent manifestation in DM being heliotrope-like changes on the skin of the eyelids [37,38]. The second most common ocular complication in DM is retinopathy, which can threaten vision and may be asymptomatic [37,38]. On ophthalmic examination, findings may include cotton-wool spots, retinal hemorrhages, proliferative retinopathy, exudative retinal detachment and choroidopathy. While the exact mechanism behind these changes is not fully understood, vasculitis is believed to play a role [37]. Because these changes are often asymptomatic, any patient suspected of having DM or PM should undergo an eye exam. Retinopathy is more common in children with DM, where vasculitis is frequently seen [38]. In rare cases, inflammatory involvement of the eyelids and extraocular muscles can occur, resulting in ptosis, proptosis, restricted eye movement, and double vision. Disorders of eyelid and eyeball mobility can lead to dry eyes secondary to increased tear film evaporation and corneal epithelial damage [37]. It is also important to consider the potential side effects of chronic corticosteroid therapy, such as cataracts and steroid-induced glaucoma [37].

Relapsing Polychondritis

Relapsing polychondritis (RP) is a rare systemic condition characterized by recurrent inflammation of cartilage-rich tissues, particularly those abundant in proteoglycans. The most common symptoms include inflammation of the ears, nose, tracheal cartilage and arthritis, though other organs such as the skin, heart and eyes can also be affected [39]. Eye involvement occurs in 20-61% of RP patients and ocular changes may even be the first indication of the disease [39]. The most common ocular manifestations include scleritis (23-47%), conjunctivitis (5-35%), uveitis (3-26%) and keratitis, including peripheral ulcerative keratitis (7%) [39]. Scleritis associated with RP is more often bilateral, recurrent, necrotizing and accompanied by visual disturbances [39]. The most common form of necrotizing scleritis is acute necrotizing scleritis with the presence of hypopyon (a leukocytic exudate in the anterior chamber) [39]. Additionally, RP patients may develop cataracts (often due to prolonged corticosteroid therapy), retinopathy (cotton-wool spots, retinal hemorrhages, retinal detachment), retinal

vessel occlusion (due to vasculitis). In rare cases, optic neuropathy or pseudotumor of the orbit may also occur [39].

IgG4-Related Disease

Ocular and orbital involvement in IgG4-related disease (IgG4-RD), which is characterized by a typical clinical presentation and distinctive histopathological features, is referred to as IgG4-related ophthalmic disease (IgG4-ROD). Among the many symptoms of IgG4-RD, involvement of the orbital structures (including the lacrimal glands) along with the pancreas, bile ducts or salivary glands is one of the most common manifestations, affecting an estimated 10 to 50% of patients [40]. Typically, IgG4-ROD manifests as painless swelling of the lacrimal glands with 62% of cases being bilateral [41]. Depending on the structures involved and the progression of the disease, symptoms may include eyelid swelling, exophthalmos, orbital pain, restricted eye movement, double vision and dry eyes [42]. Although IgG4-RD usually does not significantly impair visual acuity, there are reports of vision loss due to optic neuropathy with some theories suggesting inflammatory demyelination [43,44]. Imaging studies of orbital tissue involvement, in addition to the lacrimal glands, may show changes in other structures such as extraocular muscles, nerves (e.g., thickening of the infraorbital nerve), fat tissue, nasolacrimal duct, eyelids, and may resemble an inflammatory pseudotumor. What is more, bone destruction can also occur [45,46]. Direct involvement of ocular structures in IgG4-ROD is rare, with cases mainly describing uveitis, scleritis and conjunctival infiltration [46]. The co-occurrence of symptoms outside of the ocular system in IgG4-ROD patients is observed in 75-100% of cases and is associated with bilateral disease progression (79% vs. 14% in unilateral IgG4-ROD) [47]. In the differential diagnosis of orbital pseudotumor in IgG4-RD, among others, neoplastic diseases (especially lymphatic system-related), granulomatosis with vasculitis, sarcoidosis and myofibroblastic tumors should be considered [48]. Chronic simultaneous inflammation of the lacrimal glands and salivary glands, referred to as Mikulicz's disease, which was once considered a seronegative variant of Sjögren's syndrome, is now classified as an IgG4-related disease. Both entities require thorough differential diagnosis. The 2019 ACR/EULAR classification criteria for IgG4-RD emphasize that a definitive diagnosis requires not only serological and imaging tests but often a biopsy of the lesion and histopathological evaluation, especially since 20% of cases may have normal IgG4 serum levels [47,49].

Granulomatosis with Polyangiitis

Granulomatosis with polyangiitis (GPA) is a rare autoimmune disorder characterized by the formation of granulomas, tissue necrosis and inflammation of small and medium-sized blood vessels [50]. As a systemic disease, it can impact multiple organ systems, including the respiratory tract and kidneys. Orbital involvement occurs in 28-58% of cases, with 8-16% of patients experiencing ocular symptoms as the initial signs of the disease [50,51]. The underlying mechanism of GPA involves antibodies against neutrophil cytoplasm (ANCA), which trigger neutrophil degranulation and the release of lytic enzymes that damage the endothelium and cause vasculitis. A hallmark of GPA is the presence of c-ANCA (specifically PR3-ANCA or anti-proteinase 3) in 90% of cases, while p-ANCA (MPO-ANCA or anti-myeloperoxidase) is found in 10% of patients [50]. In GPA, any ocular structure and surrounding tissues may be involved. Approximately 8% of patients experience irreversible vision loss [51]. The most common symptom is scleritis and episcleritis [25,52]. In 16-38% of patients scleritis occurs, often with more severe progression compared to non-GPA patients, leading to scarring and even perforation of the eye [50]. The inflammatory process can spread to adjacent structures, including the cornea (ulcerations) and choroidal structures [50]. Orbital involvement is frequent, either from inflammation spreading from the paranasal sinuses or directly affecting the orbital tissues, such as the lacrimal glands, extraocular muscles and soft tissues [50]. Orbital pseudotumor is a characteristic feature, causing exophthalmos, double vision, pain, eyelid swelling and conjunctival irritation, which needs to be differentiated from a proliferative process [50,52]. Conjunctival involvement occurs in 16% of cases, with symptoms including ulceration, necrotizing conjunctivitis and scarring conjunctivitis with adhesions [50]. Inflammatory changes in the lacrimal glands lead to dry eye syndrome. Corneal involvement is also frequent, with characteristic peripheral ulcerative keratitis, which is typically unilateral but can be bilateral in 40% of cases, sometimes occurring alongside scleritis [50]. All these symptoms can lead to corneal perforation. Uveitis is a less common symptom, affecting around 10% of patients, typically as a secondary manifestation following corneal or scleritis inflammation. It can affect the anterior, intermediate and posterior segments of the eye [25,50]. Retinal and choroidal involvement is rarely observed and usually presents with arterial and venous retinal vessel occlusion due to inflammation or compression [50]. Optic neuropathy may occur as a result of vasculitis or from compression due to granulomatous changes in the orbit [50].

Eosinophilic Granulomatosis with Polyangiitis

Eosinophilic granulomatosis with polyangiitis (EGPA) is a systemic condition characterized by inflammation of small and medium-sized blood vessels, primarily affecting the lungs, paranasal sinuses, skin, heart and gastrointestinal tract. The disease mechanism involves the activation of eosinophils, which leads to excessive cytokine production, direct organ damage and the formation of necrotizing granulomas [53]. ANCA antibodies also contribute to the disease, with p-ANCA present in 30-40% of cases [53]. Ocular involvement in EGPA is relatively uncommon and typically manifests in two forms: orbital pseudotumor (also known as idiopathic orbital inflammation) and vasculitis with ischemic symptoms (including retinal artery occlusion, ischemic optic neuropathy, vasculitis, retinal swelling and transient vision loss) [53]. Orbital pseudotumor symptoms usually affect younger patients, while vasculitis-related symptoms are seen in older patients [53]. This is associated with the stage of EGPA progression: the prodromal stage, with allergy and asthma symptoms, the eosinophilic phase with granulomatous infiltration in organs and the final stage with vasculitis [53].

Behçet’s Disease

Behçet's disease is a chronic, recurrent multisystem vasculitis of unknown origin, most commonly observed in the Middle East and East Asia. It is associated with the presence of HLA-B51 in 40-80% of patients and those with this genetic marker are predisposed to develop ocular symptoms [54,55]. The disease typically presents with mucocutaneous aphthae affecting the mouth, genitals and skin, along with recurrent uveitis. The eye is the most commonly affected organ in Behçet’s disease [57]. It is one of the most frequent causes of noninfectious uveitis, affecting 50-90% of patients [55]. Uveitis typically develops 2-3 years after the onset of the disease, though in 10-20% of cases, it is the first manifestation of the disease. It leads to blindness in 16-25% of the patients [56]. In Behçet’s disease, uveitis is characterized by chronic, recurrent, bilateral, non-granulomatous inflammation of the uvea and retinal vessels [54-56]. It can affect the anterior (11%) or posterior (28.8%) segments of the eye with panuveitis being the most common manifestation (60.7%) [56]. Major complications of uveitis include cataracts, posterior synechiae and glaucoma [54]. Posterior segment complications (maculopathy, optic nerve atrophy) are the main causes of permanent vision loss in patients with severe uveitis [52]. Retinal vessel inflammation leads to retinal neovascularization, intraretinal hemorrhages and cystoid macular edema, seen in about 60% of Behçet’s disease

cases [54]. Vitreous inflammation (visible at the onset of an attack) resolves with the formation of linear, pearl-like deposits in the lower peripheral retina, which is a pathognomonic sign of uveitis in Behçet's disease [55]. A poor prognostic factor indicating severe posterior segment involvement is the presence of hypopyon (the presence of pus in the anterior chamber of the eye), seen in 5-30% of Behçet's disease cases [54,55]. What is worth mentioning, it is characterized by the absence of fibrous exudate in the anterior chamber, allowing it to move freely under gravity (in contrast to hypopyon seen in ankylosing spondylitis, where it is viscous and confined to the anterior segment) [55].

Sarcoidosis

Sarcoidosis is frequently described as a cause of inflammatory diseases within the eyes. This includes uveitis, scleritis or episcleritis, optic neuropathy and granulomatous conjunctivitis. Ocular symptoms are sometimes the only clinical manifestation of sarcoidosis and in some cases, they may precede changes seen in chest radiographs. In systemic sarcoidosis, ocular involvement is observed in 30-60% of cases [58] (79% according to Japanese studies, 17% in Turkish studies [59]), with uveitis as the most common ocular manifestation. The symptoms of uveitis vary – from an asymptomatic course, diagnosed during routine check-ups, to severe pain with redness of the eye, and the risk of complications such as posterior synechiae and cataracts. Sarcoidosis can also involve the lacrimal glands, leading to dry eye symptoms and associated complications such as conjunctivitis or corneal damage. In some cases, it may cause choroid or retina inflammation, periocular retinal vasculitis, granulomas on the optic nerve head or choroid and a worsening of visual acuity. A rare but severe complication is vision loss. The definitive diagnosis is confirmed through histological examination of a biopsy from the affected tissue. If conjunctival nodules are present, they may show granulomatous inflammation; however, in many cases, there is no ocular pathology that can be biopsied [58].

Tubulointerstitial nephritis and uveitis

Tubulointerstitial nephritis and uveitis (TINU) are diagnosed when both iritis and acute tubulointerstitial nephritis are present. Ocular symptoms usually include bilateral acute non-granulomatous iritis with conjunctivitis and endothelial deposits on the cornea [60]. This condition can coexist with diseases such as rheumatoid arthritis, Sjögren's syndrome or IgG4-related diseases, but in about half of the cases there is no association with any other autoimmune

disorder. TINU may also occur as an adverse effect of medications, such as NSAIDs. This syndrome was first described in 1975, and since then, there have been no clear guidelines for its treatment or precise data on its incidence [60].

Treatment

treatment of uveitis aims to suppress the inflammatory process locally and prevent its extension to other ocular structures [1]. If another underlying autoimmune condition is identified, systemic treatment is also indicated (including oral corticosteroids, immunosuppressive drug therapy and biologics). Patients with anterior uveitis are typically treated with topical corticosteroids, while those with posterior uveitis are managed with systemic medications. For patients with panuveitis, both topical corticosteroids and systemic treatments are usually recommended. Locally, anti-inflammatory medications, both nonsteroidal anti-inflammatory drugs (NSAIDs) and glucocorticosteroids (GCS) are used, as well as mydriatics (to dilate the pupil) and cycloplegics (to paralyze the ciliary muscle), which help prevent posterior synechiae and provide pain relief by stopping the contraction of the ciliary and sphincter muscles of the pupil [2].

Corticosteroids in their various forms are generally the first-line treatment for non-infectious uveitis. Even in cases where immunosuppressive therapy is required, corticosteroids are typically included in the initial treatment regimen. While corticosteroid eye drops are effective for treating inflammation in the anterior chamber, they have limited penetration to the posterior eye structures, making them less suitable for treating intermediate and posterior uveitis.

Topical corticosteroids, like all forms of corticosteroids, carry key risks, primarily cataract formation and increased intraocular pressure, which can lead to glaucoma. In children, cataracts also pose the additional danger of amblyopia or permanent vision loss due to insufficient visual stimuli during brain development. Untreated inflammation itself can lead to cataracts, glaucoma and other irreversible complications. Research on chronic anterior uveitis in juvenile idiopathic arthritis has shown that active uveitis is a stronger risk factor for cataract development than the use of topical corticosteroids. Additionally, a long-term study of a large uveitis cohort found that active uveitis significantly increases the risk of elevated intraocular pressure [61-64]. Topical prednisolone acetate 1%, when used three times a day, has a low risk of cataract formation, and a retrospective cohort study indicates that prednisone at doses of ≤7.5 mg/day does not raise the risk of cataract formation or intraocular pressure elevation. Thus, the

current treatment approach prioritizes controlling inflammation with the necessary corticosteroid doses, reducing long-term corticosteroid use by incorporating immunosuppressive therapy and managing any ocular complications from corticosteroid use.
In severe cases or in patients who may not comply with the prescribed treatment, sub-conjunctival GCS injections are used. In cases complicated by cystoid macular edema, GCS can be administered periocularly or intravitreally. Corticosteroids can be delivered directly into the vitreous cavity through either injection or implant. Intravitreal triamcinolone acetonide (Triescence) is frequently used for treating non-infectious intermediate, posterior and panuveitis as well as uveitic macular edema. Corticosteroid implants, available in both short- and long-acting formulations, are placed into the vitreous cavity, providing a prolonged release of the drug over time. The biodegradable dexamethasone implant (Ozurdex) releases the steroid for up to six months [65-67]. A randomized controlled trial of the dexamethasone implant in uveitis patients showed significant reductions in inflammation and improvements in visual acuity.

What is worth mentioning, a large, multicenter randomized clinical trial evaluated the effectiveness of three different regional corticosteroid injection treatments for uveitic macular edema: one being periocular triamcinolone acetonide, followed by intravitreal triamcinolone acetonide and the last being intravitreal dexamethasone implant [68]. While all three treatments were effective, intravitreal corticosteroids were found to be more effective than periocular triamcinolone. Both intravitreal triamcinolone and the dexamethasone implant showed similar levels of effectiveness and duration of action. Furthermore, all three treatments were associated with similarly low rates of intraocular pressure increase.

Systemic corticosteroids are commonly used to manage severe inflammation or inflammation affecting the posterior segments of the eye, such as in intermediate, posterior, and panuveitis. Following the treatment protocol for rheumatologic conditions, the typical starting dose is prednisone at 1 mg/kg/day, with a maximum of 60 mg/day [69,70]. If the response is satisfactory, the prednisone dose is usually tapered after 2-4 weeks. The treatment goal is to lower inflammation to a level where the dose is ≤7.5 mg/day, a dosage that is generally well tolerated over the long term with a low risk of systemic side effects [71,72]. In cases of severe, vision-threatening ocular inflammation, high-dose intravenous corticosteroids are a safe and effective short-term treatment option [73,74].

In cases of chronic or recurrent inflammation - often seen in patients with autoimmune diseases - disease-modifying antirheumatic drugs (DMARDs) are used. The decision to start DMARDs is based on progressive vision impairment despite intensive local treatment and a

recurrence of inflammation when reducing the dose of GCS (less than 7.5-10 mg of prednisone per day) [75]. The first-line drug is cyclosporine (CsA), which has been used with good results (up to 74% total remission at doses of 2-5 mg/kg/day) since the 1980s. An alternative to CsA is methotrexate (MTX), especially recommended in cases with joint symptoms of autoimmune disease. According to observations published in 2001, MTX in doses of 7.5-40 mg weekly led to remission in 76% of patients with UA [75]. The most commonly used dose is 15 to 25 mg once a week and daily doses exceeding 30 mg/week are generally not used in clinical practice. Less commonly, sulfasalazine is used for preventing recurrent UA [76]. A drug with similar effects to CsA is tacrolimus, which is primarily used in the treatment of posterior uveitis, at oral doses of 0.05-0.2 mg/kg/day. Its use is recommended in cases of CsA ineffectiveness or toxicity [77]. A currently popular drug is mycophenolate mofetil (MMF), which can be administered in combination with CsA and GCS at daily doses ranging from 500 mg to 2g [78]. MMF is well tolerated and causes few complications.

In patients treated for autoimmune diseases with TNF inhibitors, their impact on reducing the number of UA episodes has been observed. A review of the literature from 2013, summarizing over 10 years of using anti-TNF drugs, indicated that treatment with infliximab in patients with Behçet's disease led to remission in 60% of cases of uveitis within the first year of treatment, and 75% of patients showed significant improvement after the first dose (3-10 mg/kg body weight), with a decrease in the number of acute UA episodes from 40 to 5 over the course of a year in a group of 12 patients [78, 80]. In a study of children with systemic juvenile idiopathic arthritis (with recurrent UA despite systemic GCS and MTX or CsA treatment), remission was observed in 86% of patients after 10 weeks of infliximab treatment [78,79]. In an observation comparing adalimumab and infliximab, it was found that the time to noticeable improvement in visual acuity and reduction in recurrence rates was similar in patients treated with both drugs, although long-term remission was more frequent in the adalimumab-treated group [78]. In patients treated with etanercept, its efficacy in treating UA and preventing recurrence was found to be similar to that of patients treated with somatostatin analogs (SSA) [1]. Due to the lack of randomized studies and the undoubted economic burden of using this group of drugs, their use should be considered only when therapy with at least two classic drugs has failed [79] or when vision impairment is progressing, or it is not possible to discontinue GCS while using DMARDs.

The literature also includes reports on the effectiveness of other biologic agents in treating UA, such as tocilizumab (anti-IL6), daclizumab (anti-IL2Rα), abatacept (anti-CTLA4) and anakinra (anti-IL1) [78,79]. However, these are single reports, and the data concerns

heterogeneous and often small patient groups, which makes it difficult to form binding recommendations for their use. Nevertheless, these observations open up possibilities for exploring new treatment options for UA and preventing its complications, including vision loss in many patients.

Conclusions

In summary, the most frequent inflammatory eye complications seen in rheumatic diseases are uveitis encompassing over 30 distinct conditions, and the primary goal of its evaluation is to determine the underlying cause. Some forms of uveitis are linked to infections or systemic diseases, while others are thought to be immune-mediated and confined to the eyes. Diagnosing uveitis requires a combination of clinical assessment - both ocular and systemic - and targeted laboratory tests.

What is more, dry eye symptoms are a frequent complication of rheumatic diseases. In primary Sjögren's syndrome, they are one of the characteristic clinical features of the disease and result from the inflammatory process involving infiltration of the lacrimal glands, leading to a deficiency in the aqueous phase of the tear film. Corneal, scleral and episcleral inflammations are associated with systemic vasculitis, including vasculitis accompanying other systemic connective tissue diseases such as systemic lupus erythematosus or Sjögren's syndrome. Failure to recognize or late diagnosis of ocular changes in the course of rheumatic diseases can result in severe complications, including blindness. A particularly vulnerable group are children with systemic juvenile idiopathic arthritis, in whom the inflammatory process in the eye may proceed without symptoms for a long time, therefore they are especially at risk for severe complications.

Rheumatic diseases are associated with inflammation of various structures in the eye and the type of structure involved and the type of inflammation can guide clinicians toward a possible diagnosis. In a defined rheumatic disease, specific changes in the eye can be expected. This knowledge often allows for the identification of the underlying disease and prevention of severe complications, including those affecting the eye, such as worsening visual acuity or blindness, through the establishment of appropriate therapy. Treatment for non-infectious uveitis typically involves corticosteroids and, in certain cases, immunosuppressive therapy is also used. For patients with uveitis associated with systemic conditions, collaboration between ophthalmologists and rheumatologists is essential for managing both the eye and systemic

issues, which can help prevent vision loss and improve the overall quality of life for patients with rheumatic disorders.

Disclosure

Author Contributions:

Conceptualization: Agnieszka Borowiec
Methodology: Agnieszka Borowiec, Kinga Borowiec
Validation: Kinga Borowiec
Formal Analysis: Agnieszka Borowiec
Investigation: Kinga Borowiec
Resources: Kinga Borowiec
Data Curation: Agnieszka Borowiec
Writing - Original Draft Preparation: Kinga Borowiec, Agnieszka Borowiec
Writing - Review & Editing: Kinga Borowiec, Agnieszka Borowiec
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