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The Latest Research Progress on Sympathetic Ophthalmia—A Brief Literature Overview

Meinian Nong, Wenhao Ma

Graduate School of Youjiang Medical University for Nationalities, Baise, China
Email: 15278639769@163.com

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Abstract

Sympathetic ophthalmia (SO) is a rare bilateral granulomatous panuveitis, usually secondary to penetrating ocular trauma or intraocular surgery. Symptoms typically occur 5 days to 56 years after trauma or surgery, but most commonly manifest 2 weeks to 2 months postoperatively. The main clinical features include acute or chronic granulomatous uveitis, accompanied by Dalen-Fuchs nodules and choroiditis. Systemic glucocorticoids and steroid-sparing immunosuppressants remain the first-line treatments. This article systematically summarizes the latest research advances in the epidemiology, pathogenesis, and clinical management of SO, aiming to provide references for clinical practice and scientific research.

Keywords

Sympathetic Ophthalmia, Granulomatous Uveitis, Ocular Trauma, Panuveitis, Incidence

1. Historical Perspective

Sympathetic Ophthalmia (SO) is a rare bilateral granulomatous uveitis that develops following penetrating ocular trauma or intraocular surgery. This condition typically affects both eyes after trauma or surgery to one eye, particularly when the uveal tissue is involved. The injured eye is termed the "trigger eye", while the contralateral unaffected eye is called the "sympathetic eye". Symptoms may appear days to several years after injury, but most cases present within one year of the inciting event, usually insidiously, and are characterized by granulomatous inflammation. Hippocrates first described similar symptoms as early as 2000 years ago. In the early 19th century, Scottish ophthalmologist Sir William Mackenzie provided the first comprehensive description and formally named the disease [1]. The risk of bilateral vision loss due to SO should not be underestimated, as it sig-

nificantly impacts patients' quality of life. With the continuous advancement of medical science and technology, new treatment methods and strategies have emerged; however, due to the complex pathogenesis of SO, many unsolved mysteries remain.

2. Epidemiology

The incidence of SO in the general population is extremely low, making it difficult to accurately calculate its population-based prevalence. Initially, SO was thought to be exclusively associated with penetrating ocular trauma, but several studies have demonstrated an increased incidence following various ophthalmic procedures, including vitreoretinal surgery [2]-[4]. A 10-year study from a tertiary referral center in Taiwan region indicated that ophthalmic surgery has become the primary cause of SO in recent years, coinciding with the rising volume of ophthalmic surgical interventions [5]. A new study shows that the overall incidence of sympathetic ophthalmia has declined in recent years. From the 19th to 20th century, the incidence of sympathetic ophthalmia following ocular trauma ranged between 0 - 16%. However, with advancements in trauma management and surgical techniques, the modern postoperative incidence of sympathetic ophthalmia has decreased to 0.072% - 0.8%. The primary risk factors for sympathetic ophthalmia have also shifted from early ocular trauma to recent ocular surgeries. Although the reported incidence of postvitreoretinal surgery sympathetic ophthalmia is higher than that after cataract surgery (16.93% vs. 5.74%), the actual incidence after vitreoretinal surgery remains at only 0 - 0.13%, while the incidence after cataract surgery is 0 - 0.2283%. Since the beginning of the 21st century, the incidence of sympathetic ophthalmia (SO) in developed countries has dropped to 0.04% - 0.8%, while in developing countries it remains high at 0 - 1.11%. Although modern microsurgical techniques have significantly reduced SO incidence, clinicians must remain vigilant about the potential risks of postoperative sympathetic ophthalmia to further prevent its occurrence [6].

3. Etiology and Risk Factors

Existing studies generally believe that sympathetic ophthalmia is caused by a cell-mediated immune response triggered by retinal and uveal antigens exposed by trauma or surgery [7]. Risk factors for the onset of the disease include the nature of the trauma, the degree of exposure of the uveal tissue, and possible genetic susceptibility [8]. In addition to penetrating ocular trauma, several studies have reported cases of sympathetic ophthalmia after various operations and procedures [2]-[4] [9]-[24]. Ocular treatment, including penetrating ocular treatment and non-penetrating ocular intervention, has become an increasingly common risk factor for sympathetic ophthalmia, and vitreoretinal surgery should be considered as a potential trigger for sympathetic ophthalmia [25]. A cohort study found that the risk of multiple vitrectomy procedures is higher, and that the risk increases exponentially with the number of vitrectomy procedures [24]. Compared with

vitrectomy, the probability of trauma-induced SO is up to 4 - 5 times higher [11]. While there is no biological evidence that a particular age, sex or race is at higher risk, the new study suggests that women may be at risk of sympathetic ophthalmia and that older patients may be at risk of developing it earlier [26].

4. Nosogenesis

Despite its significant disability, sympathetic ophthalmia (SO) remains considered an autoimmune disorder with an unclear pathogenesis. Current research consensus indicates that the pathogenesis is closely associated with immune responses. This perspective is primarily based on the following observations: When one eye (commonly termed the "induced eye") undergoes trauma or intraocular surgery, antigens from the previously isolated uvea and retina may migrate through the bloodstream or lymphatic circulation to the healthy contralateral eye (the "sympathetic eye"). This exposure triggers a systemic immune response mediated by cellular immunity. Multiple mechanisms are involved, including the activation of the interleukin-23/IL-17 pathway [27]. Furusato *et al.* demonstrated that inflammatory granulomatous lesions exhibited elevated levels of macrophages (CD68) and Th17 cells (IL-17), whereas non-granulomatous infiltrated tissues showed aggregation of Th1 cells (IFN-γ). In the early stages of the disease, CD4+ helper T cells and CD4+ T helper cells are predominant, while in the later stages, CD8+ T helper cells and CD8+ cytotoxic T cells are predominant [28]. Research on the relationship between HLA polymorphism and sympathetic ophthalmia indicates that DRB1 *04:05 and DQB1 *04:01 alleles, along with the DRB1 *04:05-DQB1 *04:01 haplotype, may serve as potential risk factors for sympathetic ophthalmia [29]. Shindo's team found that Japanese individuals carrying HLA-DRB1*04 (relative risk 13.7), DQA1*03 (12.8), and DQB1*04 (15.4) had significantly higher risks of developing sympathetic ophthalmia compared to healthy controls [30].

5. Clinical Features and Investigations

The clinical manifestations of sympathetic ophthalmitis may vary, and the symptoms usually occur within 5 days to 56 years after trauma or surgery, but most often within 2 weeks to 2 months. In some cases, the onset may be delayed for several years, making diagnosis challenging [31]. Common symptoms include decreased vision, redness, pain, and sensitivity to light or loss of accommodation. Slit-lamp examination reveals acute/chronic granulomatous uveitis with xanthoid keratic precipitates in the anterior segment, while the posterior segment shows vitreous inflammation, exudative retinal detachment, choroiditis, optic nerve edema, and Darrell-Fox nodules (subretinal inflammatory cell aggregates). A sunset fundus pattern resembling Vogt-Koyanagi-Harada disease may also be observed. Some extraocular lesions may also occur, such as vitiligo, hair whitening, alopecia, hearing loss, or meningeal irritation signs. Sympathetic ophthalmia manifests differently in its acute and chronic phases, with distinct posterior seg-

ment involvement. Patients exhibiting MEWDS-like symptoms, rapidly progressive choroidoretinitis, or a history of contralateral eye trauma should be evaluated for active sympathetic ophthalmia. Following acute inflammation resolution, peripapillary subretinal fibrosis and perivascular ring-like atrophy may serve as key diagnostic indicators of this condition [32].

The diagnosis of sympathetic ophthalmia is mainly based on clinical manifestations and medical history. Key criteria for sympathetic ophthalmia included bilateral uveitis with 1) a history of unilateral ocular trauma or surgery and 2) an anterior chamber and vitreous inflammation or a panuveitis with choroidal involvement [33]. Although diagnostic imaging and laboratory tests can assist in diagnosis, they cannot confirm the diagnosis. Such as fluorescein fundus angiography (FFA), indocyanine green angiography (ICGA), optical coherence tomography (OCT) [15], OCT angiography (OCTA), B-ultrasound, and autofluorescence imaging. Spectral domain optical coherence tomography (SD-OCT) and others [12] [34]. The study also analyzed the choroidal vascular structure of sympathetic ophthalmia (SO) patients using spectral-scan optical coherence tomography (SS-OCT) images. The cardiovascular index showed significant elevation in SO patients, providing a novel non-invasive biomarker for assessing disease activity. Superconducting Optical Coherence Tomography (SS-OCT) provides valuable qualitative and quantitative parameters, which is expected to play an important role in the diagnosis and follow-up of SO patients [35].

6. Antidiastole

Sympathetic ophthalmia should be differentiated from the following conditions; Lenticular Allergic Uveitis: Common Features: Both conditions can present with bilateral uveitis and similar inflammatory manifestations. Key Differentiation: Lenticular allergic uveitis typically develops following lens trauma or cataract surgery, with inflammation primarily localized around the lens. When contralateral eye inflammation occurs, the original injured eye's inflammation has largely subsided. In contrast, sympathetic ophthalmia develops when inflammation persists or worsens in the injured eye (the triggering eye), while the unaffected eye (the sympathetic eye) remains unaffected. Vogt-Koyanagi-Harada syndrome (VKH syndrome) shares similarities with bilateral uveitis, presenting with fundus changes such as sunset-like opacities and Daleen-Fuchs nodules. Key distinctions include: VKH syndrome is typically not associated with ocular penetrating injuries or intraocular surgeries, and is often accompanied by systemic symptoms like meningeal irritation, vitiligo, and hearing impairment. The bilateral lesions progress synchronously, with non-granulomatous inflammation predominating, and histopathologically, plasma cell infiltration is more pronounced [36] [37]. Similarities with Behcet's disease: Both conditions may present with ocular inflammation, manifesting as redness, pain, and decreased vision. Key differentiators: Behcet's disease is a systemic immune disorder. Beyond ocular involvement, it is accompanied by oral ulcers, cutaneous lesions, and genital ulcers, with no history

of ocular trauma or surgery. The ocular inflammation is typically non-granulomatous. Symptomatic similarities between sympathetic stimulation and sympathetic ophthalmia: Following trauma to one eye, the contralateral eye exhibits photophobia, tearing, and other irritative symptoms. Key distinctions: Sympathetic stimulation manifests only as transient neurological reflex symptoms without substantial uveitis signs in the contralateral eye, and symptoms resolve upon removal of the stimulus. In contrast, sympathetic ophthalmia presents with definitive uveitis features including keratoconjunctivitis (KP), aqueous humor opacity, and retinopathy. The diagnosis of sympathetic ophthalmia requires consideration of a characteristic medical history (including ocular penetrating trauma or intraocular surgery), the sequential onset and inflammatory patterns of bilateral uveitis, and exclusion of other similar conditions. When a patient has a confirmed traumatic history with uveitis in the contralateral eye, sympathetic ophthalmia should be strongly suspected, and prompt comprehensive evaluation is essential for accurate diagnosis.

7. Management of Sympathetic Ophthalmia

The inflammation of the eye caused by sympathetic ophthalmia is devastating and can lead to permanent blindness. Therefore, early detection and timely treatment are crucial, and timely intervention can significantly alter the course of the disease. The visual prognosis of sympathetic ophthalmia is closely related to timely diagnosis and treatment. Systemic glucocorticoids (both topically and systemically) and steroid-sparing immunosuppressants remain the primary treatment options [27]. The treatment regimen usually involves immunosuppressive therapy, including glucocorticoids, and, if necessary, combination with immunosuppressants such as cyclosporine, azathioprine, methotrexate, chlorphenamine, cyclophosphamide and mycophenolate mofetil to reduce the dosage of steroids [38]. Treatment usually starts with high doses of corticosteroids to suppress the immune response and control inflammation [39]. Because of the chronic nature of sympathetic ophthalmia, many patients require long-term use of immunosuppressants to maintain inflammation control and protect vision [21]. Corticosteroid therapy may be effective for acute or refractory sympathetic uveitis, but it is not enough to control the inflammation in the long term [40]. This often requires the use of steroid-sparing drugs such as methotrexate, azathioprine or biologic agents [39]. While pulse-dose glucocorticoids and immunosuppressive agents remain the first-line treatment, tumor necrosis factor-α (TNF-α) inhibitors and other biologics represent promising new therapeutic options [8]. Biological agents (especially anti-TNF drugs) may be effective in the treatment of SO-related inflammation, which is associated with the increased expression of TNF-α, TNF-α receptor and inducible nitric oxide synthase in photoreceptors. This molecular mechanism is the underlying cause of photoreceptor damage in SO lesions, resulting in vision loss even in the absence of visible retinal changes [41]. Currently, biologics are used only in small case series as a third-line treatment for patients

with sympathetic ophthalmia who have not responded to conventional immunosuppressive therapy [42].

In contrast to drug therapy, enucleation is only indicated in cases of complete loss of light perception or in cases of severe trauma. In the past, the academic community believed that removing the affected eye within two weeks after injury could effectively prevent sympathetic ophthalmia (SO) or make the disease progression benign. The treatment of prophylactic enucleation to prevent sympathetic ophthalmia (SO) in cases of visual loss within 14 days after eyeball rupture has been regarded as the golden rule in the academic community for a century. But existing data suggest that sympathetic ophthalmia can still occur even if the eyeball is removed within 14 days of trauma [43]. In modern medical practice, doctors will try to suture the eyeball over removing it whenever sufficient tissue is available. Although the 14-day rule for severe eyeball rupture lacks scientific basis and cannot completely prevent intraocular hemorrhage, the safe time limit for prophylactic eyeball removal remains unclear [44]. For the rare cases of intraocular hemorrhage, various new drugs are available to assist in treatment. The prevailing view is to preserve the ruptured eyeball as much as possible and avoid prophylactic removal to reduce the risk of rare intraocular hemorrhage. When the eyeball needs to be removed, enucleation is a more preferable alternative to simple removal of the eyeball in the absence of intraocular malignancy.

8. Prognosis

With the improvement of medical facilities, early intervention, advanced wound repair techniques and public awareness in modern society, the prognosis of sympathetic ophthalmia has been significantly improved. The visual recovery of patients with sympathetic ophthalmia has been significantly improved by timely use of corticosteroids and long-term immunosuppressive therapy [45]. Key measures to prevent sympathetic ophthalmia or vision loss caused by it include: a) prompt and meticulous initial repair of open eyeball injuries; b) removal of the damaged eyeball when surgical repair is deemed impossible; and c) prompt control of inflammation [46]. Sympathetic ophthalmia is a rare condition that is no longer considered to cause vision loss with the use of appropriate steroids and immunosuppressants. This eliminates the need for invasive procedures such as enucleation or enucleation of the ocular contents to prevent sympathetic ophthalmia. Preventing ocular trauma is essential for managing sympathetic ophthalmia. In daily life, individuals should enhance safety awareness and avoid activities that may cause eye injuries. For high-risk occupations such as construction workers and athletes, strict adherence to safety protocols and proper use of protective eyewear are crucial to minimize the risk of eye injuries. Boosting immunity is also a key measure to prevent sympathetic ophthalmia. A strong immune system can effectively fight off pathogens and reduce infection risks. In addition to the above measures, strengthening publicity and education is also an effective way to prevent sympathetic ophthalmia. By popularizing knowledge about sympathetic ophthalmia to

the public, we can raise awareness and emphasize the importance of this disease, thereby guiding people to take preventive measures voluntarily.

9. Conclusion

With the progress of immunology and ocular imaging technology, the understanding of sympathetic ophthalmia is developing, but there are still many deficiencies and broad prospects for research. The main problem is that the pathogenesis of sympathetic ophthalmia is still like a fog that has not been fully lifted. Although the existing theories provide us with some directions, many details still need to be further explored. For instance, the precise mechanisms of immune cells in sympathetic ophthalmia, the interaction networks among different immune cells, and how these immune responses cause ocular tissue damage remain critical challenges for future research. The current mainstream research direction continues to seek better ways to predict risk factors, how to prevent progression, and how to optimize long-term management to prevent relapse and preserve vision. This article not only enhances the scientific understanding of sympathetic ophthalmia, but also provides strong support for clinical diagnosis and treatment. While numerous challenges remain—such as fully elucidating the disease mechanisms and further enhancing therapeutic efficacy—this study undoubtedly lays a solid foundation for future exploration. We anticipate more groundbreaking research in the years to come. With sustained efforts, we have every reason to believe that we will gain deeper insights into this disease, develop more effective treatments and preventive measures, and ultimately bring hope to countless patients.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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