Avicenna Anatolian Journal of Medicine

Review

Non-Traumatic Ocular Emergencies

Authors & Affiliations

Mustafa Berhuni, İbrahim Edhem Yilmaz

Gaziantep İslam Science and Technology University, Gaziantep, Türkiye

Corresponding Author: İbrahim Edhem, M.D., Gaziantep İslam Science and Technology University, Gaziantep, Türkiye
E-mail: dredhemyilmaz@gmail.com

Submitted at: 25.10.2025 - Accepted at: 12.12.2025 - Published at: 19.12.2025
Avicenna Anatol J Med. Year; 2025, Volume: 2, Issue: 3

doi 10.5281/zenodo.17931707

Abstract

Non-traumatic ocular emergencies are vision-threatening conditions requiring immediate intervention without physical injury to the eye. These emergencies, including acute angle-closure glaucoma, retinal vascular occlusions, and endophthalmitis, can result in irreversible vision loss within hours if not promptly recognized and treated. Patient history and clinical presentation provide critical diagnostic clues. Early recognition of characteristic symptoms and signs, coupled with appropriate emergency intervention and timely ophthalmologic referral, is essential for preserving visual function. This review provides emergency physicians with evidence-based diagnostic criteria, treatment algorithms, and management strategies for eight major non-traumatic ocular emergencies, emphasizing time-critical interventions that can prevent permanent visual disability.

Keywords: Eye Injuries, Emergency Treatment, Emergencies

INTRODUCTION

Non-traumatic ocular emergencies refer to vision-threatening conditions requiring immediate intervention without any physical injury to the eye. In these conditions, rapid assessment of visual function and appropriate medical intervention are crucial to prevent permanent vision loss. Patient history can be helpful in diagnosis. Therefore, detailed anamnesis should be obtained from patients. The symptoms such as decreased visual acuity, eye pain, floaters, photophobia, and red eyes should be questioned carefully. Non-traumatic Ocular Emergencies can be classified as acute angle-closure glaucoma, acute uveitis, central retinal artery occlusion, central retinal vein occlusion, optic neuritis, retinal detachment, infectious keratitis, and endophthalmitis. The correct management of these emergencies is of vital importance on the visual prognosis of patients. Non-traumatic ocular emergencies account for approximately 2-3% of emergency department visits, yet their potential for causing permanent disability is disproportionately high. The time window for effective intervention varies by condition—from hours in acute angle-closure glaucoma and central retinal artery occlusion to days in retinal detachment—making rapid triage and assessment critical. Emergency physicians serve as the first point of contact and must differentiate vision-threatening conditions from benign presentations.

Acute Angle-Closure Glaucoma

Acute angle-closure glaucoma (AACG) is an ocular emergency characterized by rapid elevation of intraocular pressure (IOP) due to obstruction of aqueous humor outflow. While multiple factors can precipitate AACG, the major predisposing factor is a narrow angle between the iris and cornea. The most common presenting symptoms include sudden-onset unilateral severe ocular or headache pain, red eye, blurred vision, nausea, and vomiting (1). Patients may also report seeing halos around lights due to corneal edema. The condition typically affects hyperopic eyes with shallow anterior chambers and shows a predilection for individuals of Asian descent, women, and those over 60 years of age (2). The diagnosis of AACG is supported by tonometric measurements showing IOP between 50 and 80 mmHg (3). Biomicroscopic findings include corneal edema, mid-dilated pupil, shallow anterior chamber, and narrow angle appearance on gonioscopy (4). Immediate treatment aims to reduce IOP to prevent irreversible optic nerve damage. Initial management includes a combination of topical beta-blockers (timolol 0.5%), alpha-2 agonists (apraclonidine 1%), and carbonic anhydrase inhibitors (dorzolamide), along with intravenous acetazolamide (500mg) or mannitol (1-2 g/kg, up to 300cc over 45 minutes) for systemic IOP reduction. Topical pilocarpine (2%) is administered after IOP begins to decrease to promote pupillary constriction. Definitive treatment

requires laser peripheral iridotomy in both eyes, as the fellow eye has a 40-80% risk of developing acute angle closure within 5-10 years without prophylactic treatment (5,6).

Acute Uveitis

Uvea consists of iris, ciliary body, and choroid. Uveitis is defined as inflammation of any or all parts of the uveal tissue. Anatomically, it can be classified as anterior, intermediate, posterior, and panuveitis (inflammation of all uveal tissues) (7). Based on disease course, it can be categorized as acute, recurrent, and chronic. Emergency presentations typically manifest as acute anterior uveitis or acute panuveitis. Acute anterior uveitis is commonly associated with HLA-B27-related conditions (ankylosing spondylitis, Reiter's syndrome, inflammatory bowel diseases, psoriatic arthritis) and can also occur in Behçet's disease (8). Panuveitis is most commonly observed in Behçet's disease (9). Acute uveitis may also present as an isolated condition without any underlying systemic disease. Symptoms of acute uveitis include sudden onset of blurred vision, red eye, floaters, and pain. Biomicroscopic examination reveals anterior chamber cells (inflammatory cells visible in the aqueous humor) and flare (protein leakage indicating breakdown of the blood-aqueous barrier), graded on a standardized scale. Additional findings include keratic precipitates on the corneal endothelium, hypopyon (layered white cells in the anterior chamber), iris nodules, and posterior synechiae (adhesions between iris and lens). In posterior and panuveitis, fundoscopy may reveal vitritis (inflammatory cells in the vitreous), retinal vasculitis (vascular sheathing), retinitis, and chorioretinal lesions. Treatment involves topical steroids and mydriatic drops, along with management of any underlying systemic condition. Initial emergency evaluation should include measurement of visual acuity, IOP, and detailed slit-lamp examination. Laboratory investigations are guided by clinical presentation and may include HLA-B27 testing for anterior uveitis, particularly in young males, and serologic testing for syphilis and tuberculosis when indicated (10).

Central Retinal Artery Occlusion

Central Retinal Artery Occlusion (CRAO) is an ocular emergency that can cause sudden and severe vision loss (11). Patients typically present with painless, unilateral sudden vision loss. CRAO represents an ophthalmic stroke requiring immediate intervention. The retina can tolerate only 90-100 minutes of complete ischemia before irreversible damage occurs, though partial perfusion may extend this window. Vision loss is typically profound, with acuity often reduced to counting fingers or light perception (12). The condition results from thromboembolism or vasospasm causing blockage of the central retinal artery, leading to impaired macular perfusion and sudden vision loss. Common embolic sources include the carotid artery, aortic arch, and heart. Giant cell arteritis is another significant cause of CRAO (13). Diagnosis is characterized by macular pallor and cherry-red spot appearance. Management remains controversial, but time-critical interventions include ocular massage, anterior chamber paracentesis to lower IOP and potentially dislodge emboli, sublingual isosorbide dinitrate for vasodilation, and carbogen (95% oxygen, 5% CO2) inhalation to promote retinal vasodilation (14). Hyperbaric oxygen therapy, when initiated within 24 hours, may improve outcomes by maintaining viable but non-functional retinal tissue (16). However, visual prognosis remains poor, with only 20-30% of patients achieving ambulatory vision (15). Concurrent evaluation for embolic sources is mandatory, including carotid duplex ultrasonography, echocardiography, and ESR/CRP to exclude giant cell arteritis in patients over 50. These patients also have increased risk of cerebral stroke and ischemic heart disease, requiring monitoring and preventive treatment (15).

Central Retinal Vein Occlusion

Central Retinal Vein Occlusion (CRVO) is a condition that can severely impact vision due to venous congestion and retinal ischemia resulting from retinal venous blockage (17). It is commonly associated with hypertension, bleeding diatheses, and Behçet's disease. CRVO can be classified into ischemic (poor prognosis) and non-ischemic (better prognosis) types based on capillary perfusion (18). Risk factors include the 'vascular triad' of hypertension, diabetes mellitus, and hyperlipidemia, along with hypercoagulable states, hyperviscosity syndromes, and systemic vasculitis. The condition results from thrombotic occlusion at the lamina cribrosa where the central retinal vein and artery share a common adventitial sheath (18). Patients typically present with painless, sudden unilateral vision loss. Fluorescein angiography characteristically shows widespread retinal hemorrhages and sometimes extensive retinal ischemia. Visual acuity is reduced in cases with macular edema. Cases with retinal ischemia require panretinal photocoagulation treatment. Cases with macular edema may show visual improvement with intravitreal anti-vascular endothelial growth factor injections (17). Some cases may require repeated injections. Ischemic CRVO carries high risk for neovascular glaucoma, a devastating complication occurring in approximately 40% of cases within 3-6 months due to anterior segment neovascularization from ischemia-induced VEGF production (19). Treatment of underlying conditions also plays a crucial role.

Optic Neuritis

A healthy optic nerve is essential for transmitting visual signals to the brain. Optic neuritis (ON) can impair signal transmission due to inflammation of the optic nerve. It typically presents as painful unilateral sudden vision loss in young individuals. The incidence ranges from 0.5 to 5 per 100,000 (20). The association with multiple sclerosis is significant: approximately 50% of patients presenting with isolated ON will develop MS within 15 years, and 70-80% of MS patients experience ON during their disease course. MRI findings of periventricular white matter lesions at initial presentation significantly increase MS risk (21). The development mechanism of ON is believed to involve autoimmune reaction

causing inflammation of the optic nerve myelin sheath (22). Some studies suggest viral etiology (23). It can also manifest as a symptom of demyelinating diseases such as Multiple Sclerosis (26). It predominantly affects women aged 20-40 years. Diagnosis relies on color vision testing, visual field testing, and optical coherence tomography. Color vision and visual field are typically impaired. Treatment with high-dose intravenous methylprednisolone (1g daily for 3-5 days) followed by oral prednisone taper accelerates visual recovery but does not affect final visual outcome. The Optic Neuritis Treatment Trial established that oral prednisone alone increases recurrence risk and should be avoided (24). Most patients recover to 20/40 or better vision within 6-12 months, though subtle deficits in color vision and contrast sensitivity may persist (24,25). Relative afferent pupillary defect (RAPD) is a hallmark sign. MRI of the brain and orbits with gadolinium is essential for identifying demyelinating lesions and stratifying MS risk. Recurrent ON attacks may occur in demyelinating diseases (26). Treatment requires a multidisciplinary approach.

Retinal Detachment

Retinal Detachment (RD) occurs when the neurosensory layer of the retina, a crucial multi-layered structure for vision in the posterior eye, separates from the retinal pigment epithelium. Patients present with unilateral sudden vision disturbance, curtain-like visual field defect, and floaters. High-risk groups include patients with high myopia (>6 diopters), history of cataract surgery, previous RD in the fellow eye (10% lifetime risk), lattice degeneration (1% annual risk), and trauma (27). Proliferative diabetic retinopathy and posterior vitreous detachment are additional predisposing factors. Classic symptoms progress from photopsia (flashing lights) indicating vitreous traction on the retina, to floaters from hemorrhage or pigment cells (Schaffer's sign or 'tobacco dust'), culminating in a progressive visual field defect described as a curtain or shadow. Central vision remains preserved until the macula detaches. The Amsler grid test may reveal metamorphopsia (distorted vision) in early macular involvement. RD is classified into three categories: rhegmatogenous, tractional, and exudative (28). Rhegmatogenous RD, the most common type, occurs when fluid enters the subretinal space through a retinal tear, causing separation of the neurosensory retina (28). The incidence ranges from 6 to 18 per 100,000 (29). It is commonly observed in high myopia and lattice degeneration cases (30). Diagnosis is made by fundus examination revealing the detached retina. Treatment approaches include pneumatic retinopexy for superior detachments, scleral buckle for phakic patients with localized tears, and pars plana vitrectomy with gas or silicone oil tamponade for complex cases. Macula-on detachments (macular sparing) require urgent surgery within 24 hours to preserve central vision, while macula-off detachments should be repaired within one week. Success rates approach 90% with primary surgery, though visual outcomes depend critically on macular involvement and duration of detachment (31).

Infectious Keratitis

Keratitis refers to inflammation of the corneal layer of the eye. Patients typically present with unilateral, sudden-onset severe pain and vision disturbance, and photophobia. Major risk factors include contact lens wear (especially extended-wear and poor hygiene), corneal trauma, ocular surface disease, and immunosuppression. Contact lens-related keratitis accounts for approximately 80% of cases in developed countries (32). Based on causative agents, it can be classified as bacterial, protozoal, fungal, and viral keratitis. Common bacterial pathogens include pseudomonas, staphylococci, and streptococci (33). Pseudomonas aeruginosa is particularly aggressive in contact lens wearers, capable of causing corneal perforation within 48 hours. Gram-positive organisms like Staphylococcus aureus and Streptococcus pneumoniae are more common in non-contact lens wearers and following corneal abrasions. The most common protozoal agent is acanthamoeba, typically contracted from swimming pools, which can cause permanent corneal damage (34). Common fungal pathogens include aspergillus, candida, and fusarium species, which are more prevalent in immunosuppressed patients and can cause irreversible severe vision loss (35). Common viral agents include Herpes simplex, Herpes zoster, and adenoviruses (36,37). Diagnosis is made through biomicroscopic observation of corneal epithelial defects, ulceration, and stromal infiltration. Diagnosis requires corneal scraping for Gram stain, culture, and sensitivity testing before initiating empiric therapy. Confocal microscopy can identify acanthamoeba cysts, and fungal stains (KOH, PAS) are essential when fungal etiology is suspected based on feathery infiltrates or satellite lesions.

Empiric treatment for bacterial keratitis begins with fortified antibiotics: fortified cefazolin (50 mg/mL) or vancomycin (50 mg/mL) for gram-positive coverage, combined with fortified tobramycin (15 mg/mL) or ceftazidime (50 mg/mL) for gram-negative coverage, administered hourly around the clock. Fourth-generation fluoroquinolones (moxifloxacin 0.5% or gatifloxacin 0.3%) provide good monotherapy for mild-to-moderate cases (38). Acanthamoeba keratitis requires dual therapy with polyhexamethylene biguanide (PHMB) 0.02% and either propamidine isethionate or chlorhexidine, administered hourly for several weeks. Fungal keratitis responds to natamycin 5% for filamentous fungi or amphotericin B 0.15-0.3% for yeast infections, though systemic antifungals (oral voriconazole or fluconazole) may be necessary for deep infections. Keratoplasty is performed for persistent corneal ulcers. Viral keratitis treatment includes topical and systemic acyclovir. Prophylactic oral acyclovir may be prescribed for up to one year in recurrent cases.

Endophthalmitis

Endophthalmitis represents an ophthalmic catastrophe with potential for complete and irreversible vision loss within days. It can be defined as inflammation involving all ocular structures due to colonization of infectious agents in the vitreous and aqueous (39). The

condition can be classified as exogenous (post-surgical or post-traumatic) or endogenous (hematogenous spread from systemic infection). Exogenous endophthalmitis accounts for >90% of cases. It is one of the most serious and significant ophthalmic emergencies where vision can be preserved with early treatment. It commonly occurs following eye surgery or trauma. The most common infectious agents are bacteria and fungi, with Staphylococcus epidermidis being the most frequent bacterial agent (40). The incidence following cataract surgery is 4 per 100,000 (41). Patient history typically includes recent eye surgery or trauma followed by sudden-onset unilateral severe pain and vision loss. Clinical severity can be stratified based on presenting visual acuity: vision of 20/40 to 20/200 suggests better prognosis, while light perception only or worse indicates severe infection with poor visual outcome despite aggressive treatment. Diagnosis is made through biomicroscopic observation of dense reaction in the anterior chamber and vitreous, hypopyon, and sometimes corneal edema. The Endophthalmitis Vitrectomy Study (EVS) established treatment protocols: immediate intravitreal antibiotics (vancomycin 1.0 mg/0.1 mL for gram-positive coverage and ceftazidime 2.25 mg/0.1 mL or amikacin 0.4 mg/0.1 mL for gram-negative coverage) are mandatory (42). Pars plana vitrectomy is recommended for patients with light perception only vision, providing both diagnostic (culture) and therapeutic benefits. Patients with better than light perception can be managed with intravitreal antibiotics and close observation, with vitrectomy reserved for non-responders. Systemic antibiotics (intravenous vancomycin and ceftazidime or fluoroquinolones) are added for severe cases. Topical fortified antibiotics, cycloplegics (atropine 1%), and corticosteroids (after 24 hours) complete the regimen. Intravitreal corticosteroids (dexamethasone 0.4 mg) remain controversial but may reduce inflammation-mediated damage. Despite optimal treatment, visual outcomes remain poor: only 53% achieve 20/40 or better vision, and 15% progress to no light perception. Prognostic factors include initial visual acuity, causative organism (Staphylococcus epidermidis has better outcomes than Streptococcus species or gram-negative organisms), and time to treatment (42-44).

Emergency Ophthalmic Examination

Emergency physicians should perform a systematic ocular examination for suspected non-traumatic emergencies:

Visual Acuity: Measured for each eye separately using a Snellen chart or near card. Document with corrective lenses if available.

Pupillary Examination: Assess size, shape, and reactivity. Test for relative afferent pupillary defect (RAPD/Marcus Gunn pupil) using the swinging flashlight test—a critical sign in optic neuritis, CRAO, and severe retinal detachment (44).

Intraocular Pressure: Measured via tonometry. Normal range is 10-21 mmHg. IOP >30 mmHg suggests acute glaucoma; asymmetric low IOP may indicate globe perforation or inflammation.

Slit Lamp Examination: When available, assesses anterior segment structures including cornea (clarity, ulceration, infiltrates), anterior chamber (depth, cells, flare, hypopyon), iris (synechiae, neovascularization), and lens (clarity).

Direct Ophthalmoscopy: Evaluates optic disc (swelling, pallor, cup-to-disc ratio), vessels (occlusions, hemorrhages, sheathing), macula (cherry-red spot, edema), and peripheral retina (detachment, hemorrhages).

Visual Field Testing: Confrontation testing can identify field defects suggesting retinal detachment or optic nerve pathology.

CONCLUSION

Non-traumatic ocular emergencies represent time-critical conditions where emergency physician recognition and intervention directly impact visual outcomes. This review has outlined diagnostic criteria, evidence-based treatment protocols, and management strategies for eight major non-traumatic ocular emergencies that emergency physicians will encounter. Key principles include: (1) systematic examination with documentation of visual acuity, pupillary function, and IOP; (2) recognition of characteristic presentations requiring immediate intervention versus urgent ophthalmologic consultation; (3) initiation of time-critical treatments such as IOP reduction in AACG or intravitreal antibiotics in endophthalmitis; and (4) prompt ophthalmologic referral while avoiding delays in definitive care.

The variable time windows for intervention—from 90 minutes in CRAO to several days in retinal detachment—underscore the importance of rapid triage and risk stratification. Emergency physicians must maintain a high index of suspicion for vision-threatening conditions in patients presenting with acute vision changes, even when symptoms appear mild. Misdiagnosis or delayed treatment inevitably leads to permanent visual disability that profoundly impacts quality of life and functional independence.

Collaborative care between emergency medicine and ophthalmology, with clear communication regarding urgency and initial management, optimizes patient outcomes. Emergency physicians should establish protocols for immediate ophthalmologic consultation and understand which conditions require emergent versus urgent evaluation. Continued education on non-traumatic ocular emergencies and maintenance of clinical competency in basic ophthalmic examination techniques remain essential components of emergency medicine practice.

DECLARATIONS

Funding: None

Author Contributions: B.M. and İ.E.Y. designed the review and performed the data collection and literature search.

Competing Interests: The authors declare no conflict of interest.
Consent Statement: None Applicable
Artificial Intelligence: Artificial intelligence is utilized in this paper.

REFERENCES

1. WWalland MJ. Acute angle closure glaucoma? Clin Exp Ophthalmol. 2018;46(3):211-212. doi:10.1111/ceo.13189
2. Sng CC, Ang M, Barton K. Uveitis and glaucoma: new insights in the pathogenesis and treatment. Prog Brain Res. 2015;221:243-269. doi:10.1016/bs.pbr.2015.06.008
3. Brusini P, Salvetat ML, Zeppieri M. How to measure intraocular pressure: an updated review of various tonometers. J Clin Med. 2021;10(17):3860. doi:10.3390/jcm10173860
4. Chua PY, Day AC, Lai KL, et al. The incidence of acute angle closure in Scotland: a prospective surveillance study. Br J Ophthalmol. 2018;102(4):539-543. doi:10.1136/bjophthalmol-2017-310725
5. Wagner IV, Stewart MW, Dorairaj SK. Updates on the diagnosis and management of glaucoma. Mayo Clin Proc Innov Qual Outcomes. 2022;6(6):618-635. doi:10.1016/j.mayocpiqo.2022.09.007
6. Lowe RF. Acute angle-closure glaucoma: the second eye: an analysis of 200 cases. Br J Ophthalmol. 1962;46(11):641-650. doi:10.1136/bjo.46.11.641
7. Jabs DA, Nussenblatt RB, Rosenbaum JT; Standardization of Uveitis Nomenclature (SUN) Working Group. Standardization of uveitis nomenclature for reporting clinical data: results of the First International Workshop. Am J Ophthalmol. 2005;140(3):509-516. doi:10.1016/j.ajo.2005.03.057
8. Werkl P, Rademacher J, Pleyer U. HLA-B27-positive anterior uveitis: clinical aspects, diagnostics, interdisciplinary management, and treatment. Ophthalmologie. 2024;121(1):12-22. doi:10.1007/s00347-023-01960-z
9. Çakar Özdal P. Behçet's uveitis: current diagnostic and therapeutic approach. Turk J Ophthalmol. 2020;50(3):169-182. doi:10.4274/tjo.galenos.2019.60308
10. Rosenbaum JT, Asquith M. The microbiome and HLA-B27-associated acute anterior uveitis. Nat Rev Rheumatol. 2018;14(12):704-713. doi:10.1038/s41584-018-0097-2
11. Cugati S, Varma DD, Chen CS, et al. Treatment options for central retinal artery occlusion. Curr Treat Options Neurol. 2013;15(1):63-77. doi:10.1007/s11940-012-0202-9
12. Hayreh SS, Zimmerman MB. Central retinal artery occlusion: visual outcome. Am J Ophthalmol. 2005;140(3):376-391. doi:10.1016/j.ajo.2005.03.038
13. Hayreh SS. Central retinal artery occlusion. Indian J Ophthalmol. 2018;66(12):1684-1694. doi:10.4103/ijo.IJO_1446_18
14. Schrag M, Youn T, Schindler J, Kirshner H, Greer D. Intravenous fibrinolytic therapy in central retinal artery occlusion: a patient-level meta-analysis. JAMA Neurol. 2015;72(10):1148-1154. doi:10.1001/jamaneurol.2015.1578
15. Park SJ, Choi NK, Yang BR, et al. Risk and risk periods for stroke and acute myocardial infarction in patients with central retinal artery occlusion. Ophthalmology. 2015;122(11):2336-2343.e2. doi:10.1016/j.ophtha.2015.07.018
16. Celebi ARC. Hyperbaric oxygen therapy for central retinal artery occlusion: patient selection and perspectives. Clin Ophthalmol. 2021;15:3443-3457. doi:10.2147/OPTH.S224192
17. Bhagat N, Goldberg MF, Gascon P, et al. Central retinal vein occlusion: review of management. Eur J Ophthalmol. 1999;9(3):165-180. doi:10.1177/112067219900900304
18. Hayreh SS, Podhajsky PA, Zimmerman MB. Natural history of visual outcome in central retinal vein occlusion. Ophthalmology. 2011;118(1):119-133.e1-2. doi:10.1016/j.ophtha.2010.04.019
19. Sivak-Callcott JA, O'Day DM, Gass JD, Tsai JC. Evidence-based recommendations for the diagnosis and treatment of neovascular glaucoma. Ophthalmology. 2001;108(10):1767-1776. doi:10.1016/s0161-6420(01)00775-8
20. Martínez-Lapiscina EH, Fraga-Pumar E, Pastor X, et al. Is the incidence of optic neuritis rising? Evidence from an epidemiological study in Barcelona (Spain), 2008-2012. J Neurol. 2014;261(4):759-767. doi:10.1007/s00415-014-7266-2
21. Toosy AT, Mason DF, Miller DH. Optic neuritis. Lancet Neurol. 2014;13(1):83-99. doi:10.1016/S1474-4422(13)70259-X
22. Soelberg K, Nilsson AC, Nielsen C, et al. Autoimmune and immunogenetic profile of patients with optic neuritis in a population-based cohort. Mult Scler Relat Disord. 2018;21:97-102. doi:10.1016/j.msard.2018.03.003
23. de Mello Vitor B, Foureaux EC, Porto FB. Herpes zoster optic neuritis. Int Ophthalmol. 2011;31(3):233-236. doi:10.1007/s10792-011-9443-y
24. Beck RW, Cleary PA, Anderson MM Jr, et al; Optic Neuritis Study Group. A randomized, controlled trial of corticosteroids in the treatment of acute optic neuritis. N Engl J Med. 1992;326(9):581-588. doi:10.1056/NEJM199202273260901
25. Beck RW, Cleary PA, Trobe JD, et al; Optic Neuritis Study Group. The effect of corticosteroids for acute optic neuritis on the subsequent development of multiple sclerosis. N Engl J Med. 1993;329(24):1764-1769. doi:10.1056/NEJM199312093292404
26. Shen T, You Y, Arunachalam S, et al. Differing structural and functional patterns of optic nerve damage in multiple sclerosis and neuromyelitis optica spectrum disorder. Ophthalmology. 2019;126(3):445-453. doi:10.1016/j.ophtha.2018.06.022
27. Mitry D, Charteris DG, Yorston D, et al. The epidemiology and socioeconomic associations of retinal detachment in Scotland: a two-year prospective population-based study. Invest Ophthalmol Vis Sci. 2010;51(10):4963-4968. doi:10.1167/iovs.10-540012
28. Kwok JM, Yu CW, Christakis PG. Retinal detachment. CMAJ. 2020;192(12):E312. doi:10.1503/cmaj.19133734
29. Mitry D, Charteris DG, Fleck BW, et al. The epidemiology of rheg5matogenous retinal detachment: geographical variation and clinical associations. Br J Ophthalmol. 2010;94(6):678-684. doi:10.1136/bjo.2009.157727
30. Gupta OP, Benson WE. The risk of fellow eyes in patients with rhegmatogenous retinal detachment. Curr Opin Ophthalmol. 2005;16(3):175-178. doi:10.1097/01.icu.0000162377.55415.f3
31. Hassan TS, Sarrafizadeh R, Ruby AJ, et al. The effect of duration of macular detachment on results after the scleral buckle repair of primary, macula-off retinal detachments. Ophthalmology. 2002;109(1):146-152. doi:10.1016/s0161-6420(01)00886-7
32. Stapleton F, Keay L, Edwards K, et al. The incidence of contact lens-related microbial keratitis in Australia. Ophthalmology. 2008;115(10):1655-1662. doi:10.1016/j.ophtha.2008.04.002
33. Tuft S, Somerville TF, Li JO, et al. Bacterial keratitis: identifying the areas of clinical uncertainty. Prog Retin Eye Res. 2022;89:101031. doi:10.1016/j.preteyeres.2021.101031
34. Maycock NJ, Jayaswal R. Update on Acanthamoeba keratitis: diagnosis, treatment, and outcomes. Cornea. 2016;35(5):713-720. doi:10.1097/ICO.0000000000000804
35. Sridhar MS, Sharma S. Microsporidial keratoconjunctivitis in a HIV-seronegative patient treated with debridement and oral itraconazole. Am J Ophthalmol. 2003;136(4):745-746. doi:10.1016/s0002-9394(03)00391-x
36. Wilhelmus KR. Antiviral treatment and other therapeutic interventions for herpes simplex virus epithelial keratitis. Cochrane Database Syst Rev. 2015;1(1):CD002898. doi:10.1002/14651858.CD002898.pub5
37. Omari AA, Mian SI. Adenoviral keratitis: a review of the epidemiology, pathophysiology, clinical features, diagnosis, and management. Curr Opin Ophthalmol. 2018;29(4):365-372. doi:10.1097/ICU.0000000000000485
38. Asbell PA, Sanfilippo CM, Pillar CM, et al. Antibiotic resistance among ocular pathogens in the United States: five-year results from the Antibiotic Resistance Monitoring in Ocular Microorganisms (ARMOR) Surveillance Study. JAMA Ophthalmol. 2015;133(12):1445-1454. doi:10.1001/jamaophthalmol.2015.3888
39. Durand ML. Endophthalmitis. Clin Microbiol Infect. 2013;19(3):227-234. doi:10.1111/1469-0691.12118
40. Gentile RC, Shukla S, Shah M, et al. Microbiological spectrum and antibiotic sensitivity in endophthalmitis: a 25-year review. Ophthalmology. 2014;121(8):1634-1642. doi:10.1016/j.ophtha.2014.02.001
41. Pershing S, Lum F, Hsu S, et al. Endophthalmitis after cataract surgery in the United States: a report from the Intelligent Research in Sight Registry, 2013-2017. Ophthalmology. 2020;127(2):151-158. doi:10.1016/j.ophtha.2019.08.026
42. Endophthalmitis Vitrectomy Study Group. Results of the Endophthalmitis Vitrectomy Study: a randomized trial of immediate vitrectomy and of intravenous antibiotics for the treatment of postoperative bacterial endophthalmitis. Arch Ophthalmol. 1995;113(12):1479-1496. doi:10.1001/archopht.1995.01100120009001
43. Durand ML. Bacterial and fungal endophthalmitis. Clin Microbiol Rev. 2017;30(3):597-613. doi:10.1128/CMR.00113-16
44. Kawasaki A. Physiology, assessment, and disorders of the pupil. Curr Opin Ophthalmol. 1999;10(6):394-400. doi:10.1097/00055735-199912000-00005