CLINICAL IMAGE

PERSISTENT RED EYE IN A PATIENT WITH IGA NEPHROPATHY

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A 37-year-old man reported to the eye clinic with irritation and pain on eye movement in his left eye for 15 days. He had associated symptoms of foreign body sensation, grittiness, itching and mild headaches. The patient reported no specific events or activities associated with the onset of his ocular symptoms, and neither fever nor recent illness were noted. Medical history included hypertriglyceridemia, obstructive sleep apnea, obesity, hyperglycemia, hypothyroidism, hypertension and gout. He also had Berger's disease—immunoglobin A nephropathy (IgA nephropathy)—resulting in stage 4 chronic kidney disease for which he was on dialysis and awaiting a kidney transplant. Five days earlier, he was seen by his optometrist who prescribed topical prednisolone acetate 1% every hour while awake for his left eye. During his current visit, he reported minimal improvement with the topical steroid therapy.

His visual acuities at this visit were 20/20 in each eye and his intraocular pressures were 21 mm Hg OD and 22 mm Hg OS. Pupils were equal, round, reactive, with no afferent pupillary defect found. Motility was normal with no restrictions. There was no proptosis, and his eyelids were normal. Anterior segment examination, with a slit lamp, of the left eye revealed 2+ temporal bulbar conjunctival injection, trace conjunctival chemosis and no staining of the cornea or conjunctiva with fluorescein dye. His left eye had no cells or flare in the anterior chamber, and his right eye was unremarkable. The posterior segment of both eyes was healthy.

FIGURE 1:

Primary gaze anterior segment photograph of the left eye showing injection of the temporal deep scleral vessels and bulbar conjunctival chemosis.

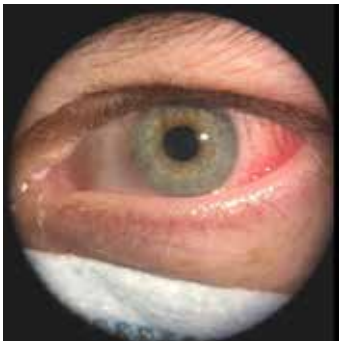

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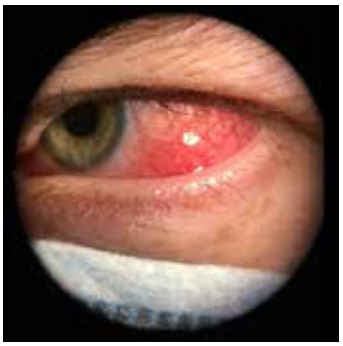
FIGURE 2:

Right gaze anterior segment photograph of the left eye showing injection of the temporal deep scleral vessels and bulbar conjunctival chemosis.



QUESTIONS

1. What is the most likely ocular diagnosis?

a. Foreign body

b. Corneal abrasion

c. Scleritis

d. Orbital cellulitis

2. What is the next best treatment plan for the most likely diagnosis?

a. Start oral nonsteroidal anti-inflammatory drugs (NSAIDs)

b. Start oral steroids

c. Start oral antibiotics

d. Start steroid-sparing immunosuppressive therapy

ANSWERS:

1. What is the most likely ocular diagnosis?

Correct Answer:
C) Scleritis

Scleritis is an ocular inflammatory condition of the sclera, often associated with an underlying systemic etiology. It can result in severe eye pain, pain on eye movement, pain of the areas surrounding the orbit, lacrimation, photophobia and potential vision loss.[1] Common signs include edema, dilation of deep scleral vessels, corneal infiltrates, corneal thinning, stromal keratitis and trabeculitis.[1] Corneal or conjunctival foreign body and corneal abrasion are the two most common forms of ocular trauma.[2]

Typically these result from lack of eye protection during high-risk activities.[2] They can present as blurred vision, redness, tearing, photophobia, extreme discomfort with or without the eyes closed and a feeling of something in the eye. Foreign body and corneal abrasion were ruled out due to the lack of any associated high-risk activity and no corneal or conjunctival staining with fluorescein dye installation. Orbital cellulitis is an infection of the soft tissue surrounding the eye that can cause conjunctival injection and severe ocular pain, especially on eye movement. However, other signs, such as fever, proptosis, restricted ocular movements, increased intraocular pressure and swelling or erythema of the eyelids, are often present as well.[3]

2. What is the next best treatment plan for the most likely diagnosis?

Correct Answer:
B) Start oral steroids

Management of anterior scleritis is largely based on the clinical presentation, severity, associated systemic conditions, and risk factors of treatment. However, the available treatment options can have severe side effects. Although topical administration of corticosteroids has limited success, it can be considered as a first line treatment of mild scleritis to reduce potential risks of systemic medications.[4] Oral NSAIDs are also considered first tier treatment for non-infectious scleritis and can be used if topicals fail.[4] For some patients, additional treatment beyond NSAIDs is needed. Second line treatment of scleritis can include oral corticosteroids and even subconjunctival corticosteroid injections if orals are unsuccessful.[4] When anterior non-infectious scleritis becomes severe, or oral steroid treatment has failed, immunosuppressive agents such as methotrexate may be used.[4] Research has shown biologics to be beneficial in scleritis as a last line treatment when all other treatments have failed. [4]

For this patient, oral NSAIDs would be contraindicated due to the association of increased mortality in patients taking oral NSAIDs while on dialysis.[5] In general, NSAIDs should be avoided in patients with kidney disease.[6] Due to lack of resolution with topical steroids, and the contraindication of NSAIDs, the next treatment option for our patient would be oral corticosteroids. Oral antibiotics would not be indicated since the patient was afebrile and there was no sign of an infectious cause. Due to this patient's mild presentation, steroid-sparing immunosuppressive therapy would not be necessary at this stage.

DISCUSSION

The incidence and prevalence of scleritis is found to be 3.4 and 5.2 per 100,000 person-years respectively.[7] Scleritis can be caused by systemic autoimmune conditions, infection, ocular surgery, trauma, chemical injury, and infiltrating ocular neoplasms.[8] Although this condition may be idiopathic, up to 50% of the time it is associated with an underlying systemic disease.[1,9] Associated systemic diseases can include rheumatoid arthritis, granulomatosis with polyangiitis, polyarteritis nodosa, spondyloarthropathies, IgA nephropathy and sarcoidosis.[1,8,9] Typically the systemic diagnosis is present prior to the onset of the associated ocular findings.[9] In the absence of a known systemic condition, lab work is indicated. However, systemic therapy is often needed whether a systemic association is present or not.[9]

In this case, the associated systemic disease was found to be IgA nephropathy, also known as Berger's disease. It is an accumulation of immunoglobulin A inside the glomeruli of the kidney. Patients can be asymptomatic, present with hematuria or proteinuria, or have reduced kidney function due to inflammation and fibrosis.[10] Our patient was diagnosed with IgA nephropathy after suffering a hypertensive emergency and acute kidney injury 1 year prior. Diagnosis of IgA nephropathy typically includes renal biopsy, which was the case in our patient. The patient then suffered acute renal failure, was put on dialysis 3 times per week and was placed on the kidney transplant list.

In a study of 116 patients with primary glomerular diseases, 6 out of 39 patients with IgA nephropathy presented with episcleritis or deep scleritis.[11] Scleritis was not found in any of the other six types of primary glomerular disease discussed in the study.[11] From this study it was thought that the deposits within the glomeruli and the accompanying scleritis could be the result of the IgA-associated immune complexes.[11]

The patient's medical history also included gout, which is a systemic condition occasionally associated with scleritis.[12] However, the patient was taking a maintenance dose of 200 mg allopurinol daily and was completely asymptomatic of any recent gout attacks. For this reason, gout was clinically ruled out as the underlying cause of the patient's scleritis.

PATIENT OUTCOME

Treatment was coordinated with the patient's primary care physician, and he was started on 60 mg daily of oral prednisone for 14 days. The patient returned to the eye clinic two weeks later with complete resolution of pain and only mild redness. Visual acuities were 20/20 in both eyes and the anterior segment findings were normal with only 1+ injection in the left eye remaining. His intraocular pressures were 19 mm Hg OD and 20 mm Hg OS. The 14-day oral prednisone treatment was completed, tapered to 50 mg daily for one week, and the patient was scheduled for follow up with his primary care physician.

REFERENCES:

1. Okhravi N, Odufuwa B, McCluskey P, Lightman S. Scleritis. *Surv Ophthalmol*. 2005;50(4):351-363. doi:10.1016/j.survophthal.2005.04.001
2. Camodeca AJ, Anderson EP. Corneal Foreign Body. In: *StatPearls*. *StatPearls* Publishing; August 10, 2020.
3. Armstrong PA, Nichol NM. An eye for trouble: Orbital cellulitis. *Emerg Med J*. 2006;23(12):e66. doi:10.1136/emj.2006.041194
4. Lagina A, Ramphul K. Scleritis. In: *StatPearls*. StatPearls Publishing; October 12, 2021. https://www.ncbi.nlm.nih.gov/books/NBK499944/
5. Lai KM, Chen TL, Chang CC, Chen HH, Lee YW. Association between NSAID use and mortality risk in patients with end-stage renal disease: a population-based cohort study. *Clin Epidemiol*. 2019;11:429–441. doi:10.2147/CLEP.S204322
6. Williams AW, Dwyer AC, Eddy AA, *et al*. Critical and honest conversations: the evidence behind the 'Choosing Wisely' campaign recommendations by the American Society of Nephrology. *Clin J Am Soc Nephrol*. 2012;7(10):1664–1672. doi:10.2215/CJN.04970512

7. Honik G, Wong IG, Gritz DC. Incidence and prevalence of episcleritis and scleritis in Northern California. *Cornea*. 2013;32(12):1562–1566. doi:10.1097/ICO.0b013e3182a407c3

8. McCluskey P, Wakefield D. Current concepts in the management of scleritis. *Aust N Z J Ophthalmo*l. 1988;16(3):169–176. doi:10.1111/j.1442-9071.1988.tb01206.x

9. Raiji VR, Palestine AG, Parver DL. Scleritis and systemic disease association in a community-based referral practice. *Am J Ophthalmol*. 2009;148(6):946–950. doi:10.1016/j.ajo.2009.07.021

10. Rodrigues JC, Haas M, Reich HN. IgA nephropathy. *Clin J Am Soc Nephrol*. 2017;12(4):677–686. doi:10.2215/CJN.07420716

11. Nomoto Y, Sakai H, Endoh M, Tomino Y. Scleritis and IgA nephropathy. *Arch Intern Med*. 1980;140(6):783–785. PMID: 7387272

12. Ao J, Goldblatt F, Casson R. Review of the ophthalmic manifestations of gout and uric acid crystal deposition. *Clin Exp Ophthalmol*. 2016;45(1): 73–80. doi:10.1111/ceo.12749