This patient presents with recurrent nodular scleritis initially unresponsive to multiple treatments. Following development of a corneal immune ring (Wessely ring) leads to the correct etiology of herpes simplex virus and successful response to treatment.

I. Case History
   a. 53 year old Native American male
   b. CC: red eye with a dull pain that started few days ago
   c. Ocular history
      a. Nodular scleritis OD, Glaucoma Suspect, CRVO OS
   d. Medical history
      a. Depressive disorder, opioid dependence, anxiety disorder, alcohol abuse, tobacco use, neck and shoulder apathy, lower back pain. Patient denies any history of MI, COPD, DM, HTN, and CVA.
   e. Medications: oral acyclovir 400mg BID, Prednisolone acetate QID OD, nicotine, hydrocodone/acetaminophen, quetiapine fumarate

II. Pertinent findings
   1. Clinical findings
      a. BCVA: 20/20\(^3\) OD, OS
      b. IOP: 21mmHg OD, 20mmHg OS
      c. Anterior segment examination was remarkable in the right eye for a corneal immune ring from 2:30-7 o’clock, mild corneal thinning with small refractile particles at 7 o’clock. Small, stable area of scleral thinning nasally and a small nodule nasally with overlying 2+ injection. Left eye unremarkable.
      d. Posterior segment examination revealed 1+ nuclear sclerosis OD, OS, C/D: 0.55V/H OD, 0.65V/H OS. Posterior pole and periphery were unremarkable OD, OS.

   2. Physical Findings

   3. Laboratory studies
      a. CBC c diff, ANA, ESR MHATP/RPR, ACE, Lysozyme, HLA, RF, quantiferon Gold, ANCA, and albumin were all normal.
      b. Conjunctival culture revealed normal flora.

III. Differential diagnosis
   1. Primary/leading
      a. Recurrent nodular scleritis secondary to herpes simplex virus
   2. Others
      a. Staphylococcal marginal keratitis
      b. Leukemia
      c. Lymphoma

IV. Diagnosis and discussion
   a. Lab work up was ordered to rule out infectious or inflammatory etiologies especially collagen vascular disorders. Lab results were normal.
   b. Albumin and CBC with differential were ordered to rule out leukemia and lymphoma, which were also normal.
   c. Peripheral, scalloped deep stromal infiltrates with a 1.5mm clear zone in between the limbus and cornea in the right eye suggested a corneal immune ring likely caused by HSV.
   d. The patient responded well to the anti-viral therapy resolving both the corneal immune ring and scleritis providing confirmation of the etiology.
   e. Studies showed the Wessely phenomenon was the result of systemically formed antibody diffusing into the cornea containing residual antigens.\(^6,8\)
   f. Another study described the corneal immune ring as the reverse Wessely phenomenon where the antigen or soluble immune complex of antigen diffused into a physiologically antibody-
sensitized cornea, and remained due to a lack of corneal vascularization.\textsuperscript{6,8}
g. Activation of complement by the immune complexes triggered cellular infiltration, resulting in the appearance of immune rings.\textsuperscript{6,8}
h. Both models share common properties including corneal manifestations, where the initial peripheral corneal haze results in a sharply demarcated arc or ring, which then migrate toward the center of the cornea.\textsuperscript{6}
i. The two theories differ in what is considered the predominant infiltrative cell type: neutrophils in the Wessely phenomenon but lymphocytes in the reverse Wessely phenomenon.\textsuperscript{6}

V. Treatment, management
a. The patient was started on oral indomethacin 25mg TID PO for treatment of his recurrent scleritis, since the patient responded well during the first episode.
b. Due to minimal response, the dosage of indomethacin was increased to 50 mg TID. Erythromycin qbedtime OD and tobramycin QID OD were added. The conjunctiva was cultured since the etiology was still not clear.
c. When the conjunctival culture revealed only normal flora, the patient was discontinued off all antibiotics. Prednisolone acetate 1% QID OD was initiated and indomethacin 50mg TID PO was continued, since staphylococcal marginal infiltrates were thought to be a co-morbidity or precipitating event for the scleritis.
d. After being lost to follow up, the patient remained symptomatic, and the clinical picture was more consistent with a corneal immune ring, usually caused by HSV.
e. The patient was administered oral Acyclovir 800mg QID, and prednisolone acetate 1% Q2H OD was continued.
f. Following a significant improvement for both the scleritis and immune ring to the initial dose of Acyclovir, the patient was tapered to a maintenance dose of 400mg BID. Anterior segment photos were taken to help aid in management.
g. Unrecognized HSV infection can cause chronic scleritis. Thus, prolonged administration of acyclovir is required for effective therapy.\textsuperscript{1}

Bibliography, literature review
VI. Conclusion

1. Clinical pearls
   a. Corneal immune rings can masquerade as peripheral staph marginal infiltrates. Thus if the patient is not responding to treatment, consider corneal immune as part of the differential.
   b. Viral causes of scleritis are under diagnosed. Consider viral etiologies when scleritis does not respond to treatment or when the laboratory work-up is negative.⁵,¹

2. Take home points
   a. Effective management and treatment of ocular HSV disease is based on an understanding of the immune pathological mechanisms of corneal inflammatory disease initiated by the virus, the immunological mechanisms involved in recovery by the disease; as well as the host's humoral and cellular immune status during virus latency and during recurrent episodes of infection.⁴