## Migratory orbital inflammation in rheumatoid arthritis

Orbital inflammatory disease is associated with several etiologies.[1] Ocular manifestations of rheumatoid arthritis (RA), such as keratoconjunctivitis sicca, have previously been noted in the literature;[2,3] however, migratory orbital inflammation is rare.

A 60-year-old female with a history of RA was off of therapy for a month while waiting to transition to adalimumab and developed conjunctival injection with foreign-body sensation OS. Symptoms resolved with erythromycin and ketotifen; 2 weeks later, the patient developed periorbital pain, edema, and monocular diplopia OD. Extraocular motility was diminished in all ductions OD, and the patient was noted to be proptotic on examination. Slit-lamp biomicroscopy revealed eyelid edema and chemosis. A short course of low-dose oral steroids was initiated. Computed tomography imaging of the orbits was negative for orbital inflammation and sinusitis. The patient was diagnosed with dacryoadenitis and prescribed an antibiotic. Four days later, the patient reported pain on the right side of her face. Given concern for posterior scleritis, the retina service was consulted.

Posterior segment examination showed inferior paracentral choroidal folds. The retina was noted to be attached on scleral depressed examination. Optical coherence tomography of the macula showed an intact posterior hyaloid membrane with choroidal folds (Fig. 1A). Fluorescein angiography showed temporal capillary leakage. B-scan ultrasound revealed diffuse choroidal thickening. Given the 1-month absence of immunosuppressive therapy, the patient's rheumatologist was contacted for possible immunosuppressive treatment.

One week later, the patient noted that her symptoms were improving OD but had reappeared OS. Anterior segment examination showed bilateral periorbital edema and chemosis (Fig. 2A). Posterior segment examination showed bilateral inferior paracentral choroidal folds and a temporal exudative retinal detachment OD. Given concern for migratory inflammation secondary to RA, the patient was hospitalized.

While admitted to the hospital, the patient's chemosis worsened, and conjunctival prolapse through the eyelid fissure OD was noted. Repeat computed tomography imaging of the orbits showed bilateral proptosis, scleritis, and orbital cellulitis, indicating panophthalmitis (Fig. 3). The patient was started on broad-spectrum intravenous antibiotics and lubricating ophthalmic ointment (1 g ceftriaxone), and 2250 g vancomycin was administered intravenously, followed by 1250 g of vancomycin every 12 hours. Infectious work-up was negative. Blood cultures showed no growth, and serum rapid plasma reagin, HIV, Lyme disease serology, and cytomegalovirus polymerase chain reaction tests were negative. After 3 days, the patient was begun on intravenous methylprednisolone 1 g daily. After 4 days, the patient had notable improvement in periorbital and eyelid edema and chemosis OU (Fig. 2B). She was initiated on rituximab while transitioning to an oral steroid taper.

On outpatient follow-up, external and anterior segment examinations were normal. No choroidal folds were noted

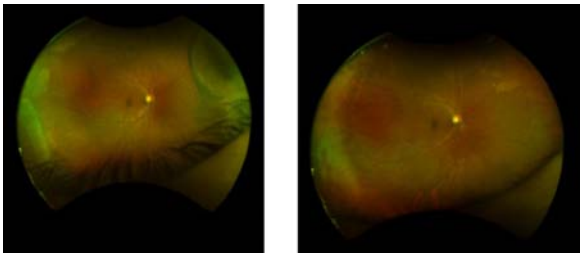

**Fig. 1—Optos (Optos, Dunfermline, UK) fundus photographs: (A) a large superotemporal and nasal exudative retinal detachment along with tortuous retinal vessels of the right eye; (B) the resolved retinal detachment approximately 6 months later.**

**Fig. 2—Chemosis of the left eye: (A) significant chemosis of the left eye causing extrusion of the eyelid speculum; (B) improved chemosis 6 days after initiation of intravenous steroids.**

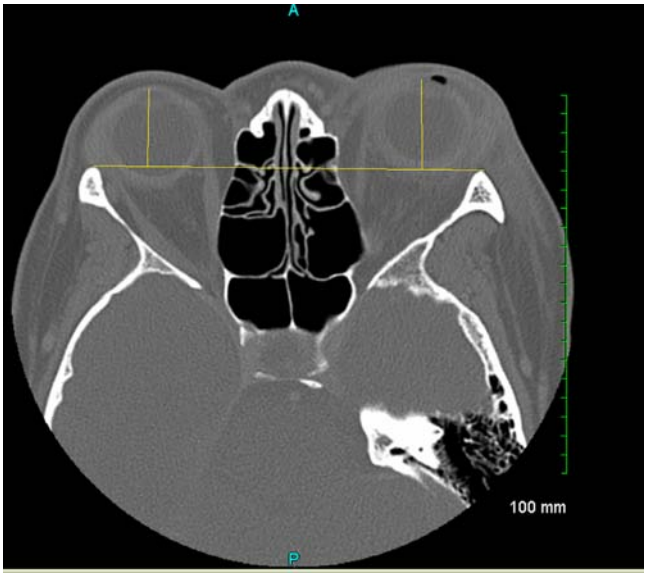


**Fig. 3—Repeat computed tomography imaging of the head showing bilateral proptosis, scleritis, and orbital cellulitis indicating panophthalmitis. There is increased circumferential thickening and enhancement of the scleral surface in the right eye and a fluid collection on the anterior surface of the right globe. Persistent stranding of the retrobulbar fat posterior to the lobe is present. In the left eye, there is proptosis of the left globe, scleral thickening, and a new fluid collection on the anterior surface.**

in either eye, and the temporal exudative detachment had resolved (Fig. 1B). The patient was subsequently placed on adalimumab and monitored for more than 1 year without recurrence.

This patient most likely had migratory orbital inflammation that occurred when she was off of immunosuppressive therapy for RA. Its association with the patient's RA is highly implied given the time of presentation and otherwise negative work-up. Clinical decline did not reverse until she was started on high-dose intravenous steroids. Previous reports of orbital myositis have been linked to psoriatic arthropathy and lupus, and management with oral prednisolone has been shown to be beneficial.[4] This suggests that systemic autoimmune conditions can have ocular manifestations and that treatment of the systemic inflammation also can alleviate ocular symptoms.

Previous case reports support the role of an inflammatory process in migratory inflammation. In one study, patients with orbital myositis of an isolated extraocular muscle and recurrent myositis of the contralateral orbit or eyelid, biopsy revealed perivascular lymphocytic aggregates and histiocytes. Treatment with oral steroids helped alleviate symptoms.[5]

In cases of suspected orbital inflammation, prophylactic antibiotic treatment should be initiated, and infectious etiologies should be ruled out prior to starting steroids because infectious and noninfectious orbital cellulitis can present initially in the same manner.

**Priya Sorab,[*,†] Arjan S. Hura,[*] Alexander Ortiz,[*] Gian Paolo Giuliari[*,‡]**

*University of Cincinnati, Cincinnati, Ohio; †St. John's Episcopal Hospital, Far Rockaway, NY; ‡Cincinnati Eye Institute, Blue Ash, Ohio.

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Correspondence to Gian Paolo Giuliari, MD; gpgiuliari@gmail.com.

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## Footnotes and Disclosure

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# Use of topical allogenic fresh-frozen plasma drops in the treatment of ligneous conjunctivitis

Ligneous conjunctivitis is a rare inflammatory condition characterized by chronic and recurrent deposition of fibrin-rich pseudomembranes on the palpebral conjunctiva.[1] Firm membranes with a characteristic 'woody' induration usually occur bilaterally presenting since childhood.[1,2] The formation of fibrinous pseudomembranes is caused by a mutation in the PLG gene, leading to deficient levels of type 1 plasminogen.[1] Plasminogen is a precursor of plasmin, a molecule involved in the degradation of fibrin.[1] Pseudomembrane formation has been reported after trauma, surgery, contact with ocular foreign bodies, and infection.[1] Lesions may also occur in other mucous membranes.[2]

There is no standardized treatment for ligneous conjunctivitis. Several topical and systemic therapeutic options have been used to prevent pseudomembrane recurrence postoperatively. These include antibiotics, corticosteroids, immunosuppressants, heparin, plasminogen and fresh frozen plasma as well as amniotic membrane transplantation, with varied outcomes.[1,2] They characteristically tend to recur shortly after surgical excision.[1] Pseudomembranes may be debilitating and may result in vision-threatening complications.[2]

A two-year-old female patient presented to our Pediatric Ophthalmology clinic with bilateral pseudomembranes and clear, watery discharge. The patient was otherwise healthy. She was born term and had no complications during pregnancy or delivery. Immunizations were up-to-date. There was no previous ocular history, and family history was not contributory. No history of consanguinity was reported.

On examination, visual acuity was 6/6 bilaterally using Cardiff cards. There was mild left upper lid swelling. Thick pseudomembranes were present along the palpebral conjunctival surfaces bilaterally. The rest of the anterior and posterior exam was unremarkable bilaterally.

Cultures of the palpebral conjunctiva were positive for *Haemophilus influenza*. The patient was treated with topical tobramycin, fluorometholone (FML), and tobramycin/dexamethasone.