Orbital Abscess in an Adult: A Hospitalist's Race to Preserve Visual Acuity

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A healthy 59-year-old man presented to the emergency room after 3 days of left eye discharge, pain, and worsening eyelid closure with swelling. He initially presented to an optometrist who prescribed moxifloxacin eye drops with some improvement in discharge. However, with continued swelling, eyelid closure, and blurry vision, he saw an ophthalmologist who noted elevated pressure of 35 mmHg of the left eye (normal ~12-21 mmHg), and given concern for orbital cellulitis, he was emergently sent to the ER.

On admission to the hospital, he was febrile with a temperature of 101.3F but with otherwise normal vital signs. His physical exam was significant for a swollen left eye with proptosis and mild periorbital tenderness to palpation. Significant conjunctivitis without purulent discharge was present in the left eye. While his right pupil was appropriately reflexive to light, the left eye had an afferent pupillary defect. Extraocular movement in the left eye was also significantly reduced. There was also subjective visual acuity loss in the left eye.

Admission labs were significant for WBC of 21.7 K/uL with 84.1% neutrophilic predominance. He had a normal complete metabolic panel and normal lactic acid level. CT of the head did not show any significant abnormalities, but a CT of the orbit revealed a preseptal and postseptal left orbital cellulitis with suspected left orbital periostitis arising from a left ethmoid sinusitis. There was a 0.6 x 2.0 cm left orbital phlegmon displacing the left medial rectus muscle.

MRI of the left orbit showed a normal globe which was displaced anteriorly by retrobulbar fat inflammation and a 1.0 by 3.0 cm elliptical fluid collection causing lateral displacement of the medial rectus. This confirmed left orbital cellulitis and a left orbital abscess laterally displacing the medial rectus muscle. The optic nerve appeared normal. Ophthalmology recommended emergent orbital decompression with lateral canthotomy/cantholysis given increased intraocular pressure and afferent pupillary defect.

The patient was initially started on parenteral broad spectrum antibiotics with vancomycin and piperacillin-tazobactam, recommended by infectious diseases. Plastic surgery and otolaryngology (ENT) also consulted, and ENT agreed that urgent endoscopic ethmoidectomy for decompression was necessary, followed by lateral canthotomy and cantholysis to be performed by plastic surgery. A left anterior and posterior ethmoidectomy and left maxillary sinus antrostomy was performed. However, the orbital abscess could not be identified during this procedure. This was followed by a successful bedside lateral canthotomy. Attempts to transfer the patient for higher level of care for oculoplastic interventions were not successful due to limited bed availability.

The patient had symptomatic improvement with significantly improved left eye proptosis, swelling and visual acuity. Intraocular pressure improved to 18 mmHg. Daily ophthalmology checks still showed a persistent left afferent pupillary defect. ENT performed endoscopy the following day for further ethmoid decompression and successfully accessed and cleared the previously identified abscess on MRI through the periorbita from the ethmoid labyrinth.

Repeat MRI the next day confirmed notable resolution of the abscess at the medial left orbit with improvement in the displacement of the left medial rectus muscle and left proptosis.

By the time of discharge, left IOP improved to 15 mmHg though the afferent pupillary defect persisted. Extraocular movements were near normal with near normalization of visual acuity to 20/25 with a pocket Snellen chart. Proptosis and conjunctivitis had largely resolved. Due to persistent pupillary defect with prolonged orbital swelling, the patient was started on methylprednisolone 1 mg/kg/day for a total of 72 hours for treatment of presumed compressive optic neuropathy.

The patient did not have immunosuppressing risk factors such as HIV or diabetes. Sepsis resolved with normalization of his white blood cell count, and blood cultures remained negative. His intraoperative cultures yielded multiple organisms including *Pseudomonas aeruginosa, Streptococcus dysgalactiae, Prevotella oralis,* and *Peptostreptococcus* species. Infectious diseases narrowed his antibiotic regimen to a 3-week course of oral amoxicillin/clavulanic acid and ciprofloxacin while ophthalmology recommended a prednisone taper to complete over 12 days for his presumed optic neuropathy. One month after his discharge from the hospital the patient underwent a left lateral canthotomy repair, canthoplasty and skin closure, but it was unclear if his afferent pupillary defect resolved as outpatient ophthalmology records were not accessible.

Discussion

Orbital cellulitis leading to an orbital abscess is an uncommon complication of bacterial sinusitis. Typically, ethmoid and pansinusitis lead to orbital cellulitis. This occurs more often in younger children than adults.¹ Common pathogens are *Staphyloccocus aureus* and streptococci species, while fungi and mycobacteria such as *Mucorales* and *Aspergillus* species can be found in immunocompromised patients, including those with poorly controlled diabetes, susceptible to diabetic keto-acidosis and patients with HIV.^{2,3}

Distinguishing between preseptal cellulitis and orbital cellulitis with complications such as abscess can be clinically challenging. Both types can present with pain, eyelid swelling, and erythema. Both can also present with sepsis, fever and leukocytosis with neutrophilic predominance. Some key signs and symptoms that favor orbital cellulitis include extraocular pain with movement, proptosis, and ophthalmoplegia with diplopia. Findings on exam including sluggish or absent pupillary light reflex or an afferent pupillary defect suggest involvement.⁴

The diagnosis of an orbital abscess is established with radiographic imaging, usually CT scan. The goal of treatment is largely two-fold: to remove and treat the source of infection, and to preserve visual acuity. Surgical drainage is recommended to achieve source control, obtain culture data, and reduce IOP. In many cases, an endoscopic approach by ENT is performed via an anterior ethmoidectomy. Several individual factors however may determine the approach, such as the patient's age as well as feasibility of access under circumstances of inflammation and swelling.⁵ For this patient, ENT's initial anterior and posterior ethmoidectomy was unsuccessful. The second surgical approach from the ethmoid labyrinth through the periorbita allowed successful drainage of the abscess.

Several organisms have been identified in orbital and periorbital abscesses, with *Streptococcus* species most common. *Peptostreptoccccus* was identified in this patient and is present in less than 3% of subgingival flora. This specific strain is rarely reported as the pathogen, with the earliest case reported in 2004.^{5,6} For this patient, we assume that its origin is likely from mild chronic sinusitis, but it is possible an occult odontogenic tract may have contributed to abscess growth. Empiric parenteral antibiotics should cover the most common pathogens, including methicillin-resistant *S. aureus* (MRSA) and in some

cases anaerobic coverage. Ideally antibiotics should be tailored after culture data. However, in the absence of culture data, initial treatment should include broad-spectrum coverage for MRSA, *Streptococcus* species, and gram-negative bacilli. There is no published consensus on the duration of treatment, which ranges from 2-4 weeks depending on severity.⁷

Preservation of the patient's vision is critical and time sensitive. In this patient, compressive optic neuropathy was the likely etiology for visual acuity compromise. Other possible etiologies include optic neuritis as a reaction to the adjacent infection and ischemia from thrombophlebitis or pressure resulting in central retinal artery occlusion.⁴ As mentioned, APD is a likely clinical manifestation of optic nerve injury which should prompt rapid treatment to salvage vision. Lateral canthotomy and cantholysis were performed to temporarily improve the IOP and the compression presumed causing optic neuropathy. Effective decompression of the orbit by abscess drainage and continuation of antibiotics. Despite adequate decompression and improvement of visual acuity, the patient still had APD, which raises questions about need for corticosteroid treatment for optic neuropathy.

Corticosteroids have been reported to have benefit in optic neuropathy including arteritic ischemic optic neuropathy or autoimmune-related optic neuritis. Intravenous corticosteroid therapy with methylprednisolone may potentially provide benefit in compressive optic neuropathy. Corticosteroid use may mask the underlying etiology of the neuropathy and provide early visual improvement due to reduction in swelling and inflammation. In the setting of infection, careful consideration of risks and benefits is needed.^{8,9}

This case demonstrates the importance of a multidisciplinary approach from different subspecialties, which were available at the hospital. Primary providers, in both the outpatient and inpatient settings, need to quickly identify the urgency of diagnosing and treating orbital cellulitis with or without an abscess. Early identification and treatment can lead to a significant decrease in morbidity, specifically blindness. It can be difficult to clinically distinguish between preseptal cellulitis and an orbital process including an abscess, but use of dynamic guidelines with diagnostic imaging and surgical intervention can guide swift and effective care.



Figure A: CT without contrast of the face and orbit, axial view. An oblong soft tissue thickening in the medial left orbit measuring approximately 0.6 cm x 2.0 cm. Left periorbital soft tissue swelling causing lateral displacement of the medial rectus muscle, ultimately causing left orbital proptosis.



Figure B: MR with and without contrast of the orbits, T1-weighted axial, post-contrast. Better visualized 3.0cm by 1.0cm elliptical fluid collection compatible with a left orbital abscess. The medial rectus muscle is displaced laterally as identified by the arrow.



Figure C: MR without contrast of the orbits, T1-weighted thin axial sectioning, post-surgical intervention.

As compared to Figure B, there is significant reduction of the abscess size in the left orbit, with near resolution of proptosis and lateral displacement of the medial rectus muscle. Mild edema noted indicating improving cellulitis.

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