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Imaging of Intracranial and Orbital Complications of Sinusitis and Atypical Sinus Infection: What the Radiologist Needs to Know

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Sinusitis is a common disease. Complications, however, are less common and can be life threatening. Major complications occur from extension of disease into the orbit and intracranial compartment and often require emergent treatment with intravenous (IV) antibiotics or operative intervention. Immunocompromised patients with acute sinusitis are susceptible to atypical infections, such as invasive fungal sinusitis, which is a surgical emergency. Therefore, it is important to accurately and promptly identify potentional complications of acute sinusitis to ensure appropriate treatment and minimize negative outcomes. This article reviews the imaging features of a spectrum of complications associated with acute sinusitis and atypical infections.

Introduction

Rhinosinusitis is defined as inflammation of the paranasal sinus and nasal cavity mucosa. It is a common disease frequently encountered by primary care physicians in both adults and pediatric patients. It affects an estimated 35 million people per year and results in substantial morbidity.[1] The annual direct medical costs of treatment are approximately $5.8 billion.[2] This results in nearly 16 million office visits, more than 500,000 surgical procedures, and is reported to be the fifth leading indication for antibiotic prescriptions by primary care providers.[1-3]

Most sinus infections are viral in origin and in the acute setting, are usually secondary to viral upper respiratory tract infections. Bacterial infection is the cause in only 2%-10% of cases[4]; an even lesser number are fungal in origin. Other etiologies include allergic and nonallergic rhinitis, chemical irritation (such as cigarette smoke), and anatomical variants that predispose to obstruction.[5] Isolated infection of the maxillary sinuses may be odontogenic in origin in up to 20% of cases.[6] Generally, children are more prone to develop sinusitis and suffer complications because of anatomical, immunological, and environmental factors.[7,8]

Most cases resolve with symptomatic treatments without antibiotics, even if bacterial in origin.[9] Viral and bacterial sinusitis often cannot be differentiated based on symptoms, although certain symptoms, such as purulent nasal discharge and pain, suggest a bacterial origin. A consensus statement published in *Otolaryngology—Head and Neck Surgery* in 2007 suggests a presumptive diagnosis of bacterial infection if symptoms last for more than 10 days following an upper respiratory infection or worsen within 10 days after initially improving.[10]

Pathophysiology and Anatomy

The paranasal sinuses are normally sterile. Mucosal edema can cause narrowing and obstruction of the sinus ostia (Fig 1), which leads to stasis of secretions and subsequent infection.

The periorbita and orbital septum are major barriers that limit spread of infection into the orbit (Fig 2). The periorbita is the periosteum of the bones that form the orbit. It is contiguous with the periosteum on the inner surface of the skull and dura at the optic foramen, superior orbital fissures, and ethmoid canals. The periorbita is tightly attached to bone at the anterior margin of the orbit and near openings for neurovascular fissures, foramina, and canals.[11] Elsewhere, the periorbita is loosely connected and creates a potential space for subperiosteal collections. Anteriorly, the periorbita extends into the eyelids and forms the orbital septum.[12,13]

The orbital septum is located deep to the orbicularis oculi muscle and attaches to the levator aponeurosis in the upper eyelid and the tarsal plate in the lower eyelid. Infection may breach the orbital septum through perforations for neurovascular structures.

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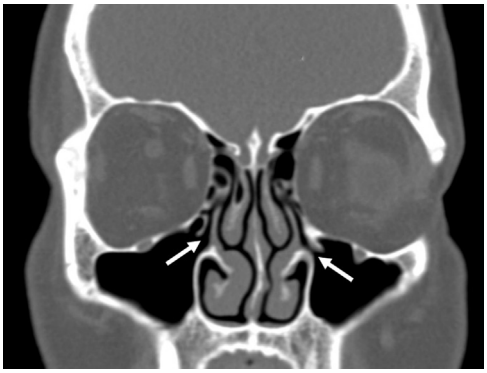

Fig. 1. Maxillary sinus ostia. Coronal CT demonstrates the maxillary sinus ostia (white arrows), the opening for the drainage pathway of each maxillary sinus.

The largest perforation is in the superomedial aspect of the orbital septum for passage of the infratrochlear neurovascular bundle and supraorbital vein to the eyelid.[12,13]

The periorbita appears low in signal on magnetic resonance imaging (MRI) and is difficult to distinguish from the underlying cortical bone.[14] It is also difficult to visualize the orbital septum on imaging. Its location, however, can be approximated as the area just anterior to the orbital fat or near where the extraocular muscles insert on the anterior globe.[15]

The orbit is bounded by frontal, ethmoid, and maxillary sinuses. The parameningeal and periorbital location of the sinuses facilitates development of intracranial and orbital complications of sinusitis. Spread of infection may be either direct or indirect.

Direct extension is often through neurovascular foramina, or congenital and acquired osseous defects (Fig 3), which are commonly seen in the lamina papyracea that forms the medial orbital walls and contains foramina for the anterior and posterior ethmoidal arteries. Occasionally, no osseous defect is found.

Hematogenous spread is a form of indirect spread via a network of valveless veins that drain the soft tissues of the face and orbits (Fig 4). Thrombophlebitis of the veins that drain the face, sinuses, and orbits are thought to be the major route for the spread of infection to adjacent structures.[7] Emissary veins located in the loose connective tissue layer of the scalp communicate with the intracranial compartment. Bidirectional flow of blood in these veins promotes spread of infection anteriorly into the scalp or intracranially. Infection commonly spreads retrograde into the orbits because of thrombophlebitis of ophthalmic veins and smaller venous tributaries that drain into the cavernous sinuses. Infection of specific sinuses demonstrates a predilection for certain complications (Table 1). Orbital complications are most commonly due to ethmoid followed by frontal sinusitis. Intracranial complications typically occur with frontal, ethmoid, and sphenoid sinus disease. Sphenoid sinusitis may result in cavernous sinus thrombosis.

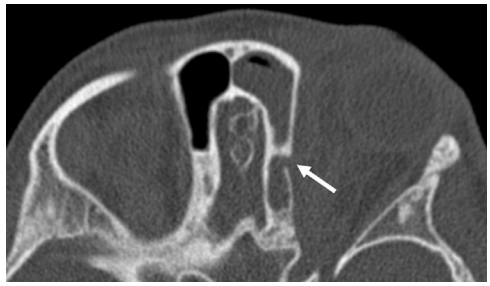

Fig. 3. Direct spread of infection. Axial CT image demonstrates a defect in the left medial orbital wall (lamina papyracea), a common location for dehiscence. Direct spread of infection occurs through neurovascular foramina or via congenital and acquired defects in bone.

Imaging Protocols

Plain radiography can detect and confirm the clinical diagnosis of sinusitis. However, radiography has shown poor interobserver agreement, a high false-negative rate and has been supplanted by computed tomography (CT).[16] CT is the initial test of choice for evaluating complications of sinusitis as it is widely available and more accurate in depicting sinus pathology, bony detail, and anatomical relationships. For orbital complications of sinusitis, helical CT should be performed ideally with IV contrast in the axial plane with submillimeter collimation to produce isotropic voxels. These data can then be reconstructed into high-quality sagittal and coronal images. Though less sensitive than MRI, contrast-enhanced CT (CECT) can be used to quickly assess for intracranial complications, such as large extra-axial collections, brain lesions, mass effect, and hydrocephalus. The patient may then be triaged appropriately for emergent neurosurgical intervention and for MRI.[7]

MRI is more sensitive in the evaluation of orbital and intracranial complications of sinusitis.[6,17] Orbital imaging should be performed using a field of view that includes the nose and orbital soft tissues anteriorly and extends posteriorly through the sella to include the cavernous sinuses. T1-weighted images (T1WI) in the axial and coronal planes can depict orbital anatomy and the stranding and soft tissue thickening that occur with inflammation and infection. Short-tau inversion recovery (STIR) or fat suppressed T2-weighted images are useful in demonstrating edema and fluid collections. Unless contraindicated, IV contrast should be administered to differentiate abscess from phlegmon, evaluate for venous thrombosis and for intracranial collections and meningitis. Dedicated images of the brain should be obtained with any sinus or orbit MRI to screen for these complications and others, including cerebral infarction. The usage of specific MRI pulse sequences is discussed as they relate to different disease entities later in this article.

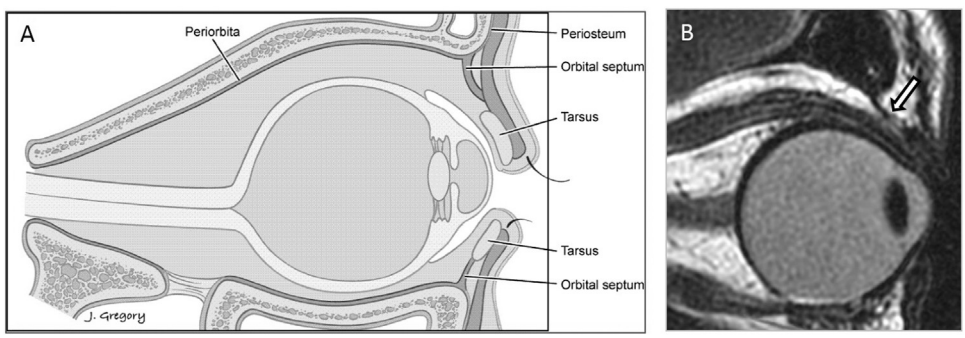

Fig. 2. Periorbita and orbital septum. (A) Sagittal illustration shows the location of the periorbita. (B) The periorbita continues anteriorly as the orbital septum (arrow) in the upper eyelid, as demonstrated on the sagittal T1WI.