



# Nontraumatic emergencies of inflammatory paranasal sinus disease

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**Inflammatory disease of the paranasal sinuses is extremely common and the course is often innocuous. However, when extrasinus spread of disease occurs life-threatening complications can arise. Intraorbital and intracranial involvement can progress rapidly either by hematogenous spread or in the case of fungal sinusitis via angioinvasion. An understanding of anatomy and appropriate imaging protocols is critical when these complications arise. The knowledge of imaging features of the different disease processes and prompt diagnosis is essential to improve patient outcomes and positively impact patient care. Semin Ultrasound CT MRI 40:125-138 © 2018 Elsevier Inc. All rights reserved.**

## Introduction

Imaging of nontraumatic emergencies of inflammatory paranasal sinus disease can be a challenging endeavor. Patients who present emergently without a history of trauma with inflammatory and/or infectious sinonasal disease should be evaluated for spread of infection into adjacent structures in the face and head. These serious and potentially life-threatening complications require prompt diagnosis in order to decrease patient morbidity and mortality. The first step to recognizing these different processes is to understand the anatomy of the paranasal sinuses as well as their common anatomic variants. We will review the appropriate imaging protocols for nontraumatic paranasal sinus disease as it is crucial to accurately detecting nontraumatic emergencies of inflammatory paranasal sinus disease. We will present the pertinent clinical presentation of each pathology and have an in-depth review of the coinciding imaging findings.

## Anatomy

### Paranasal sinuses

The paranasal sinuses are divided into paired frontal, maxillary, ethmoid, and sphenoid sinuses. These sinuses are lined with mucosa attached directly to the bone, hence the name mucoperiosteum.

The paired frontal sinuses develop superiorly to the orbits within the frontal bone and are divided by an intersinus septum. The frontal sinus drains via the frontal recess, located near midline in the floor of the frontal sinuses. Drainage from the frontal sinus then enters the nasal cavity from the anterior portion of the middle meatus.<sup>1</sup> The frontal sinus drainage pathway is commonly impacted by variant ethmoid air cells, termed frontal air cells.<sup>2</sup>

The maxillary sinuses are located inferomedially to the orbits and are the first paranasal sinuses to form. The floor of the maxillary sinus (antrum) is in close proximity to the roots of the molar teeth, which can predispose patients with periodontal disease to concurrent maxillary sinus disease.<sup>3</sup> This relationship with the molar teeth is best appreciated on sagittal and coronal views. The ostium of the maxillary sinus, the infundibulum, is located in the superior medial wall. Due to the cranially oriented drainage pathway, functional cilia are needed to properly clear the maxillary sinus. After ascending along the lateral surface of the uncinate process and exiting the infundibulum, the drainage escapes into the nasal cavity through the hiatus semilunaris which runs underneath the middle turbinate.

The ethmoid sinuses are divided into anterior and posterior groups, with the basal lamellae separating the 2 groups.<sup>4</sup> The number of ethmoid cells in each group varies from 3 to 18. The anterior group of cells is further divided into frontal recess cells which drain into the frontal recess, infundibular cells which drain into the infundibulum and hiatus semilunaris, and finally bullae cells which empty into the superior hiatus. The posterior group of cells is divided into posterior and postrema cells, draining into identically named meatuses

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which funnel into the sphenoidal recess. Of note, ethmoid ostia are the smallest of all paranasal sinuses, predisposing them to obstruction and infection.

Numerous anatomic variants of the ethmoid sinuses have been described, some of which are potentially significant in regard to inflammatory sinonasal disease. Frontal air cells are anterior ethmoid sinuses that are located along the anterior aspect of the frontal recess. The anteriormost frontal recess air cell is known as an agger nasi cell, which is seen in approximately 90 percent of the population and lies anterolateral and inferior to the frontoethmoidal recess.<sup>5</sup> Frontal air cells are described and classified as type I-IV based largely on their relationship to the agger nasi cell.<sup>6</sup> When overly prominent, the frontal air cells can predispose to sinus disease secondary to obstruction of the frontoethmoidal recess. An ethmoid air cell that lies along the inferomedial aspect of the orbit is known as a Haller cell. When large, this can narrow the ostiomeatal complex and lead to maxillary sinusitis.<sup>7</sup> Finally, Onodi cells are posterior ethmoid cells that lie superolateral to the sphenoid sinuses in close proximity to the optic nerve and internal carotid artery.<sup>8</sup> While normally asymptomatic, Onodi cells affected by sinusitis can cause early optic nerve and/or internal carotid artery involvement.

The sphenoid sinuses are located posteriorly to the ethmoid sinuses and anteriorly to the sella turcica. The roof of the sphenoid sinuses, the planum sphenoidale, is usually thin and makes up the floor of the anterior cranial fossa.<sup>9</sup> There is wide variation in the amount of pneumatization of the sphenoid sinus, with approximately 50% of the population developing lateral pneumatized recesses from the main sphenoid sinuses, involving the sphenoid wings and pterygoid process.<sup>10</sup> Pneumatization can extend into the anterior clinoid, surrounding the optic canal and predisposing the optic nerve to involvement in the setting of acute inflammatory sinusitis (Fig. 1).<sup>11</sup>

## Indications and protocol

Sinonasal inflammatory disease is extremely common, and most cases are resolved with over-the-counter treatments and supportive measures. However, certain patient presentations require further workup with imaging to evaluate for possible secondary complications. When chronic disease is suspected or there is concern for spread of disease outside the sinuses into adjacent structures, computed tomography (CT) or magnetic resonance (MR) of the sinuses are vital tools. Symptoms of extrasinus disease can be vague and include headache, facial swelling, orbital and retro-orbital pain, and proptosis. Extension of infection or inflammation from the paranasal sinuses into the surrounding structures is an emergent finding requiring rapid medical and surgical treatment.

CT is the imaging modality of choice to investigate the bone and mucosa of the paranasal sinuses. This is best accomplished with wide window settings of around 4000 Hounsfield Units (HU).<sup>12</sup> Osseous findings of sinusitis on

CT include sinus wall thickening and sclerosis. In aggressive infections, frank erosion and dehiscence may be observed in the setting of osteomyelitis. Occasionally expansion of the pterygopalatine fossa can be seen from mass effect and remodeling secondary to fungal sinus disease (Fig. 2).<sup>13</sup> The soft tissues of the face and orbits should also be evaluated at a narrow window setting of 150-400 HU.<sup>12</sup> CT is also an invaluable tool for preoperative planning in patients with recurrent or chronic rhinosinusitis because it avoids the issue of overlapping of soft tissues and bone structures seen with conventional sinus radiographs.

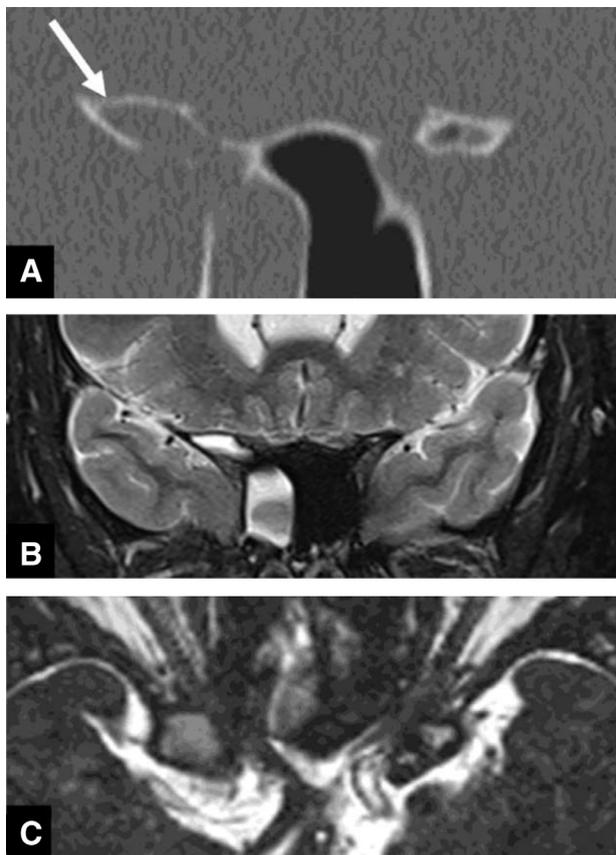
MR of the maxillofacial region with and without contrast is recommended in addition to CT when there is suspected orbital or intracranial complications secondary to inflammatory paranasal sinus disease. MR is superior to CT in the ability to delineate soft tissue material within the sinuses separate from fluid or mucous (Fig. 3).<sup>14</sup> Small extra-axial collections which can represent subdural abscess are relatively apparent on MR, whereas these are often occult on the corresponding CT images. Diffusion-weighted MR sequences can aid in detection of abscess, as these will demonstrate hyperintense signal. However, inspissated secretions can also diffusion restrict depending on the concentration of proteinaceous mucous (Fig. 4).<sup>15</sup> Therefore, additional imaging features like adjacent bone marrow enhancement and soft tissue edema should be seen to distinguish abscess from inspissated secretions.

## Inflammatory and infectious emergencies of the paranasal sinuses

### Bacterial sinusitis

The timing of inflammatory paranasal sinus disease varies from acute, subacute, or chronic. If 4 or more episodes of acute bacterial sinusitis occur within 1 year, the process is labeled recurrent acute sinonasal disease. The distribution of disease may offer a clue as to etiology, since allergic sinusitis tends to be more diffuse while bacterial sinusitis is usually localized and asymmetric.<sup>16</sup> Changes in the nasal discharge also provide information, with clear secretions implying allergic causes whereas transition to thickened mucopurulent secretions would suggest superimposed bacterial infection. The most common causative agents in acute bacterial sinusitis in the immunocompetent patient are *Hemophilus influenzae* and *Streptococcus pneumoniae*, while anaerobic bacteria such as *Peptostreptococcus*, *Propionibacterium*, and *Fusobacterium* species are associated with chronic sinusitis.<sup>17</sup> Additionally, periodontal disease and dental procedures are an important cause of inflammatory sinus disease affecting the maxillary sinuses (Fig. 5).<sup>18</sup> Immunocompromised patients with acute sinusitis may yield *Staphylococcus aureus* and *Pseudomonas aeruginosa* cultures.

Imaging findings of sinusitis vary according to timing. Acute inflammatory sinusitis will show mucoperiosteal

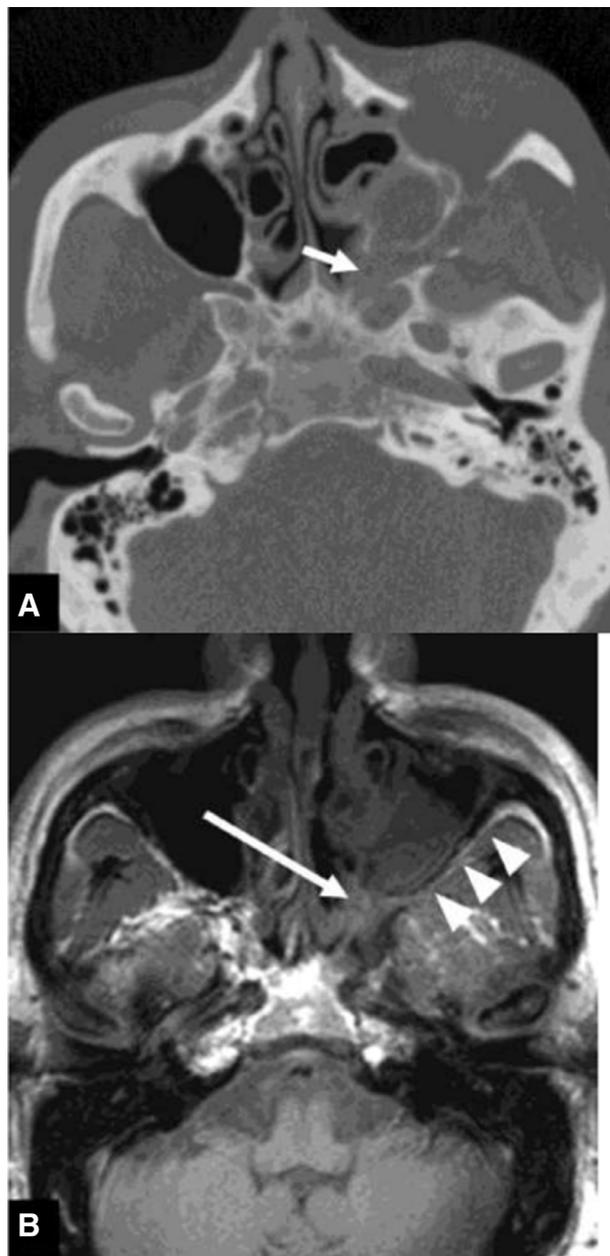


**Figure 1** Mucocoele within a pneumatized anterior clinoid in a patient presenting with vision loss.

Coronal Non-enhanced CT (NECT) (A) demonstrates an expanded and opacified right anterior clinoid (arrow). Note the normal appearing left anterior clinoid. Coronal T2WI (B) shows hyperintense signal within the right anterior clinoid. Axial T2-weighted CISS (C) shows mildly hyperintense T2 signal within the expanded right anterior clinoid with mass effect on the optic canal.

thickening, sinus fluid levels, and frothy retained secretions on CT (Fig. 6).<sup>19</sup> On MR, the mucosa will show isointense signal on T1 and hyperintense signal on T2, signifying edema and inflammation.<sup>20</sup> The fluid within the sinuses will show expected T1 hypointense T2 hyperintense signal characteristics. Postcontrast-enhanced images will demonstrate mucoperiosteal enhancement with nonenhancement of the central fluid.

Long standing or repetitive sinusitis can lead to chronic changes in the paranasal sinuses. CT findings include bony thickening and sclerosis of the sinus walls, mucosal thickening, and inspissated secretions of the affected sinuses.<sup>21</sup> Retention cysts and mucocoeles may develop within the inflamed sinuses, which can further narrow or obstruct sinus drainage pathways.<sup>22</sup> Retention cysts will appear as well circumscribed, low density intrasinus lesions on CT and demonstrate hypointense T1 and hyperintense T2 signal on MR.<sup>23</sup> The intrinsic signal within mucocoeles is variable depending on the water and/or protein ratio, with water-rich mucocoeles having similar signal characteristics as the



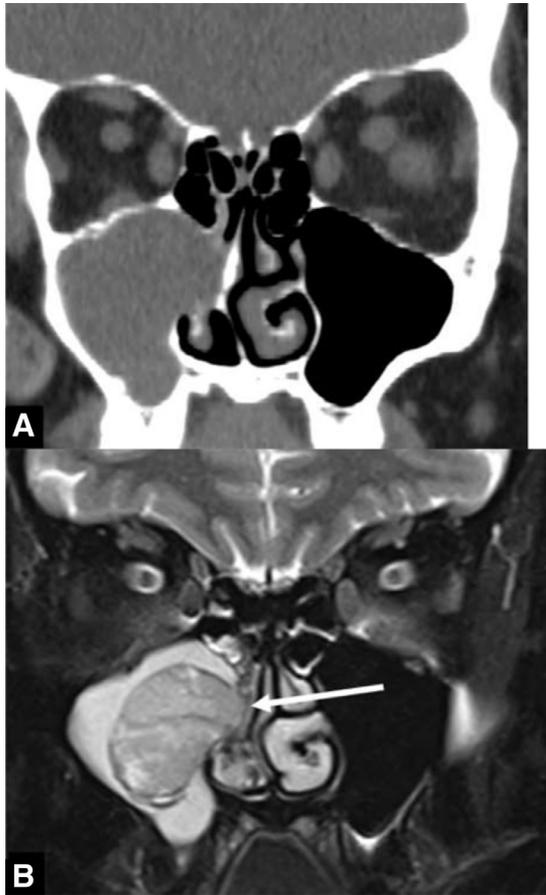
**Figure 2** Invasive fungal sinusitis causing widening of the pterygopalatine fossa.

Axial CT bone window (A) shows opacification of the left posterior ethmoid sinuses with widening of the left pterygopalatine fossa (short arrow). Axial T1WI (B) demonstrates loss of normal fat within the left pterygopalatine fossa (long arrow) and retroantral region (arrowheads). The patient was found to have mucor sinusitis.

retention cyst while protein-rich mucocoeles will be T1 hyperintense and T2 hypointense.<sup>24</sup> The distinguishing imaging characteristic of the mucocoele is bony remodeling and expansion of the affected sinus (Fig. 7).<sup>25</sup>

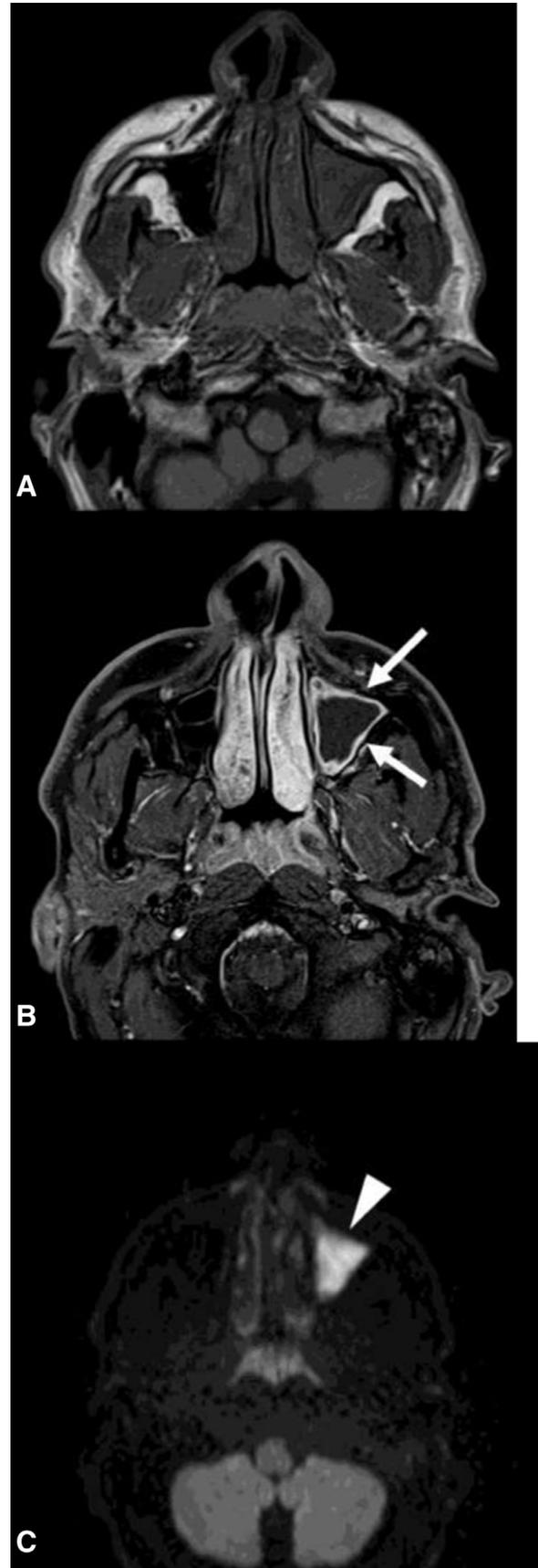
### Intracranial spread of bacterial sinusitis

Sinus pain can often be elicited upon physical exam in patients with acute inflammatory sinusitis, especially with



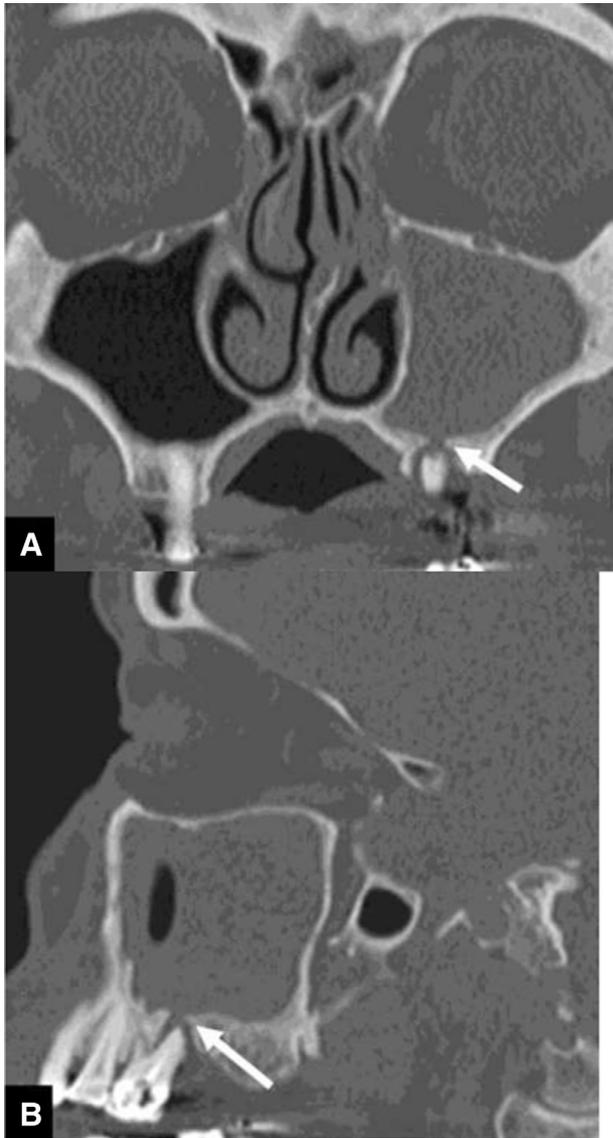
**Figure 3** MRI versus CT: Coronal NECT (A) shows complete opacification of the right maxillary sinus. Coronal T2WI (B) of the same patient demonstrates the superior delineation of fluid and soft tissue relative to CT, with a large soft tissue mass protruding from the medial wall of the right maxillary sinus (arrow) with surrounding fluid and mucous. The mass was resected and pathology showed malignant melanoma.

conversion to bacterial sinusitis. A more concerning presentation is when the focal pain becomes a generalized headache, which can indicate intracranial spread of sinonasal infection. Frontal sinusitis is the most common site for intracranial spread because of a rich venous plexus (Behcet plexus) between the frontal sinus mucosa and the meninges.<sup>26</sup> The venous plexus allows the spread of infection without bone destruction, which can make intracranial manifestations subtler. This complication of sinusitis can manifest in the form of meningitis and cerebritis. CT findings of meningitis can be understated unless contrast is administered to show leptomeningeal enhancement. On MR, prolongation of T2/FLAIR signal results in hyperintense CSF spaces, representing the increased concentration of protein and cellular materials within the CSF.<sup>27</sup> Postcontrast T1 images will demonstrate leptomeningeal enhancement, often the most striking features of meningitis (Fig. 8).<sup>28</sup> The inflammatory nature of phlegmon and pus within the subarachnoid spaces can result in vasospasm, causing arterial narrowing or occlusion which may lead to infarct.<sup>29</sup> These findings are best characterized with angiographic studies.



**Figure 4** Inspissated secretions with diffusion restriction.

Axial T1WI (A) shows hypointense signal throughout the left maxillary sinus. Axial post contrast fat sat T1WI (B) demonstrates



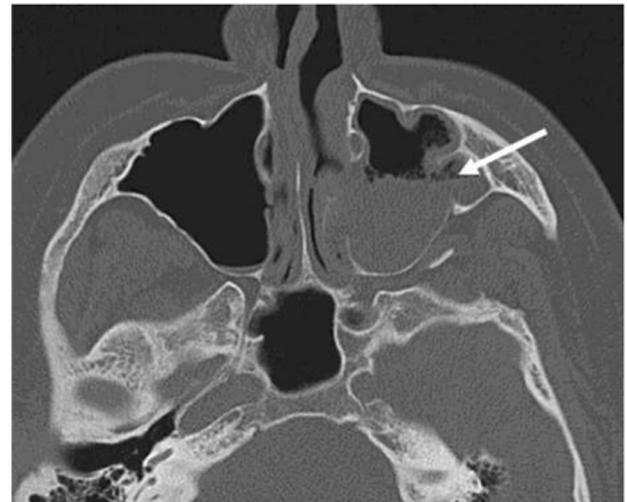
**Figure 5** Periodontal disease causing sinusitis. Coronal (A) and sagittal (B) NECT bone window show opacification of the left maxillary sinus consistent with acute sinusitis. Periapical lucency with a tract extending from the left upper first molar into the left maxillary sinus is present (arrows) consistent with periodontal spread of infection.

Although rare, acute rhinosinusitis has been implicated in acute cerebral ischemia in the absence of meningitis, in particular sphenoid sinusitis. It is postulated that the close proximity of the internal carotid arteries to the sphenoid sinuses predisposes to perivascular inflammation and resulting vasospasm.<sup>30</sup>

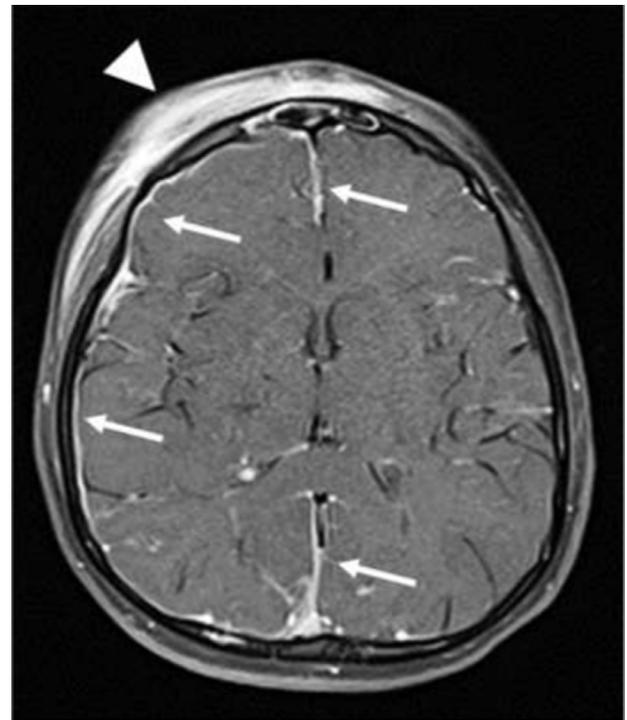
If meningitis is left untreated progression to subdural or epidural abscess or even frank cerebral abscess can occur.

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mucoperiosteal enhancement (arrows) involving the left maxillary sinus with central nonenhancement. Restricted diffusion within the left maxillary sinus (arrowhead) on axial DWI (C) indicates a high protein/low water concentration.

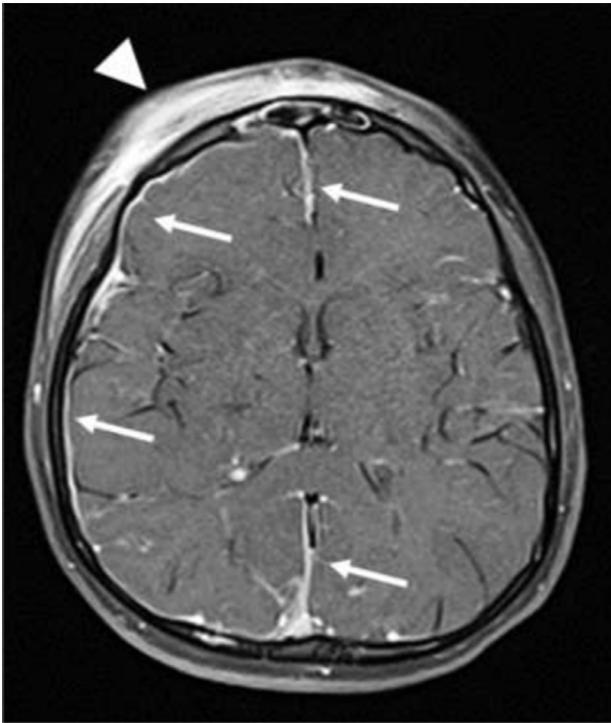


**Figure 6** Acute maxillary sinusitis. Axial NECT shows a fluid level in the left maxillary sinus with frothy air bubbles on the fluid surface (arrow), consistent with acute sinusitis. Mucosal thickening within the left maxillary sinus is present, suggestive of a chronic component.



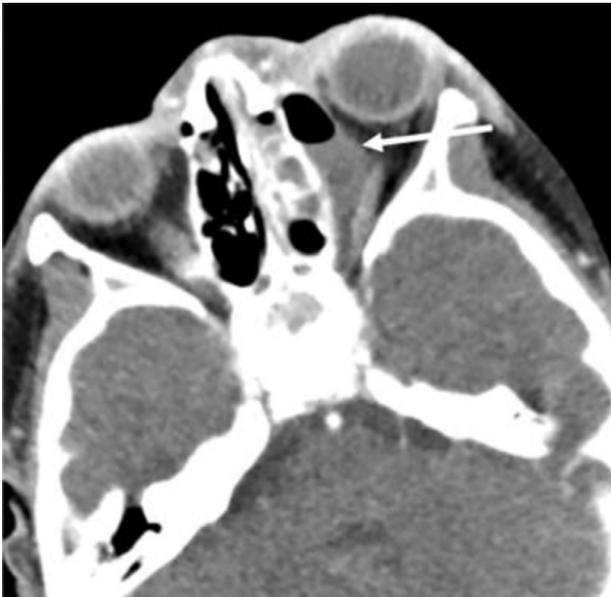
**Figure 7** Sinus mucocoele. Sagittal postcontrast fat-sat T1-weighted image through the level of the sphenoid sinus shows a hyperintense T1 signal mass filling and expanding the sinus cavity (arrow), consistent with mucocoele.

While headache is an incredibly common complaint in the emergency department, it is imperative to obtain a thorough history and when necessary perform imaging of the brain to further evaluate for serious causes of headache such as intracranial abscess. Abscess within the subdural or epidural space will show avid peripheral enhancement on CT and MR with



**Figure 8** Meningitis from sinus disease.

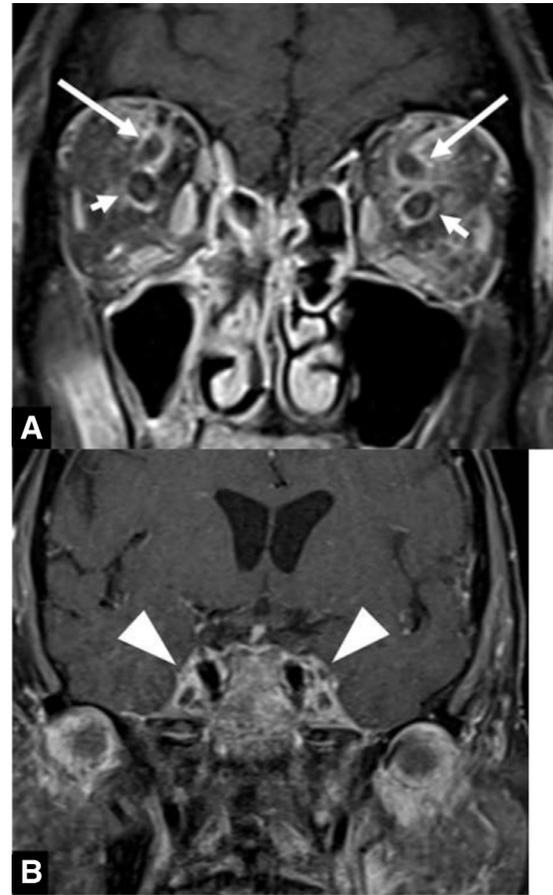
Axial postcontrast T1 of the brain shows asymmetric leptomeningeal enhancement on the right (arrows). Also present is acute sinusitis involving the frontal sinuses with cellulitis involving the forehead (arrowhead).



**Figure 9** Subperiosteal abscess in the left orbit.

Axial contrast-enhanced CT (CECT) of the face demonstrates a fluid and air collection along the medial border of the left orbit (arrow) adjacent to opacified ethmoid sinuses, consistent with subperiosteal abscess.

central T2 hyperintense signal that shows diffusion restriction.<sup>31</sup> If unable to perform MR, contrast CT can be performed to evaluate for cerebral abscess. A ring of enhancing tissue



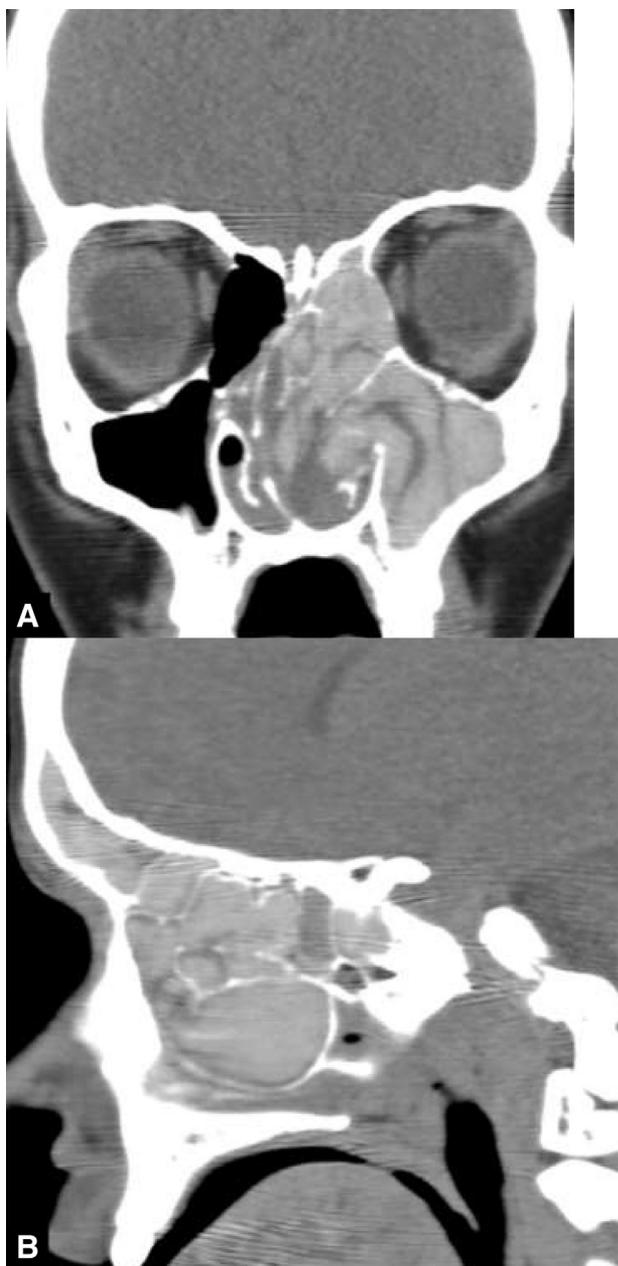
**Figure 10** Acute sinusitis with cavernous sinus thrombosis.

Coronal postcontrast fat-sat T1WI (A and B) demonstrate ethmoid sinusitis resulting in bilateral cavernous sinus and superior ophthalmic vein thrombosis. Enlargement of the bilateral cavernous sinuses with bowing of the outer margins (arrowheads) is characteristic of thrombosis. Engorged and thrombosed bilateral superior ophthalmic veins are present (long arrows) adjacent to the optic nerves (short arrows).

with central fluid attenuation and surrounding vasogenic edema will be seen, as well as possible enhancing ependyma in the form of ventriculitis and obstructing hydrocephalus. MR will show a similar ring enhancing lesion with T1 hypointense and T2 hyperintense central component. Diffusion weighted imaging (DWI) helps to differentiate abscess from most brain tumors, as abscess will demonstrate restricted diffusion centrally.<sup>32</sup> DWI is used to monitor response to treatment, as a lack of diffusion restriction will indicate resolving abscess even in the presence of persistent cavity.<sup>33</sup>

### Orbital spread of bacterial sinusitis

Due to its close proximity to the paranasal sinuses and relatively thin lamina papyracea, the orbits are prone to contiguous spread of infection from acute inflammatory bacterial sinusitis. The anterior and posterior ethmoid sinuses are most commonly implicated in secondary orbital cellulitis or abscess and it is estimated that two-thirds of all orbital infections are the result of sinusitis.<sup>34</sup> Thus, orbital infections are most commonly

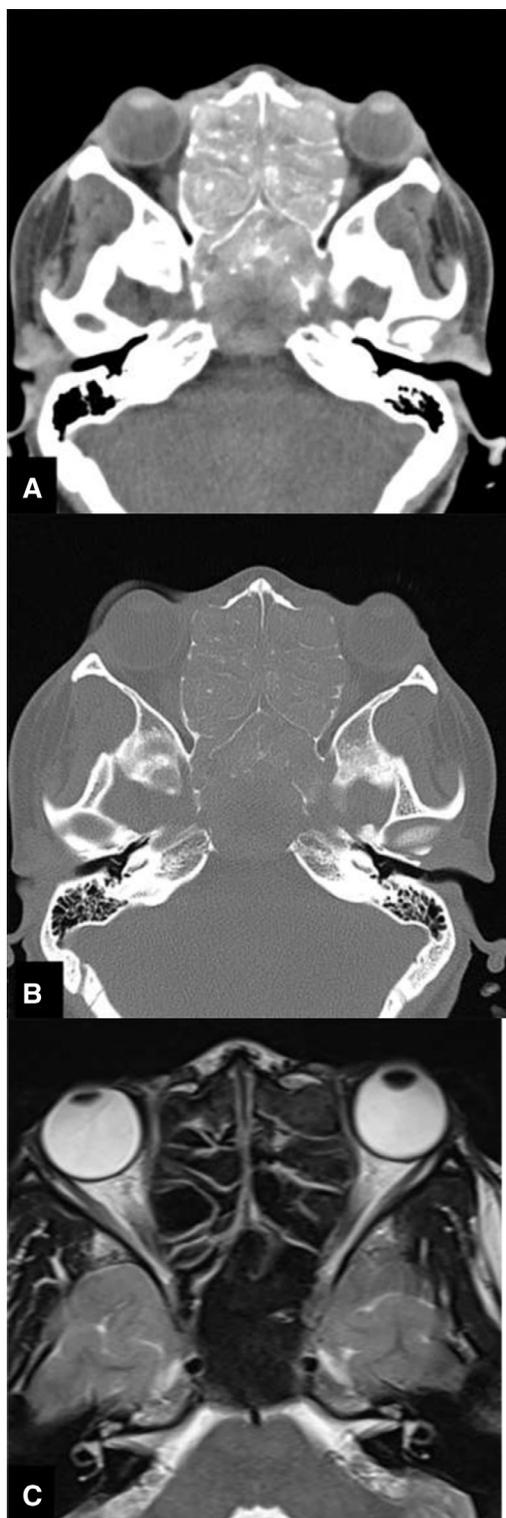


**Figure 11** Noninvasive allergic fungal sinusitis.

Coronal (A) and sagittal (B) NECT of the maxillofacial region shows hyperdense material throughout the left maxillary, ethmoid, frontal, and sphenoid sinuses with bowing of the nasal septum and widening of the left infundibulum.

visualized in the medial aspect of the orbit, directly adjacent to the lamina papyracea. Clinical manifestations vary depending on the severity and location of the infection, ranging from erythema and edema of the eyelids to proptosis, pain, and progressive vision loss secondary to extraocular muscle involvement and inflammation of the optic nerve itself.<sup>35</sup>

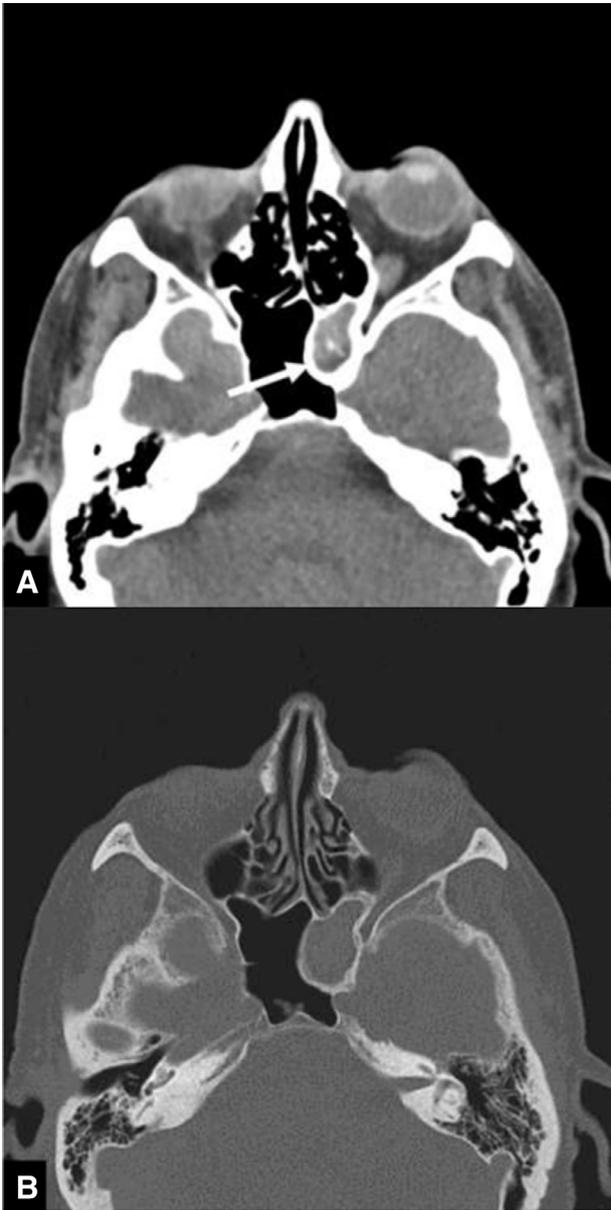
Orbital infections are characterized in regards to the orbital septum as either preseptal or postseptal.<sup>36</sup> Postseptal cellulitis is most commonly caused in the setting of inflammatory paranasal sinus disease.<sup>37</sup> CT and MR will demonstrate paranasal sinus disease of varying degree with concurrent enhancement of the postseptal soft tissues, with the greatest involvement usually



**Figure 12** Noninvasive allergic fungal sinusitis.

Axial soft tissue (A) and bone window (B) NECT shows complete opacification and sinus expansion of the ethmoid sinuses. Axial T2WI (C) demonstrates signal void noted throughout the paranasal sinuses. Note the sinus expansion and mass effect on the orbits.

seen in the extraconal fat adjacent to the affected sinus. Eventually T2 hyperintense phlegmon will replace the extraconal fat and cause displacement of the extraocular muscles.<sup>38</sup>



**Figure 13** Hyperattenuating sinus contents representing fungal ball.

Axial soft tissue (A) and bone (B) window NECT demonstrates high-density concretions within the completely opacified left sphenoid sinus (arrow). The remainder of the paranasal sinuses is clear. The lesion demonstrated stability over 4 years, with reactive hyperostosis of the left sphenoid sinus walls.

Enlargement and enhancement of the rectus muscles may also be present. If left untreated, postseptal cellulitis can progress into subperiosteal abscess. CT will show a focal fluid collection along the lamina papyracea demonstrating rim enhancement and in some cases an air-fluid level (Fig. 9). MR will demonstrate T2 hyperintense signal centrally representing necrosis and pus, with corresponding peripheral enhancement on post-contrast T1 images.<sup>36</sup> Hyperintense signal throughout the abscess on DWI aids in confirming the diagnosis. If medical treatment is delayed or the patient is immunocompromised, intraconal orbital abscess may develop. Infiltration of the extraconal and



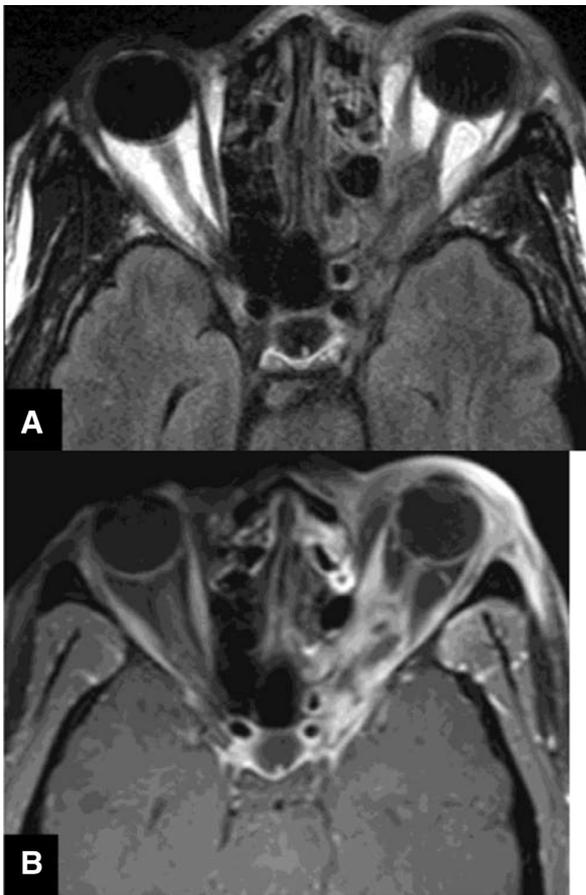
**Figure 14** Acute invasive fungal sinusitis.

Axial NECT (A) and axial T1WI (B) in a patient with rapidly progressive sinusitis. The left maxillary sinus is opacified with loss of the normal retroantral fat plane (arrows), indicative of acute invasive fungal sinusitis.

intraconal fat will be seen throughout the postseptal orbit in addition to the intraconal abscess with central hypoattenuating fluid.<sup>36</sup>

### Cavernous sinus thrombosis

The cavernous sinus is another susceptible area to complications from acute inflammatory sinusitis. Venous drainage from the sinonasal cavities, orbits, and middle-third of the face is via the cavernous sinus. Infections arising from these areas can potentially spread through these drainage pathways.<sup>39</sup> Subsequent septic thrombophlebitis and cavernous sinus thrombosis can result from spread of infection. Clinically, patients will present with headache, fever, meningitis, and often multiple cranial nerve palsies. Contrast-enhanced CT will show nonenhancement and hypoattenuation of the normally enhancing sinus, with unusually clear visualization of the internal carotid artery within the cavernous sinus.<sup>40</sup> Distortion of the cavernous sinus may be present, with lateral bowing into the middle cranial fossa. MR findings of cavernous sinus thrombosis include heterogeneous hyperintense T2 signal and lack of enhancement on postcontrast images.<sup>41</sup> A



**Figure 15** Invasive fungal sinusitis with optic nerve involvement.

Axial T2 FLAIR (A) and postcontrast fat-sat T1WI (B) of the orbits shows an isointense, enhancing lesion filling the left posterior ethmoid sinuses with invasion into the left orbit encasing the intraorbital optic nerve.

potential concurrent finding is superior ophthalmic vein thrombosis, which will manifest as an engorged vein with filling defect on contrast enhancement MR and CT (Fig. 10).

## Fungal sinusitis

Fungal sinusitis has increased in frequency and severity over the last few decades. This is likely due to a combination of increased use of immunosuppressive drugs, overuse of antibiotics, and improved diagnostic accuracy. Fungal sinusitis presentation varies widely, often in correlation with the patient's immune system status. It can range from a chronic indolent allergic infection to fulminant life-threatening angioinvasive disease. Despite the difference in presentation, aggressive surgical and medical therapy is warranted and prompt diagnosis is the key.

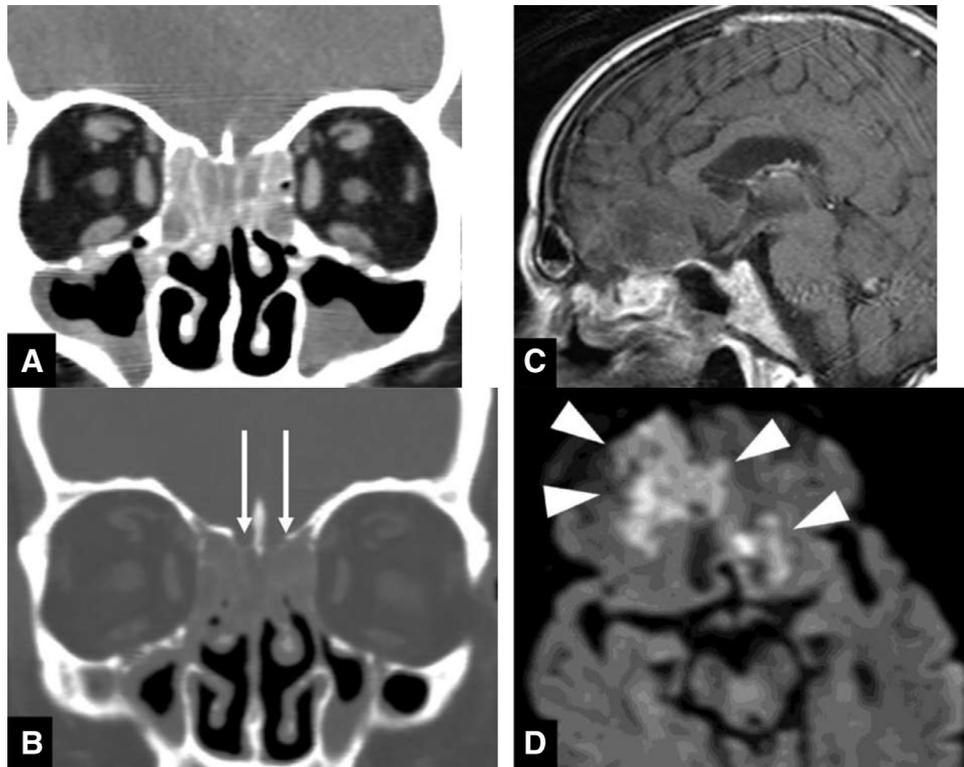
The 2 broad categories of fungal sinusitis are noninvasive and invasive. Allergic fungal rhinosinusitis and paranasal sinus mycetoma are the 2 types of noninvasive fungal sinusitis. Allergic fungal rhinosinusitis primarily affects immunocompetent people with a history of polyposis or recurrent sinusitis. Prior sinus surgery and a history of asthma are well-established risk factors.<sup>42</sup> Patients will present with chronic headaches, nasal congestion, and chronic sinusitis.

Noncontrast CT will show sinuses containing hyperdense material with expansion and thinning of the sinus walls (Fig. 11).<sup>43</sup> The disease process frequently involves multiple sinuses and can be a cause of pansinusitis. MR imaging findings will show edematous and inflamed mucosa demonstrating low-T1 and high-T2 signal. The retained secretions will exhibit variable T1 signal with classic T2 hypointense signal secondary to the high concentration of metals like magnesium and iron within the fungus itself (Fig. 12).<sup>44</sup> If longstanding, allergic fungal sinusitis can cause bony expansion and remodeling of the paranasal sinuses, potentially causing mass effect on the orbits or brain.<sup>45</sup>

Noninvasive fungal sinusitis can also take the form of mycetoma, or fungus ball. The etiology is thought to be due to inadequate mucosal clearance without evidence of fungal invasion of the mucosa.<sup>46</sup> These patients tend to have few, if any symptoms, most commonly chronic sinus pressure.<sup>47</sup> This disease is usually limited to one sinus, the most common being the maxillary sinus. CT findings include a hyperattenuating mass with internal calcification. The affected sinus may demonstrate sclerotic and thickened walls or show a sign of erosion and expansion depending upon where in the sinus the fungus ball is located (Fig. 13). The low signal on T2 sequences represents high metal content of the mycetoma, as well as a lack of free water.<sup>43</sup>

In terms of invasive fungal sinusitis, there are 3 types: acute invasive fungal sinusitis, chronic invasive fungal sinusitis, and chronic granulomatous invasive fungal sinusitis. Of these, acute invasive fungal sinusitis is the most feared, with a mortality rate of approximately 50% in patient without intracranial manifestations and up to 90% in patient with intracranial findings.<sup>48</sup> This is due in part to the angioinvasive characteristics some fungi demonstrate, particularly aspergillus and mucormycosis species.<sup>49</sup> Patients often have underlying comorbidities that contribute to an immunocompromised state, with diabetics being a commonly afflicted patient population. Clinically, patients present with sinusitis, facial pain, fever and painless necrosis of the nasal septum and turbinates.<sup>50</sup> On CT, acute invasive fungal sinusitis appears as hypoattenuating mucosal thickening with a mix of iso- and hyperattenuation of the intraluminal component. When present, osseous destruction can be marked with inflammation and fungal invasion extending into the orbital or cranial compartments. Premaxillary and retroantral fat stranding can help aid in identifying extrasinus spread of disease when osseous findings are subtler (Fig. 14).<sup>51</sup> T2-weighted MR will show opacified sinuses with scattered areas of signal hypointensity representing internal calcifications. Extension into the orbits can encase the optic nerves and cause acute blindness, MR will demonstrate diffuse enhancement surrounding the optic nerve (Fig. 15).<sup>52</sup> Intracranial spread can manifest as meningitis, cerebritis, or frank cerebral abscess. Granulomas may form, which demonstrate T1 and T2 hypointense signal and minimal enhancement.

Angioinvasive fungal sinusitis, as the name implies, can lead to vascular complications such as tissue necrosis, mycotic aneurysm, vasculitis, and infarct. With contrast-enhanced MR, a paradoxical appearance of nonenhancement



**Figure 16** Invasive fungal sinusitis leading to cerebral infarct.

Coronal NECT soft tissue (A) and bone (B) window CT findings demonstrate paranasal sinus disease with complete opacification of the ethmoid sinuses. Marked thinning of the cribriform plate and lateral lamella is present on bone window (long arrows). Sagittal T1WI (C) shows hypointense signal representing edema within the anterior inferior frontal lobes. Axial DWI (D) shows restricted diffusion in the frontal lobes (arrowheads), confirming cerebral infarct.

of mucosa and nasal turbinates has been observed, termed the black turbinate sign, indicating necrosis of these tissues secondary to angioinvasive fungi.<sup>53</sup> Angioinvasive fungi in the sinuses can directly invade into vascular structures, as opposed to hematogenous spread of fungal infection arising from the lungs.<sup>54</sup> Initially this will cause arteritis, with wall thickening and enhancement leading to luminal narrowing.<sup>55</sup> Breakdown of the vessel wall secondary to abundant elastase production of certain fungal strains can lead to mycotic aneurysm formation.<sup>54</sup> This will manifest on CT angiography or MR angiography as a saccular outpouching, often with irregular contours.<sup>56</sup> The friable aneurysm walls are prone to rupture, with spontaneous hemorrhage a serious concern. Sequelae of angioinvasive fungal sinusitis may also include cerebral infarct, whether from direct vessel wall injury or seeding secondary to septic emboli (Fig. 16). The progression of the intracranial extension is rapid, prompt diagnosis and aggressive treatment is necessary. MR imaging demonstrates increase areas of edema on FLAIR and restricted diffusion due cytotoxic edema related to the angioinvasive pattern (Fig. 17).<sup>57</sup>

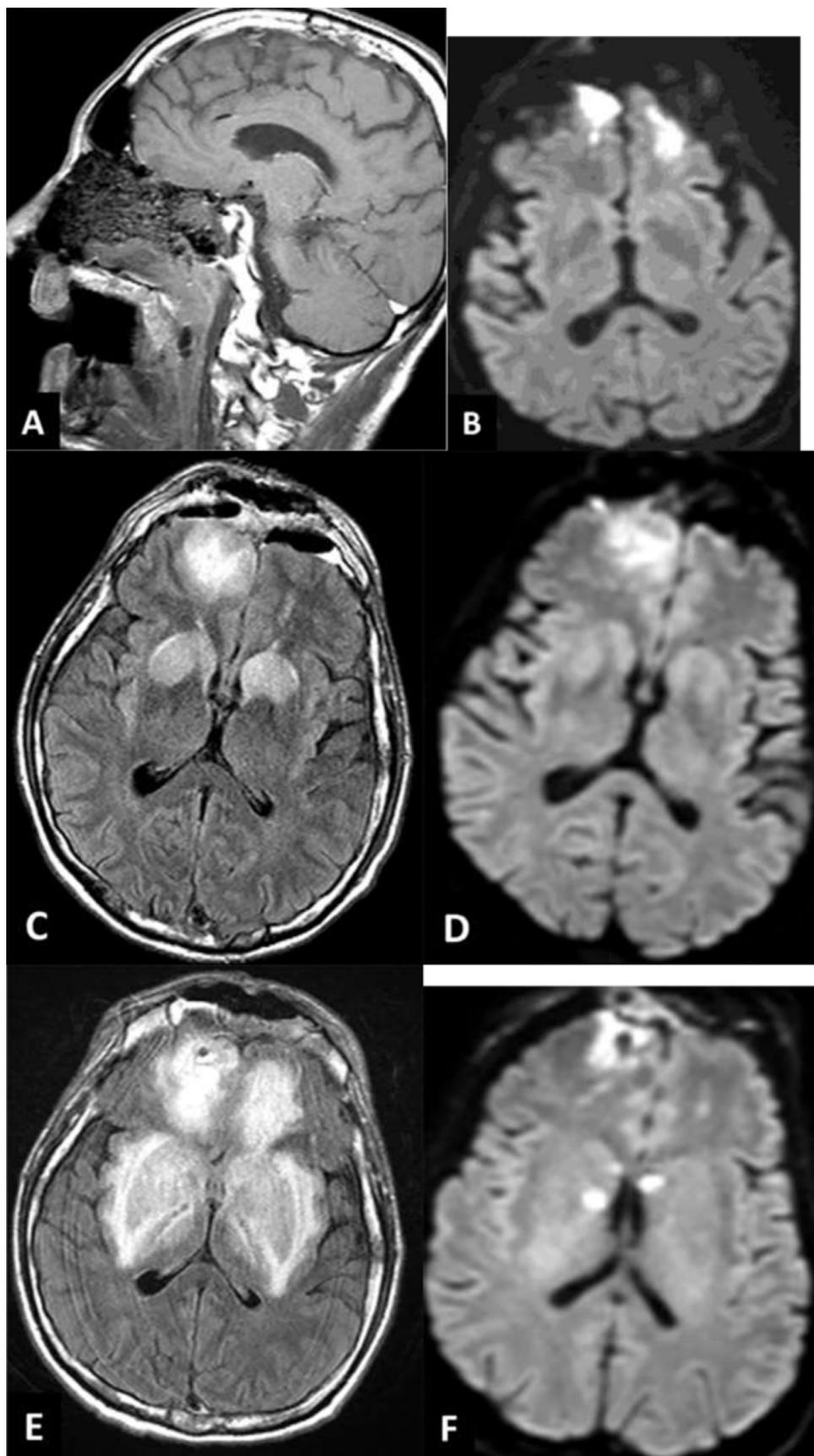
Chronic invasive fungal sinusitis follows an indolent course of disease, as opposed to the rapid progression seen with acute invasive fungal sinusitis. Patients will present with symptoms of chronic sinusitis which does not improve with standard antibiotic therapy. Comorbidities such as diabetes and immunocompromised status are common. The

appearance on CT is similar to that of acute invasive fungal sinusitis, with one key distinction that the calcification within the fungal mass will appear bulkier and denser in the chronic form as opposed to the more punctate calcifications in the acute phase.<sup>53</sup> MR findings of chronic invasive fungal sinusitis will have a varying T1 and T2 hypointense appearance secondary to the degree of fungal concretion, with some cases appearing to have normal aeration when in fact they are filled with T2 hypointense fungal elements.<sup>58</sup> There is often remodeling and expansion of the paranasal sinuses. Due to this expansile appearance and protracted presentation, chronic invasive fungal sinusitis can be easily confused for malignancy until tissue is obtained.

Finally, chronic granulomatous invasive fungal sinusitis is seen in immunocompetent patients with a history of chronic sinusitis. Imaging features are not distinct from chronic invasive fungal sinusitis, with opacification of the sinuses and extension into the adjacent soft tissues being common features.<sup>58</sup> This disease is rare in the United States, with most cases reported in Africa and Asia. Some consider this a variation of chronic invasive fungal sinusitis and not a separate entity.

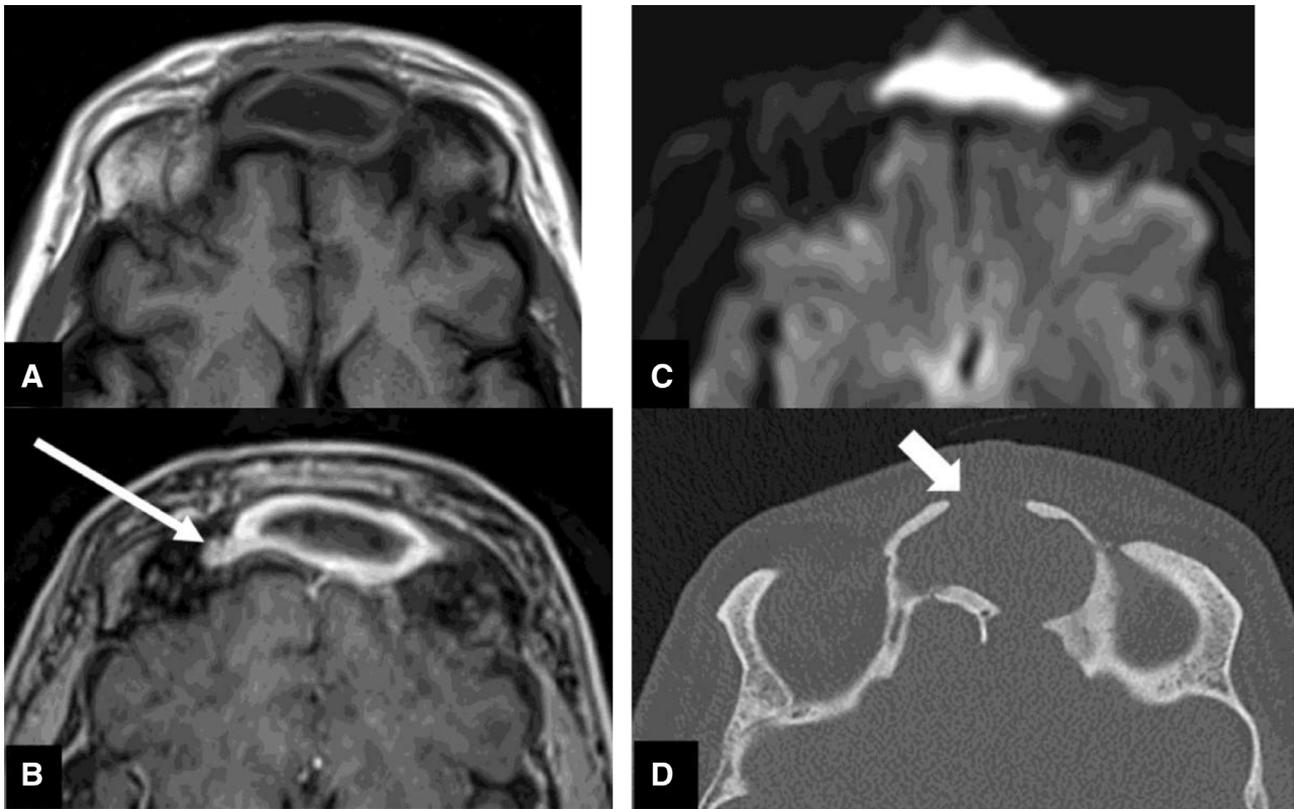
### Osteomyelitis

Chronic bacterial or fungal sinusitis can in some cases lead to osteomyelitis of the facial bones. CT findings of osteomyelitis



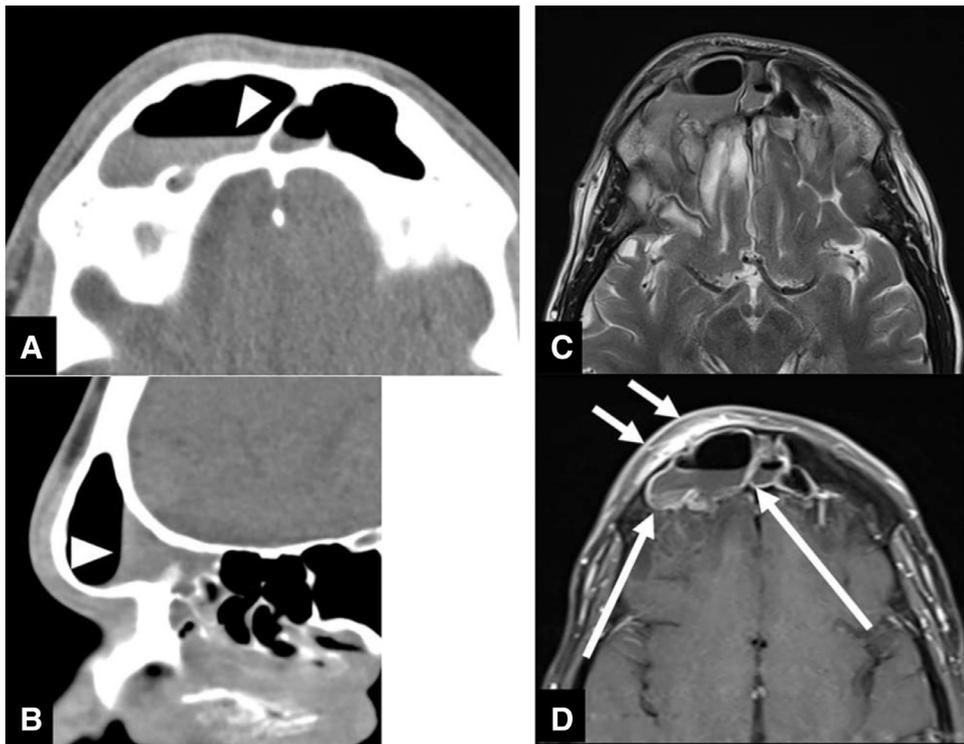
**Figure 17** Progressive invasive mucor sinusitis.

Sagittal T1WI (A) demonstrates post surgical changes in the paranasal sinus due recent debridement. Axial DWI (B) shows bilateral frontal lobes infarcts due to intracranial extension of invasive mucor sinusitis. Axial FLAIR (C) and DWI (D) after 5 days and FLAIR (E) and DWI (F) after 10 days demonstrate the rapid progression of the brain involvement. (Courtesy Toshio Moritani MD).



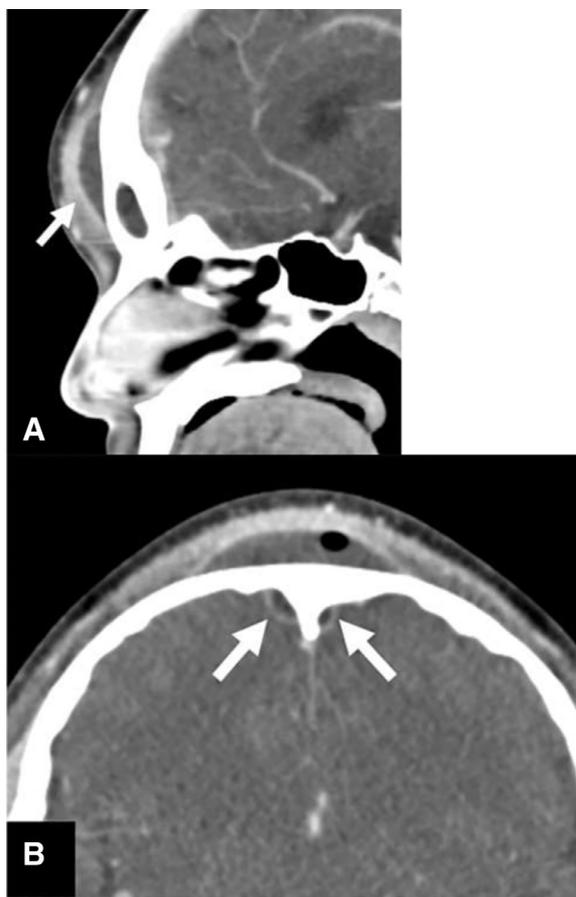
**Figure 18** Frontal sinus abscess and osteomyelitis.

Axial precontrast T1WI (A), postcontrast T1WI (B), and DWI (C) shows an enhancing and diffusion restricting fluid collection in the frontal sinus representing frontal sinus abscess. Surrounding bone marrow edema and enhancement (long arrow) indicates osteomyelitis. There is enhancement and edema of the adjacent soft tissue. NECT bone window (D) demonstrates frank osseous destruction (short arrow).



**Figure 19** Pott's Puffy tumor.

Axial (A) and coronal (B) NECT show a fluid level in the left frontal sinus (arrowhead). Axial T2WI (C) and postcontrast fat sat T1WI (D) through the frontal sinuses show frontal sinus fluid level with enhancing mucosa (long arrows), representing acute sinusitis. Inflammation and enhancement of the adjacent soft tissues (short arrows) represents contiguous spread of infection, consistent with Pott Puffy tumor. (Courtesy Wendy Smoker MD).



**Figure 20** Pott's Puffy tumor with epidural empyema.

Sagittal (A) post-contrast CT demonstrates opacification of the frontal sinus with a subperiosteal abscess (arrow). Axial (B) post-contrast CT shows an intracranial epidural collection consistent with an epidural abscess (arrows). (Courtesy Samir Noujaim MD).

will show variable degrees of bony lysis and erosion, sclerosis, and periosteal reaction depending on the duration of inflammation.<sup>59</sup> A sequestrum and/or involucrum may also be present. Osteomyelitis on MR demonstrates T1 hypointense signal in the bone marrow representing edema and marrow replacement, with corresponding hyperintense T2 signal. Post-contrast fat saturation T1 sequences will show enhancement of the affected bone, periosteum, and surrounding soft tissues.<sup>60</sup> Occasionally, abscess may be present within the sinus, serving as the nidus for osteomyelitis (Fig. 18). An associated condition is formation of a subgaleal abscess, known as Pott's puffy tumor. The mechanism of disease is through osteothrombophlebitis.<sup>61</sup> This disease process most commonly affects adolescents and presents with headaches, fever, and frontal swelling.<sup>62</sup> On contrast-enhanced CT and MR, an extracranial fluid collection adjacent to frontal sinuses or frontal bone can be seen (Fig. 19). If untreated, osteomyelitis and Pott's Puffy tumor can lead to epidural abscess, subdural empyema, meningitis, and orbital involvement (Fig. 20).

## Conclusion

Nontraumatic emergencies of inflammatory paranasal sinus disease is challenging because the presentation is often vague and symptoms may progress slowly and subclinically over time, lowering one's suspicion for a true medical emergency. However, complications of sinonasal disease can have serious consequences such as blindness, sepsis, coma, and even death. Prompt recognition of contiguous spread of infection into the adjacent structures is critical to alert the ordering physician and initiating treatment as quickly as possible. By properly employing CT and MR of the paranasal sinuses, these diagnoses can be accurately made and patient care can be positively impacted.

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