RadioGraphics

Imaging Features of Invasive and Noninvasive Fungal Sinusitis: A Review[1]

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Fungal sinusitis was once considered a rare disorder but is now reported with increasing frequency throughout the world. The classification of fungal sinusitis has evolved in the past two decades, and this entity is now thought to comprise five subtypes. Acute invasive fungal sinusitis, chronic invasive fungal sinusitis, and chronic granulomatous invasive fungal sinusitis make up the invasive group, whereas noninvasive fungal sinusitis is composed of allergic fungal sinusitis and fungus ball (fungal mycetoma). These five subtypes are distinct entities with different clinical and radiologic features. The treatment strategies for the subtypes are also different, as are their prognoses. An understanding of the different types of fungal sinusitis and knowledge of their particular radiologic features allow the radiologist to play a crucial role in alerting the clinician to use appropriate diagnostic techniques for confirmation. Prompt diagnosis and initiation of appropriate therapy are essential to avoid a protracted or fatal outcome.

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Introduction

Of the more than 400,000 known fungal species, approximately 400 are human pathogens, only 50 of which cause systemic or central nervous system infection. Many of these fungi are ubiquitous in our environment. Although many people are colonized by fungi, an intact immune system prevents subsequent infection (1). Although several fungi have been implicated to cause sinus infection, *Aspergillus*, *Bipolaris*, and *Rhizopus* are the more commonly implicated organisms causing fungal sinusitis.

Fungal sinusitis is a relatively common, often misdiagnosed disease process involving the paranasal sinuses. It is a serious condition, as certain forms of fungal sinusitis are associated with a high rate of mortality. Successful treatment requires a prompt diagnosis and frequently relies on radiologic imaging, specifically computed tomography (CT) and magnetic resonance (MR) imaging.

The classification of fungal sinusitis is ever changing, but under the most recent and widely accepted classification fungal sinusitis is broadly categorized as either invasive or noninvasive. Invasive fungal sinusitis is defined by the presence of fungal hyphae within the mucosa, submucosa, bone, or blood vessels of the paranasal sinuses. Invasive fungal sinusitis is subdivided into acute invasive fungal sinusitis, chronic invasive fungal sinusitis, and chronic granulomatous invasive fungal sinusitis. Conversely, noninvasive fungal sinusitis is defined by the absence of hyphae within the mucosal and other tissues of the paranasal sinuses. Noninvasive fungal sinusitis is subdivided into allergic fungal sinusitis and fungus ball (fungal mycetoma) (2).

To distinguish between the invasive and noninvasive forms, adequate quantities of sinus contents and biopsy specimens of diseased and healthy mucosa and bone adjacent to areas of frank necrosis must be obtained for pathologic analysis (2). Fungi do not stain well with routine stains, and special silver-impregnated fungal stains and fungal cultures are required for accurate diagnosis of the fungal sinusitis and subclassification. Fungal cultures are difficult and frequently no fungal growth is achieved despite their identification by staining the surgical specimen, and identification of the actual fungal organism is not always possible. A preoperative suggestion of fungal sinusitis is often helpful by prompting the surgeon to obtain appropriate samples during surgery and alerting the pathologist prior to histopathologic processing for proper identification of allergic mucin, and would also prompt the use of special stains and cultures for the detection of the fungal elements.

Acute Invasive Fungal Sinusitis

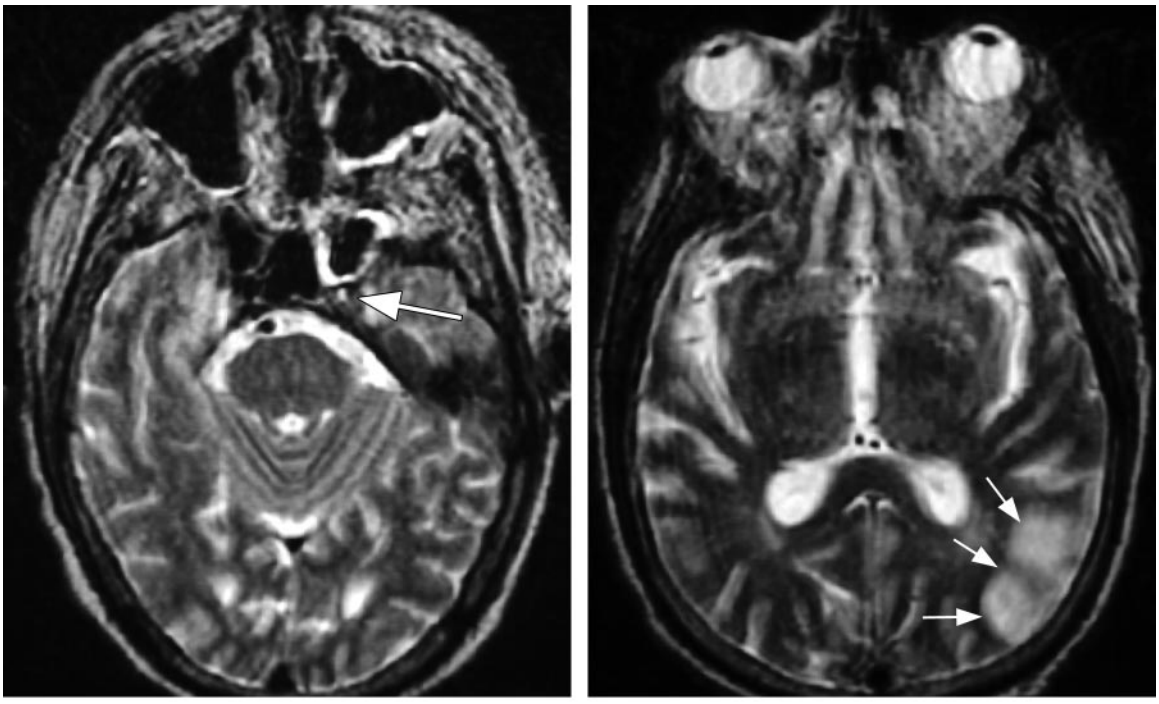
Acute invasive fungal sinusitis is a rapidly progressing infection seen predominantly in immunocompromised patients and patients with poorly controlled diabetes and rarely in healthy individuals. It is the most lethal form of fungal sinusitis, with a reported mortality of 50%–80% (3). The high incidence of associated nasal infection has prompted some to name it invasive fungal rhinosinusitis. It is also thought that the nasal cavity is the primary site of infection, with the middle turbinate accounting for two-thirds of positive biopsy results (4).

Two distinct patient populations are seen. One group is patients with diabetes, especially those with diabetic ketoacidosis. Up to 80% of invasive fungal infections in this group are caused by fungi belonging to the order Zygomycetes, such as *Rhizopus*, *Rhizomucor*, *Absidia*, and *Mucor*, and infection by these organisms is sometimes termed zygomycosis (4). The disease tends to be more rapidly progressing with relatively high mortality and morbidity, probably due to the high virulence of these fungi and also because of low surveillance for invasive fungal disease in this group as well as diagnosis at a more advanced stage of the disease.

The other group is patients with immunocompromise with severe neutropenia (fulminant invasive sinusitis or neutropenic sinusitis). This group consists of patients with hematologic malignancy; patients undergoing systemic chemotherapy, systemic steroid therapy, bone marrow transplantation, or immunosuppressive therapy for organ transplantation; and patients with acquired immunodeficiency syndrome. *Aspergillus* species are responsible for up to 80% of infections in this group (4).

Clinical Features

Invasive fungal sinusitis is clinically characterized by a painless, necrotic nasal septal ulcer (eschar), sinusitis, and rapid orbital and intracranial spread leading to death. There is fulminant progression over a few days to several weeks in which fungal organisms invade the mucosa, submucosa, blood vessels, and bony walls of the nasal cavity and paranasal sinuses. Angioinvasion and hematogenous dissemination are frequent. Symptoms include fever, facial pain or numbness, nasal congestion, serosanguineous nasal discharge, and epistaxis. Intraorbital, intracranial, and maxillofacial extension is common with resulting proptosis, visual disturbances, headache, mental status changes, seizures, neurologic deficits, coma, and maxillofacial soft-tissue swelling. Intracranial



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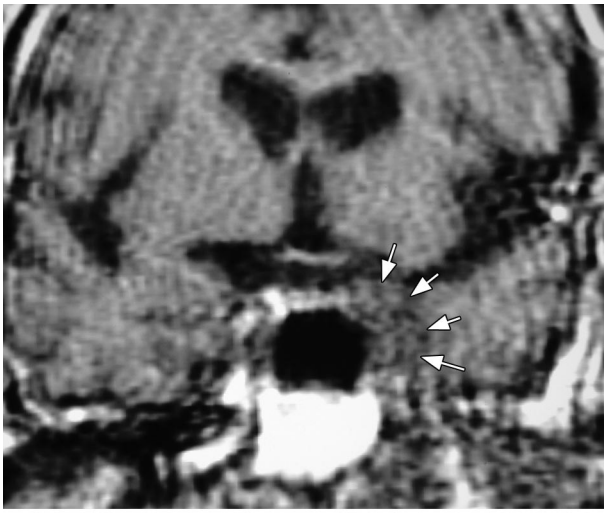


c.

Figure 1. Acute invasive fungal sinusitis due to zygomycosis in a 59-year-old diabetic man with pain and swelling of the left eye and left-sided facial droop. **(a)** Axial T2-weighted MR image shows minimal mucosal thickening in the left sphenoid sinus. The normally expected flow void of the left carotid artery is absent (arrow). **(b)** Axial T2-weighted MR image obtained craniad to **a** shows an acute infarct involving the left temporal lobe (arrows). **(c)** Coronal unenhanced T1-weighted MR image shows soft-tissue thickening in the region of the left cavernous sinus (arrows) secondary to invasion of the cavernous sinus by the sphenoid sinus disease. Despite aggressive treatment with amphotericin B, the patient died 5 days later.

spread of infection predicts higher mortality and morbidity, with up to 73% of patients dying. Patients who do not recover from their neutropenia seem to have a poor prognosis irrespective of the adjunctive therapeutic measures (5).

Imaging Features

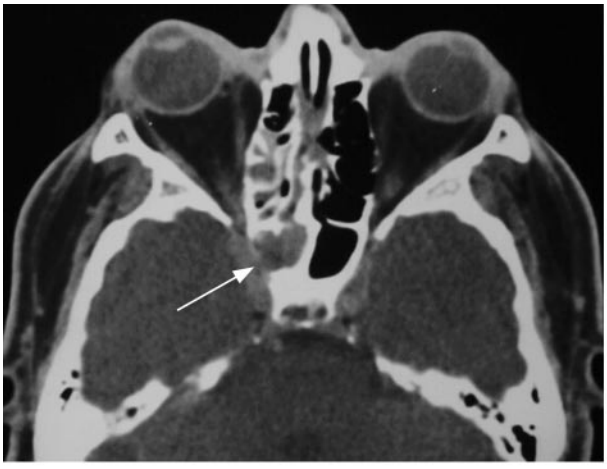
Noncontrast CT demonstrates hypoattenuating mucosal thickening or an area of soft-tissue attenuation within the lumen of the involved paranasal sinus and nasal cavity. There is a predilection for unilateral involvement of the ethmoid and sphenoid sinuses. Aggressive bone destruction of the sinus walls occurs rapidly with intracranial and intraorbital extension of the inflammation.

Teaching Point

Bone erosion and mucosal thickening may sometimes be very subtle and nonsignificant (Fig 1). These fungi tend to extend along the vessels, and extension beyond the sinuses may occur with intact bony walls. Intracranial extension of disease from the sphenoid sinus leads to cavernous sinus thrombosis and even carotid artery invasion, occlusion, or pseudoaneurysm with resulting fatal cerebral infarct and hemorrhage (Figs 1–3).

Whereas CT is better to assess for bone changes, MR imaging is superior in evaluating intracranial and intraorbital extension of the disease. Inflammatory changes in the orbital fat and extraocular muscles and resulting proptosis herald intraorbital invasion by the fungal infection (Figs 3–6). Obliteration of the periantral fat is a

Figures 2, 3. **(2)** Acute invasive zygomycosis in a 42-year-old man. **(a)** Axial contrast-enhanced CT scan shows right ethmoid and sphenoid sinusitis with destruction of the lateral wall of the right sphenoid sinus (arrow). There is invasion of the right cavernous sinus with occlusion of the right internal carotid artery. **(b)** Proton-density–weighted MR image shows the occlusion of the right internal carotid artery more clearly, with absence of the normal flow void in the artery (arrow). **(c)** Conventional angiogram obtained with injection of the left internal carotid artery shows cross flow to the right carotid circulation. **(3)** Acute invasive fungal sinusitis in a 39-year-old woman with diabetic ketoacidosis and acute left eye pain. **(a)** Axial unenhanced CT scan shows sinus disease in the ethmoid, maxillary, and sphenoid sinuses. Note the left-sided facial swelling. **(b)** Axial contrast-enhanced CT scan shows lack of enhancement in the left cavernous sinus (arrows) secondary to thrombosis from invasive fungal sinusitis. **(c)** Axial CT scan obtained craniad to **b** shows proptosis and periorbital inflammatory soft-tissue thickening on the left side (arrow).



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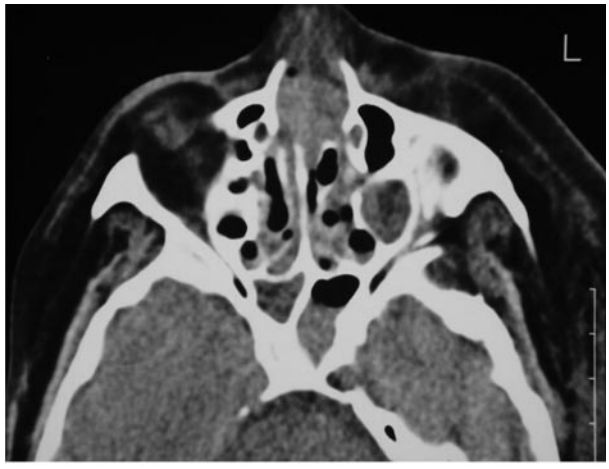

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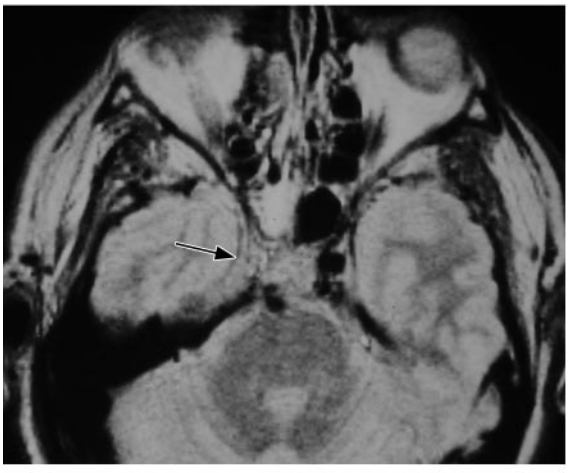

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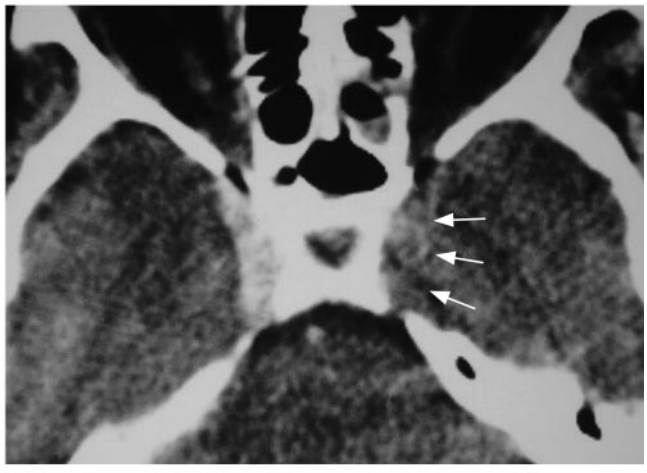

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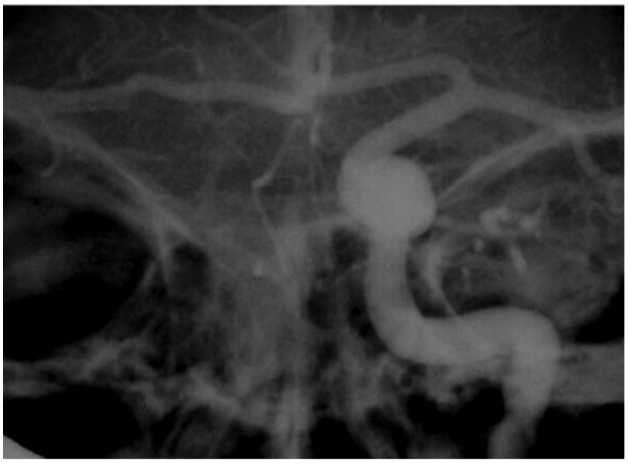

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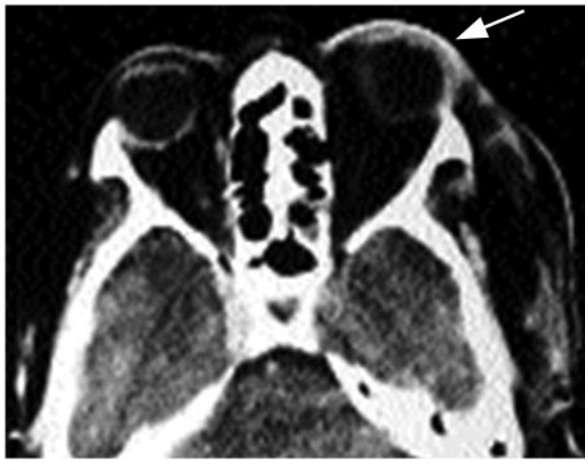

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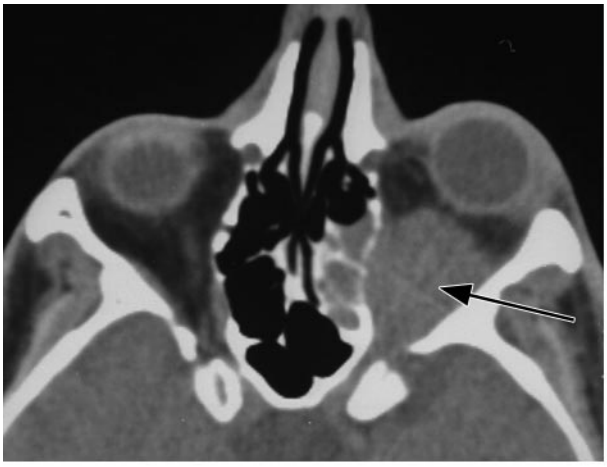
Figures 4, 5. **(4)** Acute invasive aspergillosis in a 37-year-old man with acquired immunodeficiency syndrome who presented with proptosis of the left eye. Axial unenhanced CT scans (**a** obtained craniad to **b**) show soft-tissue thickening in the left posterior ethmoid air cells, which is destroying the medial wall of the orbit and extending into the retro-orbital soft tissues (arrow). **(5)** Acute invasive zygomycosis in a 59-year-old man. **(a)** Axial unenhanced CT scan shows increased attenuation in the right anterior and posterior ethmoid air cells and right sphenoid sinus with soft-tissue thickening in the orbital apex (arrow). **(b)** Coronal unenhanced CT scan shows destruction of the medial wall and floor of the right orbit and disease extension into the orbit (arrows). **(c)** Axial unenhanced CT scan obtained caudad to **a** shows destruction of the posterior wall of the right maxillary sinus and obliteration of the periantral fat plane immediately posterior to the sinus (arrows).



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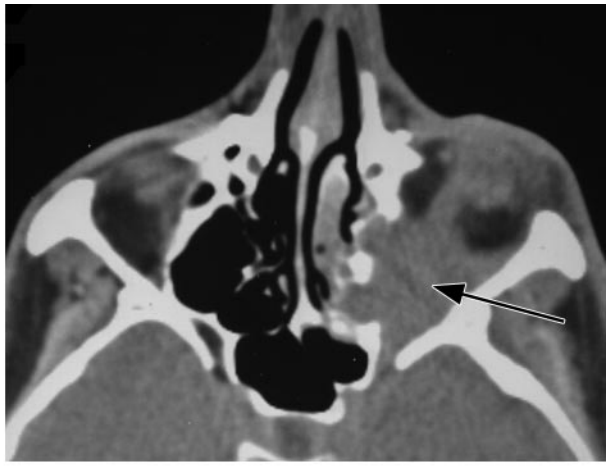

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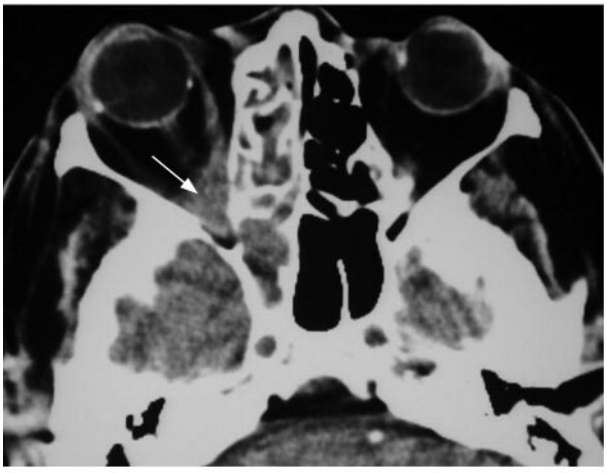

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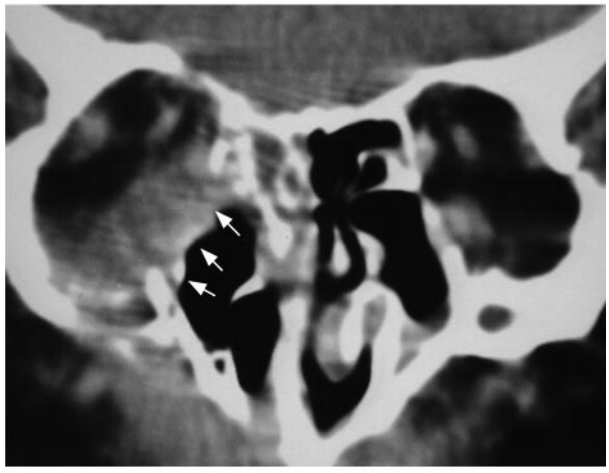

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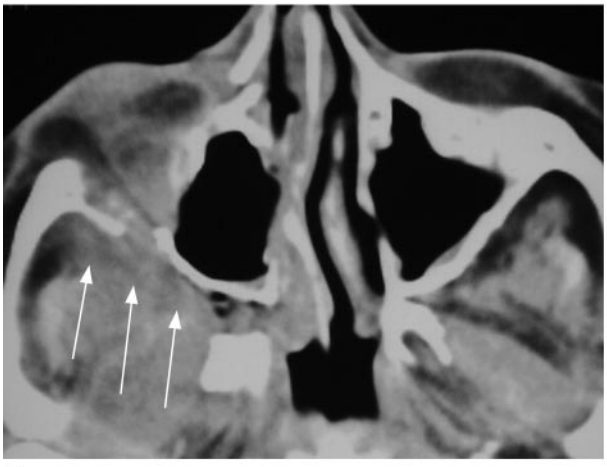

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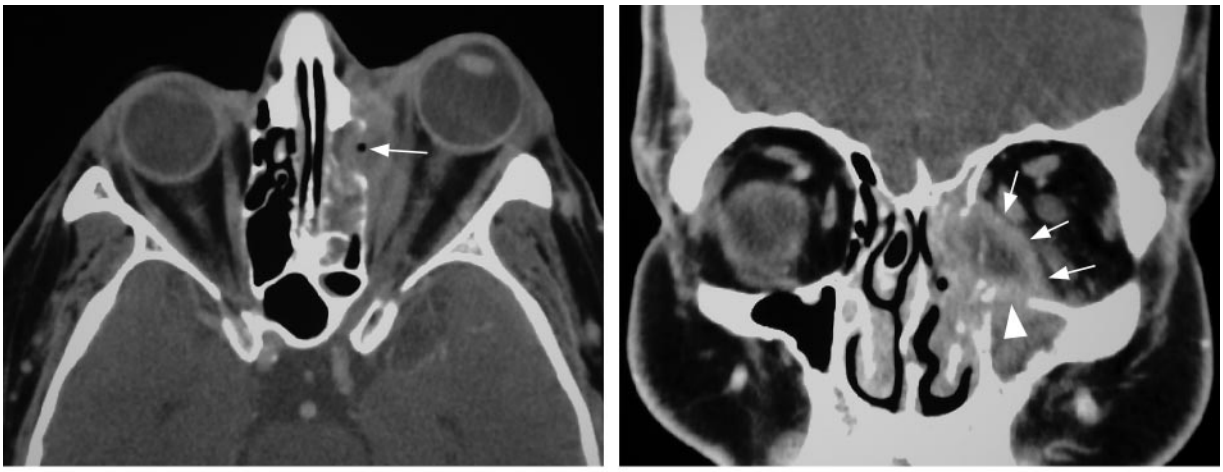


a. b.

Figure 6. Acute invasive fungal sinusitis due to zygomycosis in a 57-year-old diabetic man. **(a)** Axial contrast-enhanced CT scan shows increased attenuation in the left anterior and posterior ethmoid air cells with destruction of the medial wall of the left orbit (arrow). **(b)** Coronal contrast-enhanced CT scan shows a subperiosteal abscess occupying the inferomedial aspect of the left orbit and displacing the medial and inferior rectus muscles laterally (arrows). Note also the destruction of the orbital floor (arrowhead) and the increased attenuation in the adjacent left maxillary sinus.

subtle sign of such extension and must be sought for in all patients at risk for acute invasive fungal sinusitis (6) (Fig 5). Leptomeningeal enhancement may be seen with intracranial invasion and is subtle in the initial stages and must be diligently sought for. With progressive infection, adjacent cerebritis, granulomas, and cerebral abscess formation may be encountered. Intracranial granulomas appear hypointense on T1- and T2-weighted images with minimal enhancement on contrast-enhanced images.

Severe unilateral nasal cavity soft-tissue thickening is the most consistent, though nonspecific, early CT finding (7). More extensive changes such as retroantral fat pad inflammation, bone erosion, and orbital or intracranial invasion are more specific but late, infrequent features.

Treatment

Prompt aggressive surgical débridement of affected tissues and systemic antifungal therapy are the mainstay of treatment. Rigid nasal endoscopy and biopsy of suspicious areas are advocated for early diagnosis (4). Reversal of underlying causes of immunosuppression and administration of systemic antifungal medications such as amphotericin B should be instituted. The fatality rate tends to be in the range of 50%–80%, but active surveillance of the at-risk population, reversal of underlying neutropenia and other causes of immunosuppression, reversal of diabetic ketoacidosis, prompt aggressive surgical débridement, and systemic antifungal chemotherapy can decrease the mortality rate to 18% (5). Recovery from neutropenia is the most predictive indicator of survival, whereas intracranial spread of infection is the highest predictive indicator for mortality (5). Patients with zygomycosis probably require more aggressive surgical débridement.

Chronic Invasive Fungal Sinusitis

Inhaled fungal organisms are deposited in the nasal passageways and paranasal sinuses. Insidious progression occurs over several months to years in which fungal organisms invade the mucosa, submucosa, blood vessels, and bony walls of the paranasal sinuses. This results in significant morbidity and may even be fatal. Common organisms include *Mucor*, *Rhizopus*, *Aspergillus*, *Bipolaris*, and *Candida*.

Clinical Features

Individuals are usually immunocompetent, but those with diabetes or a low level of immunocompromise are susceptible. Patients have a history of chronic rhinosinusitis. Symptoms may include paranasal sinus pain, serosanguineous nasal discharge, epistaxis, nasal polyposis, and fever.

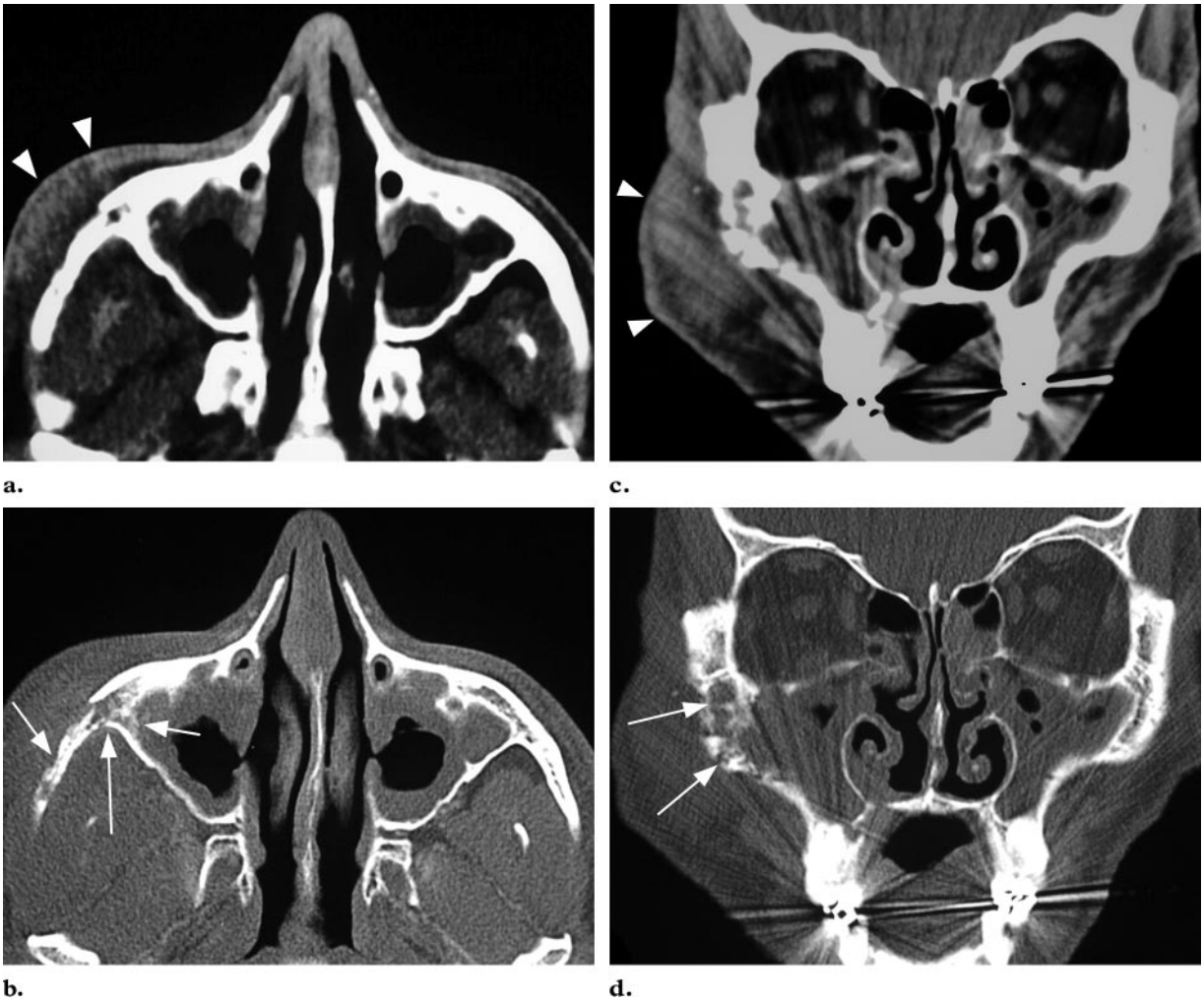


Figure 7. Chronic invasive fungal sinusitis due to zygomycosis in a 44-year-old man. Axial **(a, b)** and coronal **(c, d)** unenhanced CT scans show bilateral mucosal thickening in the maxillary sinuses. Bone invasion is noted in the form of mottled areas of low attenuation in the zygomatic process of the right maxillary bone; this finding is best visualized on the images obtained with bone windows (arrows in **b** and **d**). There is also invasion into the soft tissues of the right cheek (arrowheads in **a** and **c**).

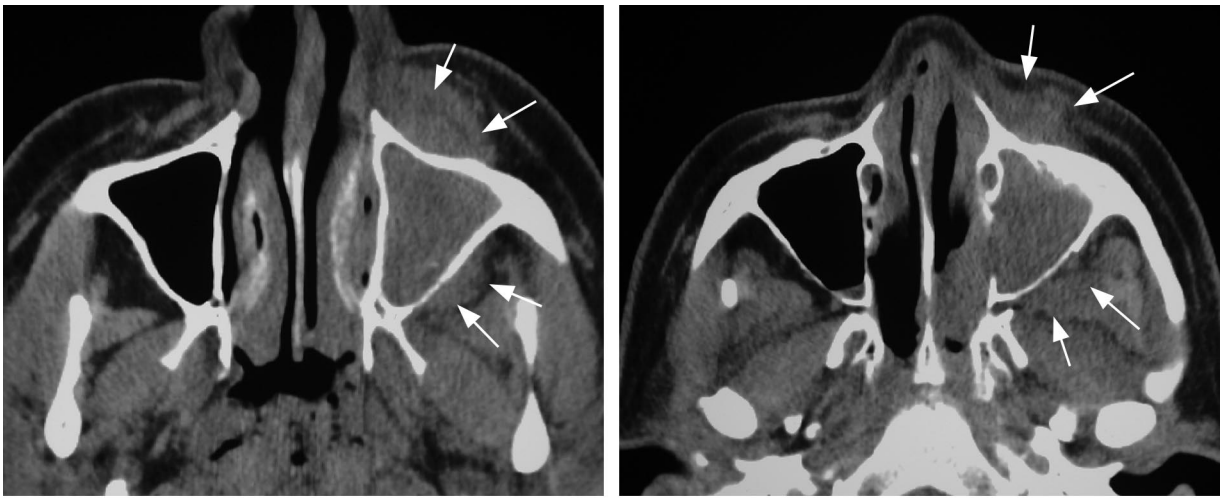
Symptoms directly related to the invasive disease take months to years to develop. The disease is usually persistent and recurrent.

Maxillofacial soft-tissue swelling develops with destruction of the bony sinus walls. Invasion of the maxillary floor leads to palatal erosions. Orbital invasion leads to orbital apex syndrome with proptosis; third, fourth, and sixth cranial neuropathy; and diminished vision. Patients may present with a clinical syndrome mimicking orbital pseudotumor and progress rapidly if steroid therapy is instituted. The cribriform plate may be eroded with resulting chronic headache, seizures, decreased mental status, or focal neurologic deficits. Invasion of the pterygopalatine fossa and infratemporal fossa as well as skull base can manifest as cranial neuropathy. Intranasal examination reveals nasal congestion and polypoid mucosa. Soft-tissue masses may be identified (8).

Imaging Features

Teaching Point

A hyperattenuating soft-tissue collection is seen at noncontrast CT within one or more of the paranasal sinuses. It may be masslike and mimic a malignancy with destruction of the sinus walls and extension beyond the sinus confines. There is decreased signal intensity on T1-weighted MR images and markedly decreased signal intensity on T2-weighted images. Mottled lucencies or irregular bone destruction may be seen in the paranasal sinuses (Fig 7).



a. b.

Figure 8. Chronic invasive fungal sinusitis due to zygomycosis in a 47-year-old woman. Axial unenhanced CT scans (**a** obtained caudad to **b**) show increased attenuation in the left maxillary sinus. Note the absence of the normal fat planes along the posterior wall of the left maxillary sinus. There is extension of infection beyond the walls of the maxillary sinus into the anterior and posterior periantral soft tissues (arrows). Corresponding images obtained with bone windows showed osseous sclerotic changes in the left maxillary sinus, findings consistent with chronic sinus inflammatory changes.

There may also be sclerotic changes in the bony walls of the affected sinuses representing chronic sinus disease (Fig 8).

Infiltration of the periantral soft tissues about the maxillary sinus is an indicator of invasive disease (Figs 7, 8). Invasion of adjacent structures such as the orbit, cavernous sinus, and anterior cranial fossa may lead to epidural abscess, parenchymal cerebritis or abscess, meningitis, cavernous sinus thrombosis, osteomyelitis, mycotic aneurysm, stroke, and hematogenous dissemination. Differentiation between chronic invasive fungal sinusitis and malignant neoplasm may not be possible based on imaging findings (8,9).

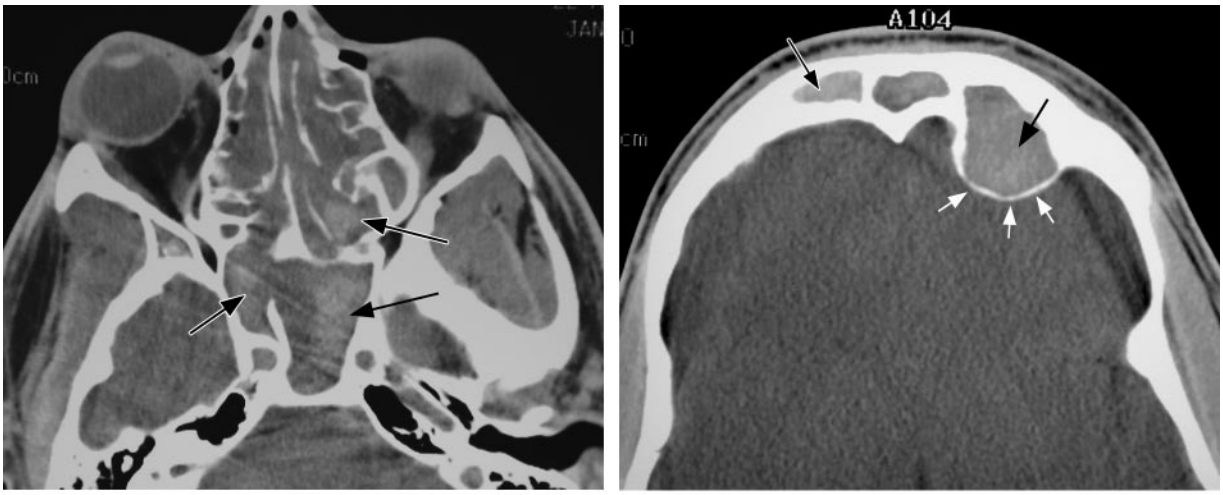
Treatment

Treatment includes surgical exenteration of the affected tissues and systemic antifungal medication. Therapy needs to be as aggressive as for acute invasive fungal sinusitis due to the high mortality and morbidity.

Chronic Granulomatous Invasive Fungal Sinusitis

Also known as primary paranasal granuloma and indolent fungal sinusitis, chronic granulomatous invasive fungal sinusitis is primarily found in Africa (Sudan) and Southeast Asia, with a few cases reported in the United States (2,8). It is usually caused by *Aspergillus flavus*. Individuals are generally immunocompetent. The disease is characterized by noncaseating granulomas in the tissues. It follows a chronic indolent course with eventual extension beyond the walls of the paranasal sinuses with orbital and intracranial extension. However, it is considered by some to be similar to chronic invasive fungal sinusitis and not a distinct entity (8).

Reports describing the imaging findings have been sparse. Cross-sectional imaging findings are expected to be similar to those of chronic invasive fungal sinusitis, with one report showing CT findings in a patient with a right maxillary and ethmoid sinus soft-tissue mass eroding the orbital walls and nasal cavity, with invasion of the orbital soft tissues and pterygopalatine fossa (8). In the three patients described in that review of chronic invasive fungal rhinosinusitis (8), the imaging features were similar to those of an invasive mass



a. b.

Figure 9. Allergic fungal sinusitis due to *Bipolaris* in a 22-year-old man with a long history of nasal obstruction. Axial unenhanced CT scans show expansion of and increased attenuation in the anterior ethmoid, posterior ethmoid, sphenoid, and frontal sinuses bilaterally. There is characteristic hyperattenuating material within these sinuses (black arrows). Note also the smooth thinning of the posterior wall of the left frontal sinus (white arrows in **b**).

lesion, with descriptive findings difficult to distinguish from those of a malignant neoplasm invading the paranasal sinuses, orbital soft tissues, infratemporal fossa, and skull base. Treatment consists of surgical débridement and systemic antifungal medication if required (8,10).

Allergic Fungal Sinusitis

Allergic fungal sinusitis is the most common form of fungal sinusitis. It is particularly common in warm, humid climates such as the southern United States. The overall prevalence of allergic fungal sinusitis is estimated at 5%–10% of all patients with chronic hypertrophic sinus disease going to surgery (11). However, the incidence of this entity may have geographic variation, and it has been reported in up to 51% of patients in northern India with chronic rhinosinusitis (12).

The underlying cause is thought to be a hypersensitivity reaction to certain inhaled fungal organisms resulting in a chronic noninfectious, inflammatory process, similar to that seen in allergic bronchopulmonary aspergillosis of the lung. Although the exact pathophysiology is not yet clear, immunologically, immunoglobulin E–mediated type I immediate hypersensitivity and type III delayed hypersensitivity are thought to be involved (11). Common implicated fungi are the dematiaceous (pigmented) fungi—*Bipolaris*, *Curvularia*, *Alternaria*—and the hyaline molds such as *Aspergillus* and *Fusarium*. Allergic fungal sinusitis is characterized by the presence within the affected sinuses of "allergic mucin"—inspissated mucus that is yellow-green, white-tan, gray, brown, or black with the consistency of peanut butter. Histologic analysis reveals eosinophils and eosinophil degradation products known as Charcot-Leyden crystals (13).

Clinical Features

Allergic fungal sinusitis tends to be a disease of younger individuals, usually in their third decade. Typically, afflicted individuals are immunocompetent. There is a frequent associated history of atopy including allergic rhinitis and asthma. Patients usually experience chronic headaches, nasal congestion, and chronic sinusitis for several years. There is often a history of sinus surgery.

Imaging Features

Teaching Point

There is usually involvement of multiple sinuses if not pansinusitis and rhinitis. Disease tends to be bilateral, and there is a frequent nasal component. The majority of the sinuses show near-complete opacification and are expanded. Noncontrast CT demonstrates hyperattenuating allergic mucin within the lumen of the paranasal sinus (Figs 9, 10) (14).

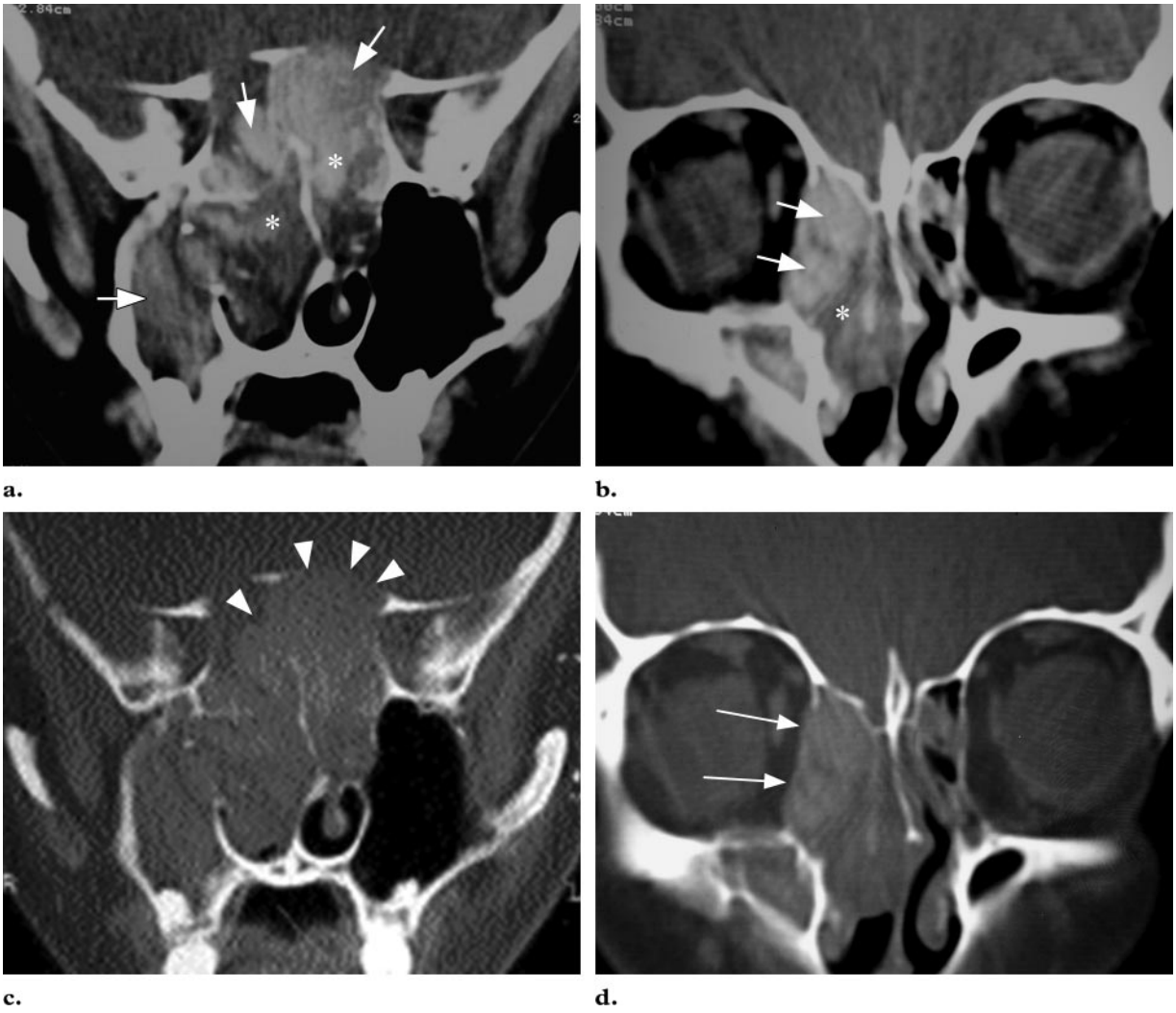


Figure 10. Allergic fungal sinusitis due to *Bipolaris* in a 26-year-old man. **(a, b)** Coronal CT scans (**a** obtained posterior to **b**) show characteristic expansile, hyperattenuating material in the sphenoid, ethmoid, and right maxillary sinuses (arrows). Extension into the nasal cavity (*) from the bilateral ethmoid sinuses and right maxillary sinus is noted. **(c, d)** Corresponding images obtained with bone windows show smooth erosion of the roofs of the posterior ethmoid sinuses (arrowheads in **c**) with intracranial extension, which is possibly limited by the dura. There is also smooth erosion of the medial wall of the right orbit (arrows in **d**) with intraorbital extension, which is possibly limited by the periosteum.

Although low signal intensity of the sinus contents has been described on T1-weighted images, we have frequently observed high signal intensity or mixed low, intermediate, and high signal intensity on T1-weighted images in these patients (Figs 11, 12). There is characteristic low signal intensity or signal void on T2-weighted images (Figs 11, 12) (15). The T2 signal void is attributed to a high concentration of various metals such as iron, magnesium, and manganese concentrated by the fungal organisms. The T2 signal void is also attributed to a high protein and low free-water content of the allergic mucin.

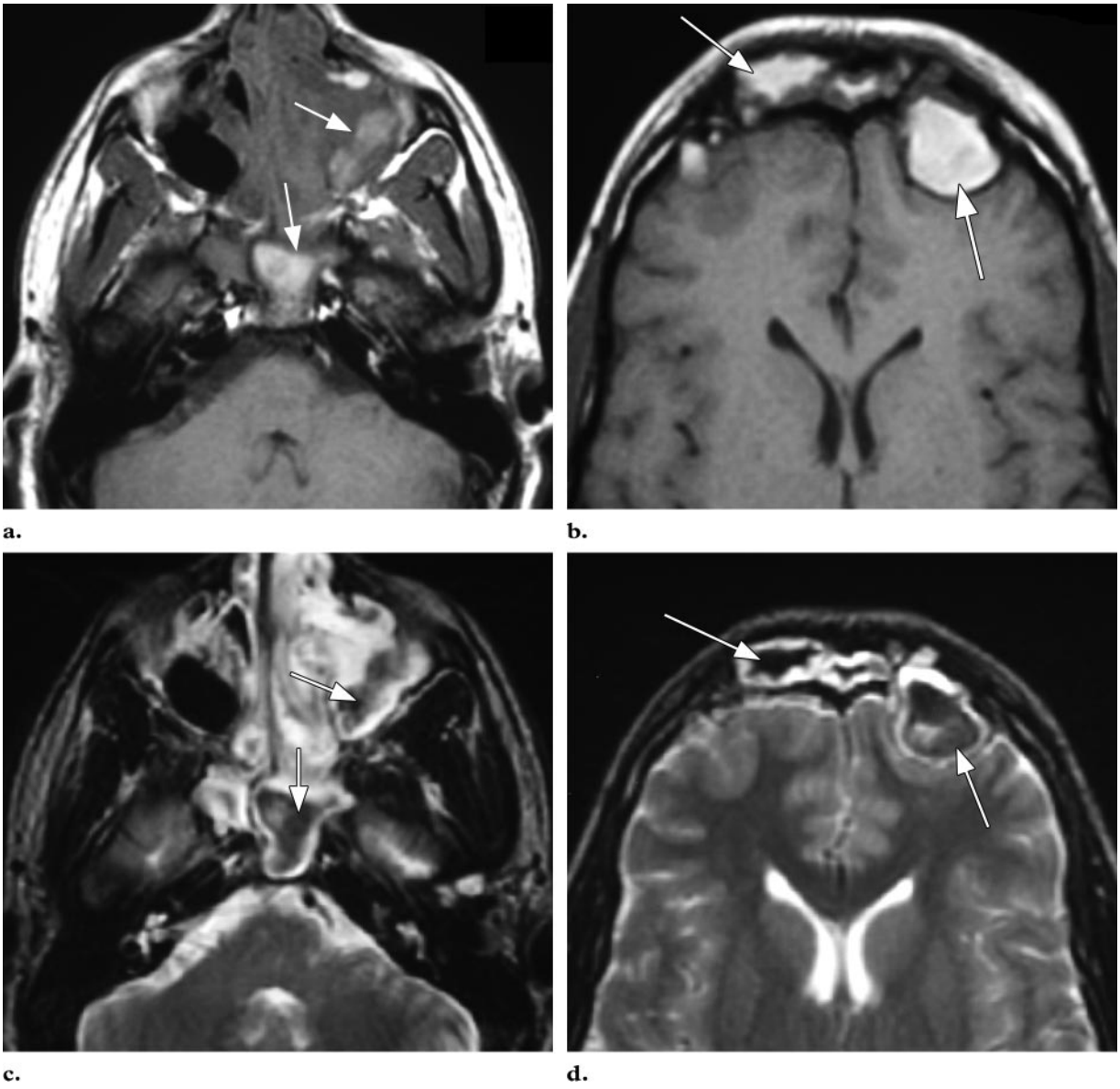


Figure 11. Allergic fungal sinusitis due to *Bipolaris* in a 22-year-old man with a long history of nasal obstruction (same patient as in Fig 9). **(a, b)** Unenhanced T1-weighted MR images show characteristic high signal intensity within the left maxillary, left posterior ethmoid, and sphenoid sinuses (arrows in **a**) and bilateral frontal sinuses (arrows in **b**). **(c, d)** Corresponding T2-weighted MR images show marked low signal intensity within the left maxillary, left posterior ethmoid, and sphenoid sinuses (arrows in **c**) and bilateral frontal sinuses (arrows in **d**).

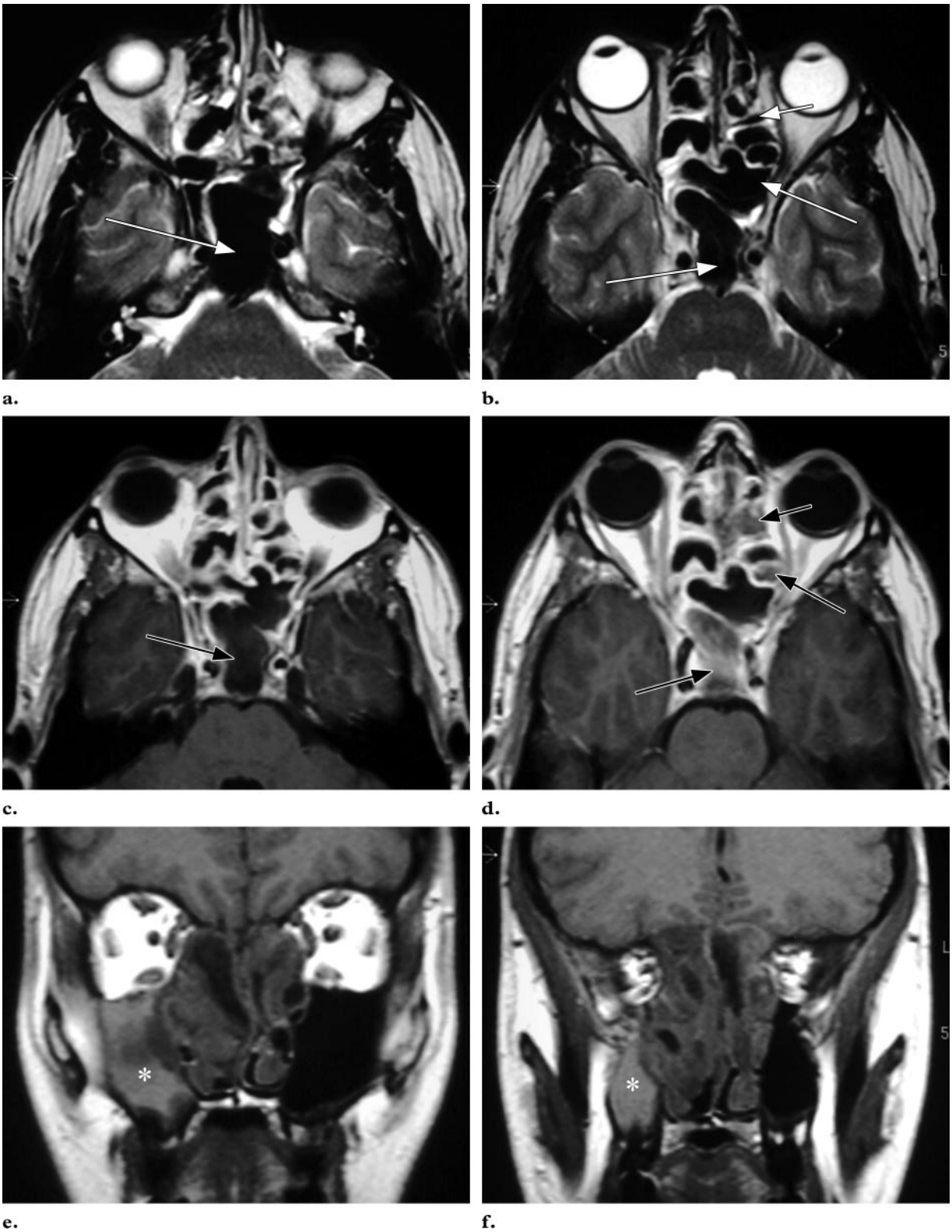
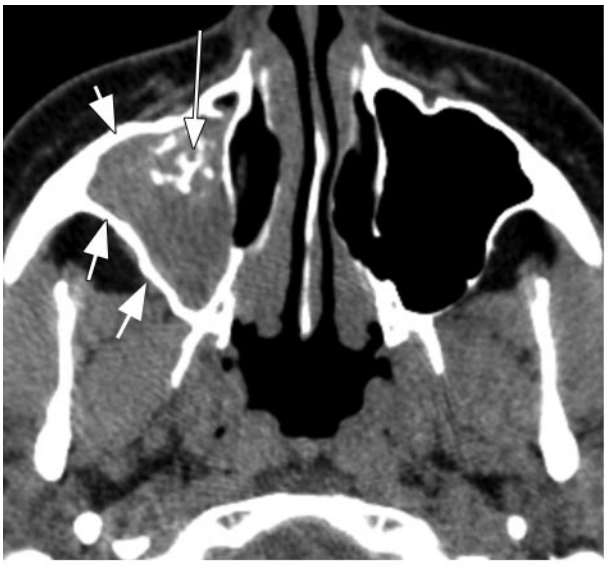


Figure 12. Allergic fungal sinusitis due to *Bipolaris* in a 26-year-old man (same patient as in Fig 10). **(a, b)** T2-weighted MR images (**a** obtained caudad to **b**) show marked low signal intensity within the hyperintense ethmoid and sphenoid sinuses (arrows), an appearance that mimics normally aerated sinuses. **(c, d)** Corresponding contrast-enhanced T1-weighted MR images show expansile hypointense to isointense material within the centers of these sinuses (arrows) with enhancing mucosa noted peripherally. **(e, f)** Unenhanced T1-weighted MR images (**e** obtained anterior to **f**) show a characteristic hyperintense collection within the right maxillary sinus (*).



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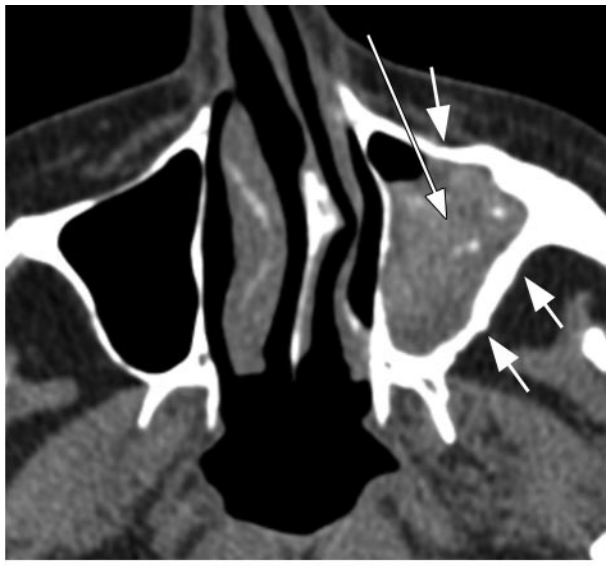

14.

Figures 13, 14. **(13)** *Mucor* fungus ball in a 49-year-old woman with chronic sinus pressure and halitosis. Unenhanced CT scan shows isoattenuating to hyperattenuating material filling the right maxillary sinus with central calcific areas of increased attenuation (long arrow). Note the circumferential thickening of the osseous walls of the sinus (short arrows), a finding consistent with a chronic inflammatory process. **(14)** *Aspergillus* fungus ball in a 60-year-old woman with mixed connective tissue disorder and a history of cryoglobulinemia and Sjögren syndrome. Axial unenhanced CT scan shows the typical hyperattenuating fungus ball with calcific foci in the left maxillary sinus (long arrow). Note the sclerotic thickening of the osseous walls of the sinus (short arrows) from chronic sinus inflammation.

The inflamed mucosal lining is relatively hypointense on T1-weighted images and hyperintense on T2-weighted images and demonstrates enhancement after intravenous administration of gadolinium contrast material. There is no enhancement in the center or in majority of the sinus contents, which distinguishes this condition from neoplastic entities (Fig 12). Although the condition is not considered invasive, if left untreated, the involved sinuses expand and there is smooth bone erosion with subsequent intracranial or intraorbital extension and resulting cranial or orbital symptoms (Figs 9–12). Intracranial extension is usually limited by the dura to the extradural space (Fig 10).

Treatment

Surgical extirpation of the allergic mucin and restoration of normal sinus drainage is the goal. Long-term use of topical nasal steroids for suppression of the immune response prevents postsurgical recurrence. Topical or systemic antifungal medication is not warranted. The use of systemic corticosteroids is controversial. Allergen immunotherapy, antihistamines, oral antileukotrienes, oral steroids, and nasal irrigation may also be helpful (11).

Fungus Ball

Also known as mycetoma, fungus ball is a relatively uncommon manifestation of fungal sinusitis. The most widely accepted pathogenesis theorizes a deficient mucociliary clearance mechanism in which fungal organisms deposited in the paranasal sinuses are inadequately cleared. Organisms germinate, replicate, and incite an inflammatory response within the paranasal sinus. The fungus ball represents a tangled collection of fungal hyphae in the absence of allergic mucin. There is no fungal invasion of the sinus mucosa, blood vessels, or bone, although chronic nongranulomatous inflammation may be observed in the mucosa. It is usually caused by *Aspergillus fumigatus*, although other fungi such as *Pseudallescheria boydii* and *Alternaria* have been implicated (16).

Clinical Features

Fungal mycetoma tends to occur in older individuals with an apparent female predilection. Afflicted individuals are usually immunocompetent. Patients are either asymptomatic or have minimal symptoms. Individuals commonly describe a chronic pressure sensation involving one of the paranasal sinuses. Other symptoms may include nasal discharge and cacosmia (2).

Imaging Features

Teaching Point

Fungus ball appears as a mass within the lumen of a paranasal sinus and is usually limited to one sinus. The maxillary sinus is the most commonly involved sinus (Figs 13, 14). However, sometimes the sphenoid sinus appears to be affected.

A fungus ball typically appears hyperattenuating at noncontrast CT due to dense matted fungal hyphae and may demonstrate punctate calcifications (Fig 13). The inflamed mucosal

lining of the paranasal sinus is frequently hypoattenuating at noncontrast CT and hyperintense on T2-weighted images with contrast enhancement. The central sinus contents do not enhance. The bony walls of the sinus may be sclerotic and thickened or expanded and thinned with focal areas of erosion from pressure necrosis. The fungus ball is hypointense on T1-weighted and T2-weighted images owing to the absence of free water. Calcifications and paramagnetic metals such as iron, magnesium, and manganese also generate areas of signal void on T2-weighted images.

Treatment

Treatment requires surgical removal and restoration of drainage of the paranasal sinus. Antifungal medications are generally unnecessary. Recurrence is rare.

Conclusions

Fungal sinusitis is an important clinical problem with diverse manifestations. It should be considered in all immunocompromised patients and in all patients with chronic sinusitis. It can be noninvasive or invasive with five major subtypes.

Acute invasive fungal sinusitis affects immunocompromised patients and patients with poorly controlled diabetes. Orbital and intracranial invasion is common, and mortality tends to be high unless the condition is detected early and treated aggressively. Imaging features are often subtle in the initial stages, and radiologists need to be alert while evaluating the sinuses in this group of patients for early signs of invasion.

Chronic invasive fungal sinusitis and chronic granulomatous invasive fungal sinusitis are characterized by a prolonged clinical course with slow disease progression. Imaging manifestations may mimic aggressive neoplastic lesions. Features of chronic sinus disease are seen in addition to invasive disease in the orbits and cranium.

Allergic fungal sinusitis tends to be a disease of young atopic individuals. There is usually pansinus disease with expansion and smooth thinning of the affected sinuses. The sinus contents tend to be hyperattenuating, and there is characteristic high signal intensity on T1-weighted images and low signal intensity on T2-weighted images. There is thin peripheral enhancement with no enhancement noted in the central sinus contents. Surgical extirpation and antiallergic medications are the mainstay of therapy without the need for toxic systemic or local antifungal therapy.

Fungus ball (mycetoma) tends to occur in a single sinus, most often the maxillary sinus, and affected individuals are usually nonatopic. The sinus contains hyperattenuating material, and there may be evidence of chronic sinus disease or smooth bone erosion. Surgical removal is generally curative and recurrence is unusual.

Teaching Point

Understanding the different types of fungal sinusitis and knowing their particular radiologic features allows the radiologist to play a crucial role in alerting the clinician to use appropriate diagnostic techniques for confirmation. Prompt diagnosis and initiation of appropriate therapy are essential to avoid a protracted or fatal outcome.

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Imaging Features of Invasive and Noninvasive Fungal Sinusitis: A Review

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Page 1285
Bone erosion and mucosal thickening may sometimes be very subtle and nonsignificant (Fig 1). These fungi tend to extend along the vessels, and extension beyond the sinuses may occur with intact bony walls.

Page 1289
A hyperattenuating soft-tissue collection is seen at noncontrast CT within one or more of the paranasal sinuses. It may be masslike and mimic a malignancy with destruction of the sinus walls and extension beyond the sinus confines.

Page 1291
There is usually involvement of multiple sinuses if not pansinusitis and rhinitis. Disease tends to be bilateral, and there is a frequent nasal component. The majority of the sinuses show near-complete opacification and are expanded. Noncontrast CT demonstrates hyperattenuating allergic mucin within the lumen of the paranasal sinus (Figs 9, 10) (14).

Page 1295
Fungus ball appears as a mass within the lumen of a paranasal sinus and is usually limited to one sinus. The maxillary sinus is the most commonly involved sinus (Figs 13, 14). However, sometimes the sphenoid sinus appears to be affected. A fungus ball typically appears hyperattenuating at noncontrast CT due to dense matted fungal hyphae and may demonstrate punctate calcifications (Fig 13).

Page 1296
Understanding the different types of fungal sinusitis and knowing their particular radiologic features allows the radiologist to play a crucial role in alerting the clinician to use appropriate diagnostic techniques for confirmation. Prompt diagnosis and initiation of appropriate therapy are essential to avoid a protracted or fatal outcome.