INVASIVE FUNGAL SINUSITIS IN THE ACQUIRED IMMUNODEFICIENCY SYNDROME

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Sinusitis is a common clinical problem for patients with AIDS. The prevalence of bacterial sinusitis in AIDS patients has retrospectively been estimated at 10% to 20%; however, recent prospective studies suggest a rate as high as 68%.[26] The incidence of fungal sinusitis also appears to be on the rise in this population. Although this may simply reflect a greater awareness and recognition of fungal sinusitis in this population, it also may be the result of changing pathophysiologic conditions associated with AIDS. Because the symptoms of chronic bacterial sinusitis and chronic fungal sinusitis are often similar, the clinical distinction between these two processes can be difficult. If a high index of suspicion is not maintained, fungal invasion may go unrecognized until localizing symptoms or physical findings draw attention to this diagnosis. Early recognition of invasion is vital to the successful management of invasive fungal sinusitis.

Four cases of invasive fungal sinusitis that have presented to the senior author between January, 1995 and April, 1997, form the basis of this article. These cases were reviewed for radiographic and clinical evidence suggesting the presence of invasion beyond the confines of the paranasal sinuses. Through a thorough literature review, 22 other well-documented

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cases of invasive fungal sinusitis in HIV-infected patients were identified.[2-5,7,9,10,13,16,18,21,23,25,29,31,33,35,36] These cases were included only if histopathologic or culture-proven presence of the fungal pathogen was determined in the setting of obvious intraoperative or radiographic tissue invasion. From these cases, patient demographics, AIDS-related illnesses, medication profiles, symptomatology, physical, laboratory, and radiographic findings, medical and surgical treatments rendered, offending organism, and survival were reviewed. Table 1 presents select information from these cases.

Case Reports

Case 1. A 34-year-old HIV-infected male presented in March, 1995, complaining of left maxillary toothache, left facial pain and intermittent swelling, fever, and chills. Following a negative dental evaluation, he was diagnosed with sinusitis. He had been treated with oral antibiotics, decongestants, and intravenous antibiotics without relief. His CD4 lymphocyte count was 4 cells per mm^3 (normal 800 to 1200 per mm^3), and his absolute neutrophil count (ANC) was 400 cells per mm^3. Because of left facial swelling, a CT scan was obtained. Left osteomeatal complex obstruction and complete opacification of the left maxillary and ethmoid sinuses was noted. A portion of the medial wall of the left antrum was eroded and there was slight bony expansion of the left ethmoid air cells (Figs. 1 and 2). Progressive facial erythema and edema, as well as the development of hypesthesia in the distribution of the infraorbital nerve, prompted surgery. At the time of left endoscopic maxillary antrostomy, thickened, grayish, polypoid mucosa with associated purulence was encountered in the maxillary sinus. Histopathologic examination revealed invasive elements consistent with mucormycosis, and cultures subsequently grew *Rhizopus oryzae*. The patient was treated with intravenous (IV) amphotericin B, and left external ethmoidectomy, sphenoidectomy, Caldwell-Luc, and medial maxillectomy were performed. Routine postoperative nasal irrigations were begun and IV amphotericin B continued for a total dose of 2 grams.

Following a brief period of symptomatic improvement, the patient again developed headaches and facial pain. MR imaging scan, performed 3 months postoperatively, revealed progression of disease into the left orbit, pterygopalatine fossa and infratemporal fossa. The patient was treated with additional amphotericin B; however, the infection slowly progressed until his death 5 months postoperatively.

Case 2. A 34-year-old HIV-infected male presented in January, 1995, with severe right facial pain and toothache unresponsive to oral antibiotic and decongestant therapy. His CD4 count was 0 cells per mm^3, and his ANC was 4000 cells per mm^3. Other than nasal septal deviation, physical examination was unremarkable. A CT scan revealed mucosal thickening in the right maxillary sinus with osteomeatal obstruction. The other sinuses were clear except for an opacified left agar nasi cell. When right cheek numbness and periorbital edema developed, a septoplasty and bilateral endoscopic ethmoidectomies and maxillary antrostomies were performed. Thick, granular mucosa was noted on the posterolateral and superior surfaces of the right maxillary sinus, and thick purulence was drained from the ethmoid cells bilaterally. Histopathologic examination of the right maxillary tissue revealed chronic inflammation, bony necrosis, and septate fungal hyphae consistent with *Aspergillus* species. Intravenous amphotericin therapy was begun. Review of the preoperative CT scan revealed a subtle dehiscence along the

Table 1. LITERATURE REVIEW

Patient (ref.)	Age	Sex	CD4/mm³	ANC/mm³	Organism	Outcome
1 (33)	45	M	<50	1440	*Aspergillus fumigatus*	Died ~ 3 mo after surgery
2 (16)	44	M	<50	2498	*Pseudallescheria boydii*	Died ~ 3 mo after surgery – unrelated cause
3 (16)	38	M	8	600	*A fumigatus*	Died ~ 1 mo after surgery
4 (16)	33	M	<10	3854	*A fumigatus*	Died ~ 3 wk after surgery
5 (16)	50	M	30	120	*A fumigatus*	Died 2 days after surgery – brain herniation
6 (4)	20	M	5	6200	*Candida albicans*	Alive 1 mo after surgery – long term N/A
7 (2)	30	M	N/A	1500	*Rhizopus arrhizus*	Died 7 mo after surgery – unrelated cause
8 (25)	62	F	150	1620	*Schizophyllum commune*	Died 2 yr after surgery – unrelated cause
9 (3)	44	M	N/A	N/A	*A fumigatus*	Died before intervention – cerebral ext.
10 (13)	30	M	5	900	*A fumigatus*	Died ~ 1 wk after surgery
11 (29)	36	M	N/A	N/A	*Aspergillus* sp	Died "few" wk after biopsy – cerebral ext.
12 (5)	N/A	M	N/A	N/A	*Aspergillus* sp	Died ~ 2 wk after surgery – cerebral ext.
*13 (18)	32	M	N/A	N/A	*A fumigatus*	Died from unrelated cause
14 (36)	32	F	N/A	N/A	*A fumigatus*	N/A
15 (7)	42	M	10	N/A	*A fumigatus*	Died ~ 3 wk after surgery
16 (21)	35	F	20	1100	*A fumigatus*	Alive 2.5 yr post onset & multiple surgeries
17 (21)	39	M	<10	N/A	*A fumigatus*	Died 2 wk after surgery
18 (35)	N/A	N/A	17	N/A	*A fumigatus*	Died 5 days after surgery – cerebral ext.
19 (23)	31	M	0	2500	*Mucor, Candida* sp	Died before surgery
20 (31)	43	F	N/A	N/A	*A fumigatus*	N/A
21 (10)	37	M	10	N/A	*Apophysomyces elegans*	Died 11 wk after surgery
22 (9)	44	M	4	N/A	*P boydii*	Died several weeks postop/cerebral ext.
23 (PR)	34	M	4	400	*Rhizopus oryzae*	Died 5 mo after surgery
24 (PR)	34	M	0	4000	*Paecilomyces variotii*	Died 4 mo after surgery
25 (PR)	36	M	0	1000	*A fumigatus*	Died 1 mo after surgery
26 (PR)	34	F	4	900	*A fumigatus*	Died 1 wk after surgery

*Case from an autopsy review study
ANC = absolute neutrophil count; ext. = extension; N/A = information not available; PR = present report

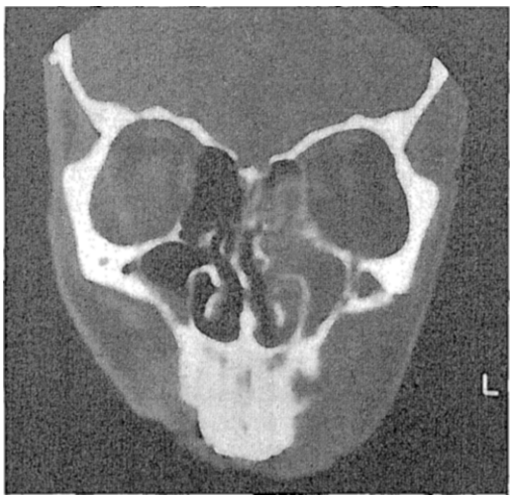

Figure 1. Coronal CT image reveals opacification of left maxillary sinus and ethmoid air cells, with slight bony expansion of ethmoid cells (Case 1).

posterolateral wall of the right maxillary sinus (Figs. 3 and 4). A right Caldwell-Luc was performed subsequently. The periosteum and soft tissue of the lateral and superior walls of the maxillary sinus were involved with an invasive inflammatory process. The infraorbital nerve and orbital periosteum also were involved grossly and infiltrated. Generous debridement was performed, sparing the orbital

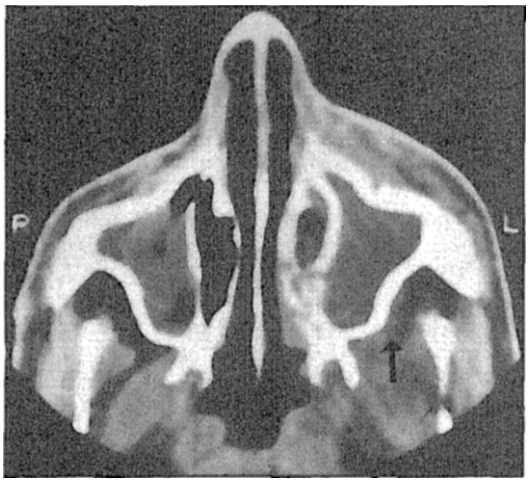

Figure 2. Axial CT image with subtle evidence of extension into infratemporal fossa. Note replacement of the normal fat plane by soft tissue density. This finding was only recognized after the diagnosis of invasive fungal sinusitis had been established (Case 1).

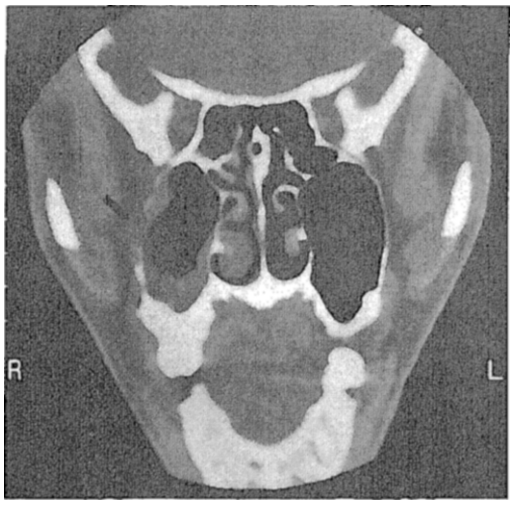

Figure 3. Coronal CT image reveals right maxillary sinus mucosal thickening with bony erosion through the posterolateral sinus wall and extrasinus inflammation into infratemporal fossa (Case 2).

contents. Culture subsequently revealed *Paecilomyces variotti* to be the etiologic agent.

The patient temporarily had symptomatic improvement; however, 3 months later he began to experience retroorbital pain. Over the ensuing month, diplopia and restricted inferior gaze developed. MR imaging revealed persistent right max-

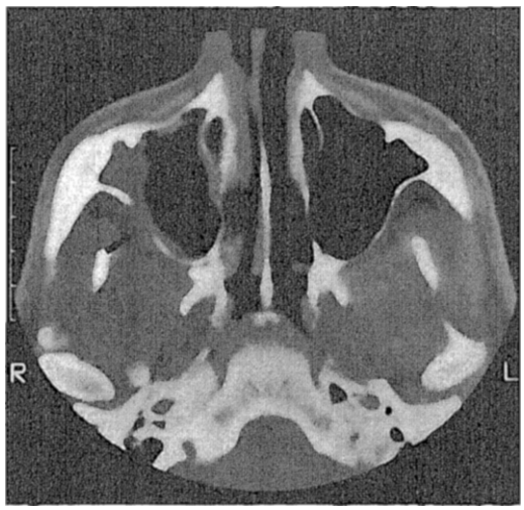

Figure 4. Axial CT image demonstrates same findings of maxillary wall erosion with disease extension into right infratemporal fossa (Case 2).

illary sinus disease with orbital invasion. The patient refused further surgery, was managed symptomatically, and died 4 months postoperatively of probable central nervous system toxoplasmosis.

Case 3. A 36-year-old HIV-infected male with a history of metastatic Kaposi's sarcoma was admitted in August, 1995, with left retroorbital and frontal headaches, fever, and rhinorrhea recalcitrant to conventional medical management as an outpatient. His CD4 lymphocyte count was 0 cells per mm^3, and his ANC was 1000 cells per mm^3. A MR imaging scan performed the previous week revealed mild left ethmoid sinusitis without intracranial pathology. A CT scan showed partial left posterior ethmoid opacification without bony erosion. All other paranasal sinuses were disease-free except for minimal mucosal thickening along the roof of the left maxillary sinus (Fig. 5). Closer examination of both the coronal and axial CT views suggested extension of the posterior ethmoid disease into the pterygopalatine fossa and pterygomaxillary space (Figs. 6 and 7). During a left endoscopic ethmoidectomy and maxillary antrostomy, polypoid mucosa was noted in the ethmoids and along the roof of the maxillary sinus. A single posterior ethmoid cell contained caseous material, and on histologic examination fungal invasion by an organism consistent with *Aspergillus* was identified in the ethmoid cell. *Aspergillus fumigatus* was subsequently cultured. Intravenous amphotericin B was begun and a left Caldwell-Luc procedure performed. Soft bone was curetted from the superior and posterior walls of the antrum. The periorbita was uninvolved. Although the sinus symptoms improved following surgery, he expired 1 month later because of multisystem failure unrelated to his invasive fungal infection.

Case 4. A 34-year-old HIV-infected woman with a history of cytomegalovirus (CMV) retinitis, *Pneumocystis carinii* pneumonia, and renal tubular acidosis was admitted in April, 1997, with a 3-day history of diplopia and decreasing visual acuity in the left eye. The patient denied fever or pain. Physical examination was significant for proptosis and opthalmoplegia of the left eye. A MR imaging scan demonstrated abnormal enhancement of the left lamina papyracea, orbital apex,

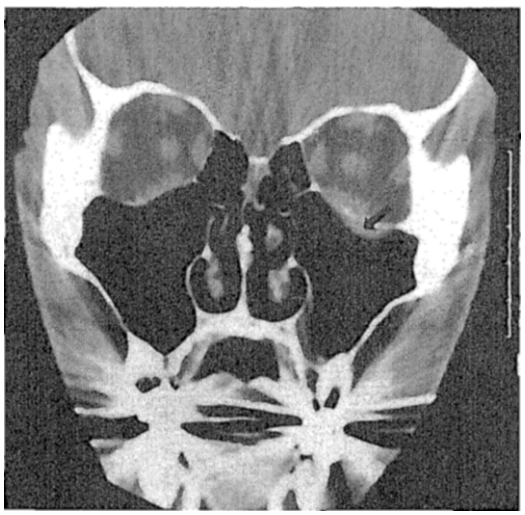

Figure 5. Coronal CT image reveals mucosal thickening limited to the roof of the left maxillary sinus at this plane. This proved to be a site of bony involvement by fungus (Case 3).

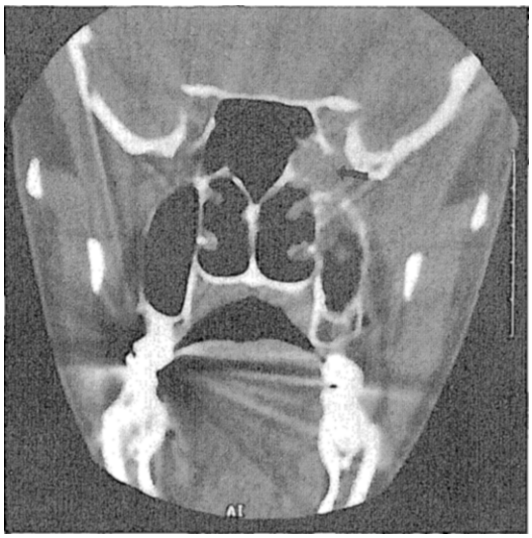

Figure 6. Coronal CT image reveals sinus involvement limited to a single posterior ethmoid cell. Note the *arrow* shows the presence of left pterygopalatine fossa invasion (Case 3).

and cavernous sinus without thrombosis. Her CD4 lymphocyte count was 4 cells per mm^3 and her ANC was 900 cells per mm^3. A CT scan revealed bilateral sphenoid sinus air-fluid levels, left ethmoid air-fluid level, and increased soft tissue density in the region of the orbital apex. Closer review identified a small defect in

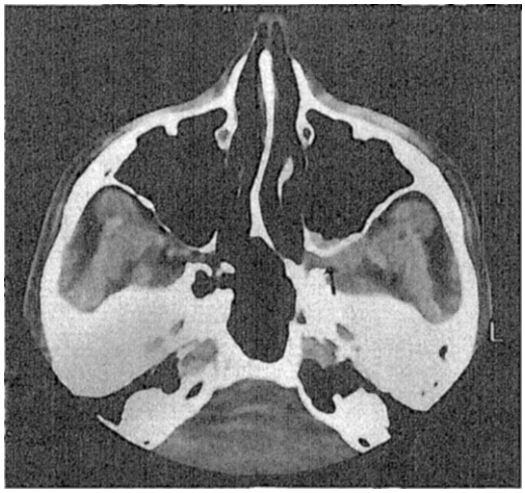

Figure 7. Axial CT image supports left pterygopalatine fossa involvement extending either from posterior ethmoid cell as described in Figure 6 or from limited mucosal thickening along the high posterior maxillary sinus wall (*arrow*) (Case 3).

the sphenoid sinus and bony erosion of the sphenoid intersinus septum (Figs. 8 and 9).

The patient underwent bilateral endoscopic sphenoidotomies, left endoscopic ethmoidectomy, and left partial middle turbinectomy. The posterior sphenoid wall was notably necrotic and mobile. Cultures subsequently grew *A fumigatus* species. Intravenous amphotericin B therapy was initiated immediately. The patient experienced improved visual acuity and decreasing proptosis in the immediate postoperative period; however, she developed multisystem failure and died 8 days following surgery.

DISCUSSION

The clinical presentation of fungal infections of the paranasal sinuses can present in a variety of ways and often depends on the host immune status. Both noninvasive and invasive forms have been recognized. With noninvasive fungal sinusitis, fungal elements are confined to the lumen of the sinus cavity. These noninvasive forms usually affect otherwise healthy individuals and include the classic fungal ball (mycetoma) and allergic fungal sinusitis as described by Katzenstein.[12] Although invasive fungal sinusitis has been described in normal hosts,[11,14,24,32,37] it is usually seen in immunocompromised patients and can present as either an indolent or a fulminant process. As the population of immunocompromised patients grows because of organ transplantations, the use of aggressive chemotherapy, and the increased prevalence of AIDS, invasive fungal sinusitis has become an increasingly common clinical problem.

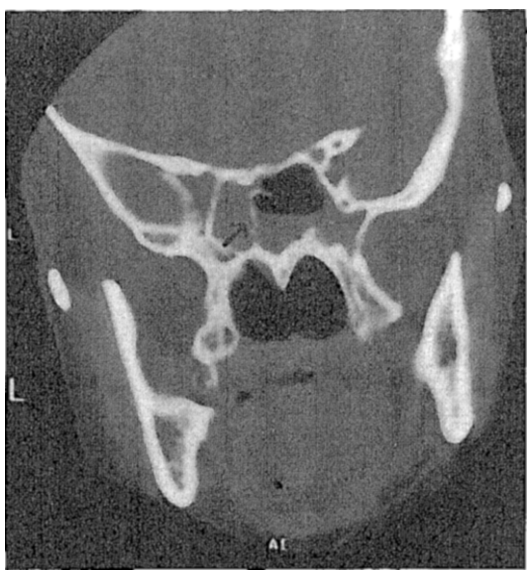

Figure 8. Coronal CT image revealing subtle evidence of erosion of right sphenoid sinusitis through the intersinus septum to involve the left sphenoid sinus (Case 4).

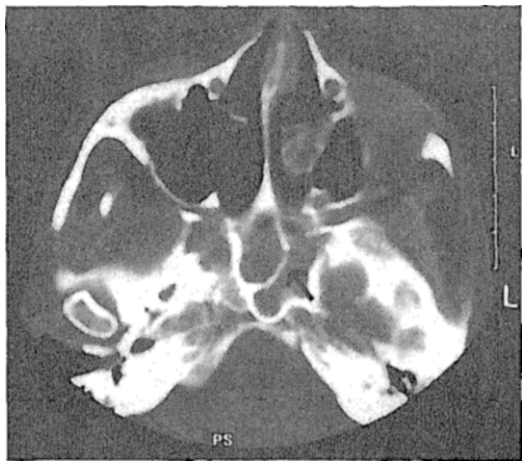

Figure 9. Axial CT image demonstrates less obvious evidence of erosion through sphenoid intersinus septum at this level (Case 4).

The most common fungal pathogen of the paranasal sinuses is *Aspergillus*. This organism is a ubiquitous, spore-forming fungus found in soil, water, and decaying vegetable matter, and colonizes sinuses by being inhaled by the host in the spore (conidia) form. When invasive, *Aspergillus* exhibits a predilection for arterial invasion, thrombosis, and subsequent ischemic necrosis of tissue.[6] *A fumigatus* is the most frequently isolated species in both invasive and noninvasive sinusitis in immunocompetent as well as in immunocompromised patients.[3,15,19,22,37] Other pathogens include those belonging to the family Mucoraceae, including the genera *Mucor, Rhizopus,* and *Absidia.*[20] Table 1 lists the fungal organisms responsible for invasive sinusitis for the four AIDS patients presented in this report, as well as those reported in the literature. *A fumigatus* appears to be the predominant fungal organism causing sinusitis in the AIDS population as well. Case 2 represents the first reported case of invasive sinusitis caused by *P variotii.* Previously, *Paecilomyces lilacinus* was noted to cause invasive sinusitis in a leukemia patient,[8] and *P variotii* has been found to cause noninvasive sinusitis.[34]

Infection by HIV causes selective depletion of CD4 (T helper) lymphocytes. Although impaired cellular immunity predisposes to fungal and intracellular bacterial infections, phagocytic polymorphonuclear cells and macrophages are the primary defenses against fungal infection, killing the mycelial and conidial forms of the organism respectively.[28] AIDS patients demonstrate both neutrophil and macrophage dysfunction.[30] Minamoto et al[18] cite neutropenia as the single greatest factor predisposing to the development of invasive *Aspergillus* in patients with AIDS. Meyer et al, in a recent review, noted that fungal sinusitis was associated with advanced AIDS and low CD4 cell counts.[16] These conditions seem to play a

role in the four cases of invasive fungal sinusitis presented here. Table 1 reviews clinical data from these four cases and existing cases in the literature. When reported, the CD4 cell counts are notably depressed, supporting this notion. The ANCs from cases in the literature inconsistently support this theory, with only three of nine (33%) of the available figures considered neutropenic (ANC less than 1000 cells/mm^3). This is in contradistinction to the authors' series where three of four (75%) patients were neutropenic. The relationship between laboratory draws and the dosing schedule of granulocyte colony stimulating factor (GCSF), a medication of prevalent use in the AIDS population, was not investigated and may affect ANC figures.

The symptoms of chronic noninvasive fungal sinusitis are often indistinguishable from those of chronic bacterial sinusitis. Invasive fungal sinusitis, however, usually produces symptoms related to the site of invasion. In three of the authors' patients, the most notable symptom was excruciating facial pain or headache, often out of proportion to findings on physical examination or radiographic studies. Disproportionate and unrelenting pain in an immunocompromised host may be the only clue as to the presence of invasive fungal sinusitis. Although the radiographic findings in Case 3 were more obscure than for Cases 1 and 2, by this time heightened suspicion facilitated an early (preoperative) diagnosis so that appropriate therapy could be initiated. Although subtle, the radiographic findings of Case 4 were interpreted preoperatively as consistent with bony erosion. Despite immediate intervention, the patient succumbed to advanced disease.

Radiographic imaging is paramount to the early diagnosis of invasive fungal sinusitis. Although MR imaging may be superior to CT scan for detecting fungal mycetomas[39] or extra-sinus extension, CT scan remains the imaging modality of choice for evaluating bony architecture and may reveal thick, sclerotic bone in the setting of chronic infection or focal erosion with an invasive process. Silverman and Mancuso concluded that infiltration of the periantral fat plane on CT or MR imaging may signal the earliest radiologic sign of invasive fungal infection.[31] Cases 1 and 2 certainly support this observation. In all four cases presented here, subtle bony erosion or extra-sinus extension was present on the initial scans; however, two of the four cases were identified only after the diagnosis of invasion had been established. Careful scrutiny of the CT scans is a vital component of establishing this diagnosis.

The appropriate treatment of invasive fungal sinusitis in the AIDS population is unclear. Treatment of such infections historically has aimed to: (1) aggressively correct any predisposing underlying condition; (2) surgically debride affected tissue; and (3) institute antifungal medication. Intravenous amphotericin B to a total dose of 2 grams seems to be the appropriate medical component. Weber and Lopez-Bernstein, however, showed improved response (including cures) with reduced drug toxicity using liposomal amphotericin B in individuals with hematologic malignancies and invasive *Aspergillus* sinusitis unresponsive to conventional amphotericin B therapy.[38] Geissmann et al propose prolonged GCSF use

(300 mg every other day) in conjunction with amphotericin B as helpful in the medical management of these patients.[7] *Pseudallescheria boydii* has been found to be amphotericin B resistant in many cases, but sensitive to azoles such as itraconazole. Thus, it is important to obtain a definitive diagnosis of this pathogen. Culture is the best method because histologic appearance of the hyphae is very similar to *Aspergillus*.[9,16] Endoscopic sinus surgery can play a role in the initial diagnosis, but may limit the ability to adequately remove affected tissue. Open surgical approaches facilitate thorough debridement of devitalized tissue, and in the authors' cases provided symptomatic improvement in three of four patients.

Treatment outcomes of immunocompromised patients suffering from invasive bronchopulmonary *Aspergillosis* improve when infection is diagnosed and treated early.[1,17,27] Whether this relationship holds true for patients with AIDS and invasive fungal sinusitis is yet to be determined. As noted by Meyer et al,[17] the most important single factor in the ultimate outcome of *Aspergillus* infection is the reversal of the underlying immunocompromising condition. As promising new drugs are developed to combat AIDS, there is a tempered enthusiasm among AIDS specialists that this ultimately fatal disease may be soon transformed into a manageable chronic condition. There continues to be a role for aggressive diagnosis, medical management, and surgical therapy of invasive fungal sinusitis in this immunocompromised population to provide palliation as well as potential irradication of disease.

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