rounded by edema and exhibit T1-weighted enhancement after intravenous Gd (Cox et al., 1992). In later stages, central necrosis and rim enhancement are the primary findings (Osborn, 1994). Sino-nasal aspergillosis may demonstrate soft tissue masses or abscesses in the nasal cavities or paranasal sinuses, from where there may be extension into the brain or orbits via the cribriform plate, sometimes associated with bone destruction or cavernous sinus thrombosis (Bowen and Post, 1991). Visceral dissemination to the liver, spleen, and kidneys is associated with solid nodular masses, abscesses, or infarcts.

Airway IPA

The less common form of invasive aspergillosis, airway IPA, most often occurs in patients with lesser degrees of neutrophil dysfunction than those in the aforementioned two groups. Many of these patients have evidence of prior airway injury, such as occurs at bronchial anastomoses in lung transplant recipients and along inflamed or infected bronchi after other infections.

Histopathology

Unlike angio-invasive aspergillosis, airway IPA develops axially in the small bronchi and bronchioles, where there is histopathological evidence of deep mycelial invasion to basement membranes (Logan et al., 1994). Among lung transplant recipients, *Aspergillus* spp. can cause airway colonization, tracheobronchitis, and invasive pneumonia. The tracheobronchitic form is common in lung transplant recipients and in patients with AIDS; the spectrum ranges from isolated simple bronchitis to pseudomembranous tracheobronchial aspergillosis (Kramer et al., 1991). In lung transplant recipients, isolated tracheobronchitis often involves the anastomotic site and tends to respond well to antifungal therapy and/or surgical debridement. In a minority of patients this can lead to dissemination. Among lung transplant recipients, only a very small fraction of patients progress from airway colonization to invasive disease, but among those who do so, fewer than half survive the infection (Mehrad et al., 2001).

Imaging

Spread of infection along the airways has the CT appearance of bronchial wall thickening, centrilobular opacities, and thickened and fluid-distended segments of small branching bronchi and bronchioli, i.e., tree-in-bud opacities and/or patches of peribronchial consolidation (Logan et al., 1994) (Fig. 5). While small-airways findings are uncommon in angio-invasive aspergillosis, they

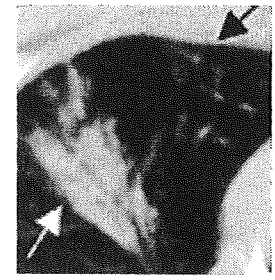

Figure 5. Peribronchovascular CT opacities in airway IPA. The CT image demonstrates a peribronchovascular opacity highlighting branching air bronchograms. There are also small branching bronchiolar opacities in the unconsolidated lung (arrows).

are the rule in airway-invasive aspergillosis, where the invasion is dominated by airway imaging findings rather than by macronodules. These findings are nonspecific because they are found in a wide variety of common community-acquired infections and in mycobacterial, cryptococcal, and aspiration pneumonias, all of which are not uncommon in lung transplant recipients and/or AIDS patients.

In the lung transplant recipient, isolated tracheobronchitis may be seen as a localized nodule or deformity at a bronchial anastomosis. CT findings of tracheobronchitis may also include bronchial wall thickening, bronchial nodularity, bronchostenosis, bronchiectasis, peribronchial consolidation, and atelectasis.

CHRONIC PULMONARY ASPERGILLOSIS

Chronic IPA (CPA) is an incompletely understood, indolent but progressive *Aspergillus* infection that occurs in preformed or new cysts (or cavities) (Denning et al., 2003; Gefter, 1992). It tends to occur in elderly and/or debilitated patients who might not be otherwise immunodeficient. Progressive constitutional symptoms are characteristic. The underlying chronic cavitary lung disease may be due to prior tuberculosis, bullous lung disease, chronic interstitial lung disease, lung irradiation, surgical lung resection, lung infarction, or cystic fibrosis (Graham-Clarke et al., 1994). End-stage sarcoidosis is a common cause of the cysts associated with CPA. CPA has overlapping characteristics with one or more of the other categories of aspergillosis from which it should be distinguished (Greene, 1981; Soubani and Chandrasekar, 2002).

Histopathology

There is pathologic evidence of local lung tissue invasion, but dissemination does not usually occur (Color

Plates 17A and B). Although the infection tends to remain local, it is resistant to eradication and usually requires long-term anti-*Aspergillus* therapy. Some view CPA as a progression from noninvasive aspergillosis to a low-grade invasive form of aspergillosis when depressed host defense mechanisms facilitate the infection. The cyst wall is usually eroded and invaded by the hyphae. There is often evidence of both acute and chronic inflammation in association with fibrosis and necrosis to various degrees.

Radiology

The characteristic imaging findings of CPA include further enlargement of preexisting cysts, the development of new lung cysts, the appearance of pericystic lung opacities, and the development of new or progressive local pleural thickening. About half of these patients have evidence of saprophytic aspergillomas within the cysts. The findings may simulate activation of post-primary tuberculosis and lung cancer (Fig. 6).

Chronic necrotizing pulmonary aspergillosis (CNPA) is an aggressive subcategory of CPA that has a more rapid course that is sometimes referred to as semi-invasive or subacute pulmonary aspergillosis (Binder et al., 1982; Denning et al., 2003; Yousem, 1997). CNPA can be recognized by the development of new progressive consolidative lung opacities that undergo de novo cyst and cavity formation. The new cysts can later become the host site for an aspergilloma. Alternatively, the cysts may develop thin walls that rapidly expand (Denning et al., 2003). CNPA may demonstrate locally invasive disease wherein mycelia may be identified invading the pleural space. Local blood vessel invasion can lead to hemoptysis. *A. fumigatus* may be recovered from the pleural space when there are bronchopleural fistula and empyema (Denning et al., 2003).

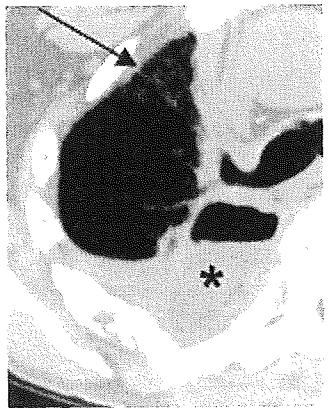

Figure 6. Illustration of CPA. A liquid-filled preexisting cavity (*) from which *Aspergillus* sp. was consistently recovered is shown. Pericystic lung opacities and new pleural thickening developed in the right upper lobe (arrows).

The main challenge in differential diagnosis is to distinguish CPA from serious but unsuspected chronic conditions that require other specific therapy. These include conditions such as upper lobe lung cancers and chronic cavitary lung infections caused by *M. tuberculosis*, nontuberculous mycobacteria, or endemic fungi. A definite diagnosis of CPA often requires biopsy proof to establish that there is local invasion by *Aspergillus* and to exclude tumor or other pulmonary infection. CPA differs from a simple aspergilloma, not only by the presence of associated constitutional symptoms but also by the development of persistent pericystic lung nodules, consolidations, or ground-glass opacities and by the development and/or progression of cavitary disease or pericystic pleural thickening. In simple aspergilloma, the development of cyst enlargement, pericystic lung opacities, and/or pericystic pleural thickening should be regarded as evidence of complicated aspergillosis consistent with CPA. Increased cavity wall thickness alone does not correlate with disease activity (Denning et al., 2003).

ABPA

Allergic bronchopulmonary aspergillosis (ABPA) is the archetype of allergic aspergillosis. The clinical constellation of ABPA consists of chronic asthma, mucus production, and elevated serum antigen levels of *A. fumigatus*. Importantly, the asthmatic component and continuing lung damage are usually responsive to corticosteroid therapy.

Histopathology

ABPA is not an infection but a damaging immune reaction caused by *A. fumigatus* colonization in large and small airways of atopic, immunocompetent patients, including some who also have cystic fibrosis (Shibuya et al., 1999a). Cytological examination of bronchoalveolar lavage fluids can demonstrate hyphae with dichotomous bifurcations in combination with numerous mucin-containing airway eosinophils. Histological studies of mucosal biopsies show evidence of allergic bronchitis without signs of fungal invasion (Color Plates 18A and B).

Radiology

Local hypersensitivity damage tends to affect the large central airways (segmental and subsegmental bronchi), small airways, and the adjacent lung, principally in the upper lobes.

Large airway damage consists mainly of severe bronchiectasis of the segmental and subsegmental bronchi. On chest radiographs large airway bronchiectasis and impaction can take on a "finger-in-glove" appearance due to central bronchiectasis emanating from the pulmonary hila (Color Plates 19A and B). On CT, the damage is seen directly as bronchiectasis filled with mucoid material. The CT attenuation of the mucoid impaction is normally low relative to soft tissue attenuation, but occasionally it is of high attenuation (Agarwal et al., 2007). High attenuation mucus does not seem to correlate with a decreased chance for complete remission. Small-airway damage causes bronchiolar dilatation and impaction that on CT is seen as centrilobular nodules and/or branching tree-in-bud opacities.

Associated damage to the adjacent peripheral lung is evident on CT as transient, migratory patches of consolidation and/or ground-glass opacities that regress spontaneously or after corticosteroid therapy. Other imaging findings include regional and overall lung hyperinflation or atelectasis distal to airway obstructions.

Differential Diagnosis

Corticosteroid-responsive ABPA-related asthma needs to be differentiated from simple chronic asthma. Three main imaging features help to distinguish these two groups of patients (Ward et al., 1999). First, varicose or cystic bronchiectasis of segmental and subsegmental bronchi is present in >90% of patients with ABPA but in only <30% of patients with simple asthma. Second, mucoid impaction of segmental and subsegmental airways occurs in 67% of patients with ABPA but only 4% of those with simple asthma. Third, small-airways abnormalities in the form of centrilobular nodules occur in 93% of patients with ABPA but in only 28% of patients with simple asthma (Ward et al., 1999). When patients with simple chronic asthma have bronchiectasis, it is usually of a mild variety and limited to the cylindrical type, i.e., bronchiectasis with parallel nontapering walls rather than with frank dilatation. Although the imaging features of ABPA described above are highly discriminatory, they do not identify ABPA before airway damage has occurred. In an effort to identify ABPA at an earlier stage, screening CT has been advocated for asthmatics with skin prick hypersensitivity to *A. fumigatus* (Eaton et al., 2000).

ASPERGILLOMA

Simple aspergilloma, or fungus ball, is the most common form of aspergillosis that is detected with imaging studies. It is the archetype of saprophytic (non-invasive) aspergillosis. It results from saprophytic proliferation of *Aspergillus* mycelia within a preformed host lung cyst (or cavity). The patient is generally asymptomatic and immunocompetent (Soubani and Chandrasekar, 2002). As mentioned above, the preformed cysts in which aspergillomas form can be caused by a wide variety of lung diseases. The four common etiologies of the chronic cysts of aspergillosis include post-primary tuberculosis (Glimp and Bayer, 1983; British Thoracic and Tuberculosis Association, 1970), end-stage sarcoidosis (Israel and Ostrow, 1969), bullous emphysema, and bronchiectatic cavities (Solit et al., 1971), such as those found in cystic fibrosis (Ryan et al., 1995) and ABPA. Aspergillomas have also been reported in immunocompetent patients who did not appear to have had a preexisting dilated lung space (Kang et al., 2002).

Histopathology

Mycelial invasion of the lung or vasculature is not a feature of aspergillomas (Rafferty et al., 1983; Tomee et al., 1995). The *Aspergillus* hyphae in the fungus ball are compactly aligned in a radial pattern, and the wall of the cyst is usually eroded or covered with metaplastic respiratory epithelium. A chronic inflammatory infiltrate is present in the wall, but no hyphal invasion occurs when the patient is immunocompetent (Fig. 7A and B).

Radiology

Initial detection is usually made during an incidental chest radiograph, often for evaluation of the underlying disease responsible for the cyst or for some unrelated reason (Glimp and Bayer, 1983). The imaging diagnosis is based on finding a well-defined mycelial

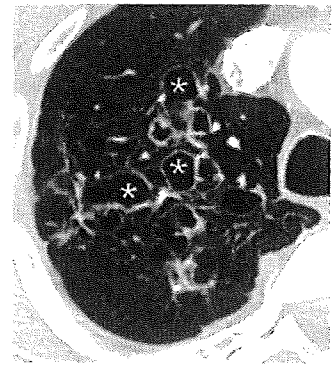

Figure 7. Imaging of ABPA. CT scan in an asthmatic patient with ABPA. There are multiple branching cystic spaces (*) characteristic of central bronchiectasis.

mass in a preformed cyst, most often solitary and in an upper lobe (Glimp and Bayer, 1983; Soubani and Chandrasekar, 2002) (Fig. 8). On CT, air may be visible within the interstices of the hyphal mass. A crescent-shaped cap of air separating the aspergilloma from the cyst wall can usually be identified (Tuncel, 1984). The fungus ball can often, but not always, be shown to move within the cyst on shifting body position from left to right or from prone to supine (Roberts et al., 1987; Soubani and Chandrasekar, 2002). Spontaneous shrinkage or even disappearance can occur in a small fraction of aspergillomas (Gefter, 1992). Enlargement of fungus balls is considered to be rare (British Tuberculosis Association, 1968). The host cysts are generally smooth and thin but may be irregular and thick. The pleural surface overlying the host cyst may be thin or thick, but it does not usually thicken over time unless or until it becomes complicated. Dystrophic, cage-like calcification over the surface of an aspergilloma can occasionally be detected. In a minority of patients, such as those with end-stage sarcoidosis, the aspergilloma may be brought to attention by potentially life-threatening hemoptysis (Glimp and Bayer, 1983; Greenberg et al., 2002). In such patients CT angiography is usually used to search for the source of the hemoptysis, i.e., hypertrophic bronchial arteries supplying the cyst wall. Occlusive metallic coils can then be placed in the dilated bronchial arteries to help control the bleeding. Bleeding may cause CT hyperdense fluid within the cyst or spread into the lung, causing ground-glass or consolidative opacities with air bronchograms.

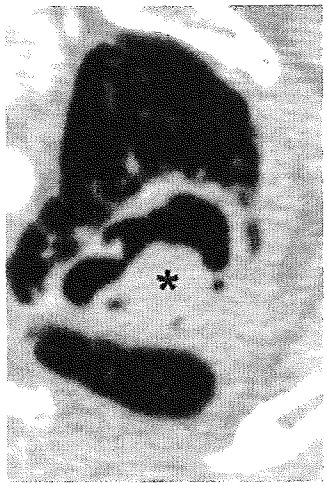

Figure 8. Image of an aspergilloma. A CT scan section demonstrates a fungus ball (*) residing within a preexisting cavity of an asymptomatic patient. The configuration simulates the air crescent sign of angio-invasive aspergillosis.

Differential Diagnosis

Definitive diagnosis requires clinical corroboration based on positive serology precipitins to *A. fumigatus* and/or recovery of *A. fumigatus* in sputum, bronchoalveolar lavage washings, or percutaneous needle aspiration biopsy material. Precipitins to *A. fumigatus* may be negative when the fungus ball is caused by *Aspergillus* species other than *A. fumigatus* or other molds, such as *Zygomycetes* or *Fusaria* (Tomee et al., 1995). The main clinical imaging challenge is to differentiate an aspergilloma (which would not usually require treatment) from other more serious lung conditions (which usually do need specific treatment). The latter includes the late phase of acute angio-invasive aspergillosis (which occurs in a totally different clinical setting) and chronic semi-invasive aspergillosis (patients with chronic, indolent symptomatology). The aspergilloma needs also to be differentiated from other cavitary lung diseases, such as those caused by lung cancer, lung abscess, and cavitary granulomatous vasculitis, such as Wegener's granulomatosis. Lung biopsy and other clinical information may be needed for a definite diagnosis. Predictors of poor prognosis for the saprophytic aspergillomas include an increase in the size or number of cysts (a finding not compatible with the diagnosis of simple aspergilloma), severe underlying lung disease, immunosuppressive therapy, AIDS, sarcoidosis, rising *Aspergillus*-specific immunoglobulin G titer, and repetitive severe hemoptysis (Stevens et al., 2000).

AIDS patients require special attention because they are at high risk to develop aspergillomas and because aspergillomas in these patients tend to develop into CPA. AIDS patients have a high prevalence of abnormally dilated host cysts because of frequent infections by *Pneumocystis jirovicii, M. tuberculosis, Mycobacterium avium intracellulare*, other bacteria, and other fungi. Aspergillomas in AIDS patients, especially those with CD4 counts of <100 cells/liter, are often discovered because of symptoms related to the aspergilloma, such as hemoptysis (Addrizzo-Harris et al., 1997), cough, or fever. Obstructing bronchopulmonary aspergillosis, a unique noninvasive, saprophytic variety of aspergillosis characteristic of AIDS, is diagnosed by massive central bronchial dilatation and mucoid impaction due to noninvasive endo-bronchial fungal overgrowth (Franquet et al., 2001).

CONCLUSION

A. fumigatus causes a wide variety of pulmonary diseases that result in significant morbidity and mortality. The diseases are of particular importance to the clinician because their unique characteristics make them

detectable through imaging even when microbiological studies are not definitive. The clinical milieu helps identify the prior probability of such disease while the histopathology is predictive of the imaging findings. When evaluated in an appropriate clinical context, imaging is likely to make the disease specifically recognizable at a time when successful treatment is possible.

REFERENCES

Addrizzo-Harris, D., T. Harkin, G. McGuinness, D. Naidich, and W. Rom. 1997. Pulmonary aspergilloma and AIDS. A comparison of HIV-infected and HIV-negative individuals. *Chest* 111:612–618.

Agarwal, R., D. Gupta, A. N. Aggarwal, A. K. Saxena, A. Chakrabarti, and S. K. Jindal. 2007. Clinical significance of hyperattenuating mucoid impaction in allergic bronchopulmonary aspergillosis: an analysis of 155 patients. *Chest* 132:1183–1190.

Aquino, S. L., S. T. Kee, M. L. Warnock, and G. Gamsu. 1994. Pulmonary aspergillosis: imaging findings with pathologic correlation. *Am. J. Roentgenol.* 163:811–815.

Armstrong, D., L. S. Young, R. D. Meyer, and A. H. Blevins. 1971. Infectious complications of neoplastic disease. *Med. Clin. North Am.* 55:729–745.

Binder, R. E., L. J. Faling, R. D. Pugatch, C. Mahasaen, and G. L. Snider. 1982. Chronic necrotizing pulmonary aspergillosis: a discrete clinical entity. *Medicine* 61:109–124.

Blum, U., M. Windfuhr, C. Buitrago-Tellez, G. Sigmund, E. W. Herbst, and M. Langer. 1994. Invasive pulmonary aspergillosis. MRI, CT, and plain radiographic findings and their contribution for early diagnosis. *Chest* 106:1156–1161.

Bowen, B. C., and M. J. D. Post. 1991. Intracranial infection, p. 501–538. *In* S. W. Atlas (ed.), *Magnetic Resonance Imaging of the Brain and Spine.* Raven Press, New York, NY.

British Thoracic and Tuberculosis Association, Research Committee. 1970. Aspergillosis and residual tuberculous cavities: the results of a resurvey. *Tubercle* 51:227–245.

British Tuberculosis Association. 1968. *Aspergillus* in persistent lung cavities after tuberculosis: a report from the Research Committee of the British Tuberculosis Association. *Tubercle* 49:1–11.

Caillot, D., O. Casasnovas, A. Bernard, J. F. Couaillier, C. Durand, B. Cuisenier, E. Solary, F. Piard, T. Petrella, A. Bonnin, G. Couillault, M. Dumas, and H. Guy. 1997. Improved management of invasive pulmonary aspergillosis in neutropenic patients using early thoracic computed tomographic scan and surgery. *J. Clin. Oncol.* 15:139–147.

Caillot, D., J. F. Couaillier, A. Bernard, O. Casasnovas, D. W. Denning, L. Mannone, J. Lopez, G. Couillault, F. Piard, O. Vagner, and H. Guy. 2001. Increasing volume and changing characteristics of invasive pulmonary aspergillosis on sequential thoracic computed tomography scans in patients with neutropenia. *J. Clin. Oncol.* 19: 253–259.

Cox, J., F. R. Murtagh, A. Wilfong, and J. Brenner. 1992. Cerebral aspergillosis: MR imaging and histopathologic correlation. *Am. J. Neuroradiol.* 13:1489–1492.

Curtis, A. M., G. F. Smith, and C. E. Ravin. 1979. Air crescent sign of invasive aspergillosis. *Radiology* 133:17–21.

Denning, D. W., K. Riniotis, R. Dobrashian, and H. Sambatakou. 2003. Chronic cavitary and fibrosing pulmonary and pleural aspergillosis: case series, proposed nomenclature and review. *Clin. Infect. Dis.* 37:S265–S280.

Eaton, T., J. Garrett, D. Milne, A. Frankel, and A. U. Wells. 2000. Allergic bronchopulmonary aspergillosis in the asthma clinic: a prospective evaluation of CT in the diagnostic algorithm. *Chest* 118: 66–72.

Franquet, T., N. L. Müller, A. Giménez, P. Guembe, J. de la Torre, and S. Bagué. 2001. Spectrum of pulmonary aspergillosis: histologic, clinical, and radiologic findings. *Radiographics* 21:825–837.

Gaeta, M., A. Blandino, E. Scribano, F. Minutoli, S. Volta, and J. Pandolfo. 1999. Computed tomography halo sign in pulmonary nodules: frequency and diagnostic value. *J. Thorac. Imaging* 14: 109–113.

Gefter, W. B. 1992. The spectrum of pulmonary aspergillosis. *J. Thorac. Imaging* 7:56–74.

Gefter, W. B., S. M. Albelda, G. H. Talbot, S. L. Gerson, P. A. Cassileth, and W. T. Miller. 1985. Invasive pulmonary aspergillosis and acute leukemia. Limitations in the diagnostic utility of the air crescent sign. *Radiology* 157:605–610.

Glimp, R., and A. Bayer. 1983. Pulmonary aspergilloma: diagnostic and therapeutic considerations. *Arch. Intern. Med.* 143:303–308.

Godwin, J. D., W. R. Webb, C. J. Savoca, G. Gamsu, and P. C. Goodman. 1980. Multiple, thin-walled cystic lesions of the lung. *Am. J. Roentgenol.* 135:593–604.

Grahame-Clarke, C. N., C. M. Roberts, and D. W. Empey. 1994. Chronic necrotizing pulmonary aspergillosis and pulmonary phycomycosis in cystic fibrosis. *Respir. Med.* 88:465–468.

Greenberg, A. K., J. Knapp, W. N. Rom, and D. J. Addrizzo-Harris. 2002. Clinical presentation of pulmonary mycetoma in HIV-infected patients. *Chest* 122:886–892.

Greene, R. 1981. The pulmonary aspergilloses: three distinct entities or a spectrum of disease? *Radiology* 140:527–530.

Greene, R. E., H. T. Schlamm, J. W. Oestmann, P. Stark, C. Durand, O. Lartholary, J. R. Wingard, R. Herbrecht, P. Ribaud, T. F. Patterson, P. F. Troke, D. W. Denning, J. E. Bennett, B. E. de Pauw, and R. H. Rubin. 2007. Imaging findings in acute invasive pulmonary aspergillosis: clinical significance of the halo sign. *Clin. Infect. Dis.* 44:373–379.

Herbrecht, R., D. W. Denning, T. F. Patterson, J. E. Bennett, R. E. Greene, J. W. Oestmann, W. V. Kern, K. A. Marr, P. Ribaud, O. Lortholary, R. Sylvester, R. H. Rubin, J. R. Wingard, P. Stark, C. Durand, D. Caillot, E. Thiel, P. H. Chandrasekar, M. R. Hodges, H. T. Schlamm, P. F. Troke, B. de Pauw, et al. 2002. Voriconazole versus amphotericin B for primary therapy of invasive aspergillosis. *N. Engl. J. Med.* 347:408–415.

Huang, S. N., and L. S. Harris. 1963. Acute disseminated penicilliosis: report of a case and review of pertinent literature. *Am. J. Clin. Pathol.* 39:167–174.

Israel, H. L., and A. Ostrow. 1969. Sarcoidosis and aspergilloma. *Am. J. Med.* 47:243–250.

Kang, E.-Y., D. H. Kim, O. H. Woo, J.-A. Choi, Y.-W. Oh, and K. H. Kim. 2002. Pulmonary aspergillosis in immunocompetent hosts without underlying lesions of the lung: radiologic and pathologic findings. *Am. J. Roentgenol.* 178:1395–1399.

Kim, Y., K. S. Lee, K. J. Jung, J. Han, J. S. Kim, and J. S. Suh. 1999. Halo sign on high resolution CT: findings in spectrum of pulmonary diseases with pathologic correlation. *J. Comput. Assist. Tomogr.* 23: 622–626.

Kramer, M. R., D. W. Denning, S. E. Marshall, D. J. Ross, G. Berry, N. J. Lewiston, D. A. Stevens, and J. Theodore. 1991. Ulcerative tracheobronchitis after lung transplantation. A new form of invasive aspergillosis. *Am. Rev. Respir. Dis.* 144:552–556.

Logan, P. M., S. L. Primack, R. R. Miller, and N. L. Muller. 1994. Invasive aspergillosis of the airways: radiographic, CT, and pathologic findings. *Radiology* 193:383–388.

Mehrad, B., G. Paciocco, F. J. Martinez, T. C. Ojo, M. D. Iannettoni, and J. P. Lynch III. 2001. Spectrum of *Aspergillus* infection in lung transplant recipients: case series and review of the literature. *Chest* 119:169–175.

Osborn, A. G. 1994. *Diagnostic Neuroradiology*. Mosby, St. Louis, MO, p. 706–709.

Primack, S. L., T. E. Hartman, K. S. Lee, and N. L. Muller. 1994. Pulmonary nodules and the CT halo sign. *Radiology* 190:513–515.

Rafferty, P., B. A. Biggs, G. K. Crompton, and I. W. Grant. 1983. What happens to patients with pulmonary aspergilloma? Analysis of 23 cases. *Thorax* 38:579–583.

Roberts, C. M., K. M. Citron, and B. Strickland. 1987. Intrathoracic aspergilloma: role of CT in diagnosis and treatment. *Radiology* 165: 123–128.

Ryan, P., D. Stableforth, J. Reynolds, and K. Muhdi. 1995. Treatment of pulmonary aspergilloma in cystic fibrosis by percutaneous instillation of amphotericin B via indwelling catheter. *Thorax* 50:809–810.

Ryu, J. H., and S. J. Swensen. 2003. Cystic and cavitary lung diseases: focal and diffuse. *Mayo Clin. Proc.* 78:744–752.

Saul, S. H., T. Khachatoorian, A. Poorsattar, R. L. Myerowitz, S. J. Geyer, A. W. Pasculle, and M. Ho. 1981. Opportunistic *Trichosporon* pneumonia. Association with invasive aspergillosis. *Arch. Pathol. Lab. Med.* 105:456–459.

Shibuya, K. 1999a. Animal models of *A. fumigatus* infections, p. 130–138. *In* A. A. Brakhage, B. Jahn, and A. Schmidt (ed.), *Aspergillus fumigatus. Contribution to Microbiology*, vol. 2. Karger, Basel, Switzerland.

Shibuya, K. 1999b. Histopathology of experimental invasive pulmonary aspergillosis in rats: pathological comparison of pulmonary lesions induced by specific virulent factor deficient mutants. *Microb. Pathog.* 27:123–131.

Shibuya, K., T. Ando, M. Wakayama, M. Takaoka, K. Uchida, and S. Naoe. 1997. Pathological spectrum of invasive pulmonary aspergillosis: study of pulmonary lesions of 54 autopsies and the relationship between neutrophilic response and histologic features of lesions in experimental aspergillosis. *Jpn. J. Med. Mycol.* 38:175–181.

Shibuya, K., S. Paris, T. Ando, H. Nakayama, T. Hatori, and J.-P. Latgé. 2006. Catalases of *Aspergillus fumigatus* and inflammation in aspergillosis. *Jpn. J. Med. Mycol.* 47:249–255.

Solit, R. W., J. J. McKeown, Jr., S. Smullens, and W. Fraimow. 1971. The surgical implications of intracavitary mycetomas (fungus balls). *J. Thorac. Cardiovasc. Surg.* 62:411–422.

Soubani, A. O., and P. H. Chandrasekar. 2002. The clinical spectrum of pulmonary aspergillosis. *Chest* 121:1988–1999.

Stevens, D. A., V. L. Kan, M. A. Judson, V. A. Morrison, S. Dummer, D. W. Denning, J. E. Bennett, T. J. Walsh, T. F. Patterson, and G. A. Pankey. 2000. Practice guidelines for diseases caused by *Aspergillus*, Infectious Diseases Society of America. *Clin. Infect. Dis.* 30:696–709.

Tomee, J. F., T. S. van der Werf, J. P. Latgé, G. H. Koeter, A. E. Dubois, and H. F. Kauffman. 1995. Serologic monitoring of disease and treatment in a patient with pulmonary aspergilloma. *Am. J. Respir. Crit. Care Med.* 151:199–204.

Tuncel, E. 1984. Pulmonary air meniscus sign. *Respiration* 46:139–144.

Wakayama, M., K. Shibuya, T. Ando, T. Oharaseki, K. Takahashi, S. Naoe, and W. F. Coulson. 2002. Deep-seated mycosis as a complication in bone marrow transplantation patients. *Mycoses* 45:146–151.

Ward, S., L. Heyneman, M. J. Lee, A. N. Leung, D. M. Hansell, and N. L. Muller. 1999. Accuracy of CT in the diagnosis of allergic bronchopulmonary aspergillosis in asthmatic patients. *Am. J. Roentgenol.* 173:937–942.

Young, N. A., K. H. Kwon-Chung, T. T. Kubota, A. E. Jennings, and R. I. Fisher. 1978. Disseminated infection by *Fusarium moniliforme* during treatment for malignant lymphoma. *J. Clin. Microbiol.* 7: 589–594.

Yousem, S. 1997. The histological spectrum of chronic necrotizing forms of pulmonary aspergillosis. *Hum. Pathol.* 28:650–656.