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SESAP Critique / Critique SESAP

Category 11, Items 34 and 35

The CT in the present case shows a single large apical lesion with an apparent thick-walled cavity. Although the lesion pictured might be malignant, the most likely diagnosis is chronic granulomatous infection. Video-assisted thoracoscopy (VATS) might provide a diagnosis, but the lesion is too large to remove by thoracoscopy. VATS would therefore need to be combined with needle aspiration or needle biopsy, and in that case offers little advantage over transthoracic needle aspiration. Thoracotomy and resection is more aggressive than would be warranted for an infectious disease, and would be dangerous if he has, for example, active tuberculosis. Transthoracic needle aspiration under x-ray control would be relatively simple and safe, and would be the least invasive procedure that is likely to produce a diagnosis. Thoracentesis could be done, but there is only a small amount of pleural fluid present, and it would be unlikely to be diagnostic. Repeat bronchoscopy is not indicated, because it has already failed to produce a diagnosis.

The differential diagnosis includes a number of possibilities. Wegener's granulomatosis typically produces multiple nodules, not large cavitary lesions. Histoplasmosis may present with a solitary mass, but would be much less likely to cavitate; further, histoplasmosis does not typically present with such a large lesion as seen here. The positive skin test is of little value in areas where histoplasmosis is endemic, such as northwest Missouri, where most of the population is histoplasmin positive. It does rule out anergy, so the negative tuberculin test is important. Tuberculosis, while always a possibility, seems less likely for three reasons; although it can produce apical cavitary lesions, the cavities tend to be more thin-walled; it does not typically grow into the chest wall, as this lesion appears to do; and the tuberculin skin test is reliable if one can rule out anergy. Aspergillosis is classically a secondary invader in a tuberculosis cavity. It is seen as a primary pathogen only in patients who are immunocompromised, which this patient was not. Actinomycosis, by contrast, can produce a large lesion, often attached to the chest wall, with small and irregular cavitary areas. There may be invasion of the chest wall, and even fistula formation to the skin of the chest. Like tuberculosis, it can attack otherwise healthy individuals. The characteristic pathologic finding of actinomycosis is the finding of yellow “sulfur granules” within the lesion.

Actinomycosis is caused by *Actinomyces israelii*, a bacterium normally found in the oral cavity, in the tonsillar crypts, and around the gums. It produces a filamentous growth pattern. This microaerophilic bacterium is notoriously difficult to isolate; anaerobic culture conditions are required. It is sensitive to many antibiotics, and penicillin remains the drug of choice. Other antibiotics such as chloramphenicol, clindamycin, tetracycline, and erythromycin can be used if penicillin fails. Treatment is begun with intravenous penicillin for several weeks, or until clinical response is noted, and then continued with oral penicillin for two to three months. Antifungal drugs such as amphotericin B and fluconazole are not indicated. Despite the somewhat fungal-sounding name, actinomycosis is a bacterial infection. Surgical therapy is usually not necessary, but resection is sometimes done for a destroyed lobe, chronic abscess, or bronchiectasis. It should be delayed until adequate antibiotic treatment has been well begun. Empyema is sometimes seen, but can usually be adequately treated with tube thoracostomy.

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