■ BRIEF OVERVIEW OF THE DISEASE

Tuberculosis (TB) is a disease likely as old as humanity itself. Aristotle is credited as being the first to recognize the contagious nature of the disease, but discovery of the specific infectious agent, the tubercle bacillus (*Mycobacterium tuberculosis*), did not occur for several more centuries, until it was isolated by Robert Koch in 1882.

Despite significant improvements in the diagnosis and treatment of tuberculosis achieved during the last 3 decades, tuberculosis still remains one of the deadliest communicable diseases worldwide. Tuberculosis is still present in all regions of the world, with a more significant impact in developing countries. Pulmonary tuberculosis (TB) is caused by infection with the aerobic, acid-fast bacillus *Mycobacterium tuberculosis*. TB is transmitted by inhalation of infected droplets and usually requires prolonged or repeated contact with infected individual(s). Those populations most commonly affected include patients of low socioeconomic status, the elderly, immunocompromised patients (i.e. AIDS patients), alcoholics, and prisoners.

Even though traditionally primary and post primary tuberculosis are considered 2 different forms of the disease based on the time of exposure, this has been recently challenged based on molecular and DNA analysis, although this terminology is still useful to describe the morphologic and imaging manifestations of the disease.

PRIMARY TUBERCULOSIS.

Primary infection may present as pulmonary consolidation. Lymphadenopathy is identified in approximately 95% of primary cases and is common in children. In the pediatric population (especially in those patients less than 1 year of age), parenchymal disease may progress and is referred to as progressive primary tuberculosis. Primary infection is usually contained by the body's normal granulomatous response and often heals without complications. Evidence of contained primary infection may include a focal region of parenchymal scarring with associated calcification (Ghon lesion). Cavitation in primary infections is rare.

POSTPRIMARY TUBERCULOSIS

Postprimary disease results from reactivation of a previously dormant primary infection in 90% of cases; in a minority of cases, it represents continuation of the primary disease. Postprimary tuberculosis is almost exclusively a disease of adolescence and adulthood with clinical complaints including cough and constitutional symptoms (weight loss, night sweats, chills).

The most common signs/symptoms include cough, pleuritic chest pain, upper respiratory infection-type symptoms, and hemoptysis.

Reactivation typically occurs in those areas of the lung with a high partial pressure of oxygen, namely the apical and posterior segments of the upper lobes or the superior segments of the lower lobes. Cavitation is an important feature of postprimary infection and indicates an active process with increased risk of transmission.

The radiologic features of postprimary tuberculosis can be broadly classified as parenchymal disease with cavitation, airway involvement, pleural extension, and other complications. Postprimary tuberculosis appears as upper lobe apical/poste-

rior segment heterogeneous consolidation with cavitation, with thick, nodular, and irregular walls. Cavitation is present in 45% of cases. Other findings include centrilobular nodules, tree-in-bud opacities, and acinar/lobular consolidations. Lymphadenopathy was not seen in this patient; however, it is present in 5% of cases.

Definitive diagnosis of active tuberculosis is based on sputum culture.

RADIOGRAPHIC FINDINGS CAN INCLUDE:

- Airspace consolidation (lobular sized and peribronchial)
- Cavitations with variable wall thickness
- Endobronchial spread to dependent portions of lung (centrilobular nodules, tree-in-bud appearance, bronchial wall thickening)
- Other findings: fibrotic changes, volume loss, adenopathy, pleural effusions
- Distribution: often segmental, apical and apical posterior segments of upper lobes and superior segments of lower lobes, gravity dependent lobes (via bronchogenous spread)
- Signs of active disease: signs of endobronchial spread, cavitation, consolidation
- Inactive disease: requires stability over 6 months
- Sequela: Consolidation and nodules resolve over 9-12 months with successful treatment. Signs of fibrosis, volume loss, calcifications of lung and lymph nodes may appear.
- If immunosuppressed, there may be progress to miliary tuberculosis, ARDS (acute respiratory distress syndrome), extrathoracic dissemination to breast, spine, kidney, meninges, bone Treatment regimens vary with drug susceptibility of organisms, image findings, and clinical factors. Multiple antituberculous drugs are administered over a prolonged time period. First-line drugs include isoniazid, rifampin, streptomycin, ethambutol, pyrazinamide. Second-line drugs include amikacin, kanamycin, and capreomycin. Hemoptysis is treated with bronchial artery embolization or surgery. Cavitation stability longer than six months is reported as stable, rather than inactive, disease.

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Answer to Radiographic Quiz

Description of the radiographies

There are patchy airspaces in both upper pulmonary lobes associated with cavitations, which appear superimposed in the lateral view. The cavitation in the right lung reveals air-fluid level.

Diagnosis: Reactivation pulmonary tuberculosis

]	L.	What is	the	most	relevant	finding?

_____ Perihilar adenopathy

__X__ Bilateral upper lobe cavitation

_____ Cystic lucencies

_____ Blunting of the posterior costophrenic angles

Peripheral atelectasis

_____Apical lung scarring

 ${f 2}$. Which infectious process would be most compatible with the findings?

_____ Primary TB

__X__ Reactivation TB

____ Cryptococcus

_____ Invasive aspergillosis

PCP (pneumocystis pneumonia)

3. The patient should be offered HIV screening?

___X___Yes

No

