Imaging Manifestations of Thoracic Tuberculosis

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KEYWORDS

• Tuberculosis • Pulmonary tuberculosis • Primary tuberculosis • Postprimary tuberculosis
• Miliary tuberculosis • Mycetoma

KEY POINTS

- Tuberculosis is one of the most prevalent infectious diseases in the world, with the second highest death rate among communicable diseases worldwide.
- Even though traditionally primary and postprimary 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.
- Imaging plays a very important role in the diagnosis and follow-up of primary and postprimary tuberculosis for both pulmonary and extrapulmonary forms of the disease.

INTRODUCTION

Tuberculosis (TB) is a disease likely as old as humanity itself.[1] 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.[2] Distinct differences in the epidemiology of TB are observed between developing and industrialized nations. In countries where the standard of living is low and health resources are sparse, the risk of infection is highest with 80% of cases involving persons in their productive years (15–59 years of age).[3] Among communicable diseases, TB is the second leading cause of death worldwide after human immunodeficiency virus (HIV)/AIDS, killing nearly 2 million people each year; approximately 13% of TB patients have coexistent HIV infection.[4] In the United States, the most important risk factors for development of disease are host immunodeficiency, immigration from or travel to an endemic area, close contact with a TB patient, exposure to untreated cases in crowded living facilities such as prisons or nursing homes, advanced age, residing in an inner city, or homelessness.[5]

Global emergence of multidrug-resistant (MDR) strains of *M tuberculosis* in recent years has greatly complicated the management and control of transmission of active cases.[3,4] MDR TB is no more infective than nonresistant TB; however, it is a more serious infection, which requires prolonged administration of more toxic medications associated with higher morbidity and mortality. In addition, these patients remain infectious for a longer period once treatment has been initiated.[6] MDR TB most commonly develops during the course of TB treatment, most frequently as a result of inappropriate treatment, patients missing doses, or patients failing to complete their treatment.[7,8]

In 2013, an estimated 9.0 million people developed TB and 1.5 million died of the disease, 360,000 of whom were HIV-positive. TB is slowly declining each year, and it is estimated that 37 million lives were saved between 2000 and 2013

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through effective diagnosis and treatment. However, given that most deaths from TB are preventable, the death toll from the disease remains unacceptably high, and efforts to combat the disease should continue to accelerate. Although most TB cases and deaths occur among men, the burden of disease among women remains high with an estimated 3.3 million cases and 510,000 TB deaths among women and an estimated 550,000 cases and 80,000 deaths among children reported in 2013.[9]

PATHOPHYSIOLOGY

M tuberculosis is a slightly curved bacillus that is an aerobic, nonmotile, non-spore-forming rod-shaped bacterium. The organism has a cell wall that has an unusually high lipid content that resists staining by the usual Gram method but accepts basic fuchsin dyes and is not easily discolorized, even with alcohol. This resistance to decolorization by acid-alcohol is termed acid-fast, a property shared only by members of the mycobacterial family and a few other organisms (*Nocardia*, *Rhodococcus*, and *Corynebacterium* species). These properties form the basis for the simple, rapid, and relatively specific traditional technique of identification by means of an acid-fast smear. In addition, of note, they are primarily intracellular pathogens with slow growth rates.[3]

Transmission of disease occurs as a result of contact to a source case in more than 80%, typically the result of exposure to sputum smear-positive cases, although smear-negative culture-positive cases have been responsible for up to 17% of new cases.[4] The presence of acid-fast bacilli in the sputum smear is the main indicator of potential for transmission. Additional source patient characteristics to consider, which increase the probability of transmission, include positive sputum culture, lung parenchymal cavitation on imaging, TB laryngitis, as well as high-volume and/or watery respiratory secretions.[3,10]

M tuberculosis is transmitted via airborne droplet nuclei produced when persons with pulmonary or laryngeal TB cough, sneeze, or speak. In a person with active pulmonary TB, a single cough can generate 3000 infective droplets, with as few as 10 bacilli needed to initiate infection. The droplets, which measure 1 to 5 μm in size and may contain up to 400 bacilli, can be kept airborne by normal air currents for prolonged periods of time, resulting in dispersion throughout a room or building. Infection occurs when a susceptible person inhales droplet nuclei that contain tubercle bacilli.[3,6] The distribution of inhaled droplet nuclei is determined by the ventilatory pattern and volumes of the various lobes of the lungs with the site of implantation preferentially in the middle and lower lung zones, although any lobe may be affected. Once lodged in the alveolus, *M tuberculosis* is ingested by alveolar macrophages. If the alveolar macrophage cannot destroy or inhibit *M tuberculosis*, the bacilli multiply within its intracellular environment, causing the host macrophage or its progeny to burst. The cycle continues as released bacilli are ingested by other alveolar macrophages and monocytes are recruited from the blood. During this period of rapid growth, tubercle bacilli are spread through lymphatic channels to regional hilar and mediastinal lymph nodes and through the bloodstream to more distant sites in the body.[3,11]

In most patients, the logarithmic phase of bacillary growth is arrested with the development of cell-mediated immunity and delayed-type hypersensitivity at 2 to 10 weeks after the initial infection.[3,12,13] Granuloma formation with a subsequent decrease in the number of bacilli is typically seen, and some of these bacilli remain viable but dormant for many years. This stage is called latent TB infection, which is generally an asymptomatic, radiologically undetected process in humans.[1,3] Sometimes, a primary complex (Ghon complex) can be seen radiographically and comprises the primary lesion and hilar adenopathy. Later, the primary lesion tends to become calcified and can be identified on chest radiographs for decades.[1,3,14] In approximately 5% of infected individuals, immunity is inadequate, and clinically active disease develops within 1 year of infection. HIV coinfection is the greatest risk factor for progression to active disease in adults, and the relation between HIV and TB has augmented the deadly potential of each disease. Other risk factors for progression to active disease include diabetes mellitus, renal failure, coexistent malignancies, malnutrition, silicosis, immunosuppressive therapies (including steroids and anti-tumor necrosis factor [TNF] drugs), and TNF receptor defects. In another 5% of the infected population, endogenous reactivation of latent infection occurs remote from time of initial infection, referred to as postprimary TB.[3,11,14]

TRADITIONAL VERSUS NEW CONCEPT OF ETIOPATHOGENESIS AND IMAGING MANIFESTATIONS OF TUBERCULOSIS

Traditionally, TB findings have been described as primary infection that occurs in patients who develop disease after the initial exposure to TB bacilli, whereas patients who develop disease as