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LITERATURE REVIEW

Pulmonary Cavities Due to Infectious Causes

Findra Setianingrum[1*], Finny Nandipinto[2], Anna Rozaliyani[1,2]

1) Parasitology Department, Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia
2) Indonesia Pulmonary Mycoses Centre, Jakarta, Indonesia

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Correspondence:

dr.findra@gmail.com

ABSTRACT

Pulmonary cavities are an important radiological finding in various infectious and non-infectious diseases. Infection-related cavities are usually associated with tuberculosis (TB), non-tuberculous mycobacterial (NTM) infection, chronic pulmonary aspergillosis, and echinococcosis, each with distinct mechanisms of formation and radiologic patterns. In TB and NTM, tissue necrosis and bronchiectasis lead to the formation of cavitary spaces that serve as reservoirs for organisms with a high burden and facilitate transmission. Residual cavities may then undergo secondary colonization by *Aspergillus* spp, resulting in a spectrum of chronic pulmonary aspergillosis with findings such as aspergilloma and thick-walled cavities. *Echinococcus* forms cysts that, when ruptured, can appear as cavitary lesions with the "water lily" sign, adding yet another differential diagnosis for pulmonary cavities. Understanding radiologic patterns, pathogenesis, and microbiological correlations can be used as an important clue to distinguish etiologies and to select appropriate management for patients.

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INTRODUCTION

Cavities are a common radiological finding in the lungs. A cavity is defined by the Fleischner Society as 'a gas-filled space within a zone of pulmonary consolidation or within a mass or nodule, resulting from the expulsion of necrotic tissue from a lesion through the bronchial channels (Gadkowski & Stout, 2008; Hansell et al., 2008). Radiologically, a cavity appears as a lucent zone of pulmonary consolidation that may or may not contain fluid and is surrounded by a wall of varying thickness (Canan et al., 2021; Gadkowski & Stout, 2008). Another definition describes a cavity as an air-filled space within a pulmonary infiltrate or mass with a wall thickness greater than 4 mm (J. H. Ryu & Swensen, 2003).

The causes of cavity formation vary, including both infectious and non-infectious diseases, such as cancer, infection, autoimmune diseases, trauma, and congenital abnormalities (Gadkowski & Stout, 2008; Ketai et al., 2018; Kim & Han, 2012). The characteristics of cavity lesions obtained from radiological imaging, such as chest X-rays and CT scans, can be used to differentiate the broad and varied etiologies of cavities (Gafoor et al., 2018). This information includes the number and location of the cavities, the thickness and contour of the cavity walls, and serial radiological images can aid in identifying the cause of the cavity (Duong et al., 2020; Nin et al., 2016). A combination of radiological, clinical, and laboratory data is required to achieve an accurate diagnosis related to the underlying disease causing the cavity (Gafoor et al., 2018). The upper lung lobes are most frequently the site of tuberculosis (Andreu et al., 2004; Urbanowski et al., 2020). In malignancies, a single cavity lesion is usually associated with primary lung cancer and lung abscesses, while multiple lesions are typically associated with septic emboli, granulomatosis with polyangiitis, rheumatoid nodules, and metastasis (Nin et al., 2016; Xue et al., 2012).

Infectious microorganisms such as *Mycobacterium tuberculosis*, *Klebsiella pneumoniae*, and *Staphylococcus aureus* tend to cause cavity lesions more frequently than other pathogens (Krutikov et al., 2019; Urbanowski et al., 2020). The COVID-19 pandemic has led to an increase in patients with residual lung cavities. Post-COVID-19 patients with pulmonary cavity lesions are at risk of lung infections, one of which is caused by *Aspergillus fumigatus*, either as a primary cause or co-infection with bacteria (Rai et al., 2022).

LITERATURE REVIEW

Non-tuberculous Mycobacteria (NTM)

Non-tuberculous Mycobacteria (NTM) consist of more than 172 species with varying virulence characteristics (To et al., 2020). These microorganisms belong to the group of opportunistic pathogens and are found in natural habitats such as water, soil, and animals (Jeon, 2019; To et al., 2020). The NTM species commonly found in humans are *Mycobacterium avium* complex (MAC), *Mycobacterium abscessus*, and *Mycobacterium kansasii* (Hoefsloot et al., 2013; To et al., 2020).

There are several phenotypes that serve as risk factors for infection in immunocompetent individuals (those without specific underlying lung diseases), including body mass index and fat levels, as well as patients with tall stature (Kartalija et al., 2013). Radiological characteristics of NTM from different species cannot distinguish the three types of NTM, so a definitive diagnosis of the causative species must be based on a combination of clinical information and radiological examination (Y. J. Ryu et al., 2016).

The 10-year mortality rate for MAC with progressive cavities is higher (46.7%) compared

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to MAC with non-progressive cavities (9.8%) (Oshitani et al., 2020). In a study involving 481 patients with lung disease due to MAC, clinical outcomes were better in patients with non-cavitary MAC (88%) compared to those with cavitary MAC (76% in fibrocavitary and 78% in nodular bronchiectatic cavities) ($p<0.05$) (Koh et al., 2017). The formation of new cavities in the non-cavitary nodular bronchiectatic MAC group occurred in 8.7% within a 4-year period after diagnosis, with risk factors including *M. intracellulare* and a history of previous TB (Han et al., 2021).

Pathogenesis

There is an association between NTM and bronchiectasis, in which NTM can exacerbate pre-existing bronchiectasis but can also cause de novo bronchiectasis (development from initially absent to present). Evidence supporting the latter theory is the finding of NTM nodules before the onset of bronchiectasis. Honda et al. explained that the formation of bronchiectasis can occur through two mechanisms (Honda et al., 2015). The first mechanism is the weakening of the airway walls. This process is caused by chronic granulomatous inflammation leading to mucosal ulceration and atrophy. The second mechanism is the dilation of the bronchi and bronchioles caused by airway obstruction due to mucous plugs.

Kim et al. proposed that some NTM cavities form from progressive cystic dilatation of the bronchi, mediated by a patent feeding bronchus connected to the cavity, as shown on both radiologic imaging and histopathologic findings (Sung Kim et al., 2005). Pulmonary MAC infection in the early stage appears as bronchial wall thickening that progresses to peribronchial thickening or peribronchial nodules. In later stages, cystic bronchiectasis develops, manifesting as masses or consolidation accompanied by cavitation on CT scans and in pathologic specimens (Honda et al., 2015; Sung Kim et al., 2005).

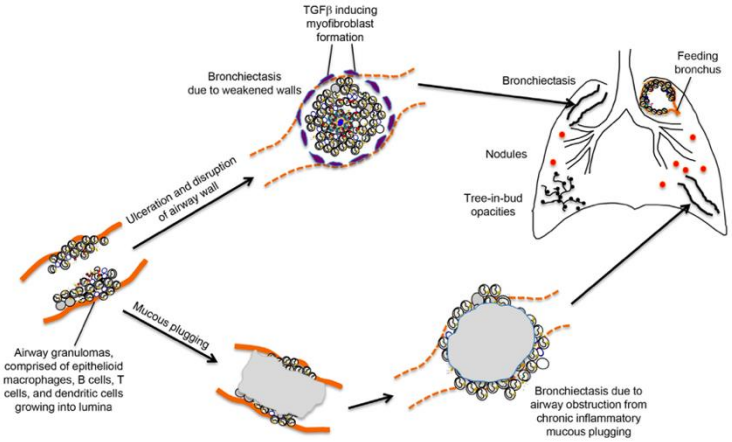

Figure 1. Histopathological-radiological correlation in lung disease caused by NTM. The main features are bronchiectasis, cavities with feeding bronchi, nodules, and tree-in-bud opacities (Source: Honda, 2014).

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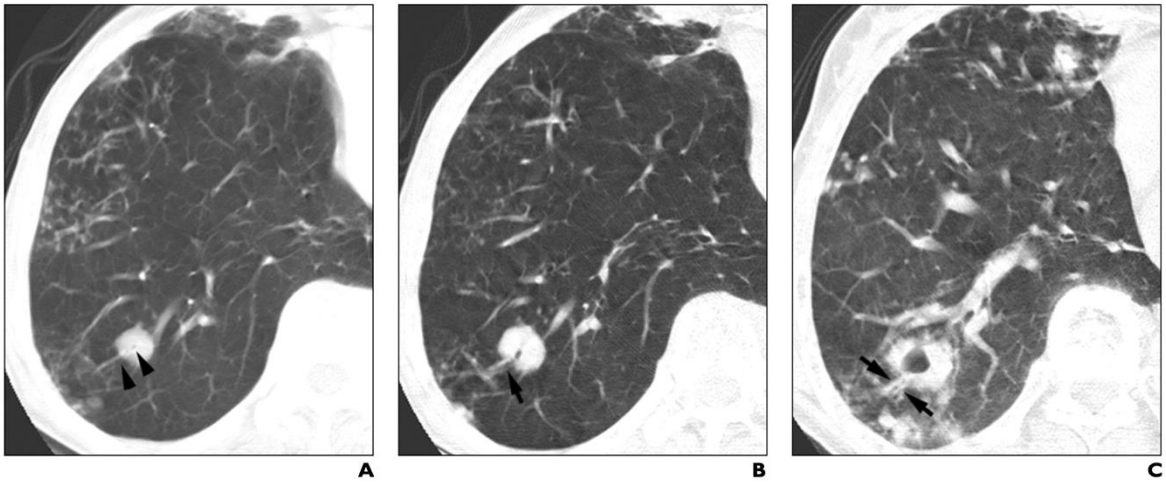

Figure 2. Serial CT images showing cavitation formation in nontuberculous mycobacterial (NTM) infection caused by *Mycobacterium avium–intracellulare* complex. (A) Peribronchial nodule in the right lower lobe; arrowhead indicates a central bronchiole appearing as a radiolucent area. (B) Six-month follow-up CT shows enlargement of the nodule with a central bronchiole extending toward the peripheral lung. (C) Twelve-month follow-up CT demonstrates a peribronchial nodule with focal cystic bronchiectasis, presenting as a large cavitary lesion. (Source: Ryu, 2016)

The formation of these central cavities begins with an inflammatory process at the center of the necrotic area, accompanied by ulceration, destruction, and detachment of the bronchial wall and cartilage. The peripheral portion of peribronchial nodules may extend into the surrounding lung parenchyma. In this condition, the still-patent central bronchus functions as a draining bronchus, removing detached central necrotic debris, resulting in cavitation and bronchogenic spread of infection (Sung Kim et al., 2005).

Radiologic Findings

Radiologic findings in NTM include bronchiectasis, tree-in-bud opacities (branching centrilobular nodules) with or without cavitation, atelectasis, ground-glass opacities, and/or consolidation (Chu et al., 2015; Daley & Winthrop, 2020; Jeong et al., 2004; Oshitani et al., 2020; Takahashi et al., 2012; Yamazoe & Yanagi, 2023). These various radiologic appearances can be broadly classified into two major patterns: fibrocavitary and nodular bronchiectatic disease (Griffith et al., 2007).

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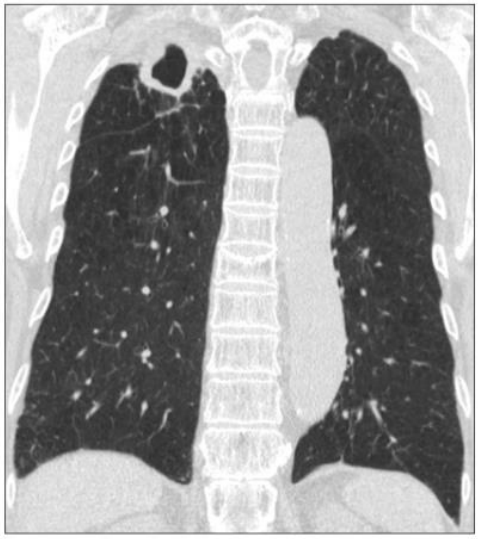

Figure 3. Fibrocavitary type of NTM. CT scan of a 73-year-old male patient with NTM pulmonary disease caused by *Mycobacterium intracellulare* shows cavitation in the right upper lobe accompanied by emphysema in both lungs. (Source: Ryu, 2016)

The fibrocavitary type of NTM resembles pulmonary tuberculosis (TB), is located in the upper lobes of the lungs, and is generally found in elderly men with underlying lung disease (Chu et al., 2015; Y. J. Ryu et al., 2016).Several radiologic abnormalities are also frequently observed, namely pleural thickening, traction bronchiectasis, and loss of lung volume in the fibrocavitary type (Y. J. Ryu et al., 2016). The nodular bronchiectatic type is more commonly found in elderly women (Canan et al., 2021). The distinguishing characteristics between TB and NTM cavities are that NTM cavities progress more slowly, have thin walls that may sometimes involve the pleura without lymph node calcification, and are not accompanied by atelectasis (Chu et al., 2015; Yuan et al., 2014). In addition, extensive bronchiectasis involving the right middle lobe and lingula is more suggestive of NTM than TB (Lynch et al., 1995).

Mycobacterium tuberculosis

Pulmonary tuberculosis (TB) is a chronic disease caused by Mycobacterium tuberculosis (Mtb), which leads to progressive damage to the lung parenchyma. There were 10.6 million people who developed TB, with 1.3 million dying, based on data from the World Health Organization (WHO) Global Tuberculosis Report 2022. Indonesia ranks second globally as the largest contributor to increases in TB cases, after India.(World Health Organization, 2023) The TB incidence rate in Indonesia in 2023 is estimated at 378 per 100,000 population.(Kementerian Kesehatan Republik Indonesia, 2024)

Pathogenesis

Mycobacterium tuberculosis enters the body through the respiratory tract. A positive acid-fast bacilli (AFB) examination on sputum serves as the primary indicator of infection transmission (Palaci et al., 2007). Other factors that increase the probability of transmission include positive culture,

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parenchymal lung cavities on radiology, TB laryngitis, and large amounts of respiratory secretions (Urbanowski et al., 2020). The effector cells of the immune system involved in eliminating Mtb are macrophages (Urbanowski et al., 2020). This elimination process produces cell aggregates consisting of lymphocytes surrounding Mtb-infected macrophages, known as caseous necrosis (Elkington et al., 2011).

In parallel with these processes, the lung parenchyma undergoes destruction and gradually changes its structure into an air-filled space known as a cavity (Ravimohan et al., 2018). There is a decrease in the concentration of the extracellular collagen matrix that contributes to cavity formation because the remaining necrotic debris is easily evacuated through the nearest bronchus. When this decrease occurs, regeneration of the basal membrane and lung tissue cannot take place. Ultimately, the cavity continues to enlarge and persist within the lung (Urbanowski et al., 2020).

Mtb then replicates exponentially within cavities that are isolated from the immune response, and it is estimated that each cavity may contain up to 10^7–10^9 Mtb organisms (Grosset, 2003; Urbanowski et al., 2020). A recent study by Ordonez et al., using PET-CT to monitor TB progression, showed that the bacterial density in the cavity wall was 100 times higher than in non-cavitary lesions (6.78 ± 0.73 versus 4.40 ± 0.61 $\log_{10}$ colony forming units (c.f.u.); $P = 0.0043$) (Ordonez et al., 2020). Cavities in TB and their proximity to the airways correlate with the occurrence of cough during TB treatment (Hales et al., 2013; Proaño et al., 2018). TB patients with cavities containing high levels of Mtb bacteria can generate more aerosols and thereby drive massive Mtb transmission (Turner et al., 2017).

Cavities often persist even after being cleared of mycobacteria and are replaced by scar tissue (Urbanowski et al., 2020). As a consequence of this process, cavitation can lead to loss of lung volume and chronic pulmonary deficits (closed healing) (Chakaya et al., 2016). Opportunistic infections resulting from secondary colonization can occur in persistent cavities (open healing) (Chakaya et al., 2016; Urbanowski et al., 2020). The combination of warm temperature, high humidity, and immune system disturbances can trigger secondary colonization, often by *Aspergillus* spp (Page et al., 2019).

Radiological appearances

Chest radiography and CT scan are imaging modalities that can be used to evaluate cavities and other pathological findings in the lungs (Urbanowski et al., 2020). The use of CT provides 91% correct diagnoses for TB and excludes non-TB cases in 76% (Lee et al., 1996). CT and HRCT help detect small foci of cavitation in areas of pneumonia and in regions with dense nodularity and fibrosis (Im et al., 1993). One study reported that the proportion of cavities successfully identified by CT scan was 58%, higher than chest radiography at 22% (Im et al., 1993).

Cavities are found in 20-45% of patients with pulmonary TB reactivation (Hales et al., 2013; Yeon & Lee, 2008). Differences in cavity wall characteristics indicate associations with disease progression (Ravimohan et al., 2018). Research by Ors et al. showed that HRCT scores based on cavity, nodule, and bronchial lesion characteristics were lower in patients with negative acid-fast bacilli (Ors et al., 2007). Additionally, MTB bacilli concentration is higher in thicker cavity walls. After TB treatment, cavity walls become thinner (Ors et al., 2007).

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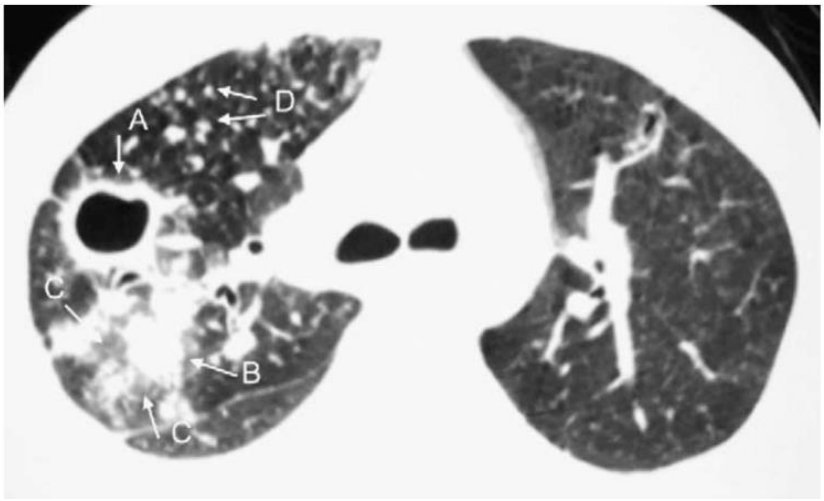

Figure 4. A patient with pulmonary tuberculosis shows HRCT findings: A (cavity), B (consolidation), C (GGO between areas of consolidation), D (micronodules). (Source: Orz, 2007)

Table 1. Comparative radiological findings between tuberculosis, non-tuberculous mycobacteria (NTM), and aspergillosis.

Feature	Tuberculosis (TB)	NTM	Aspergillosis (e.g., tracheobronchitis/CPA)
Cavities	Thick, irregular walls; upper lobe predominant; satellite nodules	Thin, even walls; often middle/lower lobes; adjacent pleural thickening	Thickened pre-existing cavities; fungal ball (aspergilloma); infiltration around cavities
Bronchiectasis	Mild/moderate; upper lobe tree-in-bud	Prominent, cylindrical; multifocal, often >5 lobes	Variable; bronchial wall thickening prominent in invasive forms
Nodules	Centrilobular/tree-in-bud; numerous ≥10 mm; miliary pattern possible	Small centrilobular; branching; fewer large nodules	Peribronchial nodules >5 mm; endobronchial nodular lesions
Airway Involvement	Endobronchial lesions rare; mucosal irregularities less prominent	Bronchial dilatation common	Circumferential bronchial/tracheal wall thickening; pseudomembranes; luminal narrowing/stenosis
Consolidation/Pleura	Pleural effusion common; consolidation with bronchogenic spread	Less effusion; contiguous spread	Peribronchial consolidation; pleural thickening near cavities
Other	Lymphadenopathy; fibrosis post-TB	Emphysema association	Air-crescent sign (invasive); halo sign (early invasive)

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Most TB cavities occur in the apical or posterior segment of the superior lobe and, to a lesser extent, in the superior segment of the inferior lobe (Gafoor et al., 2018; Urbanowski et al., 2020). Most TB cavities occur in the apical or posterior segment of the superior lobe and, to a lesser extent, in the superior segment of the inferior lobe (Urbanowski et al., 2020). The distribution of cavity lesions in the upper lung fields is linked to reduced vascular supply, higher oxygen tension, and impaired lymphatic drainage in these areas compared to the lower lobes (Goodwin & Des Prez, 1983).

Characteristics of cavities in TB can be solitary or multiple, sometimes with consolidation visible around the cavity area, fibronodular or mixed (Urbanowski et al., 2020). Some small cavities may fuse into larger ones. Imaging variations can also be classified based on patient immune status (Yeon & Lee, 2008). Most immunocompetent adult TB patients have cavities in the upper lobes (Skoura et al., 2015). Cavities accompanied by lymphadenopathy and pleural effusion are commonly found in immunosuppressed adults and pediatric patients (Rozenshtein et al., 2015). In immunocompromised patients, such as those with HIV, the TB radiologic lesions commonly found in HIV-positive patients with CD4 T-lymphocyte counts below 200/mm³ are mediastinal or hilar lymphadenopathy, cavities are rarely seen, and there is extrapulmonary involvement (Ben Romdhane et al., 1996).

Around 10% of parapneumonic effusions can become infected and progress to empyema (Kearney et al., 2000). Causes of pleural infection vary by geographic region and patient age; empyema is often due to anaerobic infections or mixed infections involving both aerobic and anaerobic bacteria. Other causes, like TB, may serve as differential diagnoses, especially in TB-endemic areas; pleural infections always result in fluid accumulation in the pleural space with pleural thickening and loculation formation, regardless of the bacterial cause (Ko et al., 2015).

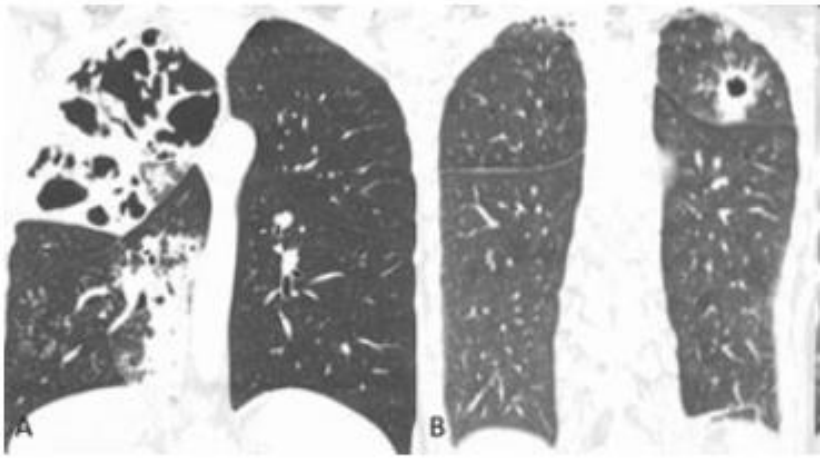

Figure 5. A. Cavitary consolidation in *M. tuberculosis*. B. Thick-walled cavity in *M. kansasii* (Source: Canan, 2021)

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Aspergillus spp

Patients with primary lung diseases such as TB, NTM, asthma, COPD, lung cancer, and pneumothorax are at risk for fungal lung disease (Denning et al., 2015; Page et al., 2019). The most common cause of fungal lung disease is *Aspergillus* spp., particularly *Aspergillus fumigatus*, which causes pulmonary aspergillosis (Howard et al., 2013). Pulmonary aspergillosis encompasses a spectrum of acute, chronic, and allergic diseases (Latgé & Chamilos, 2020). Cavities can be found across all aspergillosis spectra but are most common in chronic pulmonary aspergillosis (CPA) (Denning et al., 2011). The main risk factor for CPA is prior TB, especially with residual cavitary lesions (Page et al., 2019). CPA diagnosis is often missed due to clinical and radiologic similarities with TB (Oladele et al., 2017; Setianingrum et al., 2021).

Pathogenesis

A. fumigatus conidia (2-3 µm in diameter) are small enough to reach bronchioles or alveolar spaces after passing through the upper airways (Kwon-Chung & Sugui, 2013). Alveolar macrophages, neutrophils, and dendritic cells act as phagocytic cells against conidia in the lungs (Latgé & Chamilos, 2020). Macrophages engulf conidia, which are then digested by phagolysosomes through both oxidative and non-oxidative mechanisms. Neutrophils play a crucial role in killing germinating conidia and hyphae that escape from macrophages. *Aspergillus* conidia adhere tightly to type II alveolar epithelial cells, known as A549 cells, as well as to the extracellular matrix. The 1,8-dihydroxynaphthalene (DHN) melanin present in conidia is believed to be a virulence factor that prevents phagolysosome acidification, allowing conidia to survive inside alveolar epithelial cells (Amin et al., 2014). Subsequently, *Aspergillus* can grow on cavity walls (from prior primary lung disease) and damage lung parenchyma by releasing proteolytic enzymes, mycotoxins (e.g., gliotoxin), and possibly other metabolites secreted by *Aspergillus* (Izumikawa et al., 2014). Tissue necrosis occurs as a result of interactions between proteolytic enzymes and oxidants produced by macrophages and neutrophils (Latgé & Chamilos, 2020).

Chest X-ray is the primary imaging modality for establishing a diagnosis of chronic pulmonary aspergillosis (CPA). However, chest CT provides more detailed information on the location, distribution, and characteristics of cavities and other pulmonary lesions (Denning et al., 2015). CPA imaging patterns represent a combination of underlying lung disease and secondary changes from *Aspergillus* spp. infection (Patterson & Strek, 2014). CPA typically arises from bronchopulmonary cavities, formation of new cavities or nodules, and alveolar consolidation (Greene, 2005). Hallmarks of CPA include new and/or expanding cavities with variable wall thickness, with or without intracavitary fungal balls (aspergilloma), sometimes accompanied by pleural thickening, parenchymal destruction, and/or fibrosis (Greene, 2005; Patterson & Strek, 2014).

There are four types of chronic pulmonary aspergillosis (CPA): simple aspergilloma, chronic cavitary pulmonary aspergillosis (CCPA), chronic fibrosing pulmonary aspergillosis (CFPA), and *Aspergillus* nodule (Denning et al., 2015). Prior to aspergilloma formation, there is an area of fungal growth layering on the cavity surface with an irregular contour. Aspergilloma growth begins with fungal colonization of lung cavities or bronchiectasis, progressing to infection (Patterson & Strek, 2014). Aspergilloma represents the end-stage manifestation of the CPA clinical course. Aspergilloma is classified as simple or complex; complex aspergilloma is part of CCPA (Denning et al., 2015, 2018).

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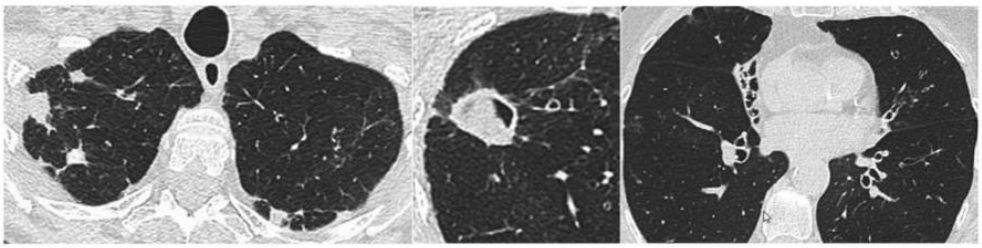

Figure 7. Multiple Aspergillus nodules of varying sizes are visible, along with a fungal ball inside a cavity with heterogeneous wall thickness, bronchiectasis, and cicatricial atelectasis in the middle lobe. (Source: Denning, 2015).

Echinococcus *spp*

Echinococcosis is a parasitic infection caused by the tapeworm *Echinococcus granulosus*, primarily found in dogs as its definitive host. The disease is widespread in Africa, Europe, Asia, the Middle East, Central and South America, and only occasionally reported in North America (Torgerson et al., 2010).

Pathogenesis

Infections often remain asymptomatic for years, but when symptoms appear, they may include chest pain, cough, hemoptysis, or pneumothorax. Other manifestations, such as wheezing, fever, urticaria, and anaphylaxis, can occur if the cyst ruptures, releasing antigenic material. Laboratory findings may show peripheral eosinophilia and positive serologic tests (Kunst et al., 2011).

Radiologic Findings

Radiographically, liver cysts are the most common finding, but lung cysts occur in 20-30% of cases. If a lung cyst ruptures and is exposed to air, it appears as a crescent-shaped lucent area or cavitary lesion, with the collapsed membrane floating within the cavity, creating the characteristic "water lily" sign. Over time, the cavity wall may calcify (Gafoor et al., 2018).

A study by Guo et al. identified the lung as the most frequently involved extrahepatic organ at 57.6%, followed by the adrenal gland (30.3%) and brain (27.3%). Pulmonary lesions appear as scattered, irregular nodules with internal vacuoles or cavities, typically peripheral in location. Other common radiologic features include multiple nodules (78.9%) and bilateral lung involvement (57.9%) (Guo et al., 2021).

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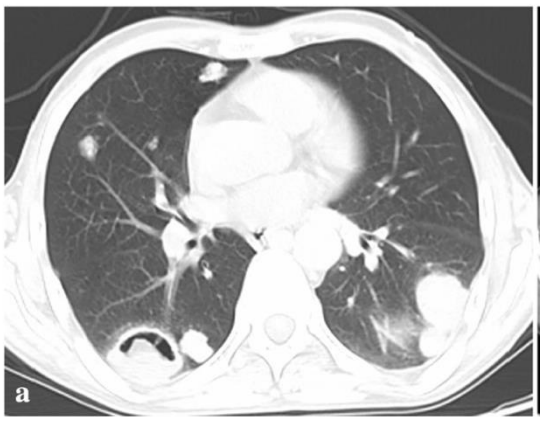

Figure 8. Multiple nodules and cavities are visible in the left lung of a patient with alveolar echinococcosis. (Source: Guo, 2021)

Conclusions

Cavities represent a common radiologic finding in lung diseases caused by infections. Radiologic evaluation, particularly with a CT scan, is essential for diagnosis as it allows detailed identification of cavity lesion characteristics. However, the lung cavities in TB and CPA are difficult to distinguish; other radiological features, such as pleural effusion, might help, as pleural effusion is found mostly in TB. The Aspergillus IgG test is positive in CPA, while the molecular rapid test or AFB is positive in TB. Combining radiologic features with clinical condition and laboratory tests is expected to yield accurate diagnoses in patients with pulmonary cavitary lesions.

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