Case Report

Double Trouble: Chronic Cavitary Pulmonary Aspergillosis with Endobronchial Chondroma, Arising Post-Tuberculosis

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DOI
10.21276/apalm.3551

Article History
Received: 17-03-2025
Revised: 03-08-2025
Accepted: 31-08-2025
Published: 20-09-2025

How to cite this article
Kulkarni S, Yadav KS, Bhalekar S. Double Trouble: Chronic Cavitary Pulmonary Aspergillosis with Endobronchial Chondroma, Arising Post-Tuberculosis. Ann Pathol Lab Med. 2025;12(9):C151-C154.

Abstract

Pulmonary tuberculosis is a common infectious condition mainly seen in developing countries like India. It is particularly important because of its long-term complications such as chronic cavitary pulmonary aspergillosis. Endobronchial chondroma is an extremely rare benign tumor which presents with nonspecific respiratory symptoms. We report a case of a previously treated tuberculosis patient in whom both chronic cavitary aspergillosis and endobronchial chondroma were diagnosed concurrently on final histopathological examination of the surgical specimen.

Keywords: Pulmonary aspergillosis; endobronchial chondroma; Tuberculosis sequelae; Pulmonary tuberculosis.

Introduction

Pulmonary tuberculosis (TB) is a common infection in developing countries. Its long-term complications include chronic pulmonary aspergillosis (CPA), which develops in cavitary TB lesions. The global burden of post-TB CPA was estimated at 1.2 million cases annually [1]. The majority of cases [22%] have chronic cavitary pulmonary aspergillosis, even at end of therapy [2].

Chondromas are benign cartilaginous tumors occurring in bone. Pulmonary chondromas are rare benign tumours with an incidence of 0.1% of all benign lung tumors [3]. These are most often asymptomatic but can cause bronchial obstruction if large and situated in the bronchial tree.

The coexistence of bronchial chondroma, chronic cavitary aspergillosis, and prior pulmonary tuberculosis is clinically significant, as post-TB structural changes and bronchial obstruction caused by chondroma can predispose to fungal colonization and complicate diagnosis and management.

Here we discuss an interesting case of a patient who was treated in the past for pulmonary tuberculosis and later presented with cavitary pulmonary aspergillosis co-existing with endobronchial chondroma.

Case Report

A 54-year-old male presented with a history of cough with expectoration and hemoptysis for 3 days. He gave a history of being treated for pulmonary tuberculosis in the past; details of which were unavailable. Chest X-ray showed homogeneous opacity in the left lung. High-Resolution Computed Tomography (HRCT) scan showed a well-defined endoluminal mixed, predominantly calcific non-enhancing soft tissue density causing significant luminal compromise and collapse of the entire left lung. Findings were suggestive of broncholith as post-TB sequelae. Bronchoscopic biopsy was inconclusive, and bronchoalveolar lavage showed mucoid material with benign squamous cells and polymorphs. No atypical cells were seen. The patient was HIV-negative, a non-smoker, and had no significant comorbidities. He was not on any long-term medications.

Left pneumonectomy was performed with removal of the endobronchial mass and sent for histopathological examination. The specimen of the left lung measured 13.5 x 6.5 x 4.8 centimetre. The pleura was thick and adherent. On cutting open, upper and lower lung lobes showed multiple cavities ranging from 0.5-5 centimetre in diameter, filled with dirty brown necrotic material [Figure 1A]. The left main bronchus had brownish friable material. A separately sent yellowish-white nodular mass measured 2.8 x 2.3 centimetre. It was gritty to cut and showed a solid white cut surface [Figure 1B].

On microscopy, sections from multiple cavities and the left main bronchus revealed large fungal colonies comprising of long, slender septate hyphae with acute angle branching suggestive of aspergillosis. Focal angioinvasion was also seen, and Grocott's methenamine silver (GMS) stain highlighted the fungal hyphae [Figure 2]. The separately sent nodular mass showed lobules of benign hyaline cartilage with areas of endochondral ossification. Admixed fatty marrow spaces were noted. There was no smooth muscle tissue or entrapped respiratory epithelium seen. No features indicative of atypia or malignancy were noted [Figure 2].

A histopathological diagnosis of Chronic Cavitary Pulmonary Aspergillosis with focal invasion with endobronchial chondroma was rendered.

Figure 1: [A] Cut surface of left pneumonectomy specimen shows multiple cavitatory lesions filled with brown necrotic material. Bronchiectasis noted in surrounding lung; [B] Cut surface of endobronchial mass is solid, firm to hard and yellowish-white.

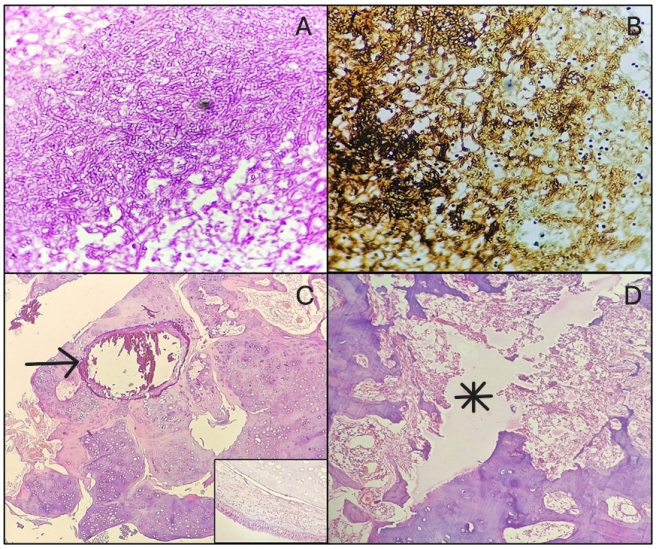

Figure 2: [A] Microscopy of cavitatory lesion shows fungal ball composed of long slender hyphae with acute angle branching (Hematoxylin-Eosin, 100x); [B] GMS special stain highlighting the fungal hyphae; [C & D] Endobronchial mass microscopy showing lobules of hyaline cartilage, endochondral ossification (arrow) and marrow elements within (asterisk). Inset shows nodular cartilage mass lined by respiratory lining epithelium.

Discussion

Tuberculosis is a communicable disease caused by *Mycobacterium tuberculosis*. Pulmonary TB can have long-lasting impacts on lung health, leading to post-TB complications such as fibrosis, bronchiectasis, cavitation, or chronic aspergillosis.

Chronic pulmonary aspergillosis (CPA) is a fungal infection caused by the *Aspergillus* genus, most commonly *Aspergillus fumigatus*. In about 20-40% of patients, a residual cavity remains even after treatment of PTB [1]. These small cavities create an environment for *Aspergillus* spores to colonize and proliferate, potentially leading to the formation of new cavities and subsequent tissue destruction and pleural changes. Sometimes, the fungal hyphae can mix with inflammatory debris and form a fungal ball called an aspergilloma. CPA patients present with chronic cough, weight loss, hemoptysis, and fever which can be confused with a recurrence of PTB, particularly in individuals with a prior history of treated PTB. Radiological findings of cavitation, fibrosis, and pleural thickening are also similar to PTB, making the diagnosis of CPA challenging.

Benign tumors of the bronchial tree include adenoma, hamartoma, papilloma, leiomyoma, and chondroma. Bronchial chondromas are rare benign tumors that originate from the cartilage of the bronchi. These can be solitary in sporadic cases or multiple in the Carney triad (comprising of gastrointestinal stromal tumor, pulmonary chondroma, and paraganglioma). They are more common in men with a mean age of 57 years. Clinically, most patients are asymptomatic, often going undetected until they lead to bronchial obstruction or affect nearby organs. The true incidence of bronchial chondroma is still unknown since cases get misclassified as hamartomas, which is the most common type of endobronchial neoplasm and also its closest differential on histology. Endobronchial chondromas are well-circumscribed, lobulated with a size of <4-centimetre diameter. On microscopy, they display moderately cellular mature cartilage tissue without atypia, and also lack the other mesenchymal elements like adipose tissue, smooth muscle, and entrapped respiratory epithelium that are typically found in hamartomas. The other differential diagnosis includes well-differentiated chondrosarcoma which shows some degree of atypia and invasion. Surgical excision is curative with extremely rare cases showing recurrence or sarcomatous transformation [4]. There is no direct causal relationship established between tuberculosis and the development of endobronchial chondromas, but the two conditions can co-exist and cause misdiagnosis. In patients with a history of tuberculosis, radiologically these are usually thought of as tuberculomas or broncholiths. In our case, the chondroma appeared as a broncholith on HRCT because of its dense calcific and chondroid composition, which radiologically mimics calcified post-tuberculous broncholiths commonly seen in chronic TB sequelae.

CPA often mimics post-tuberculous cavities, especially when radiological features overlap with old, healed TB lesions. The presence of hemoptysis or persistent cavitation may be mistakenly attributed to TB relapse. Microbiological studies are frequently inconclusive, as direct detection of *Aspergillus* in respiratory samples is difficult. Histopathology with special stains like PAS and GMS remains crucial to confirm invasive fungal elements. Clinicians must consider CPA as a differential diagnosis in patients with previous TB who present with chronic respiratory symptoms and cavitatory lesions.

Endobronchial chondromas cause bronchial obstruction leading to poor ventilation and stagnation of mucus secretions, creating an environment that may be favourable for colonization and growth of fungi like *Aspergillus*. Ozyurtkan MO et al. reported a case of multiple pulmonary hamartomas colonized by *Aspergillus* species; however, coexisting invasive pulmonary aspergillosis along with bronchial chondroma has not been reported in the literature to our knowledge [5]. This case underlines the importance of correlating radiological findings with histopathology, as benign endobronchial tumors may mask or coexist with infectious complications like aspergillosis, leading to delayed or incorrect diagnoses.

Conclusion

Post-tuberculosis sequelae are common even after treatment due to various factors. One should be careful and do a thorough evaluation for any respiratory complaints in a known case of tuberculosis. Radiological findings can be ambiguous; hence histopathology remains the gold standard for final diagnosis, paving the way for further management. In this case, we reported a co-existence of two entities—endobronchial chondroma, a rare benign tumor, and chronic cavitary pulmonary aspergillosis with focal invasion which is a known post-PTB complication.

Acknowledgements: Department of Cardiovascular Thoracic Surgery (CVTS) under Dr. Suhas Bendre and Dr. Girija Nair, Dr. D.Y. Patil Medical College and Hospital, Nerul, Navi Mumbai.

Funding: Nil.

Competing Interests: Nil.

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