

ASSOCIATION BETWEEN LUNG ADENOCARCINOMA AND PULMONARY TUBERCULOSIS: A CASE REPORT

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ABSTRACT

The association of pulmonary tuberculosis and bronchogenic carcinoma is relatively rare but serious. This coexistent statement presents diagnostic challenges and raises concerns regarding tolerance to combined antituberculous and antineoplastic chemotherapy, thereby complicating management and potentially worsening the prognosis. This highlights the importance of establishing an early diagnosis. We report the case of a 59-year-old patient with a history of chronic smoking (76 pack-years), who had quit smoking, and occasional alcohol consumption, which had also been discontinued. His symptoms had been present for three months and were characterized by the onset of chest pain, worsening dyspnea, and marked deterioration of his general condition. Thoracic imaging raised suspicion of malignancy, which was subsequently confirmed histologically by biopsies obtained during flexible bronchoscopy. Concurrently, Mycobacterium tuberculosis was detected in bronchial aspirates. The coexistence of pulmonary tuberculosis and bronchogenic carcinoma complicated the therapeutic management of our patient and was associated with an unfavorable clinical outcome.

KEYWORDS: Bronchogenic carcinoma, concomitant pulmonary tuberculosis, brain metastases, antituberculosis drugs, concurrent chemoradiotherapy.

INTRODUCTION

Pulmonary tuberculosis remains a major public health concern, particularly in developing countries. At the same time, lung cancer is a major cause of morbidity and mortality in both developing and industrialized countries. The coexistence of pulmonary tuberculosis and lung cancer is not uncommon, as these two conditions may occur simultaneously or sequentially in the same patient.^[1] Tuberculosis has long been suspected to increase the risk of lung cancer, while lung cancer and its treatment may predispose patients to tuberculosis reactivation.^[2] This coexistence presents diagnostic and therapeutic challenges because of the similarities in the clinical and radiological manifestations of both diseases.^[3]

In this report, we describe a case of concomitant pulmonary tuberculosis and bronchogenic carcinoma in a patient with a history of smoking and alcohol consumption.

CASE PRESENTATION

This is the case of a 68-year-old man with a 76 pack-year history of chronic tobacco use, who had quit smoking 4 years earlier. He reported occasional alcohol consumption, which had been discontinued 10 years previously. He had a 2-year history of chronic dyspnea and had never been treated for pulmonary tuberculosis, with no known recent history of tuberculosis exposure among close contacts.

His symptoms had begun 3 months prior to admission, with a progressive worsening of dyspnea, reaching grade III according to the modified Medical Research Council (mMRC) scale. This was associated with the onset of moderate-intensity, burning left lateral chest pain. There was no bronchial syndrome nor hemoptysis, and no other associated extrapulmonary symptoms. The clinical course occurred in the absence of fever and was accompanied by deterioration of general condition and night sweats.

Clinical examination revealed a patient in relatively good general condition (performance status: PS at 1), with a lean body habitus (body mass index [BMI]: 17.5 kg/m²; weight: 51 kg), mild digital clubbing, and poor oral and dental hygiene. The cardiopulmonary examination was unremarkable, as was the remainder of the physical examination.

A posteroanterior chest radiograph showed a dense, heterogeneous opacity in the left hilar/superhilar region, with spiculated outer margins, on a hyperinflated chest (**Figure 1**). Contrast-enhanced chest computed tomography (CT) in the mediastinal window revealed a left upper lobe mediastino-pulmonary mass associated with impaired left lung ventilation and mediastinal lymphadenopathy (**Figure 2**). The lung parenchymal window confirmed the previously described mass, with no pulmonary nodules, and showed a few areas of traction bronchiectasis with a residual/sequelae appearance, in the setting of emphysematous lung changes (**Figure 3**).

The complete blood count revealed neither neutrophilic leukocytosis nor anemia. Testing for *Mycobacterium tuberculosis* DNA by GeneXpert assay on induced sputum samples was negative.

Flexible bronchoscopy revealed a tumor-like infiltrative lesion involving the posterior aspect of the left upper lobe bronchial orifice, associated with diffuse grade 1 inflammatory changes throughout the bronchial tree. GeneXpert testing of bronchial aspirates was positive, with no rifampicin resistance detected. Histopathological examination of bronchial biopsy specimens obtained from the left upper lobe infiltrative lesion revealed a moderately differentiated, invasive non-small cell carcinoma expressing TTF-1, with negative P40 staining, consistent with a primary non-mucinous pulmonary adenocarcinoma. The patient was placed in isolation and initiated on antituberculosis drugs according to the 2RHZE/4RH regimen.

As part of the staging work-up, an 18F-FDG PET/CT scan was performed one month after initiation of antituberculosis drugs. It revealed a left upper lobe mediastino-pulmonary mass measuring 63 × 61 × 46 mm, with intense hypermetabolic activity (SUV_{max} 10.23; liver SUV_{max} 2.77), associated with a central hypometabolic area, most likely corresponding to necrosis. The lesion was contiguous with an anterior band of non-hypermetabolic atelectasis (**Figure 4**). It was also associated with hypermetabolic mediastinal lymphadenopathy involving the left lower paratracheal station (SUV_{max} 3.13) (**Figure 5**), the prevascular station (SUV_{max} 1.81), the para-aortic station (SUV_{max} 2.63), and the pre-carinal station (SUV_{max} 2.03). Brain CT showed no abnormalities. The disease was staged as T4N2bM0 (stage IIIB).

The case was discussed at a multidisciplinary tumor board, and the decision was made to initiate concurrent chemoradiotherapy one month after the initiation of antituberculous therapy.

The clinical course was favorable after one month of antituberculosis drugs, with recovery of appetite and weight (54 kg vs 51 kg) and a persistently good performance status (PS) of 1.

After 7 months, the patient had received a total of seven cycles of neoadjuvant chemotherapy, followed by concurrent chemoradiotherapy (CCRT), consisting of 33 fractions of radiotherapy combined with two cycles of chemotherapy (vinorelbine plus cisplatin). The clinical course remained favorable, with a maintained PS of 1 and no significant respiratory symptoms apart from exertional dyspnea. Radiologically, a contrast-enhanced thoraco-abdomino-pelvic CT scan (CT-TAP) was performed, demonstrating persistent left upper lobe mediastino-pulmonary disease, with a slight decrease in size, measuring 52 × 41 × 43 mm compared with the initial dimensions of 63 × 61 × 46 mm, in the setting of an emphysematous lung.

Five months later, the patient presented to the oncology emergency department with impaired consciousness and vomiting. An emergency brain MRI revealed a right parietal metastatic lesion with target sign enhancement following gadolinium administration (**Figure 6**), with marked surrounding vasogenic edema (**Figure 7**). The patient subsequently underwent four sessions of stereotactic brain radiotherapy. Molecular testing for EGFR and ALK mutations, along with an assessment of PD-L1 expression, yielded negative results. Three months later, the patient died.

The distinctive feature of this case was the concomitant occurrence of bronchogenic carcinoma and pulmonary tuberculosis in the same patient, which complicated the management and was associated with a poorer prognosis.

Figures



Figure 1: Chest X-ray showing a dense, heterogeneous opacity in the left upper hilar region, with spiculated outer margins, on a distended chest.

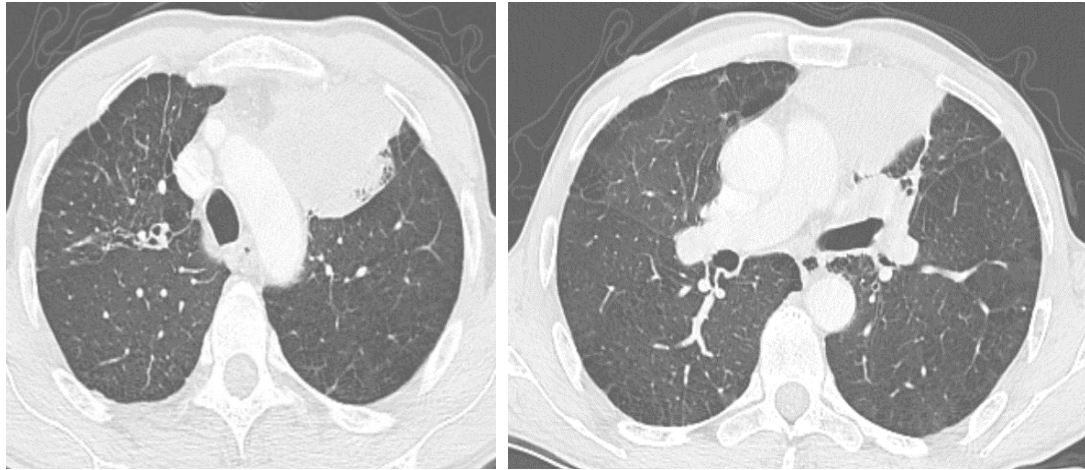


Figure 2: Chest CT scan (mediastinal window) showing a left upper lobe mediastinal–pulmonary mass associated with left-sided ventilation impairment.

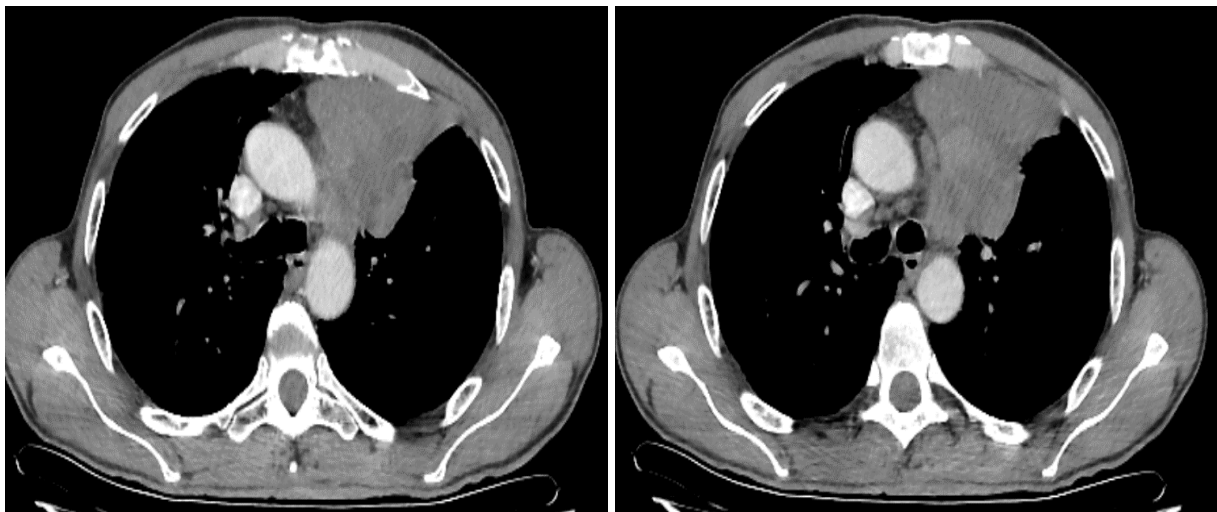


Figure 3: Chest CT scan (lung window) showing the previously described mass, with no pulmonary nodules, along with a few areas of traction bronchiectasis of presumed sequelae on a background of emphysematous lung changes.

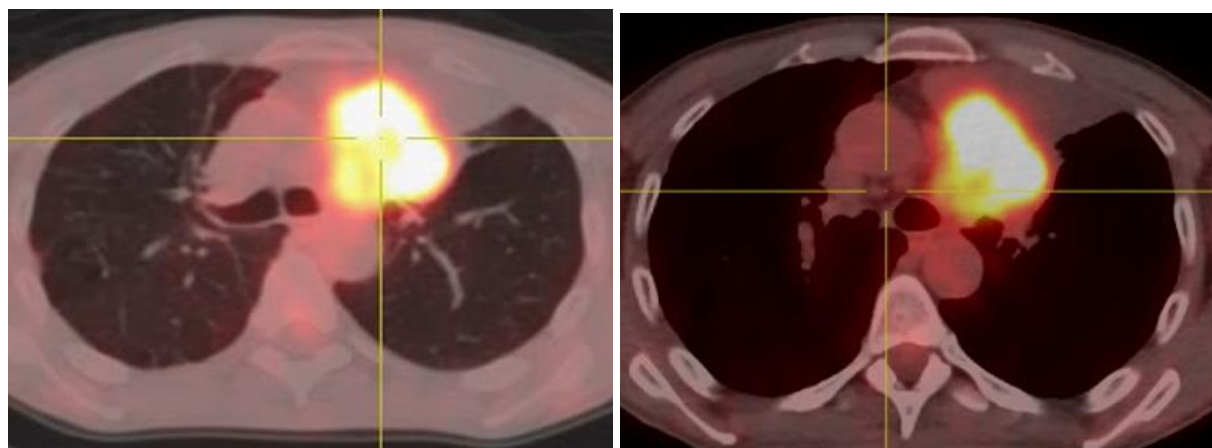


Figure 4: PET Scan demonstrating a left upper lobe mediastinal–pulmonary mass with intense hypermetabolic activity (SUVmax 10.23; liver SUVmax 2.77), associated with a central hypometabolic area, likely reflecting necrosis. The mass is contiguous with an anterior band of non-FDG-avid atelectasis.

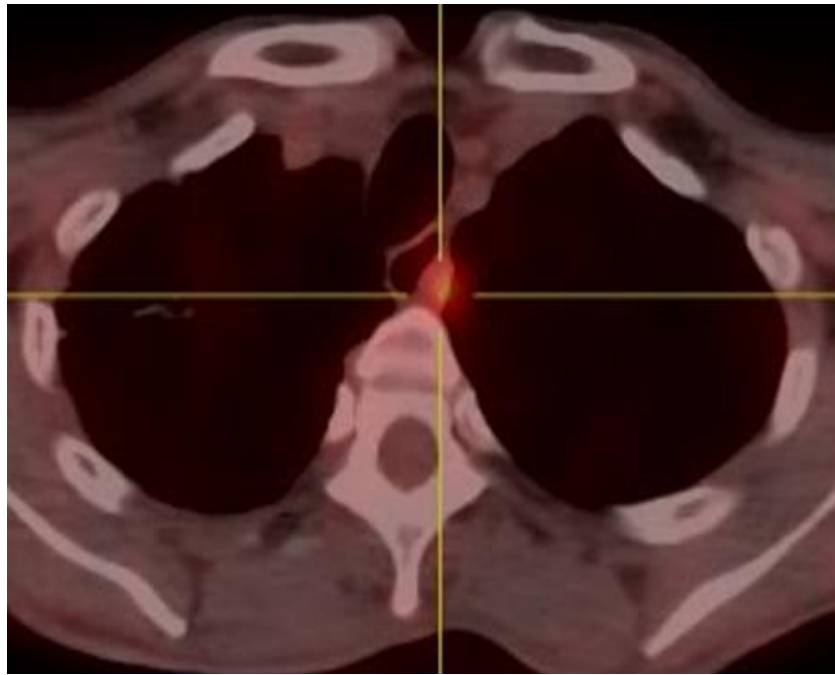


Figure 5: PET Scan showing a hypermetabolic left lower paratracheal mediastinal lymph node, with a maximum standardized uptake value (SUVmax) of 3,13.

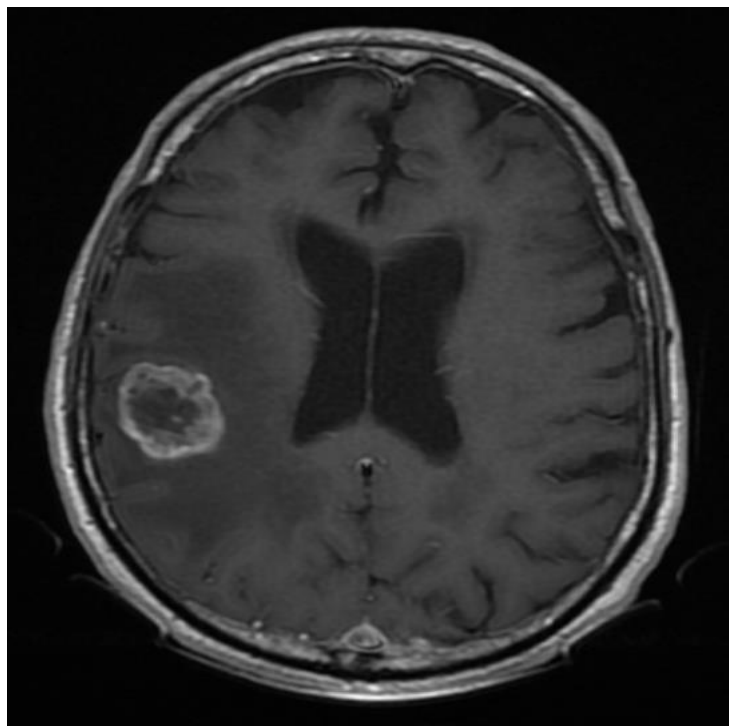


Figure 6: Brain MRI: Axial T1-weighted sequence after gadolinium administration, demonstrating a right parietal lesion with target sign enhancement.

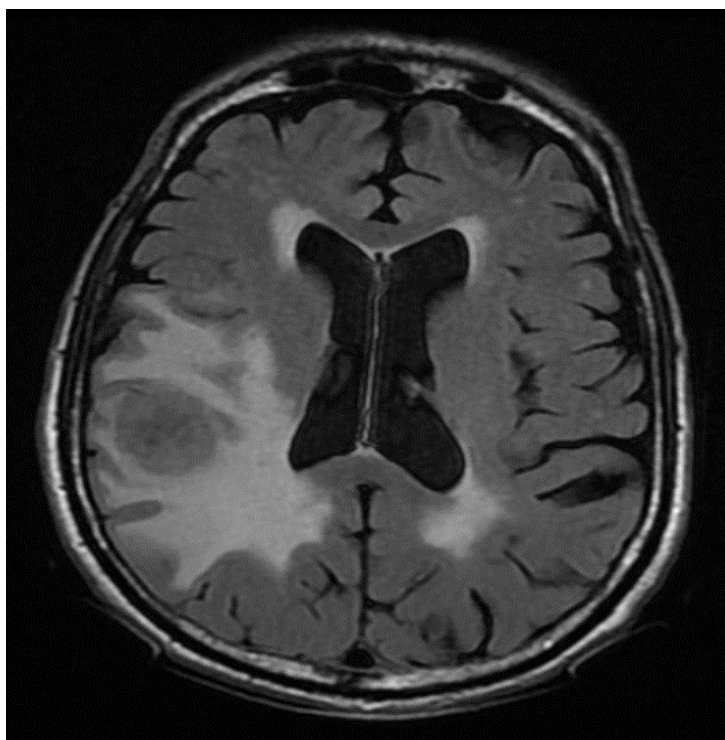


Figure 7: Brain MRI: Axial T2 FLAIR sequence demonstrating marked surrounding vasogenic edema.

DISCUSSION

The relationship between pulmonary cancer and pulmonary tuberculosis is complex. It is estimated that 1–2% of bronchial cancers are associated with tuberculosis, whereas tuberculosis is associated with bronchopulmonary cancer in 1–5% of cases.^[4] The simultaneous diagnosis of tuberculosis and bronchial cancer, as observed in the present case, is relatively uncommon. In most cases, these two diseases are diagnosed sequentially.^[5]

A study conducted in Taiwan comparing a population of 16 487 patients with cancer with a control population of 65 948 individuals demonstrated a higher incidence of tuberculosis among patients with cancer (339/100 000 inhabitants vs 202/100 000 inhabitants).^[6] Numerous studies have suggested an increased risk of lung cancer among patients with a history of tuberculosis. In particular, a meta-analysis including 37 case-control studies and 4 cohort studies demonstrated a significant association between tuberculosis and bronchopulmonary adenocarcinoma (relative risk [RR] = 1.6), whereas no significant association was found with squamous cell carcinoma or small-cell lung carcinoma.^[7]

When active tuberculosis occurs concomitantly with cancer, the clinical presentation is often dominated by a deterioration in general condition, which may manifest as prolonged fever, dyspnea, productive cough, sometimes accompanied by hemoptysis, as well as weight loss.^[8] However, the clinical presentation remains nonspecific due to the overlap in clinical manifestations between the two conditions, which may complicate the diagnosis and delay appropriate management.^[9]

Thoracic imaging findings are also nonspecific. Chest radiography may reveal suggestive abnormalities; however, it generally cannot, on its own, reliably distinguish active tuberculosis from bronchogenic carcinoma. Chest CT is more sensitive and may demonstrate a soft-tissue mass, cavitary lesions, or other parenchymal abnormalities. In a patient being followed for bronchogenic carcinoma, the development of centrilobular micronodules with a “tree-in-bud” pattern, cavitary lesions, or necrotic lymphadenopathy may suggest concomitant tuberculosis.^[10]

Thus, tuberculosis may mimic bronchogenic carcinoma, both clinically and radiologically, while the coexistence of the two conditions may also result in the underlying malignancy being overlooked.^[11] Therefore, in the event of persistent or worsening clinical symptoms, the possibility of concomitant tuberculosis and cancer should be considered, particularly as some malignant lesions may contain tuberculoid granulomas, underscoring the importance of pursuing further investigations. In cases of diagnostic uncertainty, histopathological examination and microbiological culture are essential to establish the definitive diagnosis.^[12]

Medical treatment options for lung cancer include chemotherapy, radiotherapy, immunotherapy, and targeted therapy. These treatments may be associated with drug–drug interactions and may exacerbate the adverse effects of antituberculous drugs, including skin rashes, hepatotoxicity, and renal impairment. Furthermore, cancer-related frailty may prevent completion of tuberculosis treatment, leading to

complications such as disease recurrence and increased mortality^[13], as was the case in our patient.

In both conditions, pulmonary rehabilitation is an essential component of comprehensive management, although its role remains frequently underestimated or insufficiently incorporated into therapeutic strategies. Smoking cessation at the time of diagnosis or during the management of lung cancer is also an important component of pulmonary rehabilitation programs.^[14] Smoking cessation has been consistently and significantly associated with improved overall survival in patients with lung cancer, regardless of histological subtype.

Regarding surgical management in patients with concomitant bronchogenic carcinoma and pulmonary tuberculosis, recommendations remain limited, with reported intervals ranging from 2 to 4 months. According to the Japanese Society for Tuberculosis, surgery may be considered after 4 weeks of antituberculosis drugs, provided that sputum smear microscopy or culture has become negative. In emergency situations, particularly in cases of significant hemoptysis or signs of compression, surgery may be performed without waiting for this interval, following multidisciplinary discussion and careful assessment of the potential benefits and risks.^[15]

From a prognostic perspective, patients with concomitant bronchogenic carcinoma and tuberculosis are often diagnosed with cancer at an advanced stage; approximately 30–50% of patients may present with stage IV disease. Overall, this coexistence appears to be associated with a poorer prognosis than that observed in patients with lung cancer alone. Death in these patients is most commonly related to progression of the underlying malignancy.^[10]

CONCLUSION

Bronchopulmonary cancer and tuberculosis (TB) represent two major public health challenges, and their coexistence poses significant diagnostic and therapeutic difficulties. Tuberculosis may contribute to pulmonary carcinogenesis through chronic inflammation, while cancer and its treatments may increase the risk of TB reactivation or de novo infection. The overlapping clinical and radiological features of these two conditions may delay diagnosis, whereas the concomitant use of anticancer and anti-tuberculosis therapies increases the risk of drug-related toxicity and pharmacological interactions, particularly with rifampicin. A multidisciplinary approach, combined with close clinical and therapeutic monitoring, is therefore essential to optimize the management of patients with concomitant lung cancer and tuberculosis.

Conflict of Interest: The authors declare no conflict of interest.

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