



Tuberculosis and Lung Cancer: The Challenge of Approach and Differential Diagnosis

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Abstract

Tuberculosis and lung cancer may co-exist and even increase the risk for each other, however this relationship and prevalence is not entirely known. We describe a case of a 57-year-old woman that presented with neurological symptoms and whose diagnostic work-up revealed meningeal infection by Mycobacterium tuberculosis complex. Despite antibacilar treatment, she had no clinical improvement and persisted with bilateral nodular pattern on chest-computed tomography. Bronchoscopy with bronchial biopsies revealed lung adenocarcinoma. This case emphasizes the diversity of tuberculosis presentation, including coexistence with lung neoplasms

Introduction

Tuberculosis is one of the most important inflammation-inducing factors in the lungs and has a profound role in the emergence of cancer [1]. Yu et al. [2] presented a cohort study of more than 700 000 subjects older than 20 years and free from cancers, and compared the incidence of lung cancers between tuberculosis and non-tuberculosis cohorts until 2007 and they showed an incidence of lung cancers 11-fold higher in the cohort of patients with tuberculosis [2].

Inflammation and fibroses caused by tuberculosis are crucial to predispose development of lung cancer [3]. On the other hand, neoplasms predispose to a malnutrition state and deterioration of immunity and chemotherapy and radiotherapy are likely to have a role in tuberculosis infection or reactivation [3,4].

The complex relationship between tuberculosis and cancer represents a challenging task for diagnosis and adequate treatment, especially in countries with a high tuberculosis burden.

Case Presentation

A 57-year-old black woman, previous smoker (65 package-year), presented in the emergency service due to sudden onset of neurological changes, namely dizziness, changes in speech and behavior associated with intense bilateral parietal headache. She also referred dry cough, night

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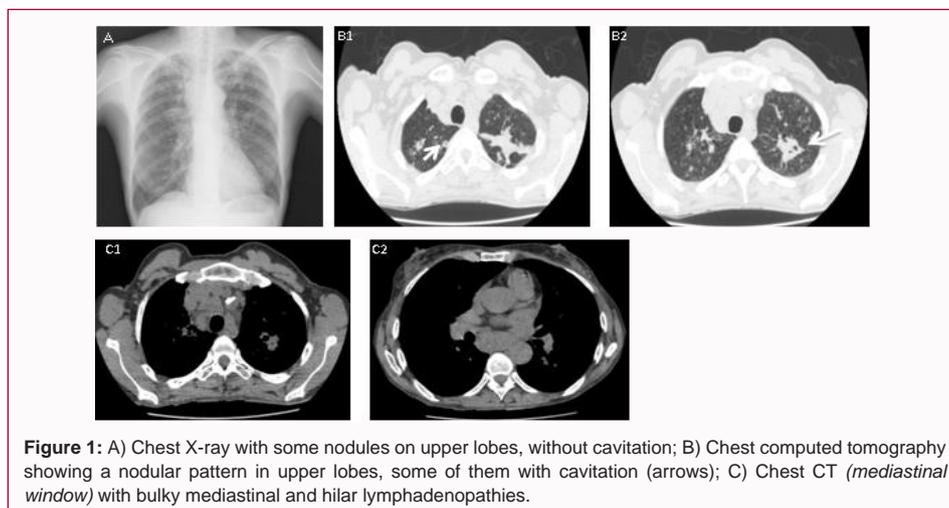


Figure 1: A) Chest X-ray with some nodules on upper lobes, without cavitation; B) Chest computed tomography showing a nodular pattern in upper lobes, some of them with cavitation (arrows); C) Chest CT (mediastinal window) with bulky mediastinal and hilar lymphadenopathies.

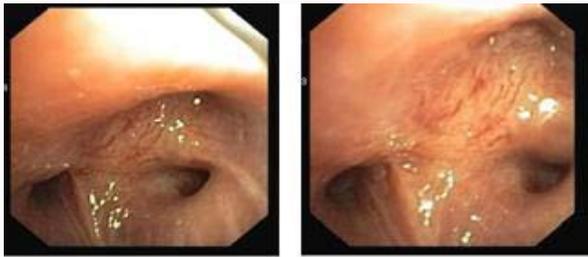


Figure 2: Bronchoscopy with signs of hypervascularization and edema of the bronchial mucosa at the right upper lobar bronchus.

sweats and weight loss quantified in 10 kg during last three months. Physical examination was normal other than anomia and dubious dysarthria. Blood samples were unremarkable. Chest X-ray showed discrete bilateral apical nodular infiltrates (Figure 1A) and chest Computed Tomography (CT) (Figure 1B) confirmed a bilateral micro nodular pattern of random distribution associated with larger nodules in both upper lobes, some partially cavitated in addition to bulky mediastinal and hilar lymphadenopathy (Figure 1C). Brain CT and magnetic resonance showed signs of microangiopathic subcortical leukoencephalopathy and some changes attributed to an infectious process. A lumbar puncture was performed which cultural exam revealed *Mycobacterium tuberculosis* complex and so she started treatment with antibacilars and steroids with slightly clinical improvement in neurological complaints. However despite antibacilar treatment, she overcomes a global deterioration with persistent anorexia and weight loss. To exclude other possible diagnosis, a bronchoscopy was performed and revealed a total occlusion of the apical segment at right upper lobar bronchus and decreased caliber of the anterior and posterior segments, due to mucosal edema (Figure 2). Bronchial biopsies were compatible with TTF-1 positive adenocarcinoma cells favoring pulmonary origin without presence of targeted mutations. Staging neoplastic disease, at this time, was not possible due to presence of lesions of uncertain origin. After three months of treatment, imagiological reevaluation showed dimensional regression of brain injuries and some of the lung lesions (Figure 3). Multidisciplinary group discussion admitted infectious origin of brain lesions. Under the confirmed neoplastic diagnosis a whole-body 18-fluor-deoxyglucose Positron Emission Tomography (18-FDG-PET) was performed and revealed hypermetabolic activity in the right upper lobe mass (SUV 14) and mediastinal and right supraclavicular adenopathies (SUV 5). She underwent Endobronchial Ultrasound Transbronchial Needle Aspiration (EBUS-TBNA) with negative results concerning malignancy and infectious disease. A video-mediastinoscopy was then performed and documented neoplastic cells in 4R ganglionar group (N2 disease). Therapeutic decision favored standard chemotherapy with carboplatin and vinorelbine. Despite initial good response, disease progression occurred and patient started second line of chemotherapy with docetaxel with no response and poor tolerance due to adverse effects, leading to hospitalization for palliative care. She died about 15 months after the initial diagnosis.

Discussion

The relationship between tuberculosis and lung cancer has brought speculation and investigation for many years [5,6]. Bayle made the first description of co-existence of tuberculosis and lung cancer in 1980 [6]. This occurrence may be explained by the increased risk that each individual entity can input to each other [4]. Wu et

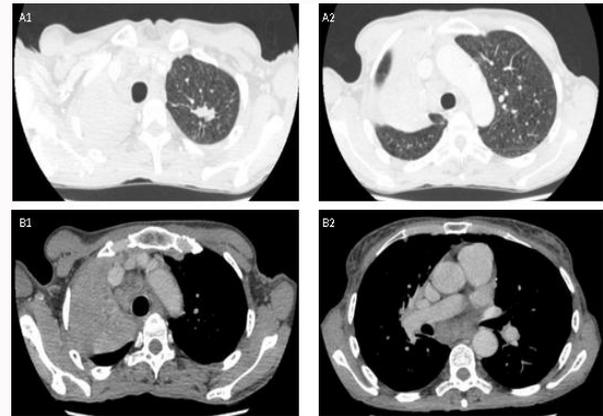


Figure 3: Chest CT after three months of antibacilar treatment showing: (A1-A2) complete collapse of the right upper lobe by occlusion of the respective lobar bronchus probably related to primary proliferative lesion. Changes in the left upper lobe with some nodules reduced in length and probably related to the infectious process; (B1-B2) Mediastinal window of chest CT showing adenopathies with slightly reduction compared to the previous exam.

al. [7] demonstrated in a population cohort study that pulmonary tuberculosis is a risk factor for subsequent lung cancer and tuberculosis patients should be carefully monitored for lung cancer [7]. In our case, clinical presentation was suggestive of neurological involvement and although the presence of a nodular pattern in chest CT, detection of *M. tuberculosis* stains in CSF became meningeal-pulmonary tuberculosis the main diagnosis. The suspicion for another co-existing diagnosis emerged from the fact that there was no clinical improvement along with CT scans alterations that imposed a differential diagnosis. In these particular situations, it may be appropriate to pursue investigation towards alternative diagnosis [8,9]. As lung neoplasm was established the main challenge was staging it, knowing that tuberculosis can also present with high metabolic activity on 18-FDG-PET [9]. According to the recommendations, we waited until completion of three months of treatment to reevaluate the response and staging with more accuracy.

Despite initial good response to chemotherapy, our patient had an unfavorable course with progression of neoplastic disease. On the other hand, the immunosuppression state caused by treatments and disease itself there was no evidence for tuberculosis worsening or reactivation. In conclusion, tuberculosis can simulates some of the radiological features and symptoms of lung cancer [3,9,10]. The delay in the diagnosis of lung cancer is substantial and a high index of suspicion is necessary, especially in low-burden developed countries [3]. Cancer awareness in patients with previous or suspected tuberculosis should be made in trying to increase the number of potentially resectable tumors.

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