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Case report

Pulmonary tuberculosis masking lung cancer – A case report



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ABSTRACT

Pulmonary Tuberculosis can co-exist with lung malignancy masking the underlying disorder leading to delay in diagnosis and management. Here we present an interesting case of a 60 year old man who on initial presentation was diagnosed with tuberculosis but on nonresponse to therapy and investigation was found to have an underlying lung malignancy.

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1. Introduction

Adenocarcinoma with lepidic pattern formerly called as Bronchioalveolar cell carcinomas (BACs), a subset of primary lung adenocarcinomas, are a diverse group of malignancies that share the pathologic feature of absence of well formed glands and lepidic growth spread along alveolar septa without stromal, pleural and vascular invasion [1]. Adenocarcinoma with lepidic pattern are pathologically classified into two subtypes: mucinous, non-mucinous [2]. Non mucinous type show ultrastructural resemblance to type II pneumocytes of Clara type are usually solitary nodules and have very good prognosis, mucinous type tend to spread and form satellite tumors or pneumonic consolidation and thus have a worse prognosis [3]. The incidence of bronchioalveolar cell carcinoma (BAC) has increased significantly from less than 5% to 24% of total lung cancers [4].

Pulmonary tuberculosis has been found in 0.7% cases of lung cancer [5]. The presence of lung carcinoma is rarely suspected in patients with active tuberculosis and the diagnosis of lung cancer may be delayed because of masking by a tuberculous lesion [6].

We present a case of an elderly male who was initially suffering from pulmonary tuberculosis but on further investigation was diagnosed to have co-existent Adenocarcinoma with lepidic pattern.

1.1. Case study

A 60 year old man, presented to our outpatient department with complaints of cough and expectoration for the past 7–8 months. The expectoration was around 100 ml/day, mucoid and non foul smelling. There was no history of associated hemoptysis. Patient did not complain of chest pain, breathlessness, fever, weight loss or anorexia. Patient was suffering from non-insulin dependent diabetes mellitus and hypertension which were under control with oral medication. Patient was a smoker with a 30 pack year history.

The physical examination revealed a moderately built, well nourished elderly male who was comfortable at rest. The vital parameters were within normal limits. There was no clubbing of the digits, no cyanosis and no lymphadenopathy. Systemic examination of the respiratory system revealed right infrascapular coarse crepitations. Remainder of the physical examination was unremarkable.

The routine blood hemogram, liver function test, renal function test were within normal limits. Chest X-ray PA view (Fig. 1) revealed air space consolidation involving the right lower and mid zones and parts of the left lower zones. Non contrast CT revealed

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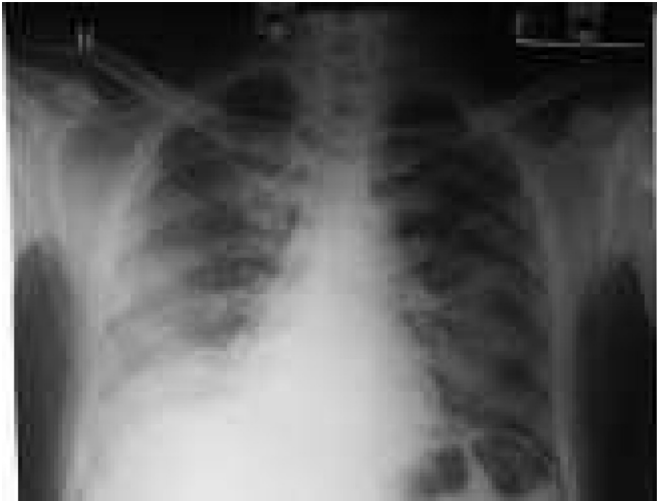


Fig. 1. Chest X ray showing right mid zone and lower zone homogenous opacities.

consolidation with air bronchogram in the right lower lobe and lateral basal segment of left lower lobe. There was no evidence of pleural effusion and mediastinal lymphadenopathy (Fig. 2).

A working diagnosis of bilateral pneumonia probably of tuberculous etiology was made. Sputum for AFB was done twice and it was negative. We proceeded with a diagnostic bronchoscopy. There were no endobronchial lesions. Bronchial wash from the right lower lobe revealed numerous acid fast bacilli.

Patient was started on a 4 drug anti tuberculous regimen and was asked to follow – up at a DOTS clinic.

Patient came back after 6 months with complaints of increased cough and expectoration of around 300 ml of thin watery sputum. There had been two or three episodes of streaking hemoptysis in the intervening period. There was no history of fever, dyspnea, weight loss or anorexia. The physical examination was similar to the earlier one with the sole difference being an increase in coarse crepitations in the infrascapular area and interscapular areas bilaterally. The patient insisted that he had continued taking anti tuberculous medication, which was verified with his DOTS card and

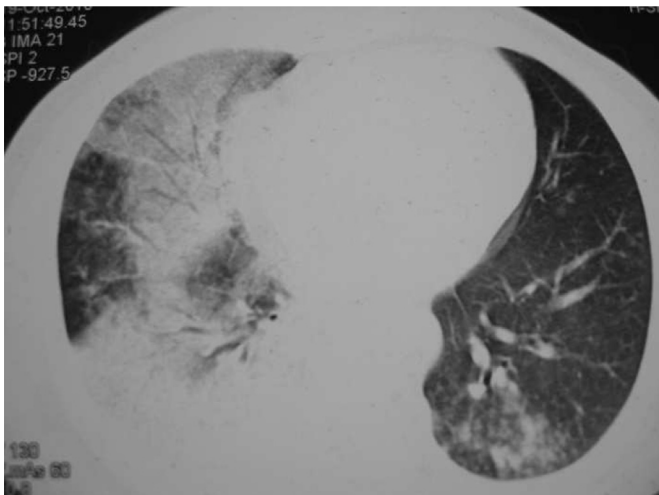


Fig. 2. CT chest showing air space opacity in the right lower lobe.

though there was initial improvement, his condition had deteriorated in the past 2 months.

Chest X-ray PA (Fig. 3) of the patient showed that the disease had worsened with the consolidation involving the lower, mid and some parts of the upper zones bilaterally. CT scan (Fig. 4) of the thorax was repeated which revealed bilateral consolidation involving the lower lobes, middle lobe and some parts of the upper lobe with air bronchogram. There was no evidence of cavitation, pleural effusion or mediastinal lymphadenopathy. A working diagnosis of slow resolving pneumonia was made and we proceeded with a bronchoscopy which was non-contributory.

Percutaneous lung FNAC was done using 21G needle which revealed three dimensional clusters of cells with mild pleomorphism. Surgical help was sought from the cardiothoracic team and patient underwent mini thoracotomy and lung biopsy was taken from right lower and middle lobe. Histopathological evaluation (Fig. 5) revealed mucinous type of Adenocarcinoma with lepidic pattern.

Our patient was referred to a cancer speciality hospital and is undergoing a combination of chemo-radiotherapy for his disease.

2. Discussion

Bayle in 1810 was the first to report on the co-existence of pulmonary tuberculosis and bronchogenic carcinoma [7]. Fontenelle et al. reviewed 90 patients over a 12 year period who had co-existent bronchogenic carcinoma and pulmonary tuberculosis. Seventy-one patients (78.8%) had active tuberculosis. One third of the 90 patients underwent thoracotomy and lung biopsy for diagnosing cancer [8].

Ting et al. proposed several plain radiographic features which should increase the suspicion of coexisting lung carcinoma in a patient with pulmonary tuberculosis the foremost of which was progression of pulmonary infiltrates while the patient was on anti tuberculosis drugs as was seen in our patient [9].

In a study to identify factors causing delay in diagnosis of lung cancer, Chandra et al. found that of 123 patients with lung cancer, 23 (17%) had been labeled initially as suffering from pulmonary tuberculosis. But of the 23, only 3 had bronchial wash positive for

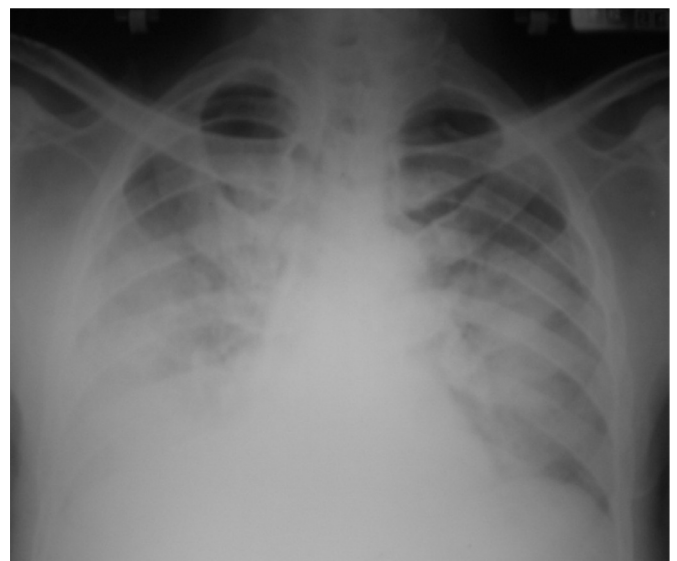


Fig. 3. Chest X ray showing bilateral worsening consolidation.



Fig. 4. CT chest showing bilateral air space opacities with air bronchogram.

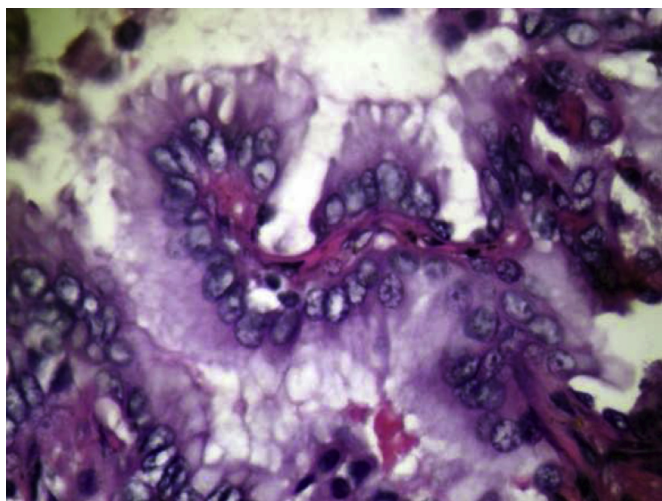


Fig. 5. Adenocarcinoma with lepidic pattern and intracytoplasmic mucin 400 \times , H&E stain.

tuberculosis [10]. In our case we had initially isolated AFB using ZN staining from the bronchial wash which was subsequently negative with treatment.

Our patient was a male smoker who had symptoms of bronchorrhoea but was not dyspneic despite extensive disease. He also had the diffuse infiltrative variety of BAC which is a less common form of presentation and has a poorer prognosis.

Wu et al. [11], proposed the theory of 'reverse causality' which explains that occult lung cancer may provoke reactivation of latent TB infection by weakening host immune mechanisms and the lung cancer is usually diagnosed in the 6–9 months of TB treatment.

3. Conclusion

In patients with non response to anti tubercular therapy and radiological worsening, a high index of suspicion has to be maintained and it is rewarding to go that extra mile, in the form of invasive procedures, in trying to establish an alternative diagnosis.

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