Case Report

ID: JCDR/2012/4067:2352

Oncology Section

Spontaneous Pneumothorax in Squamous Cell Lung Cancer

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ABSTRACT

Spontaneous Pneumothorax (SP) is usually attributed to the rupture of the sub-pleural blebs or bullae. Spontaneous pneumothorax is rarely seen in association with lung cancer. Pneumothorax can be the first sign of lung cancer. The most common possibility for SP complicating lung cancer is the tumour necrosis mechanism or, in separate cases, the rupture of the emphysematous bullae. Lung cancer should always be considered as a possible cause of SP in elderly patients or in heavy smokers. We are reporting here a case of central bronchial carcinoma with spontaneous pneumothorax, as it is a very rare complication of primary bronchogenic carcinoma.

Key Words: Pneumothorax, Spontaneous, Lung cancer

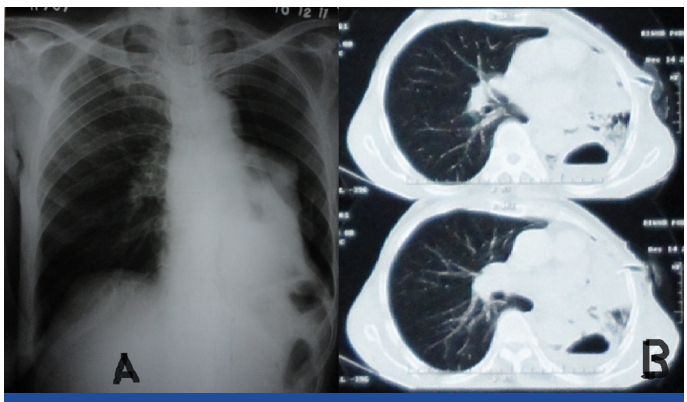
INTRODUCTION

The patients with lung cancer can present in many different ways. A majority of the lung cancers present as a result of the investigation of some new respiratory symptom or because the pre-existing respiratory state of the patients has worsened [1]. A small percentage of the patients present with no symptoms, few present with non-specific symptoms of malignancy and lastly, few present with symptoms of metastasis.

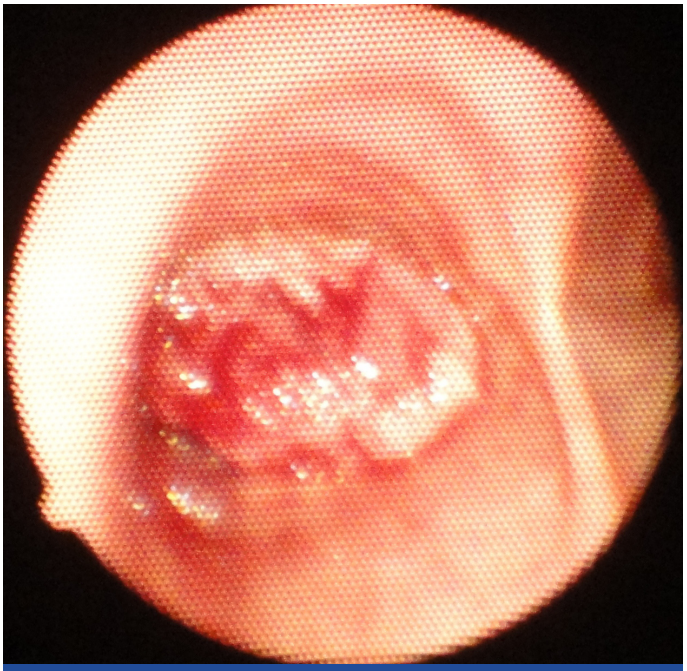
Spontaneous pneumothorax is generally attributed to a rupture of the sub-pleural blebs or the emphysematous bullae [1]. This can complicate the primary or the secondary lung tumours. Spontaneous pneumothorax as a presenting manifestation in primary lung tumour or lung metastasis is very rare. We are reporting here, a case of lung cancer with spontaneous pneumothorax as a presenting manifestation.

CASE REPORT

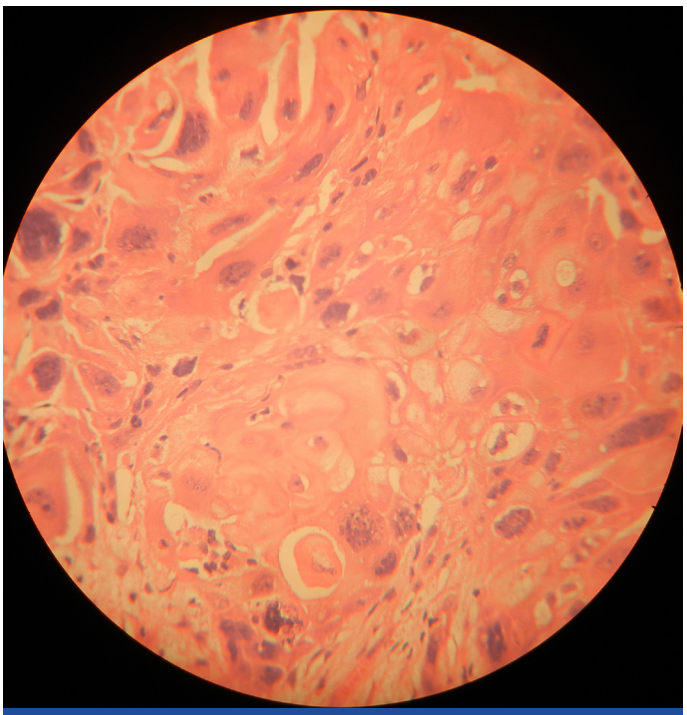
A 65-year aged farmer was suffering from cough with scanty mucoid expectoration, dull aching chest pain, anorexia and weight loss for 4 months. There also was a presence of haemoptysis on 3 occasions during this period, which subsided on conservative treatment with oral antibiotics. Suddenly, on a day when he was working on his field, there was a bout of cough followed by acute shortness of breath, for which he was admitted to our department. He was a beedi smoker for 50 years, with a smoking index of 1000. He had no past history of any anti-tubercular drug intake or any other significant past medical or surgical history. On admission, he was found to have MMRC grade IV dyspnoea with cyanosis and a hyper-resonant note on percussion on the left side of the chest, with mediastinal shifting to the opposite side. An urgent chest X-ray was done, which revealed a left sided pneumothorax and compensatory hyperinflation of the right lung, with a transmediastinal herniation to the left side [Table/Fig-1(A)]. Immediately, an intercostal chest drain was put on the left side in the 4th intercostal space, with water seal drainage. The sputum smears which were made to check for Acid Fast Bacilli (AFB) and malignant cells, both were negative for 4 consecutive days. On repeat chest X-ray, the presence of a left sided collapse with a rounded homogenous opacity was observed. A CT scan of the thorax with contrast revealed left upper lobe collapse with a fluid filled cavity in the left lower lobe, which was surrounded by consolidation and compensatory hyperinflation of the right lung, with a transmediastinal herniation to the left side [Table/Fig-1(B)]. There was no pneumothorax after putting the intercostal chest tube on the CT scan of the thorax. On spirometry, the presence of irreversible moderate obstructive airway disease without reversibility the post-bronchodilator pulmonary function parameters were FEV_1/FVC-68%, FEV_1-72% and FVC 80%) was observed. Fiber Optic Bronchoscopy (FOB) showed a cauliflower like reddish growth occluding the left main bronchus within 2 cm of the carina [Table/Fig-2]. Bronchial brush cytology demonstrated endobronchial cells along with inflammatory cells, metaplastic squamous cells, histiocytes, red blood corpuscles and a few discretely lying squamous cells with pleomorphic hyperchromatic nuclei. A biopsy from the growth revealed an infiltrating tumour which was composed of large epithelial cells with focal cellular keratinization, necrosis and stromal desmoplasia with severe



[Table/Fig-1]: Chest X-ray PA view (A) showing left sided pneumothorax and CT scan thorax with contrast (B) showing left upper lobe collapse with lower lobe fluid filled cavity with consolidation of surrounding parenchyma and compensatory hyperinflation of right lung with transmediastinal herniation to left side.



[Table/Fig-2]: Fiber optic bronchoscopy showing reddish cauliflower like growth occluding the left main bronchus.



[Table/Fig-3]: Biopsy specimen of intra-bronchial tumour showing large epithelial cells with focal cellular keratinization, necrosis and stromal desmoplasia with severe nuclear pleomorphism suggestive of squamous cell carcinoma (H &E, 400x).

nuclear pleomorphism, atypical mitosis and occasional bizarre tumour giant cells with no granuloma, which were suggestive of a large cell undifferentiated carcinoma, probably developing on a background of squamous cell carcinoma [Table/Fig-3]. On immunohistochemistry, the biopsy material was found to be positive for the cytokeratins, 5, p63 and 34 beta E12, but to be negative for the thyroid transcription factor 1. The immunohistochemistry pattern of the tumour ruled out the possibility of a large cell carcinoma and established the diagnosis of a squamous cell carcinoma. So, there was no question of a dual carcinoma. The patient was diagnosed as a case of primary squamous cell carcinoma of the lung, who was admitted with spontaneous pneumothorax as the main presenting sign.

DISCUSSION

Spontaneous pneumothorax may be of 2 types, primary and secondary. The primary spontaneous pneumothorax mostly results from the rupture of the sub-pleural emphysematous blebs and the secondary spontaneous pneumothoraxes occur in patients with the acquired immunodeficiency syndrome (AIDS), cystic fibrosis, tuberculosis, lymphangioleiomyomatosis (LAM), and Langerhans cell histiocytosis. But spontaneous pneumothorax as a complication of lung cancer is very rare [2,3].

It has been estimated that only 2% of all the spontaneous pneumothoraxes are coexistent with malignant lung diseases, either primary or secondary. This tumour complication must be especially considered in the older patients [4]. Wright et al., reported that 0.05% of the lung cancer cases which were complicated with pnemothorax and Steinhauslin et al., reported that 0.46% of the lung cancer cases were complicated with pnemothorax [5,6].

But the mechanism by which lung cancer caused spontaneous pneumothorax has not been well understood. Several mechanisms have been proposed. The first is that it may be the result of tumour necrosis-rupture of the necrotic neoplastic tissue in the pleural cavity; the second is that it may be caused by the rupture of the necrotic tumour nodule or by the necrosis of the sub-pleural metastasis. The third is the possibility of a cancer with the check valve mechanism: a tumour at the lung periphery can obstruct the bronchioles and lead to a local over distension and rupture of the lung [7, 8]. The fourth is that most of the patients with lung cancer have chronic bronchitis or emphysema bullae and that these bullae may rupture following the disturbance of the lung architecture due to bronchial cancer [9]. Pneumothorax which was related to therapy has been reported in patients who received chemotherapy and/or radiotherapy for lung cancer [10]. These theories indicate that spontaneous pneumothorax as a complication of lung cancer should not be ignored, especially in the older age group. We are demonstrating that this case report of lung cancer with pneumothorax is a rare complication of primary lung carcinoma.

The diagnosis and the classification of lung neoplasms are complex due to their diverse histopathology and tumour heterogeneity. A large number of immunohistochemical markers have recently become available, which facilitate the accurate diagnosis and the classification of pulmonary neoplasms. Among the specific markers which have been studied for the pulmonary epithelium, the thyroid transcription factor-1 (TTF-1) has received the most attention. Among the non-small cell lung cancers, upto 94% of the adenocarcinomas have been reported to express TTF-1. The small cell carcinomas are predominantly TTF-1 positive (≈ 90%), p63 negative, cytokeratin (CK) 5 negative, and CK8 positive, and the squamous cell carcinomas are typically TTF-1 negative (≈ 90%), p63 positive, CK5 positive, and CK8 negative [11]. The characteristic of classic large cell carcinoma immunophenotype is loss of staining with CK5/6.

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FINANCIAL OR OTHER COMPETING INTERESTS:
None.

Date of Submission: **Jan 28, 2012**
Date of Peer Review: **May 16, 2012**
Date of Acceptance: **Jun 13, 2012**
Date of Publishing: **Aug 10, 2012**