

Small Atypically Redistributed Pleural Effusion in Upper Lobe Collapse: An Auxiliary Differential Feature of Bronchogenic Carcinoma and Pulmonary Tuberculosis

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— Abstract —

We reviewed the computed tomography (CT) of 32 patients with upper lobe collapse to assess the significance of small atypically redistributed pleural effusion (ARPE) in distinguishing the bronchogenic carcinoma (BC) from tuberculosis (TB).

Upper lobe collapse was caused by BC in 21 and by TB in 11 of the 32 patients. Small ARPE was present in 14 of 21 patients with BC and two of the 11 patients with TB. Among 16 patients with small ARPE, CT showed mediastinal invasion in 11 (69%) patients and mediastinal lymphadenopathy in 6 (38%).

Our results suggest that small ARPE associated with upper lobe collapse can be used as an auxiliary sign in the differential diagnosis between BC and TB.

Index Words: Lung, neoplasm 60.3
Tuberculosis, pulmonary 60.23
Lung, CT 60.1211
Lung, collapse 60.74
Pleura, fluid 60.76

INTRODUCTION

The value of CT in diagnosis of BC has been reported by various literatures in the past. Even though CT has been proven superior to plain film for delineating the extent of intrathoracic neoplastic involvement (1), its role in the evaluation and staging of the BC remains incompletely defined (2).

Lobar collapse is frequently encountered in patients with both malignant and benign diseases (3-6). While a triad of obstruction of air way, a central mass causing bulging of the proximal contour and differential enhancement

of tumor versus collapsed peripheral lung was suggested as a useful differential CT sign of malignant from benign obstruction (7), there was no report about the role of small ARPE in differential feature between them. We undertook a retrospective analysis of 32 chest CTs of the patients with upper lobe collapse in the hope of distinguishing the BC from TB as the cause of collapse.

MATERIALS AND METHODS

All the chest CT of upper lobe collapse caused by BC and TB between 1984 and 1991 were retrospectively reviewed. Among a total of

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32 patients, 21 had BC and 11 had TB. Passive lobar collapse by large pleural effusion was excluded. The patients included 22 men and 10 women, 43-86 years (mean 65 years).

CT scans of all patients in our study were performed on the Somatom II, Somatom DR, and Somatom VC1 scanner (Siemens, Erlangen, Germany), using consecutive 8mm collimated scans with 10mm interval. A variety of window widths (300H to 2000H) and window levels (-30H to -800H) was used to visualize the lung parenchyma, bronchi and mediastinum. Intravenous contrast enhancement by a drip infusion of 100ml of Rayvist or Ultravist (Schering, Germany) was performed routinely.

The CT scans were evaluated jointly and agreement was reached by three radiologists (JHL, SHP and MHC) without knowledge of clinical information or pathologic diagnosis. The scans were evaluate for the presence or absence of the small ARPE between collapsed lobe and rib cage, mediastinal invsion and mediastinal lymphadenopathy. Mediastinal invasion was considered to be present if the mass involved the soft tissues of the mediastinum such as the heart, great vessels, trachea, esophgus, and vertebral bodies with loss of fat plane. Mediastinal nodes larger than 1cm in short-axis diameter were considered abnormal. The possible mechanisms of atelectasis were postulated for each patient. The criteria of obstructive atelectasis were the obstruction of the central airway and distension of peripheral small airways with retain secretions (mucous bronchogram). The criteria of cicatrization atelectasis were the patent central airway and distended air filled peripheral aiways throughout the collapsed lobe.

Diagnosis of 23 patients was made by fiberoptic bronchoscopic biopsy, one by surgical pathology, three by sputum acid fast bacillus staining and five by clinical follow-up.

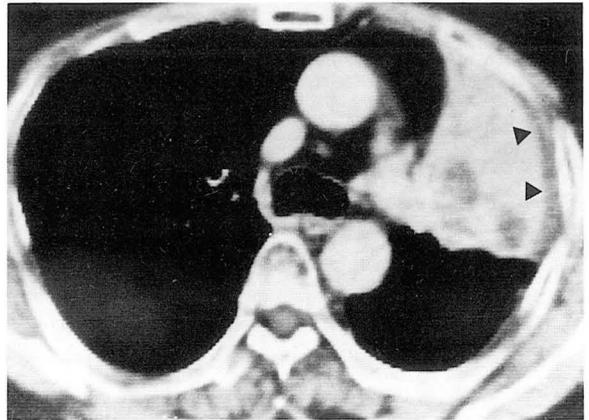


Fig. 1. Left upper lobe collapse due to squamous cell carcinoma.

Section at carina level shows triangular shaped left upper lobe collapse, mucous filled brochi, and small ARPE (arrow heads).

RESULTS

ARPE between the rib cage and collapsed lobe was observed more frequently in the patients with BC than those with TB ($P=0.026$) as 14 (67%) of the 21 patients with BC (Fig. 1) and two (18%) of the 11 patients with pulmonary TB (Fig. 2). Mediastinal invasion was more frequently observed in patents with BC than in those with TB ($P<0.005$) as among the 21 patients with BC, 13 (62%) showed mediasinal invasion while none of the patients with TB showed mediastinal invasion. Enlarged mediastinal ymph node or nodes were seen in 11 (52%) of the 21 patients with BC and 3 (27%) of the 11 patients with TB and The difference was not statistically significant ($P=0.86$).

Since among 16 patients with ARPE, CT showed mediastinal invasion in 11 (69%) patients, and among 16 patients without ARPE CT showed mediastinal invasion in only 2 (13%).

ARPE was observed more frequently in patients with mediastinal invasion than in those without mediastinal invasion ($P<0.005$). CT showed mediastinal lymphadenopathy in 6 (38%) of 16 patients with ARPE and 8 (50%) of 16 patients without ARPE. The correlation be-



Fig. 2. Right upper lobe collapse due to tuberculosis. Section at the level of aortic arch shows right upper lobe collapse, air filled bronchi, and small ARPE (arrow heads).

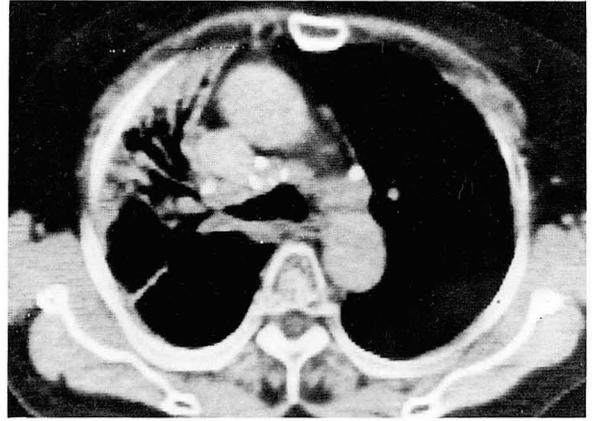


Fig. 4. Right upper lobe collapse due to tuberculosis. Section at aortopulmonary window level demonstrates marked stenosis of the right upper lobe bronchus with wedge shaped collapsed upper lobe. Multiple, dilated air-filled bronchi are seen within the collapsed right upper lobe, suggesting cicatrization atelectasis. Multiple small calcific right tracheobronchial and aortopulmonary nodes are also noted. ARPE is not present.

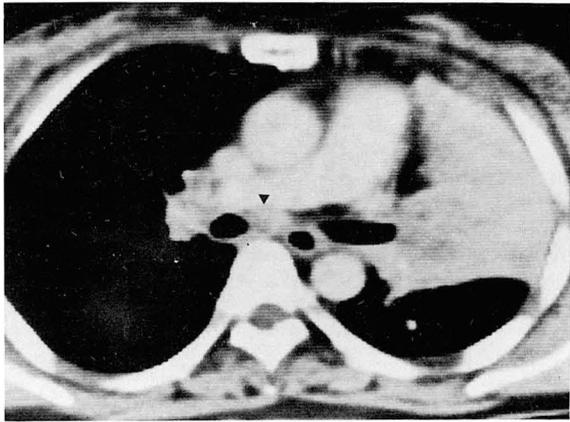


Fig. 3. Left upper lobe collapse due to squamous cell carcinoma. Section at the level of right main pulmonary artery shows left upper lobe collapse and enlarged subcarinal node (arrow head). ARPE is not seen.

tween the mediastinal lymphadenopathy and small loculated pleural effusion was not statistically significant ($P=0.62$, Yates corrected χ^2 test)

While all 21 patients with BC had obstructive atelectasis, only 6 of the 11 patients with TB had obstructive atelectasis, cicatrization atelectasis was seen in the remaining 5 patients.

DISCUSSION

Both endobronchial TB and central BC produce lobar collapse and consolidation. Several articles about endobronchial TB simulating primary BC have been published (8-12). Ip MSM (10) reported that among the patients with endobronchial TB, sputum smear was negative for acid fast bacilli in 85 of the patients and the gelatinous granulation tissue, typical bronchoscopic finding of TB, was frequently not found during bronchoscopy leading to false diagnosis of BC in 30% of the patients (10). Systemic symptom of infection was present in only 50% (10). Although it is very difficult to differentiate endobronchial TB from BC, the differentiation is very important because the former can be treated with antituberculosis chemotherapy, while the latter must be treated with chemotherapy, surgery, adjuvant radiation therapy or a combination of them.

Several CT studies reported high sensitivity

in identifying obstructing carcinoma in patients with segmental or lobar collapse (5,6,13,14), however, they also reported in inevitable high rate of false diagnosis. We observed ARPE in CT scan in order to evaluate its usefulness as an auxiliary CT sign which would lead to improvement in differential diagnosis of BC and TB. Our study shows that small ARPE between the collapsed upper lobe and rib cage on CT has been associated more frequently with BC than TB. Both BC and TB are known to be associated with pleural effusion. The mechanism of pleural fluid formation in the patient with malignancy differs from that with TB. Matthay et al (15) suggested that impaired lymphatic drainage in the pleural space appears to be the predominant mechanism responsible for the formation of pleural effusion associated with malignancy. There are several other causes of paramalignant pleural effusions such as bronchial obstruction, invasion and destruction of the thoracic duct, pulmonary embolism, low plasma oncotic pressure, radiation therapy and drug reaction. Our study shows that the small ARPE is closely associated with mediastinal invasion, although it is not closely related with the mediastinal lymphadenopathy.

Rigby et al (16) found that fluid collects in the dependent portion of the thorax and its distribution is affected by lobar collapse. When air way obstruction causes lobar collapse it creates negative local pressures secondary to distortion of the lung and chest wall. The effusion moves to this locally negative pressure area. This mechanism could explain the presence of small ARPE between the rib and collapsed lobe in our cases. However, the trapping of effusion between the collapsed lobe and chest wall would be possible only when the amount of effusion is very small if the hydrostatic pressure is greater than the locally negative pressure caused by lobar collapse as in the case of large effusion, the effusion would collect in the dependent portion rather than in the nondepen-

dent atypical site. Initially the amount of fluid should be minimal and finally increases massively as the disease progresses. Rigby et al (16) reported that when collapse of lower lobe was induced, effusion of about 2000ml moved medially and posteriorly from the subpulmonary space in an upright dog. When the lobe was re-expanded, the fluid returned to the subpulmonary position. However, collapse of the upper lobe did not alter the subpulmonary distribution. The ineffectiveness of upper lobe collapse in the redistribution of usual subpulmonic pleural effusion in their experiment could be explained by relatively smaller local negative pressure effect compared with the larger hydrostatic pressure caused by large pleural effusion in the upright position. If their experiment had been done in the setting of minimal amount of pleural effusion and upper lobe collapse in the supine position, the fluid might have shifted to upper thorax between the collapsed lobe and chest wall.

The reason for the less frequent appearance of ARPE between the collapsed lobe and chest wall in the patients with pulmonary TB than in those with BC in our study could be explained partly by the presence of 5 cicatrization atelectases in the 11 patients with TB in contrast with obstructive atelectases present in all of the 21 patients with BC. No case of pleural effusion was present in the 5 patients with cicatrization atelectasis. This finding supports the statement of Naidich et al (17) that the absence of demonstrable pleural effusion is a criterion of cicatrization atelectasis. When patient with pulmonary TB presents with cicatrization atelectasis alone without pleural involvement, no pleural effusion would be expected. The tuberculous pleural effusion results from rupture of a subpleural caseous focus in the lung into the pleural space (18). When patients with pulmonary TB presents with endobronchial TB and obstructive atelectasis, pleural effusion could be present depending on the concomi-

tant tuberculous pleurisy. With the presence of TB pleurisy, if the effusion is small, the effusion could be trapped between the collapsed lobe and chest wall in the supine position like in the case of the two patients among 7 patients in our study.

Leung et al (19) suggested the features that were helpful in distinguishing malignant from benign pleural disease such as circumferential pleural thickening, nodular pleural thickening, parietal pleural thickening greater than 1cm, and mediastinal pleural involvement. Since our patients have no such features, small ARPE may not represent malignant pleural disease itself.

When an empyema is present, the attenuation of the extrapleural fat can be increased, reflecting edema or inflammatory cell infiltration in the fat (20,21). The increased density of extrapleural fat can be mistaken for a pleural fluid collection. Since none of our cases had other CT and clinical findings of empyema, such a condition was not possible (20,21).

Major limitation of our study is that the etiology of the small ARPE was not identified by cytological examination. We only speculated that the crescentic low density (near water density) between the collapsed upper lobe and the adjacent ribs represented small ARPE. Further prospective clinical study with pleural fluid analysis in the patient showing presumed small loculated effusion adjacent to the collapsed lobe and experimental study using supine CT after introducing small amount of fluid in the pleural space before and after obstructing the lobar bronchus are needed to verify the results of our study.

We conclude that the cause of lobar collapse when associated with a small ARPE between the collapsed upper lobe and rib cage is more likely to be BC than TB.

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<국문 요약>

상폐엽허탈 결의 소량의 흉막 삼출액 : 폐종양과 폐결핵
감별의 보조적 소견

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저자들은 폐허탈의 원인중 폐암과 폐결핵을 감별하는데 있어 비특이적으로 재분포된 소량의 흉막 삼출액 소견의 역할을 알아보기 위해 상폐엽 허탈 환자 32명의 전산화단층촬영을 후향적으로 검토하였다.

32명 환자중 폐암이 21예, 결핵이 11예였다. 소량의 흉막 삼출액은 폐암 환자 21예중 14예와 결핵 환자 11예중 2예에서 관찰되었다. 소량의 흉막 삼출액을 보인 16예중 11예(69%)에서 종격동 침입이 있었고 6예(38%)에서 종격동 임프절 종대가 있었다.

결론적으로 전산화단층촬영 사진상 소량의 비전형적 흉막 삼출액이 상폐엽 허탈 결에 보일때 폐암을 시사하는 보조적 소견으로 사용될 수 있을 것이라고 생각한다.