BRONCHIOLITIS OBLITERANS IN A PATIENT WITH HYPEREOSINOPHILIC SYNDROME

Patchrin PEKANAN[1], Janjira CHATCHAVALA[1],
Somboon BUNKASEM[2], Mana ROJANAVUTINOND[3], Sopon KUMPOLPUNTH[1].

ABSTRACT

Plain chest film and CT scan of the thorax in a 30 year-old woman with bronchiolitis obliterans was presented. The patient was a known case of hypereosinophilic syndrome. The images showed ground glass appearance of both lungs. Reticular, reticulonodular and patchy lesion was observed, mainly at left lower lobe and left apex. Pleural effusion in small amount was seen in both sides of the pleural spaces. Mild volume loss was observed in left lung.

INTRODUCTION

Bronchiolitis obliterans was first described by Lange in 1901 (1). It is a descriptive term for a fibrosing inflammatory process that occludes the lumens of small airways (2,3). Histologically, bronchiolitis obliterans is defined by the presence of granulation tissue plugs within the lumen of small airways and alveolar ducts and the destruction of small airways with obliterative scarring (4).

We report the radiographic presentation of a case with bronchiolitis obliterans by plain film and CT scan.

CASE REPORT

A 30-year-old female patient had fever and blood stained sputum for 1 day. She was a known case of hypereosinophilic syndrome, diagnosed one year ago. She admitted to the hospital many times. Her previous problems were congestive heart failure, deep venous thrombosis of left arm, mitral and tricuspid valve regurgitation. She had a persistent abnormal chest film and CT scan of the chest (Fig.1,2), so that bronchoscope was performed. She had cardiac arrest during the scope and was improved after resuscitation. Lung biopsy was later performed by right thoracotomy. The lung was found to be multinodular and firm consistency. The 50 cc pleural fluid was seen with clear-yellow colour. Lower lobe biopsy was done and was compatible with bronchilitis obliterans with alveolar atelectasis. Restrictive cardiomegaly was found at cardiac catheterization.

DISCUSSION

Etiologically, bronchiolitis obliterans can be classified as (a) toxic fume bronchiolitis obliterans (2,5-7), the toxic fumes include sulfur dioxide, ammonia, chlorine, phosgene, oxides of nitrogen and ozone (b) postinfectious bronchiolitis obliterans which is usually seen in children or in young adults after Mycoplasma infection and in older adults after viral infection (c) bronchiolitis obliterans associated with connective tissue disease and organ transplantation (10-14) (d) localized lesion with bronchiolitis obliterans (6), histologically such lesions are referred to as "focal organizing pneumonia" (e) idiopathic bronchiolitis obliterans with organizing pneumonia (5).

[1] Department of Radiology, Ramathibodi Hospital, Rama 6 Street, Bangkok 10400, THAILAND.
[2] Department of Surgery, Ramathibodi Hospital.
[3] Department of Pathology, Ramathibodi Hospital.

Clinically, patients have cough, dyspnea, sputum, and fever. Both children and adults are affected, but more than 80% of patients are over 40 years old. There is a 2:1 male predominance (2,6). Good response to corticosteroid therapy, especially in the idiopathic group, is noted but recurrence after discontinuation of steroid therapy occurs in one-third.

There is a wide spectrum of radiographic patterns due to diverse causes of bronchiolitis obliterans and the varying degrees of organizing pneumonia which may be present (4). Intense exposure to the toxic-fume causes acute pulmonary edema and acute respiratory failure may develop after a 4-6 hour latency period. The chest radiograph at this stage reveals the changes seen in the adult respiratory distress syndrome (ARDS), which consist of difffuse alveolar or ground-glass opacifies uniformly distributed throughout both lungs. The heart size is normal and pleural effusions are absent. One to three weeks later, if survive, the development of irreversible airflow obstruction occurs. In mild cases the chest radiograph may be normal, whereas in severe cases hyperinflation can be identified. This stage may be characterized by multiple discrete nodular opacities of various sizes distributed uniformly throughout both lungs and may become confluent in severe cases.

The radiographic pattern in postinfectious bronchiolitis obliterans is highly variable. Most frequently the chest radiograph is either normal or demonstrates a diffuse nodular or reticulonodular pattern. The nodular pattern appears to correlate with histologic evidence of pure bronchiolitis obliterans, while the reticulonodular pattern reflects considerable interstitial fibrosis and scarring. If organizing pneumonia is extensive, patchy alveolar or ground-glass opacities may be identified. Difffuse hyperinflation is rare.

The chest radiograph in bronchiolitis obliterans associated with connective tissue disease or penicillamine treatment is normal or occasionally shows hyperinflated lungs. The histology is that of a pure bronchiolitis obliterans. Patchy areas of alveolar or ground-glass opacities are seen in organizing pneumonia in this group of patients. Course nodular or reticulonodular opacities with peribronchial thickening were seen in the long-term survivors of heart-lung transplantation.

Bronchiolitis obliterans may be associated with localized discrete radiographic opacities or nodules. They are frequently incidental radiographic findings in asymptomatic patients in whom lung biopsy specimens are obtained to exclude carcinoma. The appearance is usually that of an irregular, sublobar area of air-space consolidation or an irregular nodule.

In idiopathic bronchiolitis obliterans with organizing pneumonia, the chest radiograph shows bilateral, patchy ground-glass or air-space opacities. There is no zonal or lobar predominance. These sometimes begin as focal lesions and progressed bilaterally over time. Diffuse, small, linear and nodular opacities were uncommon. Cavities, effusions and hyperinflation were rare.

Diseases associated with eosinophilia include (1) infectious diseases, (2) allergic diseases, (3) myeloproliferative and neoplastic diseases, (4) other cutaneous diseases, (5) other pulmonary diseases, (6) connective tissue diseases, (7) immuno deficiency diseases, (8) gastrointestinal diseases, (9) occasional causes of esosinophilia e.g. post-irradiation (15).

Summary of radiographic findings of bronchiolitis obliterans are (1) normal, hyperinflation, difffuse nodular opacities in toxic-fume exposure, (2) normal, diffuse nodular or reticulonodular pattern, Swyer-James syndrome in postinfectious process, (3) normal, hyperinflation, patchy ground-glass opacities in connective tissue diseases, (4) nodule or focal irregular air-space consolidation in localized type, (5) patchy ground-glass opacities in idiopathic type (4).

Our patient had mildly hypoaerated lungs, reticulonodular pattern with ground glass appearance. Small component of patchy lesion was seen in every region. The patches could be the result of conglomuration of the reticular lesion.

REFERENCES

1. Lange W. Uber eine eigenthumliche: Erkrankung der kleinen Bronchien and Bronchiolen. Deutsche Arch Klin Med 1901;70:342-364.
2. Miki Y, Hatabu H, Takashashi M, Sadatoh N, Kuroda Y. Computed tomography of bronchiolitis obliterans. Journal of Computer Assisted Tomography 1988;12:512-514.
3. Katzenstein AA, Askin FB. Surgical Pathology of nonneoplastic lung diseases. Major problems in pathology. Philadelphia: WB Saunders, 1982, p 349.
4. McLoud TC, Epler GR, Colby TV, Gaensler EA, Carrington CB. Bronchiolitis obliterans. Radiology 1986;159:1-8.
5. Epler GR, Colby TV, McLoud TC, Carrington CB, Gaensler EA. Bronchiolitis obliterans organizing pneumonia. N Engl J Med 1985;312:152-158.
6. Gosink BB, Friedman PJ, Liebow AA. Bronchiolitis obliterans:roentgenologic-pathologic correlation. AJR 1973;117:816-832.

7. Lannuzzi MC, Farhi DC, Bostrom PD, Petty TL, Fisher JH. Fulminant respiratory failure and death in a patient with idiopathic bronchiolitis obliterans. Arch Intern Med 1985;145:733-734.
8. Laraya-Cuasay LR, DeForest A, Huff D, Lischner H, Huang NN. Chronic pulmonary complications of early influenza virus infection in children. Am Rev Respir Dis 1977;116:617-625.
9. Nikki P, Meretoja O. Valtonen V, et al. Severe bronchiolitis probably caused by varicella-zoster virus. Crit Care Med 1982;10:344-346.
19. Herzog CA, Miller RR, Hoidal JR. Bronchiolitis and rheumatoid arthritis. Am Rev Respir Dis 197;119:555-560.
11. Schwarz MI, Matthay RA,Sahn SA, et al. Interstitial lung disease in polymyositis and dermatomyositis : an analysis of six cases and review of the literature. Medicine 1976;55:89-104.
12. Epler GR, Snider GL, Gaensler EA, Cathcart ES, Fitgerald MX, Carrington CB. Bronchiolitis and bronchitis in connective tissue disease. JAMA 1979;242:528-532.
13. Ostrow D, Buskard N, Hill RS, Vickars L, Churg A. Bronchiolitis obliterans complicating bone marrow transplantation. Chest 1985;87: 828-830;
14. Burke CM, Theodore J, Dawkins KD, et al. Post transplant obliterative bronchiolitis and other late lung sequellae in human heart-lung transplantation. Chest 1984;86:824-829.
15. Wyngaarden, Smith, Bennett. Cecil textbook of medicine, 19th ed. Philadelphia: W.B. Saunders Company, 1992.

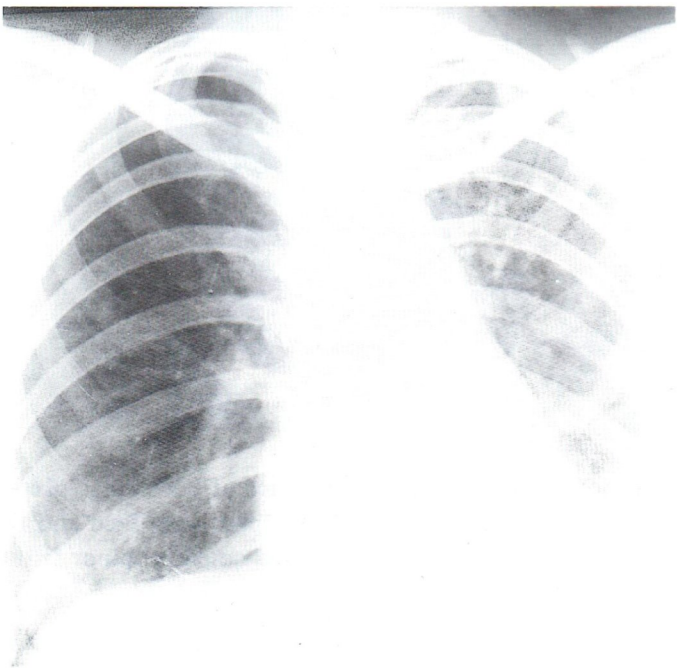

Fig.1 Plain chest film in PA view showed some volume loss in left lung. Mixed interstitial and alveolar process, mainly interstitial process involving the whole left lung, denser at lower zone. Less degree of involvement at right lung was noted.

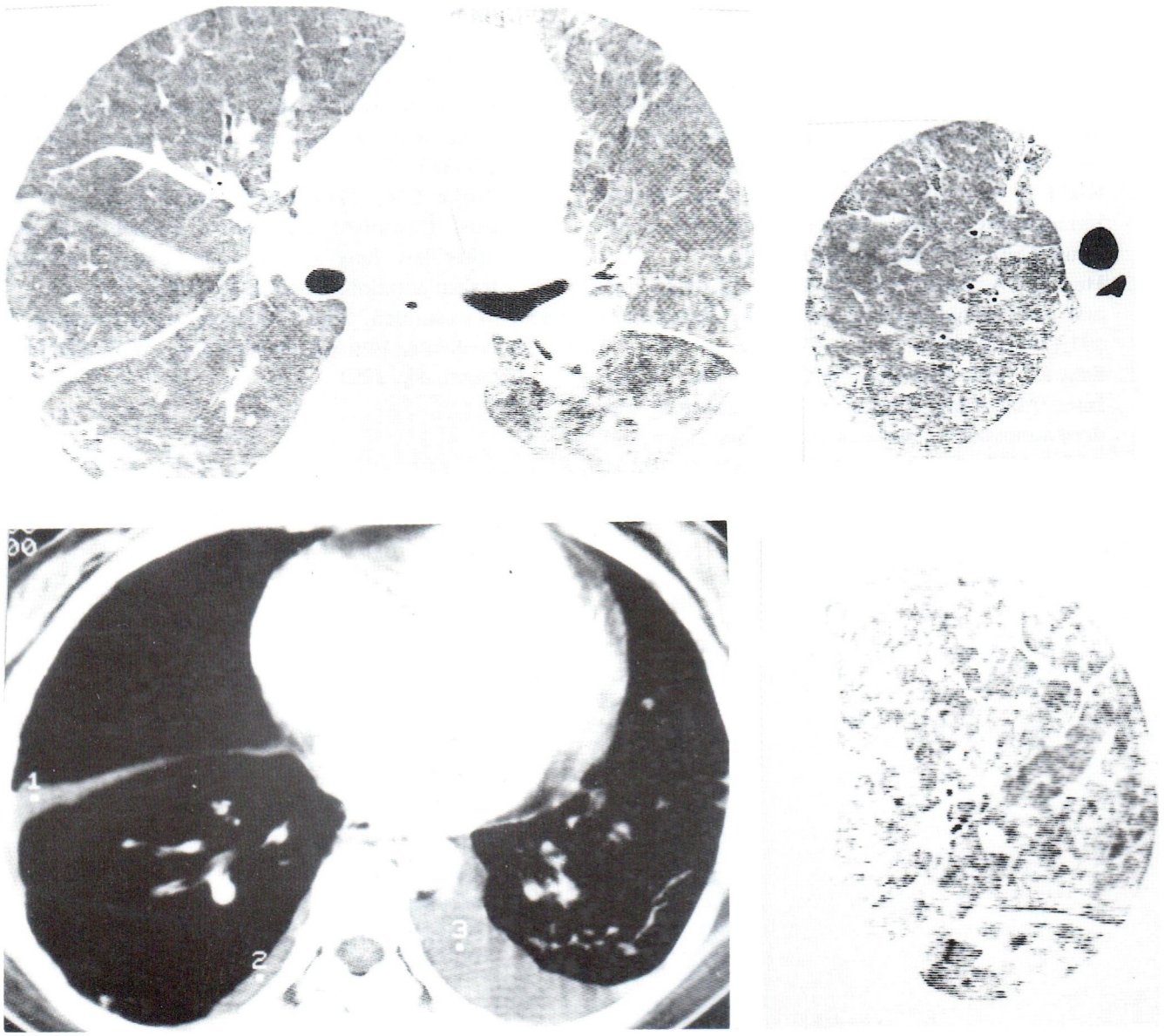

Fig. 2 I.V. contrast CT scan showed ground glass appearance of both lungs. Reticulonodular and patchy lesion was noted at left lower lung field; less dense at left upper lung field and right base. Left pleural fluid at dependent part was shown and right pleural fluid was noted at minor fissure.