Bronchiolitis Obliterans with Organising Pneumonia presenting as Non Resolving Pneumonia

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MJAFI 2006; 62 : 378-380

Key Words: Bronchiolitis obliterans; Non resolving pneumonia

Introduction

Non-resolving pneumonia is a common clinical entity [1,2]. The term non resolving pneumonia is considered when the radiograph has failed to resolve by 50% in 2 weeks or completely in 4 weeks. However pneumonia resolution is influenced by various factors like type of infection and organism [3]. The non-infectious causes of pneumonia termed as pneumonitis [4] always pose a diagnostic dilemma to the treating clinicians. We present a case of pneumonia that perplexed not only the physicians but also the pathologists which was finally diagnosed as bronchiolitis obliterans with organising pneumonia (BOOP).

Case Report

A 39 year old soldier, a welder by profession was admitted to a peripheral military hospital with short febrile illness, cough, joint aches and exertional breathlessness. Clinical examination revealed consolidation in the right side of chest and radiograph showed pneumonia in right mid and lower zones involving the lateral segment of right lower lobe (Fig 1). His haemogram, sputum for aerobic culture, anaerobic culture, acid fast bacilli and metabolic profile was normal. His human immunodeficiency virus infection (HIV) status was non reactor. The blood culture showed no growth and rheumatoid factor was also negative. He was exhibited parenteral broad spectrum empirical antibiotics. He became afebrile after two weeks, but there was no radiological resolution of the pneumonia. He was suspected of having pulmonary tuberculosis and was transferred to a chest centre. Further evaluation, revealed a solitary enlarged lymph node in the right axilla. The fine needle aspiration biopsy (FNAC) of this lymph node revealed non specific inflammation. He was subjected to trans bronchial lung biopsy (TBLB) for the persistent pneumonic patch in the right lung which suggested the diagnosis of bronchoalveolar carcinoma. To confirm the

diagnosis of bronchoalveolar carcinoma, open lung biopsy (OLB) was planned. The patient continued to be symptomatic with unproductive cough, mild joint pains and exertional breathlessness. His joint pains were attributed to paraneoplastic phenomenon. Collagen vascular disease work up in the form of anti nuclear antibody (ANA), double strand deoxy ribonucleic acid (dsDNA), rheumatoid factor, anti neutrophil cytoplasmic antibody (c-ANCA), and p-ACNA was negative. Spirometry, repeat chest radiograph and contrast enhanced computed tomography (CECT) chest were carried out which revealed persistence of pneumonia in the right lower lobe, lateral segment of middle lobe and an additional patch of consolidation in the superior segment of left lower lobe. (Fig 2, 3). In an attempt to avoid more invasive procedure of Open Lung Biopsy (OLB), a fluoroscopy-guided trans thoracic needle aspiration (TTNA) from the right lung lesion was carried out which suggested features of chemical pneumonitis (Lupoid pneumonia) on histopathological

Fig. 1 : Consolidation in the right mid and lower zones involving superior and lateral basal segments of right lower lobe and lateral segment of right middle lobe

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Fig. 2 : Persistence of consolidation after 7 months of optimal therapy

Fig. 3 : CT scan chest showing persistence of consolidation in the superior and lateral basal segment of right lower lobe and a patch of consolidation in the superior segment of the left lower lobe

examination. Since the patient was symptomatic and no specific therapy could be planned as yet, he was subjected to OLB.

The histopathological examination (HPE) report of OLB revealed proliferation of type 2 pneumocytes, alveoli and bronchioles filled with proliferating fibromyxomatous tissue and numerous foamy macrophages with infiltrates of plasma cells and lymphocytes suggesting the diagnosis of BOOP (Fig 4). The patient was managed with oral steroids (prednisolone 1mg/kg of body weight for 3 months and then with tapering dosages) and he showed a remarkable response clinically and radiologically (Fig 5).

On follow up for one year the patient remains asymptomatic, without steroids with mild restrictive ventilatory defect on spirometry.

Discussion

There have been progressive endeavours to sub

Fig. 4 : Chest radiograph showing resolution of consolidation after steroid therapy. Also seen is the minimal residual fibrosis in right mid zone

Fig. 5 : Microphotograph of open lung biopsy tissue showing interstitial fibrosis, proliferation of type 2 pneumocytes, alveoli and bronchi filled with fibromyxomatous tissue and foamy macrophages with infiltrates of plasma cells and lymphocytes

classify the pulmonary fibrotic diseases for prognostic and therapeutic purposes, especially after the observations that some of them respond well to steroids while others do not. Till 1985 all the pulmonary fibrotic diseases were clubbed under one category i.e. cryptogenic pulmonary fibrosis or idiopathic pulmonary fibrosis (IPF), having progressive course culminating into death and only few of them responding to steroid therapy. It was in 1985, when a subclass of such patients with lung fibrosis was recognised to be having different clinical, radiological, histopathological and prognostic features as compared to other pulmonary fibrotic disorders like obliterative bronchiolitis (OB) and idiopathic pulmonary fibrosis (IPF). The subset of patients with this disease was characterised by granulation tissue plugs within the lumens of bronchioles and extension of this fibroinflammatory process into alveolar ducts and alveoli mimicking organising pneumonia and having different therapeutic and prognostic implications [5]. Aetiologically it is either a primary BOOP or secondary to various pulmonary insults

like toxic fumes in welding trade as in our case, post infectious BOOP due to chlamydia, legionella, mycoplasma, adenoviruses, cytomegalo virus and influenza virus, drugs related BOOP, secondary to collagen vascular diseases and post transplant BOOP[6].

Various chest radiographic findings have been described in BOOP but the typical chest radiograph shows bilateral peripheral patchy acino nodular infiltrates, migratory in nature in 10 to 25% cases [7]. BOOP may also present as solitary pulmonary nodule, with or without cavitation or linear basal opacities, and may be unilateral. BOOP has also been reported to be of acute onset and progressive in course making it indistinguishable from acute interstitial pneumonia or may have an acute onset and an indolent course thereafter as in our case [8]. BOOP is also a reversible cause of respiratory failure, responsive to steroids therapy when it presents as diffuse acute interstitial pneumonia [9]. Radiologically and histopathologically BOOP may be confused with bronchoalveolar carcinoma and lupoid pneumonia as our case [10].

To conclude BOOP is a pattern of lung injury seen in many pulmonary conditions and is responsive to steroids in 65 to 80% of cases.

Conflicts of Interest

None identified

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