



BRONCHOLITIS OBLITERANCE ORGANISING PNEUMONIA IN A KNOWN CASE OF NEUROCYSTICERCOSIS: A RARE CASE REPORT

Pulmonary Medicine

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ABSTRACT

The etiology of bronchiolitis obliterance organizing pneumonia remains controversial. While it has been associated with several disorders, there are only rare reports of BOOP associated with Neurocysticercosis. Here in we report a 57 year old male patient who presented with sudden onset of dyspnea at rest with high grade fever as initial symptoms. The diagnosis of BOOP was confirmed by Transbronchial lung biopsy. His respiratory symptoms and radiological findings significantly improved following prednisolone treatment.

KEYWORDS

Boop,neurocysticercosis,steroids

INTRODUCTION:

Bronchiolitis obliterans organizing pneumonia (BOOP) is characterized histologically by the formation of plugs of fibrous tissue in the alveolar ducts, alveoli, and the distal bronchiole. The etiologies of BOOP were reported, associated with various disorders including infection, inhalation exposure to toxins, drugs, and collagen vascular diseases. Notably, patients with BOOP respond well to systemic steroid therapy. Therefore, such patients may heal promptly without further complications. Herein, we present a known case of neurocysticercosis presented as BOOP. Previously published case reports were collected and distinctive features were compared with the current case.

CASE REPORT:

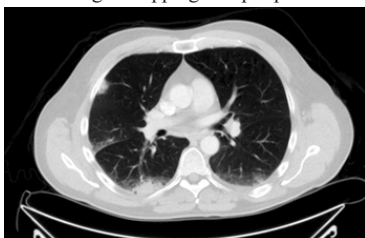
A 57-year-old hindu male patient was admitted to our hospital for sudden onset of dyspnea at rest, high grade fever, dry cough since 15 days. He was known case of neurocysticercosis since 2013 and was taking sodium valproate and tab. clonazepam but he was on irregular treatment. Chest examination showed an alveolar patch in both lower lung fields. Peripheral white blood cell count on admission was $3.93 \times 10^9/L$ with 64% neutrophils, 22% lymphocytes, and 13% monocytes. The hematocrit value was 32.7%.

CXR PAVIEW:



On admission, physical examination revealed bilateral basal crackles over the lung field. The heart rate was 82 beats/min in regular heart sound. He had no history of smoking, chemical-agent exposure, alcohol consumption, betel nut chewing, and animal breeding. He also denied any family history of such respiratory disease.

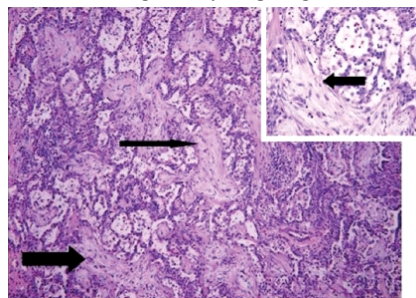
HRCT THORAX: showing air trapping with peripheral consolidation.



His MRI report was showing: multiple cystic area in right cerebellar hemisphere, bilateral basal ganglia, right frontal, left temporal, left parietal and bilateral occipital lobes suggestive of neurocysticercosis. For this he was taking Tab. sodium valproate and clonazepam since 2014.

After admission to the ward, intravenous empirical antimicrobial therapy including cefosalbactam 1.5gm 8 hourly and clindamycin 0.6gm 8 hourly for 7 continuous days was prescribed for atypical pneumonia. The serological tests for clinical pathogens including Mycoplasma, Legionella, and Cryptococcus were negative. Sputum examination revealed no acid-fast organisms or fungus. After one-week observation, no effect on symptoms of fever, cough, and dyspnea on exertion. Then he was started inj. Colistin according to his sputum culture sensitivity report.

In this case, the patient's symptoms did not improve with the above treatment. Further laboratory studies revealed leukopenia ($1.94 \times 10^9/L$). A high-resolution computed tomography (HRCT) showed ground glass opacity with interlobular septal thickening (crazy paving pattern) in both lung fields in perihilar region with subpleural sparing. Although BOOP was the radiologist's first impression, bronchoalveolar cancer, acute hypersensitivity pneumonitis was excluded. His biopsy examinations confirmed BOOP. The patient showed continued improvement in his dyspnea on exertion and radiological opacity after giving injectable methylprednisolone 125mg 8 hourly in tapering dose.



Case of idiopathic BOOP, shown on low power [magnification $\times 10$] - pale staining areas of elongated branching fibrosis, involving bronchiolar lumen and peribronchial airspaces [solid arrow]. The alveolar septae [inset] shows mild chronic inflammation.

Then he suddenly developed right sided upper and lower limb paralysis and on MRI brain found to having left sided acute infarct most probably due to septic emboli. Then he was started on tab. Aspirin, atorvas. Patient improved after 20 days of treatment and then was continued on oral Tab. Prednisolone 30mg on opd bases in tapering dose. Patient symptomatically improved and maintained SPO2 95%.

DISCUSSION:

Bronchiolitis obliterans organizing pneumonia (BOOP), is an inflammation of the bronchioles (bronchiolitis and surrounding tissue in the lungs). It is often a complication of an existing chronic inflammatory disease such as rheumatoid arthritis, dermatomyositis, or it can be a side effect of certain medications such as amiodarone. In our case it was idiopathic.

BOOP is originally differentiated from organizing pneumonia, which is characterized by formation of granulation tissue in the distal air space, but when it presented with granulation tissue plugs within lumens of small airways, the term bronchiolitis obliterans was added to OP. The precise mechanism of injury leading to the formation of BOOP remains controversial. However, it was reported that injury to the alveolar epithelium plays an important role. Al-Ghanem et al. indicated that the pathogenic mechanism of BOOP is that of an inflammatory lung disease rather than a fibrosing process.

The clinical features and radiological imaging resemble infectious pneumonia. However, diagnosis is suspected after there is no response to multiple antibiotics, and blood and sputum cultures are negative for organisms. In our case similarly, radiographic features were resembling like acute viral pneumonia but multiple antibiotics trial was failed. Multiple causes are found like:

- Pulmonary infection by bacteria, viruses and parasites
- Drugs: antineoplastic drugs, erlotinib, amiodarone
- Toxic fumes
- Ionizing radiations^{[7][8]}
- Inflammatory diseases
- Systemic lupus
- Rheumatoid arthritis (RA-associated COP)
- Scleroderma
- Bronchial obstruction
- Proximal bronchial squamous cell carcinoma^[9]

It was identified in 1985, although its symptoms had been noted before but not recognised as a separate lung disease. The risk of COP is higher for people with inflammatory diseases like lupus, dermatomyositis, rheumatoid arthritis, and scleroderma. Mostly it is idiopathic as it was found in our case.

The classic presentation of COP is the development of nonspecific systemic (e.g., fevers, chills, night sweats, fatigue, weight loss) and respiratory (e.g. difficulty breathing, cough) symptoms in association with filling of the lung alveoli that is visible on chest x-ray. In our case the presentation was similar with acute history of dyspnea and low grade fever since 15 days.

The preferred diagnosis method of BOOP is lung biopsy. However, conventional radiography and CT serve as a guide in determining the biopsy site. Moreover, pathologic examination is needed to exclude infection and other pulmonary manifestations. We noticed that inflammatory response in BOOP seemed different from other pulmonary inflammatory diseases such as asthma, chronic obstructive pulmonary disease (COPD), and granulomatous lung disease. Early differential diagnosis is important because treatment might be ineffective against the wrong type of inflammatory response. We in our case found acute history with CT finding meeting the criteria of BOOP confirmed by lung biopsy.

BOOP may resolve spontaneously; however, corticosteroids are the current standard treatment. The majority of patients with BOOP recover with treatment, symptoms resolving within days or weeks. Similarly the radiographic findings show improvement in 50-86% of patients; however, in a minority of patients, the disease may persist. Approximately 30% of the patients experience relapse upon withdrawal of treatment. Patients with asymptomatic mass lesions or nonprogressive disease can be observed and treated at a later time if needed. There is no consensus regarding the optimal doses of prednisone and optimal treatment duration. The dosage is generally 0.75 mg/kg/day for 1 to 3 months, then 0.50 mg/kg mg/day for 3 months, then 10 to 20 mg/day or every other day for a total of 1 year. Every-other-day scheduling can be successfully used for this disorder. A shorter 6-month course may be sufficient in certain situations. However, this duration can extend up to 12 months or even longer due to relapses. A total and permanent recovery is seen in most patients, but it is also dependent on the cause or associated systemic disorders.

erythromycin, inhaled triamcinolone, azathioprine, cyclosporin and cyclophosphamide have been used to treat BOOP. We gave injectable steroid at time of admission and then tapered as patient got improved and discharged patient with oral steroid 5mg bd.

CONCLUSION:

We described a patient with neurocysticercosis who manifested respiratory illnesses due to BOOP. BOOP is an important treatable inflammatory lung disease. Idiopathic BOOP has become an important differential of focal lung nodular lesions. In addition, HRCT and pathological examinations can be used for early differential diagnosis of pulmonary disease in patients with neurocysticercosis.

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