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TB or Not TB*

Cavitary Bronchiolitis Obliterans Organizing Pneumonia Mimicking Pulmonary Tuberculosis

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Two patients with subacute symptoms and signs compatible with pulmonary tuberculosis (TB) had right upper lobe cavitary infiltrates shown on chest radiography. In both patients, purified protein derivative and microbiologic testing excluded TB, and tissue examination yielded typical histologic changes of bronchiolitis obliterans organizing pneumonia (BOOP). Glucocorticoid therapy led to clinical and radiologic resolution. Though probably rare in this situation, BOOP should be considered in the differential diagnosis of patients presenting with clinical and radiologic features of pulmonary TB.

Key words: bronchiolitis obliterans organizing pneumonia; radiography, thoracic; tuberculosis, pulmonary

Abbreviations: ANCA = antineutrophil cytoplasmic antibody; BOOP = bronchiolitis obliterans organizing pneumonia; ESR = erythrocyte sedimentation rate; TB = tuberculosis

Patients with bronchiolitis obliterans organizing pneumonia (BOOP) present with clinical and radiologic manifestations that are not specific to this entity. Depending on the clinicoradiologic presentation, a number of other diseases may have to be considered in the differential diagnosis of BOOP. Tuberculosis (TB), however, is not usually included in this differential diagnosis. We describe two patients who presented with clinical and radiologic features suggestive of pulmonary TB but turned out to have BOOP instead.

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Case Reports

Case 1

A 66-year-old man was hospitalized with a 2-month history of productive cough, anorexia, and weight loss. One month prior to hospital admission, fever, night sweating, and malaise ensued. He had a history of mild chronic obstructive lung disease and benign prostatic hypertrophy. On hospital admission, the patient looked ill with a temperature of 39°C and a regular pulse rate of 110 beats/min. BP was 140/85 mm Hg, and respiratory rate was 18 breaths/min. Tubular breathing and rales were heard in the upper region of the right lung. The physical examination was otherwise normal.

A chest radiograph (Fig 1, top) showed a massive right upper lobe alveolar infiltrate with multivacitary lesions. CT of the chest (Fig 1, bottom) performed at the same time showed a “Swiss cheese” alveolar infiltrate with multiple cavities in the posterior segment of the right upper lobe.

The erythrocyte sedimentation rate (ESR) was 14 mm/h. With the exception of mild normocytic anemia (hemoglobin, 11.7 g/dL) and a thrombocyte count of 585,000/μL, all routine laboratory test results, including a CBC count, liver and kidney function tests, serum proteins, and urinalysis, were normal. Blood, sputum, and urine culture findings were negative.

Sputum examination results for microorganisms including acid-fast bacilli were repeatedly negative. Results of purified protein derivative skin test and polymerase chain reaction of the sputum for Mycobacterium tuberculosis were negative. Serologic test results for Mycoplasma, Legionella, cytomegalovirus, Candida, Aspergillus, and Nocardia were negative. Antiinflammatory antibodies and antineutrophil cytoplasmic antibodies (ANCAs) were not found. Bronchoscopy revealed diffuse inflammatory bronchial changes. No bacteria, acid-fast bacilli, or fungi were seen in the bronchial secretions, and culture findings for TB organisms were negative.

Treatment with IV cefuroxime and erythromycin did not improve the patient’s condition. Transbronchial biopsy (Fig 2) showed myxoid fibroblastic tissue and intrabronchiolar aggregates of mononuclear cells invading alveolar spaces. These findings were considered to be consistent with BOOP.

Treatment with prednisone was started at a dosage of 60 mg/d. Rapid lysis of the fever resulted. Within a few days, the other symptoms regressed as well. The patient was discharged receiving prednisone, 60 mg/d with slow tapering, and was followed up in the outpatient clinic. Six months later, he was asymptomatic, and a chest radiograph (Fig 3) showed only moderately severe hyperinflation and minimal residual infiltrate in the right apex.

Case 2

Ten weeks after the hospital admission of Case 1, a 76-year-old man was admitted to the hospital for progressive dyspnea, pleuritic pain, and malaise of 1-month duration. Cough, fever, and increased sweating were denied. Medical history consisted of peptic disease, diaphragmatic hernia, heavy smoking, and stable ischemic heart disease. On hospital admission, the patient was cachectic, with no fever, pallor, or cyanosis. Respiratory rate was 30 breaths/min, BP was 150/65 mm Hg, and the pulse rate was 88 beats/min. Rales were heard in the right apical region of the lungs, the examination findings being otherwise unremarkable. The ESR was 70 mm/h, a leukocyte count showed 13,200 cells/μL, and the serum albumin level was 3.0 g/dL. Findings of all other routine laboratory tests were normal.

A chest radiograph (Fig 4, top) showed severe hyperinflation, and alveolar and interstitial infiltration involving mainly the right upper lobe, with multiple cavities. CT of the chest (Fig 4, bottom) demonstrated severe bullous emphysema with massive alveolar infiltration and multiple cavities. Treatment with IV cefuroxime and erythromycin was started.

Results of sputum examinations, including Ziehl-Nielsen preparations, were negative, as were a skin tuberculin test and cultures of the blood, sputum, and urine. As in the first case, serology findings for infectious etiologies were negative. The result of testing for antinuclear antibodies was weakly positive, and cytoplasmic ANCA findings were positive, at a low titer of 1:20.

Copious purulent secretions were seen on bronchoscopy, which microscopically showed few Gram-negative and Gram-positive bacteria and no acid-fast bacilli or fungi. Culture of these secretions grew Pseudomonas aeruginosa but was negative for mycobacteria. When the result of the sputum culture became available, treatment with cefuroxime was switched to ciprofloxacin. There was no clinical response to antibiotic therapy.
A transbronchial biopsy (Fig 5) was diagnostic of BOOP, showing findings similar to those described in the first case. Treatment with antibiotics was discontinued, and prednisone, 60 mg/d, was started. This led to rapid clinical improvement, and the patient was discharged 4 days after initiation of treatment without fever or dyspnea. One month later, he was readmitted to the hospital with epigastric distress. His respiratory symptoms had disappeared and, other than his abdominal complaint, he was well. A chest radiograph (Fig 6) showed mild-to-moderate improvement of the right upper lobe findings seen at the time of the previous hospital admission. Famotidine, 40 mg bid, was administered with good clinical response, and the patient was again discharged. Two weeks later, despite medical advice to the contrary, the patient discontinued prednisone and was unavailable for follow-up.

**DISCUSSION**

The main differential diagnosis of cavitary lung infiltrates includes pyogenic abscess from a variety of bacterial organisms, pulmonary TB, fungal infection, and parasitic disease of the lungs. In addition to these infectious sources, pulmonary cavities have been well documented in patients with primary or secondary malignancies, collagen disease, Wegener granulomatosis, and less frequently other vasculitides. Rarely, lymphoma, sarcoidosis, and cysts may also present in this fashion.1

Our two patients presented with cavitary infiltrates that were confined to the right upper lobe. This radiologic pattern, especially when accompanied by compatible clinical manifestations as in these patients, suggests TB rather than most of the other diagnostic contenders listed above. Pulmonary TB was therefore the working diagnosis in both patients at hospital admission. Testing results for TB were negative, however, and tissue biopsy demonstrated BOOP.

The clinical manifestations of BOOP can account for all of the complaints our two patients presented with. Cough, dyspnea, fever, night sweating, and weight loss are...
all common in this entity, being observed in ≥ 50% of the cases at presentation. Pleuritic pain occurs in one of four patients with BOOP. An ESR > 60 mm/h is seen in 40% of patients, and leukocytosis in seen in 50% of the patients at presentation. Thrombocytosis, which we observed in our first patient, has been described in 20% of cases.

In addition to its cryptogenic variant, BOOP may be associated with conditions such as pulmonary infection or tumor, collagen disease, hematologic malignancy, vasculitis, hypersensitivity reactions, and other rare diseases. No evidence for any of these was found in our first patient, who therefore qualifies for a diagnosis of cryptogenic BOOP.

Two points need clarification with regard to the second case. First, other than the pulmonary infiltrates, no additional cardinal features of Wegener granulomatosis were present and findings characteristic of this disorder were not seen on biopsy. Thus, the low titer of classic pattern ANCA in this case was considered a false-positive finding. Secondly, the patient’s clinical presentation, as well as his response to steroids after adequate antibiotic therapy had failed, do not suggest Pseudomonas pneumonia, and a MEDLINE search identified only one description of BOOP associated with this organism. We therefore believed that the positive culture result could be yet another incidental finding, but a remote possibility remains that in this case BOOP developed from a Pseudomonas pneumonia.

BOOP may present with a variety of radiologic patterns, including multiple patchy alveolar infiltrates, diffuse interstitial infiltrates, solitary opacities that may sometimes resemble tumors, and rarely other patterns. Although cavitary infiltrates are not usually included in textbook descriptions of the disease, BOOP presenting with cavitating infiltrates has indeed been described, albeit rather rarely. Two of the 42 cases of cryptogenic BOOP in the series published by Epler et al in 1985 appear to represent the earliest mention of a cavitary radiologic pattern in this disorder. Several additional cases demonstrate that this pattern occurs in the cryptogenic form of BOOP, as well as in BOOP associated with disorders such as essential mixed cryoglobulinemia and lymphoma.

In view of these cases, Cordier in a recent editorial stressed that BOOP must be included in the differential diagnosis of cavitary lung disease. However, only one of the patients described to date presented with upper lobe cavitation, and, to our knowledge, there are no descriptions of BOOP actually mimicking TB in both its clinical and radiologic aspects. Our two patients presented with subacute symptoms and radiologic findings characteristic of pulmonary TB. When this strong first impression could not be corroborated by laboratory test results, the diagnosis of BOOP by tissue examination was surprising. In our view, this course of events proves that BOOP not only presents sometimes with cavitary disease but may also resemble, both clinically and radiologically, typical pulmonary TB, a possibility not emphasized in the literature. Whether this is a very rare occurrence or, as might be suggested by the short interval in which these two cases presented, a more common phenomenon, remains to be seen.

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Respiratory Control During Independent Lung Ventilation*

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We describe the case of a lung transplant patient with primary graft failure and an emphysematous native lung, who displayed different respiratory rates between the transplanted lung and the native lung. Inflation of the native lung delayed the next inspiratory effort relative to inflation of the denervated transplanted lung. Synchronous inflation of both lungs required more pressure in each lung than when that lung was inflated with the contralateral lung near functional residual capacity, suggesting the two lungs compete for space within the thoracic cavity.

Key words: emphysema; independent lung ventilation; single lung transplant

Abbreviations: Paw = airway pressure; PEEP = positive end-expiratory pressure; TLC = total lung capacity; Vt = tidal volume.

Respiratory frequency and tidal volume (Vt) are normally controlled by the respiratory center integrating excitatory stimuli from chemoreceptors and inhibitory stimuli from receptors in the lung and chest wall. During assisted mechanical ventilation in patients with hypopneic respiratory failure, patients commonly make inspiratory efforts that do not trigger the ventilator, either because they lack sufficient force or they occur at a time during expiration when alveolar pressure is sufficiently high that an inspiratory effort that would trigger the ventilator at a lower volume is not sufficient to stop expiratory flow. Ventilatory strategies for respiratory failure due to ARDS and COPD are quite different. Patients with ARDS receive ventilation with a positive end-expiratory pressure (PEEP) and relatively slow inspiration in order to prevent alveolar collapse and to prevent overdistention of areas that fill more rapidly respectively. Of necessity, this produces a short expiratory time. However, in these patients with stiff lungs and reasonably normal airways, there is no difficulty expiring the Vt, so that at end-expiration, alveolar pressure equals extrinsic PEEP. In contrast, patients with COPD receive ventilation to allow maximal expiratory time without extrinsic PEEP, so when the Vt has been expired, the difference between

<table>
<thead>
<tr>
<th>Variables</th>
<th>Results</th>
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<tr>
<td>FEV1, L</td>
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<tr>
<td>FVC, L</td>
<td>2.56 (49)</td>
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<tr>
<td>FEF25–75%, L/s</td>
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<td>RV, L</td>
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<td>Alveolar volume, L</td>
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<td>P(a–a)O2, mm Hg</td>
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*Data are presented as No. (% predicted) unless otherwise indicated. FEV1, FEF25–75% = forced expiratory flow, midexpiratory phase; PEF = peak expiratory flow; SVC = slow vital capacity as measured during plethysmography; TGV = thoracic gas volume as measured during plethysmography; RV = residual volume; Dlco = corrected diffusing capacity of the lung for carbon monoxide; DLV = diffusion capacity of the lung corrected for hemoglobin; (CHEST 2001; 120:678–681)
