The Role of Two-Segment Bronchoalveolar Lavage in the Diagnosis of Pulmonary Infections*

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Study objective: To determine if performing bronchoalveolar lavage (BAL) from more than one lung segment augments the diagnostic yield in patients with pulmonary infection.

Patients and study design. Seventy-six patients who underwent bisegmental BAL for the diagnosis or exclusion of pulmonary infection were studied prospectively.

Main results: In patients with AIDS, the concentration of Pneumocystis carinii was higher in the upper lobes than in the middle lobes, regardless of whether the patients had been receiving pentamidine prophylaxis. In patients without HIV infection, the number of P carinii clusters was much lower. In 2 of 5 HIV-negative patients, P carinii organisms were detectable but in one of two lavage specimens, whereas in only 1 of 19 AIDS patients, P carinii organisms were not found in both lavaged segments. In bacterial pneumonia, BAL fluid from a segment of the radiologically most involved area had a much higher cell concentration, percentage of neutrophils, and concentration of bacteria than from the segment that was not or less severely involved on chest radiograph. In two of nine patients with AIDS and cytomegalovirus (CMV) pneumonia, cytopathogenic CMV effects were not found in both lavaged segments. In one of eight patients, mycobacteria could be cultured only from one of two radiologically involved segments.

Conclusion. An increase in the diagnostic sensitivity by performing BAL in two lung segments is limited to cases where P carinii pneumonia is a relevant consideration in immunocompromised patients without HIV infection. In bacterial pneumonia, BAL can be performed in a single radiographically involved lung segment without a loss in diagnostic sensitivity. Since our study population of patients with CMV pneumonitis, mycobacterial infections, and fungal infections was small, no reliable conclusions are possible and BAL of more than one lung segment seems justified until more information is available.

Key words: bronchoalveolar lavage fluid; microbiology; pneumonia

Bronchoalveolar lavage (BAL) is an established procedure in the evaluation of pulmonary complications in immunosuppressed patients, particularly in the diagnosis or exclusion of certain types of pulmonary infections such as Pneumocystis carinii pneumonia (PCP) and nosocomial bacterial pneumonias. There is, however, scant information about the value of performing BAL in more than one lung segment for the diagnosis or exclusion of pulmonary infection.1–3

In this prospective study, we evaluated if sampling of alveolar specimens from a larger portion of the lung improves the sensitivity of BAL for the diagnosis of pulmonary infections. We furthermore determined if regional differences in the density of P carinii in patients with different types of underlying immunosuppression were detectable.

METHODS

Patients

Patients were included into this prospective study if they underwent bronchoscopy, including BAL, for the evaluation of suspected pulmonary infection. Seventy-six patients (59 men and 17 women) with a mean age of 40 ± 13 years (range, 20 to 67 years) were studied. Most of the patients were immunosuppressed (n=71) and belonged to one of the following risk groups: HIV infection (n=54), patients receiving chemotherapy and/or corticosteroids for the treatment of hematologic malignancies (n=5), and patients who had undergone organ transplantation (n=12). Five patients had no evident immunosuppression.

Diagnostic Techniques

Fiberoptic bronchoscopy was performed in a standard fashion. The first BAL was performed by wedging the bronchoscope into a subsegmental bronchus of the right or left upper lobe. Three portions of 50 ml of physiologic saline solution were instilled and aspirated back by gentle manual suction. If the patient tolerated the first BAL without pulmonary symptoms (ie, labored breathing, persistent cough), without a fall of the transcutaneously measured oxygen saturation below 85 percent, without hemorrhage or hemodynamic instability, a second BAL with the same

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volume was performed in a subsegment of the middle lobe or the lingula, of the same lung site, respectively (50 patients). This particular sequence of BAL was carried out when PCP was suspected. In 13 cases, the BAL was performed in another combination of segments of the same lung, including a segment with the most prominent radiologic involvement. In 13 patients, BAL was performed from the middle lobe as well as the lingula.

Transbronchial biopsy specimens were obtained consecutively when appropriate under fluoroscopic guidance from the different segments of the same lung.

**Laboratory Processing**

The fluid from either lavage was processed separately and first strained through a double layer of surgical gauze into a sterile graduate cylinder. The fluids were cultured for aerobic and anaerobic bacteria, Legionella, Nocardia, fungi, mycobacteria, and cytomegalovirus (CMV). The number of cells was determined by hemocytometer and expressed per microliter of BAL fluid (BALF). Cytocentrifuge preparations were made with aliquots of original BALF using a cytocentrifuge (Cytospin Shandon, 1,000 rpm, 5 min) and stained with May-Grünwald-Giemsa and Gram. Differential cell counts were performed on 400 nucleated cells per slide. Normal values for BAL cell differential counts were taken from the Report of the European Society of Pneumology Task Group on BAL. Three different detection methods for *P. carinii* were used. The number of clusters of *P. carinii* in relation to 500 nucleated cells was noted for each slide by observers blinded to the patient’s clinical status. Two cytocentrifuge slides with 500 μl of original BALF were prepared (800 rpm, 5 min) and stained by toluidine blue O. *Pneumocystis carinii* clusters were counted per each slide and the mean values were taken. Another cytocentrifuge slide was examined for *P. carinii* using indirect immunofluorescence technique (DAKO A/S, Glostrup, Denmark). The CMV early antigen in BALF was detected with monoclonal antibodies using the shell vial assay.

Quantitative bacterial cultures were performed by plating 1 μl of the BALF specimen on sheep blood agar, chocolate agar, CN-Agar (blood agar containing colistin and nalidixic acid), and MacConkey agar. After inoculation, plates were incubated at 37° C in 5 percent CO2. MacConkey plates were incubated aerobically. Estimates of the number of bacteria in the original fluid were made by colony counts and were expressed as colony-forming units (CFU) per milliliter of BALF. Papanicolaou-stained slides were examined for the presence of intracytoplasmic or intranuclear inclusion bodies and malignant cells.

**Diagnostic Criteria**

The diagnosis of PCP was established by demonstrating cysts by one of the applied methods. A CMV pneumonitis was diagnosed if cells obtained by BAL showed typical intranuclear or intracytoplasmic inclusions, BALF was positive for CMV early antigen, and the clinical picture was consistent with the diagnosis of a pulmonary CMV infection.

The diagnosis of a bacterial pneumonia was made on clinical grounds if all of the following criteria were present: fever >38.5° C, a localized radiographic infiltrate, and improvement after appropriate antimicrobial therapy. The CFU per milliliter of BALF of all detectable bacteria were determined for each individual patient. A CFU of ≥10^3/ml for a single bacterial strain or a sum of ≥10^7/ml of different microorganisms was considered to support the diagnosis of bacterial pneumonia. The diagnosis of legionellosis was made if *Legionella pneumophila* could be cultured in BALF independent of the CFU per milliliter.

Aspergillus pneumonia was diagnosed if the organism was identified by microscopy and culture in a patient with fever, prolonged (>1 week) neutropenia, and multiple pulmonary infiltrates, or if lung tissue invasion by aspergillus hyphae was identified by transbronchial lung biopsy specimen. The diagnosis of pulmonary cryptococcosis was made if Cryptococcus was found by smears and/or by culture of BALF.

Tuberculosis was diagnosed if *Mycobacterium tuberculosis* was cultured in BALF. Nontuberculous mycobacterial infection (mycobacteria other than tubercle) was diagnosed according to standard criteria. If BALF provided evidence of alveolitis and no specific pathogen was detectable, the diagnosis of nonspecific interstitial pneumonia was made.

**Statistical Analysis**

Most data were not normally distributed and were, therefore, analyzed using nonparametric statistics. The Mann-Whitney U test was used for unpaired samples and the Wilcoxon-signed-rank test was used for paired samples. The relationship between parameters was assessed by means of Spearman correlation coefficient. The significance level was taken as p<0.05.

**RESULTS**

The BAL in two lung segments was well tolerated by all patients. In none of them subsequent respiratory failure attributable to the procedure developed. Table 1 summarizes the pulmonary diagnoses in the study population.

A diagnosis of PCP could be established in 24 patients, 19 of whom were HIV infected and 5 of whom were receiving immunosuppressive medications after transplantation. In 16 patients with AIDS, it was the first episode of *P. carinii* infection, in 2 the second, and in 1 patient the third episode. All three patients with recurrent PCP and one with the first episode had received prophylaxis with aerosolized pentamidine.

In one of them the chest radiograph (CXR) showed predominant involvement of the upper lung lobes.

**Table 1—Pulmonary Diagnoses Established in the Study Population**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>All Patients</th>
<th>HIV Positive</th>
<th>HIV Negative</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Pneumocystis carinii</em></td>
<td>24</td>
<td>19</td>
<td>5</td>
</tr>
<tr>
<td>Pneumocystis carinii</td>
<td>16</td>
<td>10</td>
<td>6</td>
</tr>
<tr>
<td>Bacterial pneumonia</td>
<td>9</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>CMV</td>
<td>10</td>
<td>8</td>
<td>2</td>
</tr>
<tr>
<td>Mycobacterial lung infection</td>
<td>5</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Fungal pneumonia</td>
<td>4</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Non-specific interstitial pneumonia</td>
<td>4</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Fever without pulmonary infiltrates</td>
<td>9</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Diagnosis undetermined</td>
<td>22</td>
<td>22</td>
<td></td>
</tr>
</tbody>
</table>

*Five patients had more than one diagnosis.*
the others showed disseminated bilateral lung infiltrates. Of the 15 patients not receiving pentamidine prophylaxis, 3 had predominant CXR changes in the upper lobes, 1 had a normal CXR, and in the other 11 patients, bilateral diffuse pulmonary infiltrates were found. Four HIV-negative patients showed diffuse bilateral lung infiltrates; one had a normal CXR.

Two patients suffered from a concomitant pulmonary infection: one patient had concurrent CMV pneumonia and another had infection with *M. avium-intracellulare*. The PCP was the only infection in four of five without AIDS; one patient had Legionella pneumonia as well. The number of *P. carinii* clusters assessed by the May-Grünwald-Giemsa stain was compared with the number of *P. carinii* clusters found by the more sensitive toluidine blue stain. Since *P. carinii* clusters seen in the May-Grünwald-Giemsa-stained slides were expressed per number of nucleated cells but were counted per slide in the toluidine blue-stained smears, these values were not expected to be identical. The two different methods, however, correlated well in the assessment of the *P. carinii* density (*r=0.83, p<0.0001*).

In two of the five HIV-negative patients, *P. carinii* clusters were detectable in only one of the two lavaged lung segments. By the less sensitive Giemsa stain, *P. carinii* clusters were found in both lobes in only one of the patients. In one patient with negative Giemsa as well as negative toluidine blue stain (after transplantation of kidney), a single *P carinii* cluster was found by immunofluorescence in both segments. The number of clusters per 500 nucleated cells found in HIV-negative immunosuppressed patients ranged from 0 to 11 (median, 0) in the upper lobes and between 0 to 17 (median 0) in the middle lobes. These concentrations of *P carinii* clusters were significantly lower compared with those of the patients with AIDS: 0 to 165 (median, 20) in the upper lobe and between 0 to 73 (median, 11) in the middle lobe or lingula, respectively (*p<0.01*).

In patients with AIDS, the median number of *P carinii* clusters per 500 nucleated cells was higher in the upper lobes (20 clusters) than in the middle lobe or lingula (11 clusters) (*p<0.05*), regardless of whether the patient had been receiving prophylaxis with aerosolized pentamidine. In a single patient without pentamidine prophylaxis, no *P carinii* clusters were found in the BAL from the middle lobe by all three stains whereas few *P carinii* clusters were found in the upper lobe. In two patients with AIDS, no clusters were found by Giemsa stains in both lobes; in one they were found only in the middle lobe. The cases with negative Giemsa slides showed single clusters in the toluidine and immunofluorescence stains.

The BAL cell differential cell counts in patients with AIDS are shown for the two different lobes in Figure 1. We did not find any significant interlobar differences. The percentage of neutrophils in the BAL correlated with the density of pneumocysts, but...
this correlation was weak (r = 0.58, p < 0.05). In HIV-negative patients with few clusters, we found the same increased percentage of BAL lymphocytes (32.5 and 34.5) and neutrophils (8.5 and 7.0) in both lobes. There were no interlobar differences in alveolar cellularity and in P carinii density, as well.

**Bacterial pneumonia** could be diagnosed by clinical criteria in 16 patients. Ten patients were HIV positive, and six were HIV negative (four after organ transplantation, two without evident immunosuppression). Ten patients had received antibiotics for 1 to 7 days prior to BAL, and six were untreated. The BALF from a segment of the radiologically most involved area contained a much higher percentage of neutrophils (median value, 64.0 percent) than from the segment which was not or less severely involved on the CXR (median value, 14.3 percent; p < 0.0001) (Table 2). In six patients the concentrations of bacteria were above 10^5 cfu/ml in the most affected lobe; three of them had been treated by antibiotics. In five patients pretreated by antibiotics the CFU were less than 10^5/ml in the most involved area; in two the BALF remained sterile. In only one of the patients CFU reached 10^5 CFU/ml in the radiologically not involved lunge segment.

All our patients with CMV pneumonitis (n = 9) had HIV infection. The BAL specimens showed many cells with characteristic intracytoplasmic or intranuclear inclusions in both lobes in seven patients, whereas few cells with positive cytopathogenic effects were found in only one of the two lavaged segments in two patients. All patients had either normal radiologic findings or diffuse bilateral CXR changes. Early antigen was found in all lavaged lung segments. We found normal cell distributions in a single lobe, in both lobes, or mild to moderately increased lymphocytes and/or neutrophils in both lobes independent if the cytopathogenic effects were found in few

<table>
<thead>
<tr>
<th>Table 2—Interlobar BAL Differences in Bacterial Pneumonia*</th>
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<tbody>
<tr>
<td><strong>Radiologically Most Involved Segment</strong></td>
</tr>
<tr>
<td>Pt</td>
</tr>
<tr>
<td>-----</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
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<td>4</td>
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<td>15+</td>
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<td>16+</td>
</tr>
<tr>
<td>16+</td>
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<tr>
<td>Median</td>
</tr>
</tbody>
</table>

*Pt=patient; ++=pretreated by antibiotics; Segm=lung segment; RBl=segment B1 right lung; LB4=segment B4 left lung, etc; N (%)=percentage of neutrophils; P=Pseudomonas; St=streptococci; S=Stomatococcus; E=Enterobacter; C=Citrobacter.

fp<0.0001.
1p<0.005.
or many cells.

Our population with *Mycobacterial infection* (n=10) consisted of eight HIV-positive and two HIV-negative patients. Acid-fast smears were positive in four patients, in all of them in both lobes. In the three patients with tuberculosis, *M tuberculosis* was identified by culture in both lavaged segments (one non-HIV, two HIV positive). Mycobacteria other than tuberculosis (three *M avium intracellulare*, two *M xenopi*, one *M kansasii*) were present in both lavaged segments in six patients, all being HIV positive. In a single patient (HIV positive) *M simiae* was found but in one of the two lavaged segments. There were no characteristic changes in the cell differential count; four patients had normal cell distribution, in three the BAL neutrophils were increased, and in three others a BAL lymphocytosis was found in both lobes. In eight patients the cell distribution in both lung segments were similar.

Three patients suffered from *invasive aspergillosis*, two from *pulmonary cryptococcosis*. One patient with cryptococcosis had AIDS and one was treated by corticosteroids after heart transplantation. The three patients with aspergillosis were receiving chemotherapy for the treatment of leukemia. Fungi were identified from both lobes in all patients. Two patients with cryptococcosis showed a comparable increase of BAL lymphocytes in both lung segments. The patients with aspergillosis had slightly to moderately increased neutrophils and lymphocytes with differences in the cell distribution (>10 percent) not related to the radiologic involvement.

All patients with *nonspecific interstitial pneumonia* (n=4) were HIV positive and all had bilateral radiologic changes. The cell differential count showed a marked (between 33.5 and 78.5) increase of BAL lymphocytes in both lung segments. One patient had slightly increased percentage of neutrophils as well.

The studied population comprised four HIV-infected febrile patients without pulmonary infiltrates, in whom BAL was not diagnostic. Despite infiltrates on CXR, no microorganisms could be found in nine additional patients (four HIV positive, two receiving chemotherapy or corticosteroids, two after solid organ transplantation, and one without immunosuppression). Four of them showed interlobar differences (>10 percent) in the percentage of neutrophils or lymphocytes, which were in accordance with the radiologic changes. In one patient, an increased percentage of lymphocytes was found only in the radiologically most clearly involved segment. The diagnosis remained undetermined in these cases since no further invasive procedures were performed and also the follow-up did not give any hint in regard to etiology.

**Discussion**

The uniformity of alveolitis in pulmonary infections and the additional information obtainable by performing BAL in more than one lung segment has not been systematically evaluated (to our knowledge), except in HIV-infected patients with PCP. It is unknown whether the number of *P carinii* clusters in the lung correlates with the functional impairment or the survival of patients with PCP. Colangelo et al showed that in patients with AIDS with PCP, treated with one of the usual regimens, *P carinii* cluster counts were reduced by more than 50 percent in 24 of 25 cases within 21 days. The five patients who died early in the course of PCP had significantly higher initial cluster counts than patients who survived. Orholm et al found no correlations between the number of *P carinii* and the severity of PCP. We found a weak but significant correlation between the percentage of BAL neutrophils and the number of *P carinii* clusters (r=0.58, p<0.05) in both lobes in patients with AIDS. Our patients showed only mild neutrophil alveolitis and their PCP was moderate or mild. Only one of our patients died within 3 weeks after diagnosis. He had the highest number of *P carinii* clusters in our studied population (165 in the upper lobe and 73 in the middle lobe).

As other investigators, we found much lower numbers of *P carinii* clusters in patients without HIV infection compared with patients with AIDS. In some of them, single cysts were found only by monoclonal antibodies. Furthermore, the percentages of neutrophils recovered from patients with AIDS were substantially less than in the non-AIDS group.

It is notable that 7 of 19 AIDS patients had markedly increased proportions of eosinophils (3.0 to 11.3 percent) in comparison to none in the non-AIDS group. This is in agreement with other observations.

In patients with AIDS receiving prophylaxis with aerosolized pentamidine, a preferential PCP involvement of the upper lobes has been reported and is thought to be due to a poor apical aerosol deposition. However, several patients without inhalational pentamidine prophylaxis presenting with apical PCP were recently described. Only four of our patients had inhaled pentamidine prophylactically. We found, however, higher upper lobe *P carinii* concentrations in 17 of the 19 patients with AIDS. These findings are, therefore, not attributable to the aerosol prophylaxis. An upper lobe predominance in the CXR was found only in four patients (21 percent). Similar results have recently been reported by Baughman et al in a larger population.

In only 1 of 19 patients with AIDS with PCP, the organisms were not found in both lobes, whereas in
2 of 5 immunocompromised patients without HIV infection, P carinii clusters were detected in BALF from one lobe only. Although our population of non-HIV PCP is small, we believe that this difference is most likely attributable to the higher load of P carinii in patients with AIDS. Meduri et al found in a mixed population of HIV-positive and negative patients two-segment BAL more sensitive for the diagnosis of PCP. Since they performed BAL in the right middle lobe and the lingula and not unilateral in the upper lobe and middle lobe or lingula, respectively, as we did, their results cannot be compared with our findings.

We conclude that in patients with AIDS with suspected PCP, there is no need to perform BAL in two lung segments. The yield for P carinii may be increased by performing BAL in the apical segments because the number of P carinii clusters is highest in the upper lobes even in patients without inhalational pentamidine prophylaxis. Two-segmental BAL, however, increases the sensitivity of BAL for PCP diagnosis in immunosuppressed patients without HIV infection.

Although differences in BAL findings between the radiographically involved and noninvolved lobes were expected in bacterial pneumonia, we are not aware of any study addressing this issue. The BAL sampling area is usually chosen according to the radiographic localization of a new or progressive pulmonary infiltrate. We found a markedly increased number of cells and percentage of neutrophils in the BAL of the most involved lung segment compared with an unaffected or less involved lobe. However, the differential cell count of BALF in bacterial pneumonia is of limited value. In patients not having received antibiotics, a reliable distinction between bacterial colonization and pneumonia is possible by quantitative culture of BALF. In only one of seven patients, bacteria in a concentration of >10^5 CFU/ml of BALF could also be cultured from a radiologically less involved area. These findings confirm that a single BAL in the radiographically most affected area is the adequate procedure for the diagnosis of bacterial pneumonia.

Only few publications contain information about the BALF cellularity in pulmonary CMV infections. Woods et al reported that immunosuppressed patients (three quarters of them were liver transplant recipients) showed an alveolar lymphopenia compared with those without CMV pneumonia and normal control subjects. These results differ from the findings of an increased number of BAL lymphocytes in allogeneic bone marrow transplant recipients with CMV pneumonia. All our studied patients with CMV pneumonia were HIV positive. The cell distribution pattern varied widely and showed no consistent pattern. In two of nine patients in whom the cytopathogenic effects were present in only one of the lavaged segments, the diagnosis of CMV pneumonitis might have been missed by a single segment lavage.

Several groups studied BALF from tuberculous lesions and found an increased number of lymphocytes as the predominant abnormality. Ainslie et al found the highest number of lymphocytes in patients with miliary tuberculosis. In localized pulmonary tuberculosis, the absolute number of lymphocytes was elevated in the BALF of the affected as well as the unaffected lobe with a trend toward more lymphocytes in the affected lobe. A modest increase in the percentage of neutrophils was seen in the affected lobe of localized pulmonary tuberculosis as well. Our population with mycobacterial infections is too small and too heterogeneous to draw any conclusions in regard to the significance of the cell distribution of the lavaged lung segments. In four patients in whom mycobacteria were numerous enough to be detectable by microscopy, they were visible in both lavaged segments. In one of the eight patients with AIDS the diagnosis of mycobacterial infection (M kansasi) would have been missed in BAL from a single lung segment.

In all our patients with fungal pneumonia, a marked increase in the percentage of lymphocytes in at least one lobe and in three of them, the percentage of neutrophils in BALF was found. This is in agreement with a study of Baughman et al. Furthermore, the fungi could be cultured from both lung segments. Our studied population, however, was too small to draw any reliable conclusions in regard to the role of two-segment BAL in the assessment of this type of infection.

In conclusion, an increase in the diagnostic sensitivity by performing BAL in two lung segments is limited to cases where PCP is a relevant consideration in immunocompromised patients without HIV infection. In bacterial pneumonia, BAL can be performed in a single radiographically involved lung segment without a loss in diagnostic sensitivity. Since our study population of patients with CMV pneumonitis, mycobacterial infections, and fungal infections was small, no reliable conclusions are possible and BAL of more than one lung segment seems justified until more information is available.

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