The role of bronchoscopy in the diagnosis and treatment of pulmonary disease in HIV-infected patients

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Abstract

Background: Pulmonary disease is the most common reason for presentation and the major cause of death in HIV-infected patients. There has been an evolution in the optimal approach to the investigation of a pulmonary infiltrate in HIV-infected patients since the introduction of induced sputum for the diagnosis of Pneumocystis carinii pneumonia (PCP).

Aims: To evaluate the usefulness of flexible fibreoptic bronchoscopy (FFB), bronchoalveolar lavage (BAL), transbronchial biopsy (TBB) and bronchial brushings (BB) in the diagnosis of pulmonary disease in HIV-infected patients and to examine the effect of FFB on changes in therapy and survival.

Methods: The histories of all HIV-infected patients referred to Fairfield Hospital for FFB between January 1990 and June 1993 were examined retrospectively.

Results: Forty-two FFB were performed on 41 patients (40 male and one female). Definitive diagnoses made at FFB included Kaposi's sarcoma (KS) (n = 9), invasive aspergillosis (n = 5), PCP (n = 4), Mycobacterium avium complex (MAC) pneumonia (n = 2), cytomegalovirus (CMV) pneumonia (n = 1), Cryptococcus neoformans pneumonia (n = 1), microsporidium (n = 1) and Pseudomonas aeruginosa pneumonia (n = 1). TBB and BB did not provide a diagnosis for diseases not seen macroscopically at FFB or diagnosed by BAL. FFB findings altered diagnosis in 21/42 (50%) presentations and changed therapy in 26/42 (62%) cases.

Conclusions: FFB together with BAL altered the working diagnosis and changed therapy in a significant number of patients. TBB and BB should not be routinely performed in all patients as these procedures are of limited value in this setting. (Aust NZ J Med 1995; 25: 133-139.)

Key words: Bronchoscopy, HIV, AIDS, pulmonary disease.

INTRODUCTION

Pulmonary disease is the commonest reason for presentation1 and the commonest cause of death2 in HIV-infected individuals. In the 12 years since the HIV epidemic began there has been an evolution in the mode of investigation of pulmonary disease in such patients. Initially, the approach taken was similar to that used in the investigation of pulmonary disease in other immuno-suppressed individuals. Many patients underwent fiberoptic bronchoscopy or even proceeded to open lung biopsy to achieve a diagnosis.1

In 1986 the introduction of induced sputum with its high sensitivity in diagnosing Pneumocystis carinii (PC)
infection led to a decline in the frequency of bronchoscopy in this group of patients.

This study retrospectively evaluated the usefulness of flexible fibreoptic bronchoscopy (FFB), bronchoalveolar lavage (BAL), transbronchial biopsy (TBB) and bronchial brushings (BB) in the diagnosis of pulmonary disease in HIV-infected patients during a 30 month period after the introduction of induced sputum collection at a single centre. The effects of the bronchoscopic results on alterations in therapy and on patient outcome were also examined.

PATIENTS AND METHODS

The histories of all HIV-infected patients who underwent FFB at Fairfield Hospital, Melbourne between January 1990 and June 1993 were examined retrospectively. Fairfield Hospital is a major referral centre for HIV-infected persons in the state of Victoria and 1675 HIV-infected patients attended this hospital between January 1981 and June 1993. During the study period, 62% of Victorian AIDS notifications were from Fairfield Hospital (personal communication, Dr S. Thompson, Macfarlane Burnet Centre for Medical Research). Pneumocystis carinii pneumonia prophylaxis for patients with CD4 lymphocyte count < 200 × 10^6/L was introduced at Fairfield Hospital in 1986.

Patients underwent FFB to investigate pulmonary symptoms with or without chest radiographic changes. All patients with suspected PCP had produced at least three PC-negative induced sputum specimens prior to FFB and had either failed to improve or had deteriorated during a therapeutic trial of conventional anti-PCP treatment. The other patients had either negative induced sputum results or were failing to improve on apparently appropriate therapy.

FFB were performed using an Olympus BF-IT10 bronchoscope. Briefly, all patients were monitored with pulse oximetry and received intranasal oxygen during the procedures. Patients were premedicated with 50-100 μg intravenous fentanyl and 0-5 mg intravenous midazolam. The bronchoscope was passed transnasally after the application of topical 4% lignocaine (Astra, Sydney) to the nose and pharynx. Further lignocaine (4%, topical) was applied to the cords prior to intubation, and then to the trachea and both main bronchi (2%, topical). After examination of the bronchial tree the tip of the bronchoscope was wedged into an appropriate lobar segmental bronchus and BAL was performed. Twenty five mL aliquots of warmed sterile 0.15 M NaCl were instilled and then immediately aspirated using low pressure suction, the total instillate not exceeding 100 mL. When indicated, BB using a sheathed brush (Olympus BC-15C) and TBB with or without endobronchial biopsy (Olympus biopsy forceps BF-19C) were also performed.

BAL fluid was cultured for bacteria, mycobacteria, fungi, and, when indicated, viruses (CMV, adenovirus, herpes simplex, varicella zoster, enterovirus and influenza). Cytocentrifuge preparations of BAL fluid were examined after gram, toluidine blue and Ziehl-Neelsen staining. BB were air dried and stained with Ziehl-Neelsen staining or were fixed with Cytofix and stained with silver methenamine and Papanicolaou stains. TBB and endobronchial biopsies were fixed in formalin and examined microscopically after haematoxylin-eosin, Grocott's methenamine silver and Ziehl-Neelsen staining. Cultures for viruses, fungi and mycobacteria were performed as for BAL.

In evaluating the effect of FFB on clinical practice a change in diagnosis was defined as a situation where the working differential diagnosis and final clinical diagnosis differed as a result of FFB.

Definitive diagnoses were made according to the following criteria. Briefly, a diagnosis of PCP was made if organisms were identified by silver staining of induced sputum, BAL, BB or TBB; a diagnosis of bacterial pneumonia was made if the patient had fever or pleuritic pain with focal or diffuse radiographic abnormalities with culture of a bacterial pathogen; invasive aspergillosis was diagnosed as probable or definite according to accepted criteria; CMV pneumonia was diagnosed by the presence of at least one characteristic intranuclear inclusion body in association with some features of interstitial inflammation of lung tissue; MAC and cryptococcal pneumonia were diagnosed on the basis of growth of the organism plus recovery of the organism from at least one other non-pulmonary site. KS was diagnosed on the basis of visual identification at bronchoscopy of typical red or violaceous raised or plaque like lesions in the bronchial tree with or without histological confirmation. Definitive diagnoses were not based solely on an appropriate response to treatment.

### TABLE 1

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>D</th>
<th>F</th>
<th>N</th>
<th>Total</th>
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<tr>
<td>Kaposi's sarcoma</td>
<td>9</td>
<td></td>
<td></td>
<td>9</td>
</tr>
<tr>
<td>Invasive aspergillosis</td>
<td>3</td>
<td>2</td>
<td></td>
<td>5</td>
</tr>
<tr>
<td>PCP</td>
<td>2</td>
<td>2</td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>MAC pneumonia</td>
<td>2</td>
<td>1</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>CMV pneumonia</td>
<td>1</td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Cryptococcus neoformans</td>
<td>1</td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Microsporidum</td>
<td>1</td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Pseudomonas aeruginosa pneumonia</td>
<td>1</td>
<td></td>
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<td>1</td>
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<tr>
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<td>21</td>
</tr>
<tr>
<td>Total</td>
<td>45</td>
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<td></td>
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</tr>
</tbody>
</table>

Abbreviations: D = Diffuse infiltrate; F = Focal infiltrate; N = Normal; PCP = Pneumocystis carinii pneumonia; MAC = Mycobacterium avium complex; CMV = Cytomegalovirus.

NB. Total diagnoses include three patients with more than one diagnosis.
RESULTS
Between January 1990 and June 1993, 230 definitive diagnoses of HIV-associated PCP and 87 presumptive diagnoses of PCP (according to CDC criteria) were made. Forty-two bronchoscopies were performed on 41 patients (40 males). The mean age was 34 years (range 24-48 years). HIV risk factors were homosexual and heterosexual acquisition (n = 40), blood transfusion (n = 1) and heterosexual acquisition (n = 1). The mean CD4 count was 38 x 10^6/L (range 0-410 x 10^6/L). Thirty patients were receiving anti-retroviral chemotherapy at the time of bronchoscopy. Chest radiographs (CXR) were normal in 2/42 (5%) of cases, and the abnormalities in the CXR from the other patients are described in Table 1.

FFB was performed to confirm or refute working differential diagnoses of PCP (19 patients), KS (12 patients), bacterial pneumonia (six patients) and tuberculosis (three patients); and for investigation of fever (one patient), cough (one patient) and unexplained dyspnoea (one patient).

Bronchoscopic Results (Tables 2 and 3)
BAL was performed in all cases, with TBB with or without endobronchial biopsy (EB) in 32 of 42 and BB in 23 of 42 cases. BAL yielded at least one organism in 31 of 42 cases. In 18 of 42 BAL only one organism was found. Two pathogen were found in 11 BAL and three pathogens in two BAL. No organisms were found in 11 BAL (25%).

At least three TBB specimens were obtained. Specimens were of adequate size (at least 2 x 1 mm) in 30/32 cases. TBB diagnosed KS (two patients), CMV pneumonia (one patient), PCP (two patients), pulmonary MAC (two patients) and C. neoformans (one patient). No diagnostic features were found in 22/32 (69%) biopsies although chronic inflammatory changes were seen in two and evidence of fibrosis in another.

BB diagnosed PCP in one/23 patients and no other diagnoses were made via this technique.

P. carinii Pneumonia
Although a provisional diagnosis of PCP was made in 19 cases prior to FFB, PCP was only diagnosed in four cases; in two patients from BAL and TBB, in one patient from BAL and BB and in one patient from BAL only. The CXR of these patients revealed focal infiltrates in two and diffuse infiltrates in the remaining two (Tables 1-3). All four patients were taking anti-PCP prophylaxis.

Cytomegalovirus
CMV was cultured from 15/31 (50%) specimens submitted for viral culture, in 7/15 in the setting of polymicrobial culture (but never in conjunction with PCP). TBB was performed on 12 of these patients and showed evidence of CMV pneumonitis in only one patient. No patients were treated for CMV pneumonitis (Tables 1-3).

Bacterial Infection
Pyogenic bacteria were isolated in four patients and in three cases the isolate was considered to be clinically significant. The organisms were E. coli, S. pneumoniae, S. aureus and P. aeruginosa. In two patients, the organisms (E. coli and P. aeruginosa) were also detected on induced sputa obtained prior to bronchoscopy. In all four of these patients, other organisms were also isolated: C. albicans in three patients and CMV in one patient. No patient had concomitant positive blood cultures for these organisms. CXR showed a diffuse infiltrate in three patients and was normal in one patient (Tables 1-3). E. coli was isolated in the patient with a normal CXR and appropriate response to antibiotic therapy resulted in the diagnosis of acute bronchitis. S. aureus was isolated from a patient thought to have possible pulmonary KS on the basis of nodular pulmonary opacities and numerous cutaneous lesions. He presented with progressive dyspnoea and a dry cough, was treated empirically for PCP and improved. He received no specific anti-staphylococcal

<table>
<thead>
<tr>
<th>TABLE 2</th>
<th>TABLE 3</th>
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<tbody>
<tr>
<td>Microbiological Results of Bronchoalveolar Lavage, Transbronchial Biopsy and Bronchial Brushings</td>
<td>Diagnoses from Transbronchial and Endobronchial Biopsy (from 32 Patients)</td>
</tr>
<tr>
<td></td>
<td>BAL</td>
</tr>
<tr>
<td>Number of specimens</td>
<td>42</td>
</tr>
<tr>
<td>Cytomegalovirus</td>
<td>15</td>
</tr>
<tr>
<td>Candida albicans</td>
<td>8</td>
</tr>
<tr>
<td>Aspergillus fumigatus</td>
<td>7</td>
</tr>
<tr>
<td>Pneumocystis carinii</td>
<td>4</td>
</tr>
<tr>
<td>Mycobacterium avium complex</td>
<td>3</td>
</tr>
<tr>
<td>Other pyogenic bacteria</td>
<td>4</td>
</tr>
<tr>
<td>Cryptococcus neoformans</td>
<td>1</td>
</tr>
<tr>
<td>Microsporidium</td>
<td>1</td>
</tr>
<tr>
<td>Candida krusei</td>
<td>1</td>
</tr>
<tr>
<td>Rhinovirus</td>
<td>1</td>
</tr>
<tr>
<td>No organism</td>
<td>11</td>
</tr>
<tr>
<td>Kaposi's sarcoma</td>
<td>2</td>
</tr>
<tr>
<td>Pneumocystis carinii</td>
<td>2</td>
</tr>
<tr>
<td>CMV pneumonia</td>
<td>1</td>
</tr>
<tr>
<td>Cryptococcus neoformans</td>
<td>1</td>
</tr>
<tr>
<td>MAC pneumonia</td>
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</tr>
<tr>
<td>Chronic inflammation</td>
<td>2</td>
</tr>
<tr>
<td>Insufficient specimen</td>
<td>2</td>
</tr>
<tr>
<td>No diagnostic features</td>
<td>20</td>
</tr>
</tbody>
</table>

Abbreviations: BAL = Bronchoalveolar lavage, TBB = Transbronchial biopsy, BB = Bronchial brushings.
treatment. Although no microscopic or macroscopic abnormality was seen at bronchoscopy he received chemotherapy and died three months following bronchoscopy. *P. aeruginosa* was isolated from a patient with large volumes of purulent sputum who was treated with ciprofloxacin alone with a reduction of sputum volume although the organism was never cleared from his sputum. At bronchoscopy he was found to have macroscopic abnormalities consistent with KS but no TBB was taken as he was thrombocytopenic. He died three months following bronchoscopy and at autopsy was found to have extensive pulmonary KS, acute bronchitis and focal bronchopneumonia. *S. pneumoniae* was isolated from a patient who went on to have an open lung biopsy which revealed pulmonary KS. He was treated with broad spectrum antibiotics including imipenem, doxycycline and amphotericin.

*Mycobacterium avium* complex was isolated in three patients, each in association with MAC bacteraemia. Two of the three patients had MAC isolated from induced sputum in the month prior to FFB. No cases of *Mycobacterium tuberculosis* were diagnosed by FFB.

**Fungal Infection**

*Candida* spp. were isolated from BAL in nine patients. In two patients it was a sole isolate. In one of these the patient had a diffuse pulmonary infiltrate with minimal symptoms. The isolate was not treated and a repeat FFB six weeks later failed to isolate the organism again. In the other patient, white plaques were seen on the mucosa of the bronchial tree and hence the candida was thought to be clinically significant and the patient was treated with itraconazole. In the other seven patients only the copathogen was treated. *Aspergillus* spp. were isolated from BAL in seven patients. Five patients were treated, three with oral itraconazole and two with intravenous amphotericin. In the two untreated cases the fungi were isolated with the copathogens *E. coli* and MAC. Appropriate treatment of these copathogens induced an improvement in both cases. *C. neoformans* was isolated from BAL and TBB of one patient in the setting of cryptococcaemia, meningitis and a diffuse pulmonary infiltrate. This was treated with intravenous amphotericin.

**AIDS-Related Pulmonary Neoplasia**

Nine patients had cherry red, raised macroscopic endobronchial abnormalities consistent with KS. EB together with TBB were performed on two patients; EB alone on two patients; TBB alone on four patients; and neither EB nor TBB on one patient. Only two patients had TBB evidence of KS. Of the other seven, TBB with or without EB failed to show any diagnostic abnormality. In none of the biopsies was a ‘non diagnostic’ result consequent upon the biopsy specimen being of inadequate size (<1 x 2 mm in diameter), and in each case two to three biopsy specimens were submitted for histological examination. In one patient, FFB and TBB were normal but the patient was subsequently shown to have KS on open lung biopsy. Six of the nine patients were given chemotherapy. Only one patient with an antemortem diagnosis of KS underwent autopsy and the diagnosis of pulmonary KS was confirmed.

**Complications**

Asymptomatic pneumothoraces requiring intercostal drainage because of their size occurred in 2/32 (6%) patients after TBB. Neither patient had PCP.

**Clinical Use of Bronchoscopy**

Diagnoses made at FFB together with their CXR appearances are shown in Table 1. FFB changed the provisional working diagnosis in 21/42 (50%) cases.

Changes in therapy following FFB were made in 26/42 (62%) cases (Table 4).

**Other Investigations**

Two patients underwent fine needle aspiration (FNA) of pulmonary lesions under computer tomographic control after FFB had not yielded a diagnosis. In neither case was FNA diagnostic and one FNA was complicated by a small pneumothorax which did not require treatment. In both patients autopsies were subsequently performed, providing retrospective diagnoses of a lung abscess with gram positive cocci in chains, which failed to grow on post-mortem culture, and pulmonary non-Hodgkin’s lymphoma. Only one patient came to open lung biopsy which was successful in providing a diagnosis of pulmonary KS. In this patient FFB with TBB and pleural aspiration of an effusion had failed to determine the cause of a rapidly progressive pulmonary infiltrate.

**Survival**

The mean survival after FFB was 112 days (range two-521 days). The one month survival was 72%. The mean survival of patients with a diagnosis of KS was 123 days. There was no significant difference in mean survival when patients who had received chemotherapy

| TABLE 4 |
| Changes in Therapy Following Bronchoscopy |
| Change | |
| PCP therapy started | 3 |
| PCP therapy ceased | 4 |
| Chemotherapy | 7 |
| Other antibiotics | 4 |
| antifungals | 4 |
| palliative care | 4 |
| No change | 17 (38%) |

Abbreviation: PCP – Pneumocystis carinii pneumonia.
were compared to those who had received supportive care alone (Mann-Whitney, $p > 0.06$).

DISCUSSION
The role of FBB, BAL, TBB and BB in the investigation of pulmonary disease in the HIV-infected patient has changed during the AIDS epidemic. Induced sputum examination is now clearly the primary investigation of choice for diagnosing PCP and mycobacterial disease. However, the subsequent approach when induced sputum is non-diagnostic is less clearly defined. In our institution a trial of empirical PCP therapy is begun if the clinical pattern is consistent with PCP. If the patient deteriorates or fails to improve, FFB is performed. This approach is less invasive for the patient, has been shown previously to be cost effective and is associated with the same one month survival rate as immediate FFB.

The findings of FFB in HIV-infected patients have been reported previously, however, all these studies were performed either prior to or during the introduction of routine induced sputum collection at each of several institutions. In previous studies the most frequent diagnosis following FFB was PCP with a frequency ranging from 40-60%. In this study, PCP was diagnosed in only four/42 (10%) patients, but it is notable that sputum induction for detection of PCP was introduced at Fairfield Hospital in 1986, and was in routine use during the study period. Two hundred and thirty definitive and 87 presumptive diagnoses of PCP (on CDC criteria) were made at Fairfield Hospital with only four cases of PCP diagnosed by FFB during this time period. This clearly suggests that few patients with PCP now require FFB for diagnosis provided that induced sputum is used.

CMV was isolated in 50% of cases. There is wide variation in the reported isolation rates of CMV from BAL in HIV positive patients, ranging from 19% to 49%. The differences may relate to variability of culture techniques, to the degree of contamination by saliva or to the degree of immunosuppression of the population studied. CMV excretion increases with increasing immunosuppression. Despite guidelines for establishing the diagnosis of CMV pneumonitis in HIV-infected patients, the role of CMV as a pulmonary pathogen in HIV/AIDS remains unclear.

Most clinicians and researchers in this area regard CMV as a rare cause of pulmonary disease in the HIV-infected population which infrequently requires treatment. Millar et al. showed prospectively that failure to treat CMV isolated from BAL in HIV-infected patients with pulmonary infiltrates does not alter outcome and that many patients improve when the copathogen alone is treated or no treatment is given. In contrast, however, are case reports of CMV pneumonitis responding to gancyclovir and autopsy series reporting death from respiratory failure secondary to CMV pneumonia. In the same autopsy series, antemortem diagnosis was shown to be insensitive and of low specificity. Ten out of 13 patients with significant CMV pneumonitis were not diagnosed by TBB and six/nine were not diagnosed with open lung biopsy (OLB).

New pyogenic bacteria were uncommonly isolated in our patient group. Only two/four patients had pyogenic bacteria not previously isolated on induced sputum. Two patients had clinical evidence of bacterial pneumonia and one patient had purulent bronchitis with a normal CXR. The role of pyogenic bacteria in pneumonia in HIV-infected patients varies in different series. Jiminez et al. reported the isolation of pyogenic bacteria from BAL in 12% of cases and legionella in 5% of cases in a population containing 80% injecting drug users (IDUs). However, other series report an absence of pyogenic bacteria in BAL.

Heurlin et al. isolated bacteria from BAL in 16/82 FFB but commented that only three/16 patients had clinical evidence of pneumonia. The higher incidence of pyogenic bacteria in Heurlin et al.'s study may be explained partly by the high number of IDUs in their study population. A higher incidence of bacterial infections is known to occur in IDUs than in other HIV-infected individuals although the reason for this is unknown. Alternatively, the true incidence of bacterial pneumonia may depend on the definition of bacterial infection and how it is distinguished from oropharyngeal contamination. This has been defined in immunocompromised hosts by quantitative culture together with a cell differential for detection of squamous epithelial cells. This technique is not used at our institution.

In this study no cases of pulmonary tuberculosis were diagnosed by FFB. In Victoria there have been only 22 cases of tuberculosis in HIV-infected patients since the beginning of the AIDS epidemic. Fifteen of these had pulmonary involvement and 14/15 were diagnosed by sputum examination. One patient had a positive culture from bronchial washings prior to 1990 (personal communication, Mycobacterial State Reference Laboratory, Fairfield Hospital).

The frequency of isolation of aspergillus in this study is higher than in other reported series. Pulmonary aspergillosis is rare in HIV/AIDS and two forms of disease are described: invasive aspergillosis and obstructing bronchial aspergillosis. Five out of seven patients fulfilled the criteria for 'probable invasive aspergillosis' as defined by the presence of pulmonary infiltrates with a positive bronchoscopic culture of aspergillus species without the identification of other significant pathogens. Three of the seven patients were suspected of having acquired the organism nosocomially as there were seven cases of *A. fumigatus*.

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in the same HIV ward over a five month period. However, despite extensive environmental sampling this was not substantiated. The other four patients were distributed evenly over the time period studied.

The role of TBB in HIV/AIDS patients is of particular interest as it contributes significantly to the morbidity of FFB and to the risk of blood exposure to the bronchoscopist. In this study, TBB confirmed the diagnosis of KS in two/nine patients with endobronchial abnormalities suggestive of KS but four/four EB and four/six TBB were negative. Only one of the patients with a bronchoscopic diagnosis of KS underwent postmortem which confirmed the presumptive diagnosis of KS based on the appearance of macroscopic abnormalities of the bronchial tree. However, the mean time to death was consistent with the known poor prognosis of pulmonary KS. Previous studies suggest that KS is difficult to diagnose by TBB due to its focal nature. In this study changes in therapy such as commencement of chemotherapy or institution of palliative care were made according to the macroscopic endobronchial abnormalities observed by the bronchoscopist, with or without a positive biopsy result. Diagnostic yield for PCP was not enhanced by TBB in contrast to previous reports where TBB is reported to have a sensitivity of 87%11,12 to 100%13 and to be complementary to BAL. We were, however, unable to assess the frequency of false negative diagnoses of PCP. As only three patients underwent postmortem examination (none of these had a missed diagnosis of PCP), this study does not have a true gold standard to enable evaluation of the true specificity and sensitivity of TBB in our institution.

The complications of TBB are severe haemorrhage and pneumothorax. In this study there was no significant haemorrhage but a pneumothorax rate of 6% (two/32) was recorded. This is comparable with complication rates in other series of HIV-infected patients where the incidence of pneumothoraces after TBB ranges from 6-9%. These figures are higher than those quoted for HIV-negative populations and may be considered to be unacceptable high when comparable diagnostic yields may be obtained via BAL. BB were not of diagnostic value in this study, confirming previous reports in which BB has not contributed to the diagnostic yield of BAL and has added significantly to the cost of FFB.4 There have been reports however, of the benefit of BB in diagnosing tuberculosis25 and cryptococcosis26 in the HIV-infected population.

In this study only two patients, both with diffuse lung opacities, required subsequent CT-guided FNA of pulmonary lesions and one required OLB. FNA is of limited value in the diagnosis of diffuse infective processes and the risk of pneumothorax is higher than for TBB. Neither FNA was diagnostic in this study and the pneumothorax rate was one/two (50%). Although OLB is supported by some authors as an appropriate investigation after a non-diagnostic FFB, others have reservations about its usefulness in the investigation of pulmonary disease in patients with HIV infection if the preceding FFB has included a TBB. The use of less invasive surgical techniques such as videoscopically assisted thoracoscopic surgery may make OLB a more feasible diagnostic procedure in highly selected patients where FFB with BAL and TBB has proved unhelpful.

FFB altered the clinical diagnosis in 50% of patients and, more significantly, altered management in 62% of patients. Thus, FFB appeared to have an impact on patient care, allowing the withdrawal of toxic drugs or the institution of appropriate antibiotics or chemotherapy where indicated. In instances where the diagnosis was of a condition which responds poorly to treatment, both the patient and the treating physician were in a better position to decide upon withdrawal of therapy where appropriate. The true clinical efficacy of these decisions and their effect on morbidity and mortality are difficult to assess adequately retrospectively. The multitude of diagnoses, the small numbers of patients with each diagnosis and the high likelihood of concurrent non-pulmonary AIDS-related complications in these patients further compounds this difficulty. We hope to answer this complex question in a prospective study.

FFB is clearly useful in the evaluation and treatment of the HIV patient with a pulmonary infiltrate and negative induced sputa. BAL appears to be the most sensitive method of establishing an infective aetiology. The role of TBB should be considered carefully and we recommend its use be reserved for those patients without classical endobronchial lesions of KS in whom KS is suspected clinically, or in patients where tuberculosis or cryptococcosis are suspected. BB does not contribute significantly to diagnostic yield and is not recommended as a routine.

Our approach to the investigation of the HIV-infected patient with a pulmonary infiltrate and negative induced sputa is to perform FFB and BAL only as the initial investigation. If the BAL is non-diagnostic or the patient fails to improve with treatment, then repeat FFB with TBB is recommended.

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References


