

Mycobacterial contamination of fiberoptic bronchoscopes

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Abstract
Contamination of bronchoalveolar lavage specimens by environmental mycobacteria in hospital water supplies may lead to diagnostic confusion, particularly in immunocompromised patients. Mycobacteria may become concentrated in the tubing of bronchoscope disinfecting machines. It is very difficult to eradicate these organisms once contamination has occurred.

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Specimens taken by bronchoalveolar lavage at bronchoscopy are valuable in the investigation of mycobacterial infection caused either by *Mycobacterium tuberculosis* or opportunistic mycobacteria. As the population of immunocompromised patients increases, these infections are likely to become more common. However, some *Mycobacterium* spp are ubiquitous in the environment and have been found as contaminants of specimens and cultures.¹ It is not usually possible to differentiate between pathogenic and contaminating organisms when they are first seen on microscopic study of a Ziehl-Neelson stained smear of the specimen. In view of the long period required to culture mycobacteria the clinician must decide whether or not to treat before culture results are known. In immunocompromised patients, or patients in whom tuber-

culosis is thought possible, this may mean that several months of potentially toxic treatment is given.

Methods and results

In the 14 month period from March 1991 to May 1992 we experienced 15 episodes of contamination of bronchoalveolar lavage specimens with an environmental *Mycobacterium* sp. During this time 180 bronchoscopies were performed and 76 bronchoalveolar lavage specimens were sent for microscopy and culture for mycobacteria. Details of the 15 Ziehl-Neelson positive patients are shown in the table. Nine of the 15 patients were immunocompromised and were under investigation for pulmonary infiltrates or respiratory symptoms. The remaining six patients presented with symptoms requiring exclusion of tuberculosis. Eight of the patients were treated with anti-tuberculous therapy when Ziehl-Neelson films were positive, as it was not possible to exclude mycobacterial infection on clinical grounds. In six patients other diagnostic tests were positive, as shown, although in four of these coexisting mycobacterial infection could not be excluded for certain and treatment was continued.

Subsequently all but one of the mycobacterial cultures of clinical specimens were negative, even after prolonged incubation (12 weeks), suggesting perhaps that the mycobacteria seen were not viable. However, as contamination of bronchoalveolar lavage specimens was considered likely, the Olympus EW-20 machine used to disinfect all our fiberoptic endoscopes was investigated. *M. chelonae* and *M. fortuitum* were isolated from debris found inside the water inlet and all drainage tubes of the machine (figure). These tubes were replaced. Later, *M. chelonae* was also isolated from samples of sterile water flushed through the bronchoscopes. *M. xenopi* was isolated from a bronchoalveolar lavage specimen from one patient with haemoptysis and cavitating upper lobe changes seen on the chest radiograph.

Patient details of positive Ziehl-Neelson stained films from bronchoalveolar lavage specimens

Sex	Age (y)	Underlying disease/symptoms	Ziehl-Neelson	Culture	Other positive findings	Treatment with antituberculous drugs
M	56	Renal transplant	+	Negative	Lymphoma	Yes
F	46	Renal transplant	+	Negative	Cytomegalovirus	Yes
M	81	Haemoptysis	+	Negative		No
M	46	Renal transplant	+	Negative	<i>Pneumocystis carinii</i>	Yes
M	83	Haemoptysis	+	Negative		No
M	31	AIDS	+	Negative		Yes
M	63	Cough, weight loss	+	Negative		No
F	62	Rheumatoid arthritis with alveolitis	+	Negative		Yes
F	52	Renal transplant	+	Negative	<i>Pneumocystis carinii</i> / Cytomegalovirus	No
F	28	Status asthmaticus	+	Negative		No
M	74	Haemoptysis	+	Negative		Yes
M	61	Upper lobe shadowing on chest radiograph	+	<i>M. xenopi</i>		Yes
M	55	Lymphoma, pulmonary infiltrates	+	Negative	<i>Pneumocystis carinii</i>	No
M	19	Relapsed acute myeloblastic leukaemia	+	Negative		No
M	40	AIDS	+	Negative	<i>Pneumocystis carinii</i>	Yes

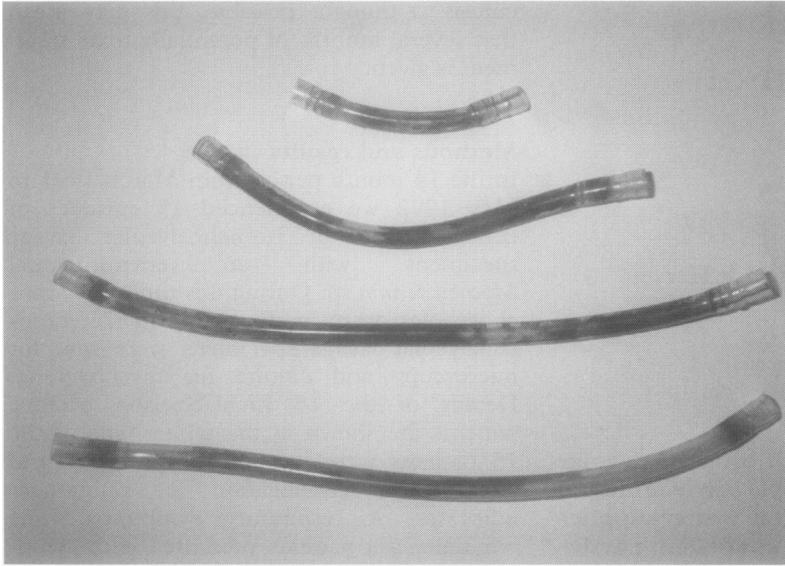
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Tubing containing debris from the bronchoscope disinfecting machine.

Discussion

Mycobacteria are ubiquitous in the environment and may be recovered from mains water supplies.¹ The rapidly growing *M fortuitum* and *M chelonae* are normally of low pathogenicity, but have been described as a cause of infection in patients undergoing renal dialysis and those with neutropenia.^{2,3} There are now several recent reports of contamination of bronchoscopes with *M chelonae*, each associated with mains water cleaning.⁴⁻⁶ Many of the patients described in these reports received antituberculous treatment showing, as in our cases, the therapeutic consequences of a false positive bronchoalveolar lavage result. The diagnostic confusion is further emphasised by the isolation of *M xenopi* from one of the patients in our series. This may have been clinically significant.

Contamination of bronchoscopes may also lead to transmission of infection to the patient. In the letter by Pappas *et al*⁷ contamination by *M chelonae* occurred in bronchoalveolar lavage specimens from 72 patients. Nine patients were transiently colonised by the organism following bronchoscopy, and two immunocompromised patients were thought to have been infected with it. Active infection following transmission of *M tuberculosis* by bronchoscopy has also occurred.⁸

It seems likely that contamination of bronchoalveolar lavage specimens results from exposure of bronchoscopes to mycobacteria in mains water during cleaning. Automatic disinfecting machines contain pipes and drains that are difficult to clean and may provide an ideal environment in which organisms may survive and multiply, significantly increasing this exposure. In experimental conditions some environmental mycobacteria have been shown to be able to survive despite long periods in 2% glutaraldehyde.⁸ This is of particular concern as the concentration of glutaraldehyde in the disinfecting machine may fall to well below 2% with normal use.⁹ Thus, the disinfecting machine may become a

reservoir of mycobacteria to which the bronchoscope is repeatedly exposed. Persistent colonisation of the bronchoscope is possible if organic matter is left after incomplete cleaning, or if the bronchoscope is damaged in some way.^{4,7}

Colonisation of the disinfecting machine may be very difficult to eradicate, despite regular cleaning according to the manufacturers' recommendations. Tubing in the machine may need to be replaced regularly if it becomes coated with organic debris. Prolonged exposure of bronchoscopes to 2% glutaraldehyde is already advised before and after instrumentation of immunocompromised patients.¹⁰ It is also possible to dismantle and autoclave parts of the bronchoscope^{4,8} or to decontaminate them with alcohol^{6,10} or ethylene oxide.⁵ However, these procedures do nothing to remove the source of contamination—that is, mains water—and may be insufficient if some organisms survive, or if dead organisms are subsequently flushed into bronchoalveolar lavage specimens. This may have occurred in our patients, as the Ziehl-Neelson films were positive but 14 of the 15 cultures were negative.

An ideal solution would be to prevent exposure of the bronchoscope to any mycobacteria by using only sterile water during cleaning. Unfortunately, the large volumes of water used make this impractical. We therefore recommend rinsing the bronchoscope with sterile water immediately before use, and sending an aliquot of this with the bronchoalveolar lavage specimen for separate microscopy and culture for mycobacteria. This may flush out organisms introduced during cleaning, and is helpful in the interpretation of subsequent culture results. Unfortunately, if mycobacteria are still present in the disinfecting machine in large numbers, rinsing alone will not completely prevent contamination of bronchoalveolar lavage specimens as we have found recently following the isolation of *M chelonae* from three of 13 such specimens. We therefore suggest that bacterial filters are fitted to the mains water inlet, despite their expense and need for regular replacement. They would need to be fitted before any contamination of the bronchoscope disinfecting machine had occurred, or immediately after the machine has been thoroughly cleaned. Bacterial filters have not yet been evaluated in this setting, but need serious consideration.

As more environmental organisms are associated with opportunistic infection, problems with interpretation of culture results are likely to appear in many settings. This will have implications for clinical practice and also for the design of equipment such as bronchoscopes and machines to disinfect them.

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Unilateral wheeze caused by pseudomembranous aspergillus tracheobronchitis in the immunocompromised patient

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Abstract

Unilateral wheeze in the immunocompromised patient with unremitting fever may be the first localising sign of aspergillus tracheobronchitis. Two such cases are presented.

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With the improved range of antibacterial and antiviral agents currently available, fungal infection has become a more common cause of mortality in the immunocompromised host. Superficial fungal infections, particularly candida, can be prevented or treated with various drugs including fluconazole. However, deep seated fungal infections are a major problem, particularly invasive aspergillosis for which prophylaxis is difficult, so the cornerstone of a successful outcome is early diagnosis and treatment. The diagnosis of invasive pulmonary aspergillosis is based upon clinical features and radiographic evidence with confirmation by culture or microscopy of bronchoalveolar lavage fluid or biopsy of lung tissue. In invasive aspergillus tracheobronchitis, however, where the disease is confined to the airways with only superficial mucosal invasion, the diagnosis is typically delayed because of insidious onset, non-specific signs and symptoms, and lack of radiographic abnormalities.¹⁻³

We report two cases of aspergillus tracheobronchitis in which unilateral wheeze was a

distinctive physical sign preceding any radiographic abnormalities. It is therefore suggested that, in immunocompromised patients with apparent infection not responding to broad spectrum antibiotics, such a sign should raise the suspicion of aspergillus tracheobronchitis and prompt early bronchoscopy and culture to clarify the diagnosis.

Case reports

PATIENT 1

A 57 year old man presented with a six week history of weight loss, anorexia, non-productive cough, and pyrexia. The spleen was considerably enlarged with mild hepatomegaly and minimal lymphadenopathy. There was a peripheral blood pancytopenia and a diagnosis of diffuse immunoblastic non-Hodgkin's lymphoma was made following splenectomy. Cultures were sterile but fever persisted, despite broad spectrum antibiotics, until initiation of cytotoxic chemotherapy including corticosteroids. He required ventilation briefly for a dramatic cyanotic hypothermic reaction immediately after his second pulse of chemotherapy and recovered in 48 hours. However six days later, when neutropenic, he became pyrexial and dyspnoeic and, on auscultation of the lungs, was noted to have left sided expiratory wheeze with basal crackles. The chest radiograph remained normal, blood cultures were sterile, but sputum grew an alpha haemolytic streptococcus. Despite antibiotics his respiratory function deteriorated, and at bronchoscopy two days later thick white plaques forming a pseudomembrane were seen in the trachea and left main bronchus and its divisions with almost complete obliteration of their lumen. The right bronchial tree appeared relatively clear. Microscopy of bronchial secretions and brushings revealed numerous hyphal elements and culture confirmed the presence of *Aspergillus fumigatus*. Despite initiation of high dose (1 mg/kg/day) intravenous and nebulised amphotericin B he died from respiratory insufficiency within 24 hours. Post-mortem examination was not performed.

PATIENT 2

A 61 year old man presented with a four week history of weight loss, polyarthralgia, night sweats, and pyrexia. There were no focal

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