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*CASE STUDY*

# Endobronchial lesions in a non-AIDS patient with disseminated *Mycobacterium avium-intracellulare* infection

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*Endobronchial lesions in a non-AIDS patient with disseminated* Mycobacterium avium-intracellulare *infection. J-Y. Shih, H-C. Wang, I-P. Chiang, P-C. Yang, K-T Luh. ©ERS Journals Ltd 1997.*

**ABSTRACT: A 34 year old female developed *Mycobacterium avium-intracellulare* infection with generalized lymphadenopathy, hepatosplenomegaly, pulmonary infiltration, pleural effusion and endobronchial polypoid lesions.**

***M. avium-intracellulare* was identified by means of sputum cultures, pleural effusion culture and lymph node culture.**

**The anti-human immunodeficiency virus (HIV) antibody was negative. The CD4+ cell count was normal. Bronchoscopic examination revealed multiple polypoid lesions, which had nearly occluded the right main bronchus, right middle lobe and left lower lobe bronchi.**

**Neodymium yttrium aluminium garnet (Nd-YAG) laser and antimycobacterial therapy were used effectively to relieve the airway obstruction. The clinical symptoms and signs responded favourably to antimycobacterial therapy.**
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*Mycobacterium avium-intracellulare* (MAI) is ubiquitously distributed in the environment. Before the era of the acquired immunodeficiency syndrome (AIDS) epidemic, pulmonary and disseminated infections with MAI [1] were extremely rare. The pulmonary manifestations of nontuberculous mycobacterial infection have been well described in medical literature [2, 3]. Pulmonary and disseminated MAI infections are common in patients with AIDS [4]; endobronchial lesions caused by MAI infection are rare in AIDS patients [5, 6]. However, the occurrence of endobronchial lesion caused by MAI infection has not, to our knowledge, been reported in a non-AIDS patient in the literature (in English). The case of a 34 year old, non-AIDS female who had disseminated MAI infection with endobronchial polypoid lesions is described.

## Case report

In November 1994, a 34 year old nonsmoking woman developed nontender lymphadenopathy over the right neck. From February 1995 she suffered from intermittent productive cough, night sweating and fever. Chest radiography revealed infiltration in the right middle lobe (RML). The patients was treated with antibiotics, with poor response. She was admitted to this hospital in September 1995 with worsening fever, productive cough and generalized lymphadenopathy.

On admission, the findings on physical examination were: body temperature 39°C; blood pressure 110/70 mmHg; pulse 110 beats·min$^{-1}$; respiratory frequency 30 breaths·min$^{-1}$; movable, tender lymph nodes over the bilateral neck, axillary and inguinal areas, 1–2 cm in size; coarse breathing sound; and marked hepatosplenomegaly.

The white blood cell count was 21,200 cells·mm$^{-3}$, with 4% band forms, 82% neutrophils, and 12% lymphocytes. The absolute CD4+ cell count was 2,035 cells·mm$^{-3}$. Haemoglobin was 9.7 g·dL$^{-1}$. The pleural effusion was exudative, with lymphocytosis.

Laboratory results were as follows: immunoglobulin A (IgA) 598 mg·dL$^{-1}$ (normal 177–342 mg·dL$^{-1}$); immunoglobulin G (IgG) 2,050 mg·dL$^{-1}$ (normal 1,139–1,699 mg·dL$^{-1}$); immunoglobulin M (IgM) 400 mg·dL$^{-1}$ (normal 88–233 mg·dL$^{-1}$); C-reactive protein (CRP) 14.2 mg·dL$^{-1}$ (normal <1.0 mg·dL$^{-1}$); and tuberculosis antigen A60 IgG >10,000 EU, IgM positive. Anti-HIV antibody was negative. The purified protein derivative (PPD) (tuberculin) test and multiple skin test for cell-mediated immunity were nonreactive. Lymphocyte subsets determined by immunofluorescent flow cytometry showed: 85% CD3+ (normal 74.2±2.5%); 1.2% CD19+ (normal 11.3±2.4%); 80% CD4+ (normal 46.1±1.6%); 74.5% CD4+CD45RA- (normal 13.7±2.0%); 12.8% CD8+ (normal 31.4±2.5%); 90% αβ-T-cell receptor (TCR) (normal 66.8±2.3%); 0.3% γδ-TCR (normal 5.6±2.9%). The mitogen-stimulated proliferative responses of T-cells to concanavalin A, phytohaemagglutinin, pertussis toxin and tetanus toxoid (assayed by $^{3}$H-thymidine incorporation) [7] were normal.

Findings on a chest radiography showed mediastinal lymphadenopathy, infiltration at the right upper lobe (RUL), atelectasis at the RML, and pleural effusion at the right side (fig. 1). Abdominal computed tomography (CT) scan revealed retroperitoneal lymphadenopathy and hepatosplenomegaly.

Sputum examination revealed acid-fast bacilli (AFB). Cervical lymph node biopsy showed necrotizing inflammation with polymorphonuclear infiltration, and scattered

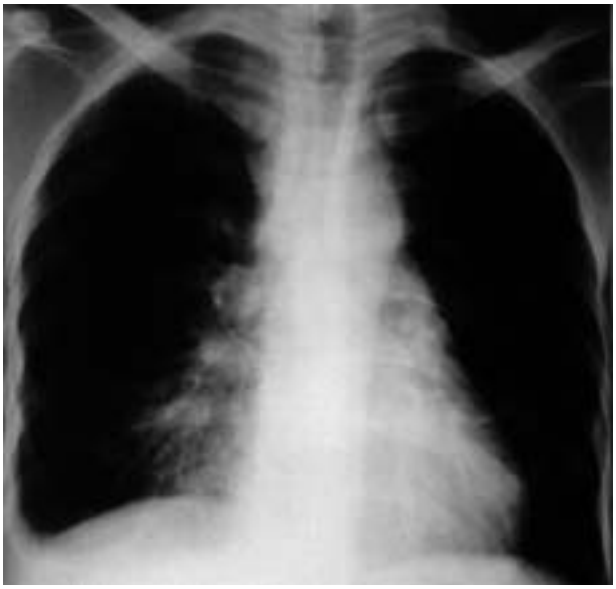


Fig. 1. – Posteroanterior chest radiograph, showing mediastinal lymphadenopathy, infiltration at the right upper lobe, atelectasis at the right middle lobe, and pleural effusion at right side.

macrophages. Acid-fast stain demonstrated bacilli in the macrophages (fig. 2).The bacterial and fungal cultures of blood, pleural effusion, and lymph node tissue were all negative. The cultures of five separate sputum specimens all yielded *M. avium-intracellulare*; pleural effusion culture and lymph node culture yielded the same pathogen. Susceptibility testing of the isolate, determined by standard proportional method, showed resistance to isoniazid (>1.0 μg·mL$^{-1}$) and ethambutol (>10 μg·mL$^{-1}$). Minimum inhibitory concentrations (MICs) of the following agents by using Etest [8] were: amikacin 1.0 μg·mL$^{-1}$; imipenem 2.0 μg·mL$^{-1}$; ciprofloxacin 4 μg·mL$^{-1}$; clarithromycin 1.0 μg·mL$^{-1}$; and rifampin 16 μg·mL$^{-1}$, respectively.

Antimycobacterial therapy was given with ofloxacin 600 mg·day$^{-1}$, clarithromycin 1,000 mg·day$^{-1}$, imipenem 2 g·day$^{-1}$, and amikacin 500 mg·day$^{-1}$. Bronchoscopy was performed because of stridor. This study revealed multiple polypoid tumours in both main bronchi, right intermediate, RML, RUL and left lower lobe (LLL) bronchi (fig. 3a and b). Orifices of the right main bronchus, LLL and RML bronchi were nearly occluded. The biopsy specimen revealed granulomatous inflammation infiltrated by neutrophils, lymphocytes and plasma cells, and necrotic debris, as in the lymph node specimen. Neodymium-yttrium aluminium garnet (Nd-YAG) laser treatment was performed twice, successfully, for relief of the airway obstruction. Sputum culture became negative for MAI after 2 months of treatment. Subsequently, fever subsided, generalized lympha-denopathy and hepatosplenomegaly regressed, breathing sound became clear, pleural effusion resolved and the RML re-expanded. Antimycobacterial therapy was continued with ofloxacin 600 mg·day$^{-1}$, clarithromycin 1,000 mg·day$^{-1}$, and rifampin 450 mg·day$^{-1}$ after discharge. Follow-up bronchoscopy after 10 months of therapy revealed that the endobronchial lesions had almost completely resolved, with anthracosis at the previous laser therapy site. The patient was still under treatment.

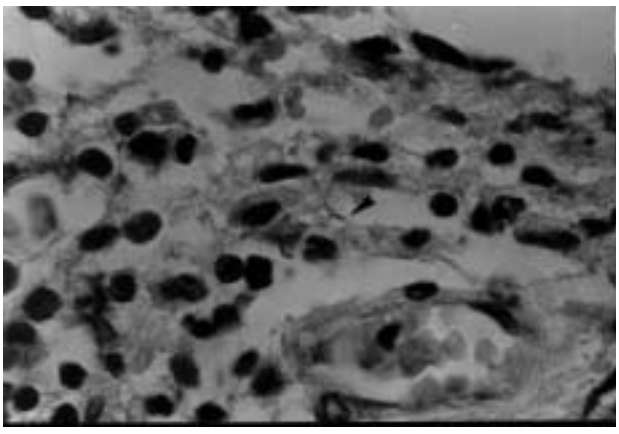
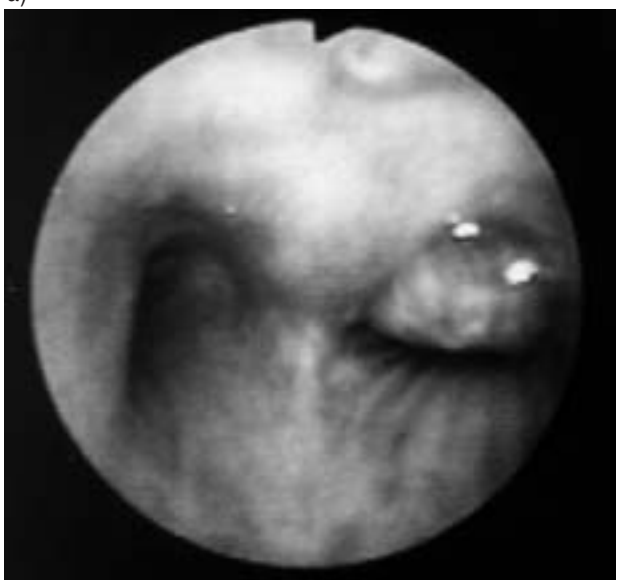


Fig. 2. – Microscopic examination of lymph node specimen shows polymorphonuclear cells infiltration. Acid-fast bacilli are visible at the centre of the picture (Acid-fast stain; scale bar = 10 μm).

a)



b)

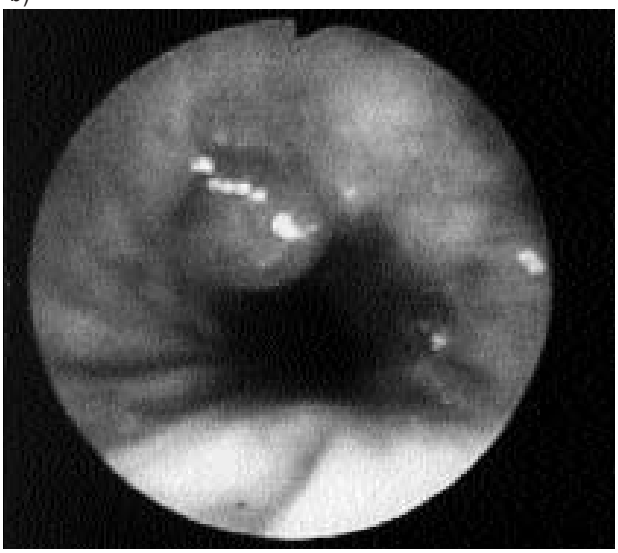


Fig. 3. – a) Bronchoscopic photograph of carina, revealing the orifice of the right main bronchus nearly occluded by a polypoid lesion. b) Endobronchial photograph of right intermediate bronchus revealing another polypoid lesion.

## Discussion

This 34 year old woman, without known underlying disease, developed disseminated *M. avium-intracellulare* infection with generalized lymphadenopathy, hepatosplenomegaly, pulmonary infiltration, pleural effusion and endobronchial polypoid lesions. Disseminated infection with MAI in non-AIDS patients is rare [1, 2, 4]. About one third to a half of patients with disseminated MAI infection have underlying immunodeficiency, such as use of steroid or chemotherapy, haematogenous malignancy, or post-transplantation [1–4]. We could not identify any acquired or primary immunodeficiency in the present patient. The clinical features of disseminated infection with MAI in non-AIDS patient were dominated by systemic symptoms, such as fever and loss of body weight. The physical findings were those of generalized involvement of reticuloendothelial system, such as lymphadenopathy and hepatosplenomegaly [1, 2]. HORSBURGH *et al*. [1] reported that cough and night sweats were relatively uncommon, and pleural involvement was absent in their series. In the present patient, the clinical pictures of irritating cough and night sweats, pleural effusion with positive culture result, and multiple polypoid endobronchial lesions with collapse of the RML were markedly different from previous reports.

Endobronchial lesions caused by MAI have been reported in three patients in the literature (in English) [5, 6]. All of them were patients with AIDS. Two cases reported by PACKER *et al*. [5] had isolated pulmonary involvement and presented with endobronchial obstruction and hilar lymphadenopathy. The lesions responded to biopsy forceps resection and antimycobacterial therapy. MEHLE *et al*. [6] reported the case of a patient who presented with repeated haemoptysis and multiple endobronchial lesions; biopsy forceps resection of the endobronchial lesions was performed to relieve obstruction. To our knowledge, there is no report in English of endobronchial polypoid tumour caused by MAI in a non-AIDS patient. The endobronchial lesions may have been caused by erosion of mediastinal nodes, similar to endobronchial tuberculosis, from the evidence of marked mediastinal lymphadenopathy. When endobronchial MAI lesions induced symptoms of obstruction, the lesions were usually removed by forceps [5, 6]. However, using the thermal effect of laser, endobronchial lesions can be destroyed effectively, with very low risk of bleeding as shown in this case [9].

In summary, this case demonstrates that disseminated *M. avium-intracellulare* infection in a non-acquired immune deficiency syndrome (AIDS) patient may occur with manifestations of pleural effusion and endobronchial lesions. Bronchoscopic examination is necessary in the disseminated *M. avium-intracellulare* infection with symptoms and signs of bronchial obstruction; neodymium-yttrium aluminium garnet laser can be used effectively to relieve the obstruction.

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