

## CASE REPORT

# ‘Black bronchoscopy’: a case of active mycobacterial tuberculosis

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## Abstract

A 63-year-old male presents with chronic cough and hemoptysis. Computed tomography of the chest revealed a left lower lobe (LLL) area of consolidation with prominent ipsilateral hilar lymphadenopathy. Bronchoscopic airway examination revealed black mucosal discoloration and airway narrowing at the superior segment of the LLL. Bronchoalveolar lavage from the corresponding site grew mycobacterial tuberculosis. The patient’s symptoms subsided with anti-tuberculous therapy with a significant decrease in the size of the LLL mass.

## INTRODUCTION

‘Black bronchoscopy’ refers to the presence of black discoloration involving the mucosa of the endobronchial tree. It was first described in early 1940s in association with occupational exposures [1]. Since then, various etiologies have been noted to produce black discoloration of the airways, including malignancies, inborn errors of metabolism and infections [1, 2]. Although most commonly seen in the setting of healed endobronchial mycobacterial tuberculosis (MTB), active tuberculous infection has been described as a rare cause of dark pigmentation of the airways [1–4]. We describe a patient with active pulmonary tuberculosis, presenting as a left lower lobe (LLL) mass abutting the hilum. Bronchoscopy revealed black discoloration of the mucosa at the corresponding endobronchial site. We believe that black mucosal pigmentation is the sequela of the MTB organisms. He showed a significant response to anti-tuberculous therapy.

## CASE REPORT

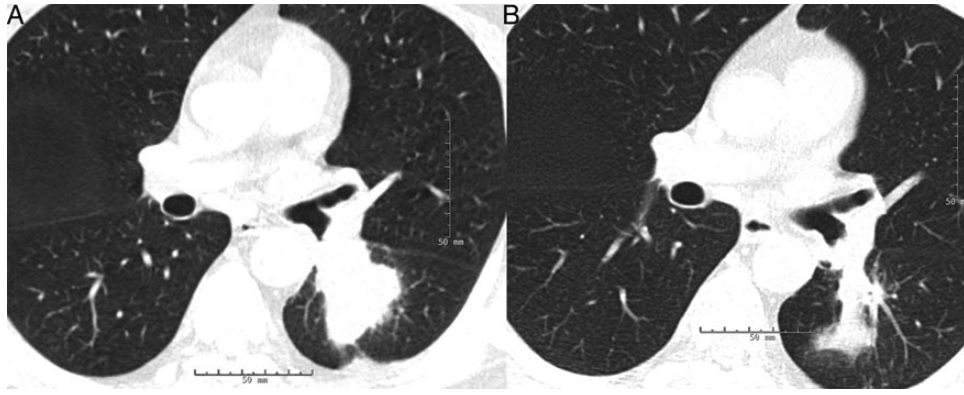
A 63-year-old, non-smoker, male presented for an evaluation of chronic cough and hemoptysis of 8-week duration. He had

emigrated from India 21 years ago and last travelled to his homeland 5 months prior to the presentation. He denied occupational exposure to mining or industry and had no history of exposure to soot. Computed tomography (CT) of the chest revealed a 4-cm area of consolidation involving the superior segment of LLL, extending to the left hilum and associated with prominent hilar and para-aortic lymph nodes (Fig. 1). Prior imaging, 3 years earlier, it was normal. Blood QuantiFERON gold testing (Cellestis, Inc., USA) for *Mycobacterium tuberculosis* was positive. Our differential diagnosis included primary lung cancer and infection, mostly mycobacterial tuberculosis. The bronchoscopic examination revealed an area of black mucosal discoloration and irregularity at the level of the superior segment of the LLL, with narrowing of the segmental airway. The mucosal abnormality was raised and velvety in appearance (Fig. 2). The adult bronchoscope could not be negotiated beyond the narrowing and hence, a pediatric bronchoscope was used. Bronchoalveolar lavage (BAL) and endobronchial biopsies were performed from the site. Subsequently, EBUS-guided fine needle aspiration of the left hilar and subcarinal lymph nodes was performed. Cytology from the aspirate was negative for malignancy. Histology sections of the

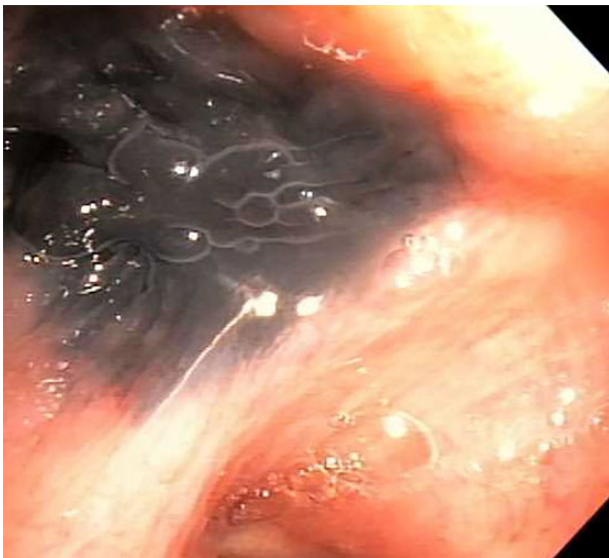
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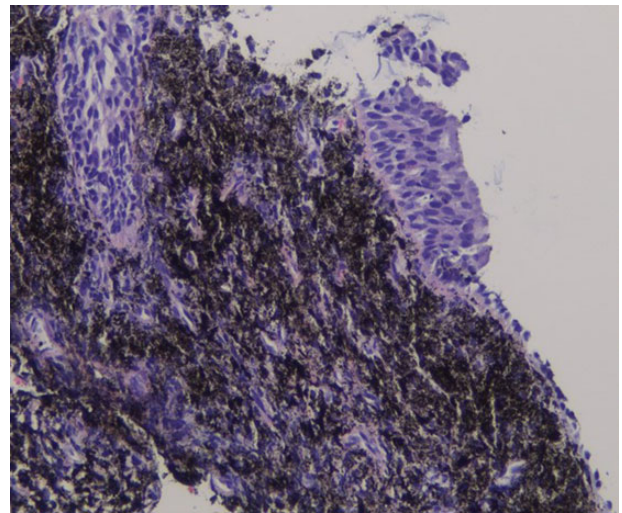
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**Figure 1:** (A) LLL area of consolidation measuring 4 cm, located in the superior segment of the LLL and abutting the hilum. (B) CT scan of the chest after 3 months of anti-tuberculous therapy showing a significant decrease in size of the LLL lung mass.



**Figure 2:** Bronchoscopic image featuring velvety irregular dark discoloration of the mucosa located at the superior segment of the LLL.



**Figure 3:** Endobronchial biopsy, H&E stain  $\times 20$ . Histologic sections of the endobronchial biopsy show an unremarkable bronchial mucosa and a submucosal accumulation of pigmented macrophages/histiocytes. No malignancy or granulomas were identified.

endobronchial biopsies revealed a submucosal accumulation of macrophages/histiocytes with abundant intracytoplasmic black pigments. No granulomas or malignant cells were identified (Fig. 3). To exclude the possibility of involvement by a malignant melanoma, immunohistochemical staining was performed. S100 and Melan-A stains were negative, thus ruling out melanoma. *Mycobacterium tuberculosis* complex grew in BAL fluid. Thus, the patient was diagnosed with active MTB infection and was placed on RIPE therapy (rifampicin, isoniazid, pyrazinamide and ethambutol) along with pyridoxine. A repeat CT of the chest showed a significant decrease in the size of the LLL infiltrates and associated lymphadenopathy, confirming response to therapy.

## DISCUSSION

The term 'black bronchoscopy' was first introduced in 2003 to describe the endobronchial appearance of malignant melanoma [2]. However, multiple etiologies are known to cause black discoloration of the airways; therefore, bronchoscopists should be knowledgeable of the differential diagnosis. In a recent review by Tunsupon et al. [1], congenital causes, inborn errors of

metabolism, infections, neoplasms and iatrogenic causes were described causing hyperpigmentation of the airways. In addition, environmental exposures to cigarette smoking or to heavily polluted atmosphere can also lead to deposition of carbon particles in the bronchial mucosa or the lung parenchyma and are phagocytosed by macrophages, leaving dark plaques of distinct sizes and shapes, without mucosal irregularity. Endobronchial anthracosis is the term used to describe this benign finding [1, 5].

On the other hand, anthracofibrosis and anthracostenosis are terms used to describe airway stenosis associated with dark mucosal pigmentation in patients with or without history of environmental exposures [1, 3, 5]. This predominantly occurs in Asian elderly females, who are thought to be exposed to wood smoke while using biomass fuels for indoor cooking. Inhaled anthracotic particles alone does not typically lead to airway abnormalities as carbon is an inert substance. The fibrotic response in 'anthracofibrosis' is thought to be associated with exposures to substances such as silica or wood smoke [1, 3, 5]. Airway narrowing can lead to segmental or lobar collapse. The right middle lobe is the most commonly affected lobe, followed by right upper, left upper, right lower and LL lobes [3].

A similar condition has been described in patients infected with mycobacterial tuberculosis. Both active and healed *Mycobacterium tuberculosis* (MTB) are known to cause dark pigmentation and often associated narrowing of the airways, as described by Chung *et al.* [6] In prior studies, 20–60% of patients with anthracofibrosis were found to have MTB. Multiple mechanisms have been proposed. Bronchial stenosis is thought to be an immunologic response to tuberculous antigens in the peribronchial lymphatics or contiguous lung, or extrinsic compression from adjacent intrathoracic lymphadenopathy [3, 7, 8]. However, the pathogenesis of dark mucosal pigmentation is debatable. The black pigment is likely residing in infected lymph nodes adjacent to the airways. When the necrotizing process subsequently perforates into the bronchus, the black pigment gets incorporated into the airway walls [1, 3, 7]. Bircan *et al.* described a case of mycobacterial tuberculosis presenting with an enlarged right paratracheal lymph node impinging on the recurrent laryngeal nerve, causing left vocal cord paralysis. Pathological examination of the lymph node demonstrated caseating granulomas surrounded with anthracotic pigmentation, further supporting the hypothesis that anthracotic pigments reside within lymph nodes infected with MTB [9].

In most instances of healed TB, the black pigmentation is restricted in the area of known lymph node stations. Although some authors believe that carbon particles could be coming from an unknown concomitant exposure such as wood smoke, which was not the case in our patient. We presume that the black pigments are, in fact, sequelae of the tuberculous organisms [1, 5]. This is similar to non-tuberculous mycobacteria (scotochromogens and photochromogens) producing pigments of different colors. Subsequently, healing and proliferation of fibroblasts may lead to airway stenosis.

The radiologic and bronchoscopic findings of our patient were concerning for neoplasm. However, endobronchial biopsies and EBUS-guided fine needle aspiration of adjacent lymph nodes were negative for malignancy. Growing mycobacteria in BAL fluid lead to the diagnosis of active MTB, and response to anti-tuberculous therapy further supported this diagnosis.

Thus, we conclude that airway hyperpigmentation, associated with enlarged adjacent lymphadenopathy, should raise the suspicion for active MTB, especially in patients with no history of occupational exposure, after ruling out malignancy. The airway narrowing and atelectasis usually respond, at least partly, to therapy; however, hyperpigmentation is irreversible [1, 5, 7, 8].

## CONFLICT OF INTEREST STATEMENT

None declared.

## FUNDING

There are no sources of support for this study.

## ETHICAL APPROVAL

Ethical approval exempt in this case. Not applicable.

## CONSENT

Informed consent was obtained from the patient.

## GUARANTOR

Dr. Atul Mehta is the guarantor of the work.

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