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## CASE REPORT

# *Ascaris*-induced eosinophilic pneumonitis in an HIV-infected patient

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A case of *Ascaris*-induced eosinophilic pneumonitis in an HIV-infected patient is described. Owing to his HIV status and the absence of peripheral blood eosinophilia on admission, the initial diagnosis was incorrect until the passage of two worms in his stool. The patient developed eosinophilia subsequently, and examination of his sputum also showed increased eosinophils. The patient gradually improved with inhaled bronchodilators, steroid and mebendazole. As peripheral blood eosinophilia may be transient and the larval migration phase occurs before eggs are present in stool, a high index of suspicion is required in making the diagnosis of *Ascaris* pneumonitis. Examination of sputum for larvae or increased eosinophils should be performed in patients suspected of having pulmonary infiltrates from endemic areas irrespective of peripheral blood eosinophil counts.

Pulmonary infections are a major cause of morbidity and mortality in patients infected with HIV. As the potential causative agents of these patients presenting with pulmonary infiltrates are broad, the clinical syndrome often represents a diagnostic challenge. Therefore, different investigation tools, such as sputum induction and bronchoscopy, are often required to identify the specific aetiological agent and initiate appropriate antimicrobial treatment. Nonetheless, outcomes of many patients remain poor, especially in those with undetermined aetiologies. We present a case of pneumonitis in an HIV-infected patient with an unusual aetiology, for which epidemiological clues and simple investigations can be the key to diagnosis and proper management.

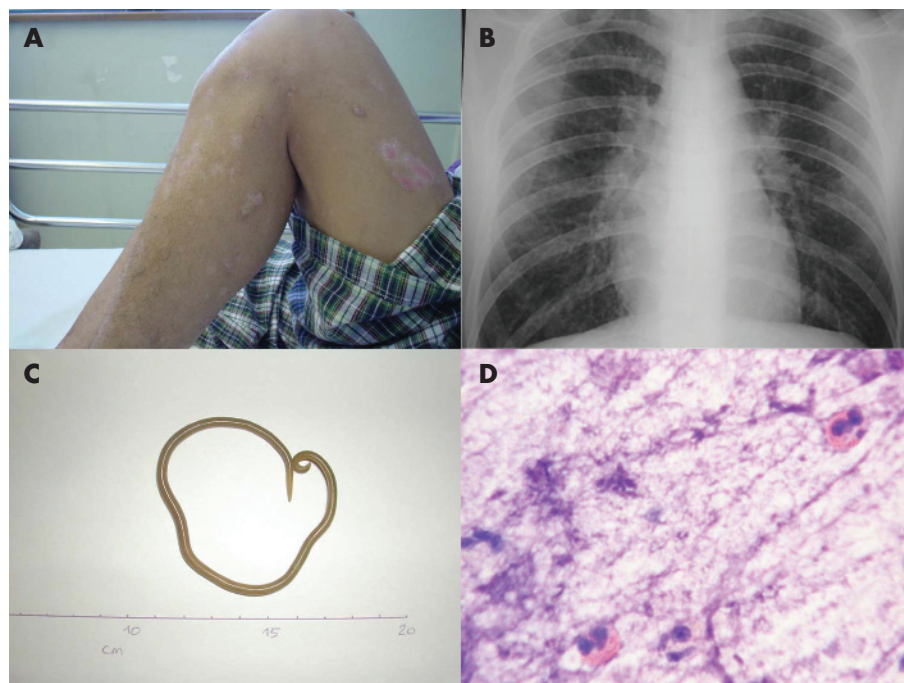
## CASE REPORT

A 27-year-old Vietnamese man was admitted in July 2003 with shortness of breath and chest tightness for 3 days. He also reported having had dry cough, fever and sweating in the past 1 week. He was an intravenous drug user and an illegal immigrant. Since he left Vietnam 1 month previously, he had noticed gradual development of a pruritic rash over his body and limbs. His medical history was unremarkable. On examination, he was found to be febrile and tachypnoeic, with prolonged expiratory phase and diffuse wheeze in both lungs. A generalised pale macular rash was present with fairly defined scales (fig 1A). Arterial-blood gas analysis showed type II respiratory failure and respiratory acidosis. His complete blood counts (including differential counts), and renal and liver function tests were normal on admission. Test for HIV-1 antibody was positive. Chest radiograph disclosed diffuse reticulonodular shadows (fig 1B). In addition, computed tomography and pulmonary angiogram of the thorax showed mediastinal and hilar lymphadenopathy, but no evidence of pulmonary embolism. Urgent Gram smear of his sputum showed numerous leucocytes. Ziehl–Neelsen stain was negative for acid-fast bacilli. In view of his demographic factors, radiological findings and HIV status, the provisional diagnosis was miliary tuberculosis, with differential diagnoses being bacterial, viral and fungal pneumonia. Treatment with empirical isoniazid, pyrazinamide, ethambutol, levofloxacin and azithromycin was begun.

Over the next 2 days he developed repeated episodes of bronchospasm and desaturation requiring biphasic intermittent positive airway pressure. The bronchospasm responded to nebulised bronchodilator and systemic steroids. Sputum for bacterial and fungal cultures recovered only normal oral flora. On day 3 after admission, he passed two brownish round worms (each about 20 cm long; fig 1C) in stool, which were identified as male adult worms of *Ascaris lumbricoides*. Ova of *Trichuris trichura*, but not of *Ascaris*, were also found in his faecal specimen on examination for parasites. A Wright stain of his sputum showed an increased number of eosinophils (accounting for 15% of all leucocytes; fig 1D). He gradually developed peripheral blood eosinophilia (highest eosinophil count  $3.0 \times 10^9/l$ ). A diagnosis of eosinophilic pneumonitis complicating *Ascaris* infection was made. He was treated with mebendazole, with gradual improvement of symptoms and pulmonary shadows. His rash also improved with topical steroids. Treatment for tuberculosis was discontinued. The peripheral blood eosinophil count normalised gradually. His absolute lymphocyte and CD4 counts were normal (1676 and  $510/\mu l$ , respectively), with a reduced CD4:CD8 ratio (0.58). Serological tests for antibodies against mycoplasma, chlamydia, penicillium marneffeii,<sup>1</sup> influenza and parainfluenza viruses were negative. Sputum and blood for mycobacterial culture were negative. The patient was discharged after 3 weeks of hospitalisation. Subsequent stool examination after treatment was negative for parasites.

## DISCUSSION

Although intestinal *Ascaris* infection is common in people infected with HIV in tropical countries regardless of immune status,<sup>2</sup> *Ascaris* pneumonitis in patients infected with HIV has been reported only rarely.<sup>3</sup> This is related to the overall low prevalence of symptomatic pulmonary ascariasis even in endemic areas, which is believed to be due to the high degree of natural tolerance in these populations.<sup>4</sup> Moreover, establishing a diagnosis of *Ascaris* pneumonitis can be difficult. In previous reports of acute eosinophilic pneumonia or bronchiolitis in patients infected with HIV, often no aetiological diagnosis could be identified.<sup>5–7</sup> Pneumonitis due to infection by geohelminths, including *Ascaris* and hookworms, occurs during the pulmonary larval migration phase as a result of a hypersensitivity reaction towards the larvae, and often manifests as respiratory symptoms mimicking episodic asthmatic attacks, pulmonary infiltrates and peripheral blood eosinophilia.<sup>8</sup> Because *Ascaris* is unable to complete its life cycle within the human host, it does not behave as an opportunistic pathogen in patients infected with HIV or other immunocompromised hosts. Therefore, disease manifestations of *Ascaris* infestations in patients infected with HIV are similar to those in the general population, unless their immune status is severely impaired. As the larval migration phase precedes the return of the larvae to the intestines, where they develop into mature worms with egg production, stool examination for parasites is not useful unless there is preexisting infestation in the intestinal phase, as in the present case. The presence of only adult worms, but not of *Ascaris* eggs, in the stool examination of our patient may



**Figure 1** Pale macular rash on the left leg (A), chest radiograph showing diffuse reticulonodular shadows (B), brownish, male, *Ascaris* worm with curved tail passed in stool (C), and haematoxylin and eosin stain of sputum specimen showing the presence of eosinophils (D).

be explained by pre-existing infestations by only male but not female worms. Demonstration of increased eosinophils in the sputum, as in our patient, may be a useful clue to diagnosis. Larvae can sometimes be found in the sputum or gastric aspirates. As peripheral blood eosinophilia might be transient, the diagnosis may solely rely on a high index of suspicion.

On the basis of the epidemiological background, clinical presentations, presence of eosinophils in the sputum and adult worms in stool, and the prompt response to corticosteroids, *Ascaris*-induced eosinophilic pneumonitis was the most likely diagnosis, although other causes of eosinophilic pneumonia cannot be excluded. In retrospect, the clinical presentation in our patient was typical and his rash was also probably a form of cutaneous manifestation of the infection.<sup>9</sup> However, peripheral blood eosinophilia was absent on admission and the initial diagnosis was partly misled by his HIV status. The presence of both intestinal and larval migration phases of the infestations, coexisting *Trichuris* infestations and the chronicity of his rash suggest that the patient acquired the *Ascaris* infestation in Vietnam before his arrival in Hong Kong, with subsequent reinfection. The patient probably had repeated exposure to *Ascaris* eggs, which, after being ingested, hatched in the small intestine and released larvae that migrated via venous blood to the liver, heart and finally the lungs, where they caused the pneumonitis. The larvae then ascended the tracheo-bronchial tree and were swallowed back to the intestines for development into mature worms to produce eggs. This whole cycle takes around 2 months. As the patient left Vietnam only a month ago, it is likely that he acquired *Ascaris* in his own country, although

reinfection could have occurred subsequently. As helminth infestations usually manifest no differently in patients infected with HIV, they should be considered in the differential diagnoses in those with compatible clinical presentation. Examination of the sputum for increased eosinophils or larvae should be performed in patients suspected of having pulmonary infiltrates from endemic areas irrespective of peripheral blood eosinophil counts.

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#### Take-home messages

- *Ascaris* pneumonitis is rare in HIV positive patients.
- Pneumonitis is encountered during the pulmonary larval migration stage.
- It mimics asthma with pulmonary infiltrates and peripheral blood eosinophilia.
- Patients who have pulmonary infiltrates should have their sputum examined for increased eosinophilia and larvae.