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Ascaris-induced eosinophilic pneumonitis in an HIV-infected patient

Susanna Kar Pui Lau, Patrick C Y Woo, Samson S Y Wong, Edmond S K Ma and Kwok-yung Yuen

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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 infilrates from endemic areas irrespective of peripheral blood eosinophil counts.

**DISCUSSION**

Although intestinal Ascaris infection is common in people infected with HIV in tropical countries regardless of immune status, Ascaris pneumonitis in patients infected with HIV has been reported only rarely. 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. 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.

Pneumonitis due to infection by geoemninthis, 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 infilrates and peripheral blood eosinophilia. 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
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. 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 tracheobronchial 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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REFERENCES

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.