Bronchial Nodules Produced by *Strongyloides stercoralis* as the Cause of Bronchial Obstruction

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**Introduction**

Infection by *Strongyloides stercoralis* occurs when filarial larvae penetrate the skin and are carried to the lungs through the bloodstream. After burrowing into the alveoli, the larvae migrate through the airways where they are coughed up and swallowed, eventually maturing in the walls of the small intestine. Unlike other nematodes, *S. stercoralis* are capable of autoinfection of the host.

During the migratory cycle, pulmonary infestation may cause abscess, hemorrhaging, acute respiratory distress syndrome, pleural effusion, or asthma-like symptoms. When asthma-like symptoms present, the patient is likely to be classified as asthmatic and receive inhaled or systemic corticosteroids. Steroid use, as well as immunocompromised status, may lead to the uncontrolled spread of *S. stercoralis* to other organs or to unusually large numbers of them in the organs that are usually involved in the parasite’s life cycle (hyperinfection). Such conditions may lead to death.
Although asthma-like symptoms caused by *S. stercoralis* are well described in the literature, their mechanism has not yet been elucidated. The purpose of this study is to report an additional possible obstructive mechanism that causes asthma-like symptoms: the presence of multiple nodules protruding into the lumen of the airways due to massive invasion by *S. stercoralis* larvae.

**Clinical Observation**

A 55-year-old male farm worker with a 2-year history of illness characterized by cough, dyspnea, and wheezing was treated in our hospital. He had been diagnosed with asthma and received bronchodilators and oral corticosteroids (30 mg/day for the last 5 months). In spite of treatment, his symptoms persisted, and he was referred to our hospital for severe dyspnea. Physical examination showed blood pressure of 110/70 mm Hg, respiratory rate at 24 breaths/min, and wheezing. Chest x-rays revealed air trapping mainly in the right side (Figure 1A). Arterial blood gases were within normal range for the altitude of Mexico City, with pH of 7.42, arterial carbon dioxide pressure of 31 mm Hg, and arterial oxygen pressure of 66 mm Hg. Pulmonary function testing demonstrated moderate obstruction: forced vital capacity (FVC) was 4.7 L (95% of the predicted value), forced expiratory volume in the first second (FEV₁) was 2.2 L (62%), the ratio of FEV₁/FVC was 45.7%, forced expiratory flow between 25% and 75% of vital capacity was 1.4 L (58%); the bronchodilator test was negative.

During the first 5 days after admission, the patient was treated as having severe asthma, with intravenous methylprednisolone (125 mg/12 h), intravenous aminophylline (0.7 mg/kg/h), salbutamol, and also ceftazidime (1 g/12 h) due to suspicion of bacterial bronchitis. The patient did not respond to treatment; on the contrary, 3 to 4 days after admission, he presented with pain and diffused abdominal distention, nausea, and vomiting.

Bronchoscopy was performed because pulmonary function tests demonstrated an irreversible obstruction pattern. Yellowish, friable mucous with engorgement of the vascular bed was observed along with irregular contours due to

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Figure 1. Radiographic and bronchoscopic images of the patient. A: initial chest x-ray where slight air trapping was observed, especially in the right lung. B: image observed when the fiberoptic bronchoscope was in the main carina. The main right bronchus can be observed at the top right of the image. Main findings were yellowish mucous, engorged vascular bed, widening of the carina and multiple nodules protruding into the airway lumen and partially obstructing the opening to the left bronchus.

Figure 2. *Strongyloides stercoralis* larvae. A: larva observed in the bronchial brushing (hematoxylin-eosin ×400). B: larva (arrow) found in a lymphatic vessel of the bronchial submucosa. The cellular infiltrates are composed primarily of mononuclear cells (hematoxylin-eosin ×150).
Discussion

It has been well documented that *S. stercoralis* are capable of producing asthma-like symptoms, yet little research about the mechanism has been carried out, and what we do know remains speculative. Infection due to *S. stercoralis* may cause severe exacerbation of asthma symptoms in subjects suffering from asthma, and it has been suggested that the cause of such a change in status is increased bronchial inflammation, local invasion of the larvae, the exacerbation of the allergic process, or an increased load of T-helper-2 cytokines. Other studies describe relatively sudden onset of asthma-like symptoms in previously healthy people, but mechanisms have not been clearly established.

In this article we describe another mechanism which may explain the asthma-like symptoms. This mechanism consists of protruding, swollen nodules caused by *S. stercoralis* larval invasion in airway submucosa. In our case, nodules were observed by fiberoptic bronchoscopy, thus, fully explaining the obstruction pattern resistant to salbutamol observed in the respiratory function tests. Migration to the bronchial submucosa indicates that hyperinfection had begun, probably due to the prolonged use of corticosteroids. The route the larvae took was most likely direct penetration into the bronchial tubes.

In at least 2 reported cases, bronchoscopy was performed, and in both cases mucosal inflammation without nodules was observed. Therefore, it is likely that the development of nodules explains only some cases with refractory symptoms similar to asthma. However, this possibility should always be considered, especially wherever this parasite is endemic.

We conclude that infection by *S. stercoralis* should be taken into account in the differential diagnosis of asthma-like symptoms and, at least in some cases, the production of nodules in the airways may be the cause of the obstruction.

REFERENCES