CASE REPORT

Hypereosinophilic syndrome secondary to strongyloides infection: a case of recurrent asthma exacerbations

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SUMMARY

Hypereosinophilic syndrome is a disease characterised by a persistently elevated eosinophil count. The syndrome can be reactive to infections, autoimmune disease, cancers, etc. Multiple organ involvement can occur including cardiomyopathies, pulmonary involvement and neuropathies. We describe a case of a patient who presented with signs and symptoms of asthma with recurrent asthma exacerbations, but in fact proved to be hypereosinophilic syndrome secondary to strongyloides infection.

BACKGROUND

This patient who had no history of childhood asthma developed symptoms over a 3-year period. In spite of adequate inhaler cover involving combined long-acting β -agonist and inhaled steroids, she had recurrent admissions for asthma exacerbations. She was also noted to have raised eosinophil counts and nasal symptoms throughout these admissions. She had a baseline peak flow of 350. She had significant use of steroids over the period of these exacerbations. Her symptoms would improve acutely during treatment with steroid therapy, but she would then be readmitted several times throughout the year with exacerbations of her asthma. The reason why we wrote this article is that this is a diagnosis that could easily be missed and for anybody who has returned from an area endemic to strongyloides and who has a raised eosinophilic count, the diagnosis of strongyloides should be considered.

CASE PRESENTATION

A 35-year-old Asian nurse presented to the accident and emergency department with a 1-week history of diarrhoea, vomiting and abdominal pain. This was also associated with a flare up of her asthma with increasing wheeze and breathlessness.

She had no significant medical history other than flare-ups of her asthma. She started with increasing nasal symptoms and was with diagnosed allergic rhinitis, and over the ensuing 2 years she had multiple presentations to accident and emergency with asthma exacerbations. She reported the benefit from short courses of steroids and was currently on seretide 250 volumatic and ventolin inhaler. Her best peak flow was 350 L/min and her lung functions showed an FEV1/FVC ratio of 63% which correlates with obstructive airway disease consistent with asthma and the patient was on 20 mg of

prednisolone at the time of presentation. For the last 1 year, she had mild to moderate elevation of eosinophils (figure 1). This was a persistently elevated eosinophil count spanning over a period of 6 months which would meet the definition of hypereosinophilic syndrome. Her original serum IgE was 310 ku/L and her IgE to common allergens was negative. She also had a number of sputum cultures sent for infections such as tuberculosis and all of these were negative. Her chest X-ray during her admission showed new linear atelectasis. Her previous X-rays had been normal.

Three sets of stool culture were negative for common bowel pathogens and microscopy was negative for parasites, ova and cysts. Her serum eosinophilic count was raised markedly at $3.61 \times 10^9/L$ and as a percentage of 30 of the total blood differential count. The haematocrit was normal. A malaria screen was negative and a quantiferon gold test was positive. Coeliac screen was negative.

She continued to deteriorate with increasing breathlessness and wheeze not responding to standard asthma treatment including magnesium and intravenous hydrocortisone. Oral steroids were avoided during initial admission due to gastrointestinal symptoms.

Further investigation included auto antibodies screen and vasculitic screen which were negative. Her CT of the thorax/abdomen was consistent with uncontrolled asthma features, showing mild bronchial wall thickening, mucus plugging and atelectasis (figure 2). Her abdomen scan was normal though there was mild thickening of the distal oesophagus indicating a degree of oesophagitis.

Following this she had bronchoscopy, endoscopy and colonoscopy. Her bronchial alveolar lavage showed eosinophil count of over 90% with trans-bronchial biopsy showed eosinophilic infiltrate with alveolar tissue. Similar biopsy of the oesophagus revealed eosinophilic infiltrate, and colonoscopy was normal. Screening for tuberculosis on lavage samples and culture was negative for *Mycobacterium*.

The initial working diagnosis was Churg Straus/hypereosinophilic syndrome.

The diagnosis of hypereosinophilic syndrome secondary to strongyloides infection was confirmed.

INVESTIGATIONS

Serology for strongyloides was then found to be positive. This Elisa test for Strongyloides antibodies has high specificity and sensitivity. The sensitivity for the test is around 97%, with a specificity of 99%.

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Figure 1 Graph showing the normalisation of eosinophil count post-treatment for strongyloides.

DIFFERENTIAL DIAGNOSIS

The diagnosis of hypereosinophilic syndrome secondary to strongyloides infection was confirmed.

TREATMENT

She worked in the healthcare service and her age group was associated with an increasing risk of activation of latent tuberculosis. The use of high dose of steroids would also lead to potential reactivation of tuberculosis.

She was started on prednisolone 5 mg/kg body weight reducing dose and chemoprophylaxis for latent tuberculosis with isoniazid for 6 months and ivermectin for strongyloides.

OUTCOME AND FOLLOW-UP

With 3 weeks of treatment, much of her symptoms improved. Her eosinophil count returned to normal (figure 1).

Her oral steroids were gradually weaned down over a 6-month period. Her asthma was generally poorly controlled requiring treatment between steps 3 to 4 (British thoracic society guidelines on asthma management). There has been considerable improvement so far as she is presently only using a short-acting β 2-agonist. This had been stepped down from her combination inhaler and leukotriene antagonist. She had repeat endoscopy with oesophageal biopsy which showed only mild chronic inflammatory infiltrate with eosinophils.

DISCUSSION

Hypereosinophilia can be caused by a variety of diseases. It may be idiopathic or secondary to other conditions. Some of these include asthma, drug allergies, allergic skin diseases, parasitic

infections, lymphoma, autoimmune conditions such as systemic lupus erythematosus and vasculitis such as Churg Strauss.

Owing to multiorgan involvement, the signs and diagnosis of strongyloides infection can be quite difficult to establish. Immunosuppression in strongyloidiasis can lead to hyperinfection syndrome and substantial disseminated disease.

Strongyloides is prevalent in the tropical and subtropical regions of the world.¹ In the tropical areas, the overall incidence may exceed to over 25%. In countries such as the USA the highest rates of infection tend to be among individuals who have been in endemic areas. The high risk groups tend to be immigrants, refugee travellers and military personnel. Two studies in particular highlighted this fact. A Canadian study looked at the seroprevalence of newly arrived immigrants to Canada.² The Kampuchean population of immigrants had nearly 76% seroprevalence when tested followed by Laotians (56%) and Vietnamese (12%). In the other study, nearly up to 40% on new Cambodian immigrants to Australia tested positive for strongyloides serology.³

Patients usually present with symptoms relating to nausea, diarrhoea, weight loss, abdominal pain, dyspnoea, wheeze and breathlessness.⁴ Patients in some instances may develop hyperinfection syndrome or disseminated disease with severe life-threatening symptoms leading to shock or alveolar haemorrhage.⁵ The diagnosis can be quite difficult. Case reports relating to hypereosinophilic syndrome secondary to strongyloides suggest that patients tend to have a high yield of parasites during an infection from biopsy samples from the gut or lung.⁴ Hence, for any patient with significant eosinophilia and recent travel to endemic areas of strongyloides infection, a high index of suspicion should be maintained.³

Albendazole and ivermectin are the main therapies to treat strongyloides. This treatment can achieve approximately 80% eradication rates.⁶ Eradication is difficult to confirm; hence, in some cases, when symptoms persist, therapy is repeated again. Case reports are usually related to respiratory and gut symptoms; however, patients with significant immunocompromise may present with neurological symptoms such as meningitis, etc.

Our patient presented with recurrent asthma exacerbations requiring steroids. The treatment only partially resolved symptoms and it was only when she presented with abdominal pain and vomiting with persistent eosinophilia that strongyloides was considered as a possibility.

Figure 2 CT showing bronchial wall thickening and mucus plugging consistent with asthma.

Learning points

- ▶ A diagnosis of strongyloides should always be considered in patients presenting with persistent eosinophilia from areas endemic to strongyloides.
- ▶ Untreated strongyloides may lead to life-threatening features of shock and pulmonary haemorrhage.
- ▶ Patients should be followed up and repeated courses of treatment may be required if there are ongoing symptoms.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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