A CASE OF LOEFFLER'S SYNDROME MIMICKING PULMONARY TUBERCULOSIS - AN UNUSUAL PRESENTATION
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Abstract: Loeffler’s syndrome is a clinical syndrome characterised by mild respiratory symptoms, peripheral eosinophilia, elevated total IgE and transient migratory pulmonary infiltrates. SCARIS LUMBRICOIDES is a common parasitic infestation in developing countries. Immune hypersensitivity to ascaris is the likely cause. It affects all age groups usually self limited resolves within two weeks. Ascaris larva can be found in sputum or gastric aspirate. Stool examination for ova and parasites is positive only after eight weeks of respiratory symptoms. A 55 years old male presented with complaints of cough with expectoration, dyspnea, fever for fifteen days. On examination bilateral wheeze. Past history of similar complaints six years back. For that he was diagnosed as smear negative pulmonary tuberculosis and treated with empirical ATT. Now his sputum for AFB is negative during the course of advanced investigations, We measured peripheral eosinophilia, transient migratory pulmonary infiltrates in CT chest, high level of total IgE and observed ASCARIS LUMBRICOIDES eggs during stool examination [after 2 month]. The patient was given a diagnosis of Loeffler's syndrome and treated symptomatically. Mebendazole 100mg twice daily for three days was given to prevent gastrointestinal manifestations of Ascaris infestations.

INTRODUCTION
Loeffler’s syndrome is a clinical syndrome characterised by mild respiratory symptoms, peripheral eosinophilia and transient migratory pulmonary infiltrates. SCARIS LUMBRICOIDES is a common parasitic infestation in developing countries. Immune hypersensitivity to ascaris has been recognised as the likely cause. It affect all age groups. Usually self limited, resolves within two weeks. Stool examination for ova and parasites is positive only after eight weeks of respiratory symptoms treated with oral mebendazole 100mg twice daily for three days to prevent gastrointestinal manifestations.

CASE REPORT
A 55 year old male presented with complaints of cough with expectoration for one month, dyspnea, fever for fifteen days. Past History of similar complaints present 7 years back, for that he was diagnosed as smear negative pulmonary tuberculosis and treated with CAT 1 ATT-2009, CAT 2 ATT - 2011. Not a known Diabetes/Hypertension, not a known asthmatic, no family history of asthma. Not a smoker. Not an alcoholic. On examination wheeze present in bilateral interscapular, and infrascapular area. Other system examinations normal. His sputum examination negative for AFB, His absolute eosinophil count 2890 cells/cmm., HRCT Chest shows migratory pulmonary infiltrate, total IgE 278.90 IU/ml, and observed ASCARIS LUMBRICOIDES EGGS during stool examination [after 2 month]. The patient was given a diagnosis of LOEFFLER'S SYNDROME and treated symptomatically. Mebendazole 100mg twice daily for three days was given to prevent gastrointestinal manifestations of Ascaris infestations.

June 2015. chest x-ray showing bilateral upper and mid zone heterogeneous opacity. Right upper zone opacity > Left upper zone opacity.


July 2015. CT chest showing bilateral upper lobe and right middle lobe consolidation peripheral and subpleural location, with GGO
DISCUSSION

In 1932, Loeffler first described a clinical syndrome characterized by mild respiratory symptoms, peripheral blood eosinophilia, and transient, migratory pulmonary infiltrates (1,2). The term Loeffler's syndrome, or simple pulmonary eosinophilia, has been used to define the numerous similar cases reported subsequently. Immune hypersensitivity to Ascaris lumbricoides has been recognized as the likely cause of most of the earliest reported cases, although several other parasitic infections and exposures to numerous drugs and other agents have also been recognized to induce a Loeffler's-like syndrome (3,4,5). An identifiable etiologic agent may be lacking in up to one-third of patients (6). Loeffler's syndrome affects people of all ages. It is characterized clinically by the presence of low-grade fever, nonproductive cough, dyspnea (mild to severe), and occasionally hemoptysis. The respiratory manifestations of Loeffler's syndrome are usually self-limited, typically resolving in 1-2 weeks (7). Laboratory examination of peripheral blood from patients reveals moderate to extreme eosinophilia, which may be at peak levels as respiratory symptoms resolve. Expectorated sputum, if present, frequently contains eosinophils. Transient, migratory, nonsegmental interstitial and alveolar infiltrates (often peripheral or pleural based) are evident on the chest radiograph. Pulmonary function evaluation typically reveals a mild to moderate restrictive ventilatory defect with a reduced diffusing capacity for carbon monoxide (DLCO). When Loeffler's syndrome is due to A. lumbricoides, the pulmonary manifestations are believed to result from a hypersensitivity reaction to the Ascaris larvae. Following ingestion of ova, larvae hatch within the small intestine, then cross the intestinal wall to enter the splanchnic and ultimately the pulmonary circulation. Subsequently, the larvae migrate across pulmonary capillaries into alveoli, mature into adult worms, ascend the large airways, and are swallowed into the gastrointestinal (GI) tract, where they complete their life cycle. The pulmonary manifestations of Loeffler's syndrome begin approximately 9 to 12 days following ingestion, and occur during the migration of larvae through the lung. During the pneumonic stage of the illness, Ascaris larvae may be identified in sputum or gastric aspirates. In keeping with the life cycle of Ascaris, stool examination for eggs, and parasites is typically negative until 8 weeks after the onset of the respiratory syndrome (8). Histological evaluation of lung tissue is not required for confirmation of the diagnosis. When tissue has been obtained, a characteristic and striking eosinophilic infiltration of interstitium and alveolar-capillary units has been noted. Increased numbers of macrophages have also been appreciated. Tissue necrosis and vasculitis are not features of the disorder. Since Loeffler's syndrome may be induced by a variety of exposures, a search for an etiologic agent (e.g., parasitic infection or drug reaction) should be undertaken. Bronchodilators and rarely corticosteroids may be used for alleviation of pulmonary symptoms, although these are usually self-limited. In cases due to Ascaris, treatment with oral mebendazole (100mg twice a day for 3 days) should be given to prevent late GI manifestations of Ascaris infestation, which may include malnutrition, diarrhea, abdominal pain, and/or intestinal obstruction typically 8 weeks or more after onset of respiratory symptoms. Since stool specimens are negative for ova and parasites early in the illness, clinical follow-up over a 2- to 3-month period is indicated.

CONCLUSION

We suggest that Loeffler's syndrome must be considered in patients who live in endemic areas for parasitic disease with peripheral eosinophilia, transient migratory pulmonary infiltrates in CT Chest and ASCARIS LUMBRICOIDES eggs in stool examination.

References
