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Acute Eosinophilic Pneumonia Due to Round Worm Infestation

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ABSTRACT

We report a case of an 18 month old child who presented with severe eosinophilic pneumonia requiring ventilation as a result of round worm infestation. This child presented with symptoms alike acute severe asthma and had high absolute eosinophil count of 9,234/cmm. A course of steroids, albendazole and diethylcarbazine were followed by rapid recovery and a decrease in eosinophil counts to 616/cmm within one month of treatment. This is the first reported case of a child with severe eosinophilic pneumonia requiring ventilation as a result of roundworm infection [Indian J Pediatr 2008; 75 (3) : 296-297] E-mail : drsmonika@gmail.com

Key words: Ascariasis; Eosinophilia; Eosinophilic pneumonia

Roundworm infestation is a fairly common problem in developing countries, such as ours. Roundworms along with other nematodes are known to be associated with visceral larva migrans and pulmonary eosinophilia with mild and transient symptoms. Severe forms of eosinophilic pneumonia have been seen in adults and have been associated with the more severe entity, idiopathic hypereosinophilic syndrome, with multisystem involvement. We report a case of severe eosinophilic pneumonia with extra pulmonary involvements associated with ascariasis infestation.

CASE REPORT

An 18 month old male child, presented to us with history of fever and cough for 10 days and respiratory distress for 3 days. Investigations done prior to

hospitalization showed, Hb 9.0 mg%, TLC 35,500/cumm, N71%, L 20%, M05%, E04% (Absolute eosinophil count-AEC- 1,420 per cu.mm). At admission, the child had respiratory distress with a respiratory rate of 90 per minute, heart rate of 180 beats per minute and oxygen saturation of 80%. Apart from wheezing in the chest, pushed down liver and spleen, rest of the systemic examination was unremarkable. Considering a diagnosis of acute severe asthma, the child was started on bronchodilator therapy and had to be ventilated for respiratory failure. Chest X ray showed bilateral fluffy opacities with hilar clustering of the confluent shadows (Fig 1). Hb was 9.3 gm%, TLC of 34,200/cumm, N30%, L42%, M01%, and E27%. AEC was 9234. Serum sodium was 128 meq, serum potassium 4.3 meq. Urinary spot sodium was 22 meq. Syndrome of inappropriate secretion of anti diuretic hormone (SIADH) was considered and managed with fluid restriction. Repeat serum sodium gradually improved to 130 meq on day 2 and 134 meq on day 3 of admission. Blood urea and creatinine were within normal limits.

ECG, done in view of persistently high heart rate of 200/min revealed paroxysmal supraventricular tachycardia (PSVT) which improved with adenosine.

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Tay Syndrome

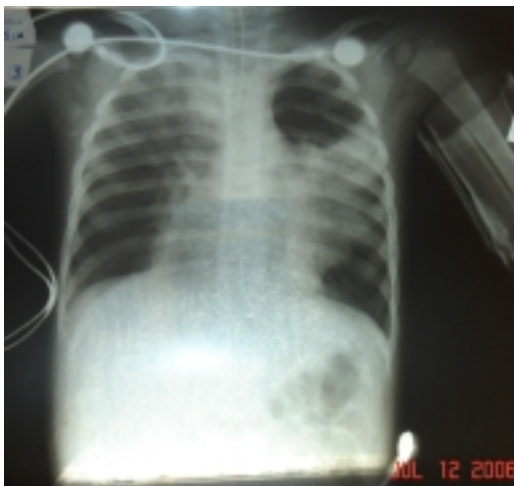


Fig. 1. Bilateral confluent hilar shadows.

AEC repeated on 5th day of admission, was 16,380/cumm. Presence of severe respiratory symptoms and PSVT, in the background of peripheral eosinophilia, raised a suspicion of hypereosinophilic syndrome. Serum IgE was 3094 units. Bronchoalveolar lavage did not show presence of eosinophil or fungal infection. Serologic tests for microfilaria were negative. Stool examination showed presence of ova of *ascaris lumbricoides*

The child was started on steroids (IV betamethasone) and albendazole, followed by a reduction in AEC to 1197. Administration of steroids was followed by a dramatic improvement, with a reduction in ventilator requirement, which could be weaned off within 2 days of starting steroids. Meanwhile, the child also passed round worms in stool, further confirming ascariasis as the culprit. AEC gradually came down to 8194 on day 12 and 5060 on day 14. By discharge, the child was symptom free, had no tachypnoea or tachycardia, and had occasional wheezing in chest. The child was also given diethylcarbazine for 14 days and a course of oral steroids followed by tapering doses. On follow up, the AEC gradually decreased and was 616 on the last visit one month after discharge. The patient is on regular follow up and has been symptom free.

DISCUSSION

Our patient presented with severe respiratory symptoms, peripheral eosinophilia and evidence of intestinal round worm infestation. Severe respiratory distress in the presence of hyper-eosinophilia was evaluated on lines of the possible differential diagnosis-severe asthma, Idiopathic hypereosinophilic syndrome, pulmonary aspergillosis and filariasis¹. Presence of evidence of intestinal round worm infestation explaining the peripheral eosinophilia and a gradual reduction in eosinophil counts over the next 3 months, negated the diagnosis of idiopathic HES¹. Bronchoalveolar lavage and

serological tests ruled out pulmonary aspergillosis and filariasis. Lack of response to adequate bronchodilator therapy and symptom free follow-up over the next one year does not favour a consideration of bronchial asthma.

Ascariasis, was considered as the likely diagnosis on clinical grounds with the background of eosinophilia and radiological picture. Rapid response to steroids and albendazole also supports the diagnosis.

The only other case reported with severe eosinophilic pneumonia due to roundworms is report of a 50-year-old male with eosinophilic pneumonia caused by visceral larva migrans due to *ascaris suum*, from a rural district of Japan.² The diagnosis in this case was based on the respiratory symptoms, radiological picture, pulmonary eosinophilia (confirmed by broncho-alveolar lavage) and a positive ELISA for the specific parasite. Our case, presenting with similar clinical features and radiology, was confirmed by a positive stool examination and ruling out other possible etiologies. Specific serological testing for *ascaris lumbricoides* could not be done.

During a parasitic infestation, the parasitic antigens activate the eosinophils, which are concentrated in the sub-epithelial surfaces (e.g., respiratory epithelium during lung migration). These activated eosinophils release cytokines such as IL-5 and stimulate T helper cells, which in turn stimulate the bone marrow to produce and release more eosinophils³. The first stage of local activation results in the local toxic effects and explains pulmonary, cardiac and neurologic manifestations in various hypereosinophilic states. The second stage of bone marrow stimulation, which occurs a little later, explains the peripheral eosinophilia.

The treatment is directed towards reduction of the eosinophil counts with steroids and anti-parasitic drugs. Hydroxyurea and vicristine have been suggested in patients unresponsive to steroids⁴.

To our knowledge this is the first report of a severe eosinophilic pneumonia with hypereosinophilic syndrome-like picture, in a child with round worm infestation, the previous one being a 50-year-old.

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