A case of eosinophilic meningitis

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Introduction
Eosinophilic meningitis (EM) is a rare clinical entity in children which is diagnosed when there are more than 10 eosinophils/cu mm or >10% of cerebrospinal (CSF) leucocytes[1]. Parasitic infection, neoplasm and hyper-eosinophilic syndrome (HES) are the leading causes of EM[1]. Here we report a case of eosinophilic meningitis without any evidence of parasitic infection which dramatically responded to steroids.

Case report
A 15 month-old Sinhalese boy from Nakiyadeniya, Galle, presented with fever, vomiting and drowsiness of 6 days duration. On the day of admission child had developed an episode of short lasting generalized tonic clonic seizures. There was no history of trauma and the rest of his systemic inquiry was normal. On examination, he was febrile and irritable. He did not have neck stiffness or Kernig sign. There was no lymphadenopathy, skin rashes or hepatosplenomegaly. He did not have any focal neurological signs and bilateral fundi were normal. He was commenced on intravenous cefotaxime and acyclovir thinking of possible meningoencephalitis.

His initial complete blood count revealed a white blood cell (WBC) count of 26,500/cu mm with 32% neutrophils, 38% lymphocytes and 23.7% eosinophils. Blood picture showed mild lymphocytosis with reactive lymphocytes and severe eosinophilia (6095/cu mm) and no abnormal cells were detected. His basic biochemical investigations and inflammatory markers were within the normal range. Lumbar puncture was done on the 13th day of the illness and the CSF revealed 470 polymorphs, 25 lymphocytes and 5 red cells. CSF protein was 72mg/dl. CSF cytology showed many eosinophils with scattered neutrophils suggestive of eosinophilic meningitis. Due to the investigation findings, a focused history was obtained. There were no pets in the surrounding home environment nor was there a history of pica. He did not have any history of eczema, wheezing or atopy. He has never consumed pork, lizard meat, snails or beef but 2-3 times a week he consumes sea fish. There was no recent travel history.

Bone marrow examination was done on the 13th day of the illness and did not reveal any features of malignancy. Stools full report was nil for ova and cysts. Toxoplasma, toxocara and filarial antibodies were negative and thin and thick blood films did not show evidence of malaria. Retroviral screening was negative. Ophthalmological assessment did not reveal any ocular evidence of toxocariasis. Ultrasound scan of abdomen, 2D echocardiogram and chest x-ray were normal. Contrast enhanced computer tomography (CECT) of the brain done prior to lumbar puncture was normal.

Since the child continued to have fever, he was treated with albendazole 2 doses 1 week apart, mebendazole for 3 days and diethylcarbamazine for 21 days (from day 11 to day 32 of illness). Though there was a reduction of the total eosinophil count following anti-helminthic therapy, his clinical profile remained unchanged with on and off fever spikes, but he was not drowsy. Repeat lumbar puncture done at the end of anti-helminthic therapy showed marked CSF eosinophilia. The child was started on 2mg/kg/day oral prednisolone on the 45th day of the illness, continued for 2 weeks and tailed off over 4 weeks, for which the child clinically responded, being afebrile thereafter. After the completion of steroids, repeat lumbar puncture and blood picture were done which showed complete resolution of the CSF and peripheral blood eosinophilia.

Serial full blood counts and blood pictures are shown in Table 1. Serial CSF values are shown in Table 2.

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Table 1: ***Serial full blood counts and blood pictures***

	Day 6	**Day 13**	**Day 41**	**Day 56**	**Day 90**
Total white blood cell count/cu mm	26,500	11,700	16,200	13,400	11,930
Neutrophils	32%	45%	50%	30%	28.7%
Lymphocytes	38%	23%	32%	56%	60%
Eosinophils	23.7%	24%	9.6%	7.7%	4.6%
Total eosinophils/cu mm	**6281**	**2808**	**1555**	**1032**	**548**
Haemoglobin (g/dl)	10.7	9.2	10.7		10.2
Platelet count/cu mm	472,000	556,000	598,000		348,000
Blood picture (eosinophilia)	Severe		Moderate		No

Table 2: ***Serial cerebrospinal fluid (CSF) values***

	Day 13	**Day 42**	**Day 90**
CSF polymorphs	470	255	Nil
CSF lymphocytes	25	12	01
Red blood cells	05	790	46
CSF protein (mg/dl)	72	457	28.2
CSF glucose (mg/dl)	17	34	42
CSF cytology	Many eosinophils with scattered neutrophils	Large amount of eosinophils with few neutrophils	No eosinophils
CSF culture	No growth	No growth	No growth

Discussion

CSF eosinophilia is defined as >10 eosinophils/ml or >10% of the total CSF leucocytes. Helminthic infections such as *Angiostrongyliasis, Gnathostomiasis* and Schistosomiasis are the leading causes of eosinophilic meningoencephalitis followed by rare causes like neoplasms, drugs, prosthetic reactions and idiopathic miscellaneous conditions such as hyper-eosinophilic syndrome (HES)[1].

HES initially required 3 diagnostic criteria viz. blood eosinophilia $\geq 1.5 \times 10^9$/L lasting for > 6 months, no evidence for an underlying condition known to cause hyper-eosinophilia, and existence of eosinophil-mediated organ damage and/or dysfunction[2]. Currently, the duration as well as the presence of end organ damage are not required to make a diagnosis of HES[3]. Clinically, HES includes potentially lethal multi-system disorders, characterized by eosinophilic infiltration of a variable spectrum of target organs, predominantly the skin, heart, lungs, gastrointestinal tract, central and peripheral nervous systems.

Angiostrongylus cantonensis is the commonest cause worldwide causing EM and transmission occurs mainly through consumption of raw snails infected with 3rd stage larvae. Other sources of infection include frogs, fresh water prawns, crabs and lizards[4]. Diagnosis is aided by history, magnetic resonance imaging (MRI) of brain revealing multiple micronodular enhancements in brain tissues and linear enhancement in the pia mater and supportive serological evidences by ELISA to detect antibodies against *Angiostrongylus cantonensis*. It is well documented that treating these patients with anthelmintic drugs worsens the condition because the killed worms exacerbates the inflammatory response[5]. Though the exact treatment modality is unknown, it is recommended to treat with oral prednisolone for 2 weeks and evidence is not yet established in combining steroids with albendazole or mebendazole.

In our patient, despite extensive questioning we could not establish the mode of transmission, to support the possibility of helminthic origin meningitis. Though there was haematological improvement of peripheral eosinophilia with anthelmintic therapy, clinical and CSF clearance of eosinophilia was not seen till the commencement of steroids. Therefore he needs regular follow up as HES is still a possibility.

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