

neutropenia increases the risk of infection. Tocilizumab, in our experience, is a safer option even in infants and it provides immediate relief to the dramatic symptoms.

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Eosinophilic Meningitis in a Toddler

Eosinophilic meningitis (EM) is a chronic aseptic meningitis often caused by helminthic infestation. EM is defined as eosinophils >10 per mm³ in CSF or >10% of total CSF leukocyte [1,2]. The most common infectious cause of EM worldwide are *Angiostrongylus cantonensis*, *Gnathostoma spinigerum* and *Baylisascaris procyonis* [1,2]. Non-infectious causes include malignancy like non Hodgkin lymphoma, multiple sclerosis, hypereosinophilic syndromes, malfunctioning ventriculo-peritoneal shunt and adverse drug reactions [2]. Even though EM has been reported in adults and children from India, parasitic etiology has not been confirmed in those cases [4]. We report a one year old child, resident of South Kerala, India, who reported with prolonged fever due to confirmed helminthic infestation.

A 1-year-old female child presented with 3 weeks history of irregular fever, irritability and poor oral intake. Child was started on oral cefixime from suspecting UTI. On admission, she had continuous fever for 5 days with irritability and episodes of inconsolable cry for 3 days. No history of vomiting or seizure, ear infection, head trauma, recent vaccination or contact with Tuberculosis, or exposure to any drugs or allergens. On examination, vitals were stable with no features of raised intracranial pressure or signs of meningeal irritation and with a normal CNS examination. On investigation, white blood cell count was $14.8 \times 10^9/L$ (36% neutrophils, 42% lymphocytes, 22% eosinophils) with peripheral smear showing eosinophilia and no parasites or abnormal cells. C-reactive protein was negative. Stool and urine examination did not reveal ova or cysts. In view of non remission of prolonged fever and history of irritability and headache, CSF study was done on second day of admission which revealed increased CSF pressure of clear fluid with 1150 whole blood cells, (30% neutrophils, 70% lymphocytes) with a protein 115 mg/dL and sugar 30 mg/dL (blood sugar- 89 mg/dL) suggestive of meningitis. Cultures of blood, CSF and urine were sterile.

Tuberculosis PCR, CSF biofilm for bacterial and viral panel were negative. India Ink and CSF biofilm were negative for Cryptococcus, KOH wet mount did not reveal any fungal elements. Mantoux test and HIV ELISA were negative and Chest Roentgenogram was normal. MRI contrast study of brain showed multiple cortical infarcts with sub cortical and cortical hyper intensities in T2W/FLAIR, leptomeningeal enhancement suggestive of meningitis. In view of meningitis, she was initially treated with ceftriaxone, then upgraded to vancomycin, meropenem and acyclovir. Since fever and irritability persisted even after 7 days of antibiotics, repeat sepsis screen was done, which was negative, and had similar findings on repeat MRI. Therefore, correlating the peripheral eosinophilia with this history, EM was suspected. On revisiting the history, mother gave history of a pet dog at home with rat and snail infestation in the locality. Absolute eosinophil count was 3080/mm³ on day 1, 5500/mm³ on day 7, 2880/mm³ on day 12, 3102/mm³ on day 17. Repeat CSF study revealed 295 white blood cell/mm³ (5% neutrophils, 85% lymphocytes, 25% eosinophils) with protein 104 mg/dL, sugar 34 mg/dL (blood sugar-103mg/dL). Real Time PCR was

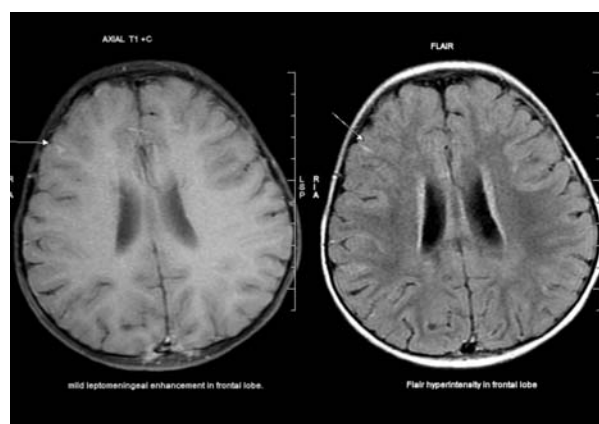


Fig.1 T1W/FLAIR images showing leptomeningeal enhancement/hyperintensity in frontal lobe.

done in CSF, which came positive *A. cantonensis*. She was started on prednisolone 2 mg/kg/day along with Albendazole 15 mg/kg/day for 2 weeks. Within 48 hours of treatment she became afebrile, her sensorium improved with improvement in activity and appetite. After 2 weeks of hospital stay she was discharged in a stable condition with an absolute eosinophil count of 625/mm³ on day 20.

Peripheral eosinophilia is an immunologically mediated response to various conditions like allergic (atopy, medications), neoplasms (leukemia, lymphoma, tumor associated) drug induced hypersensitivity and infectious diseases (parasite, fungal). It is essential to look for non infectious causes of hyper-eosinophilia symptoms, before looking for parasitic infection.

Overall parasitic meningitis is rare, but exact incidence and prevalence is not reported. Among the three major helminthes that cause EM [1,2], *A. cantonensis* is the most common. Neurocysticercosis, although rare, also is known to cause EM in endemic areas.

Similar infections have been described in Southeast Asia, South Pacific, Taiwan, Africa, Caribbean, Australia and North America [1]. Shipboard travel of rats is the most common cause for the spread of parasite to other continents as rats are the definitive host. [2,3] and human beings are the accidental hosts. Infection occurs due to ingestion of third stage larvae in raw or undercooked snails or fish and children who play in the dirt in endemic areas are prone for infection. Eosinophilic meningitis is diagnosed presumptively based on travel or exposure history with CSF analysis showing >10% eosinophils, mildly elevated protein and normal glucose or hypoglycorrhachia [5]. Peripheral eosinophilia peaks about 5 weeks after exposure. MRI brain demonstrates high signal intensities, leptomeningeal enhancement, hyper intense signals on T2W image. For detection of parasite in CSF, ELISA is sensitive and specific but is limited by commercial non availability and cross reactivity between helminthic parasites. PCR based studies are sensitive in detecting the parasite DNA.

Treatment is mainly supportive with analgesics, corticosteroids and anthelmintic drugs [6,2]. There is higher

incidence of neurological sequelae among children and prognosis is good with 70% improvement within 1-2 weeks and mortality is <1%.

This case is presented to sensitize the clinician, the rarity of EM, caused by helminthic infection. The possibility of eosinophilic meningitis/parasitic meningitis must be considered in a patient with fever, peripheral eosinophilia, headache with or without meningeal signs. In such patients, CSF eosinophil staining is recommended along with demonstration of parasite antigen through real time PCR will help to establish parasitic etiology and also in prognostication and appropriate follow up.

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Undifferentiated Nasopharyngeal Carcinoma and Paraneoplastic Leukemoid Reaction

Nasopharyngeal carcinoma is a rare pediatric malignancy [1]. In this report, we describe a nasopharyngeal carcinoma in child presenting with paraneoplastic leukemoid reaction (PLR). Adult patients with solid tumors presenting with PLR have been reported in the past, but very few pediatric cases have been described [1].

A 11-year-old boy, with a history of global developmental delay, presented with bilateral neck swelling that progressively increased over two months, associated with loss of weight, increased frequency of fever spikes, tiredness and difficulty in swallowing solid feeds. There was no history of contact with tuberculosis. Developmental age was 4 years and his antenatal, natal and post-natal history was uneventful. On examination, he was awake, alert, cooperative, and responded to verbal commands. He was pale, febrile, and had bilateral cervical lymphadenopathy of 15x10x3 cm on the right side and 12x10x3 cm on the left side, non-tender, immobile and firm to hard in consistency. The child was underweight and stunted and head circumference was below 2 SD when compared to age- and sex-matched controls. At presentation, the child was febrile with