# Eosinophilic Meningitis: Report of Three Cases from a Hilly Area in Kerala

**Bindu Varghese, Murali Krishna Menon, Thara Pratap[1], A. Moheen, Jomy Mathew[2], T. P. Vijayan[3], M. P. Tomy[4], Muhammed Jasim Abdul Jalal[2]**

Departments of Neurology, [1]Radiology and [2]Family Medicine, VPS Lakeshore Hospital, [3]Departments of General Medicine and [4]Ophthalmology, General Hospital, Kochi, Kerala, India

## Abstract

Normal cerebrospinal fluid (CSF) does not contain eosinophils. The presence of >10 eosinophils/μL in CSF or at least 10% eosinophils in total CSF leukocyte count confirms eosinophilic meningitis. We present three patients with eosinophilic meningitis from the same locality with peripheral eosinophilia.

**Keywords:** Angiostrongyliasis, eosinophilic meningitis, gnathostomiasis, peripheral eosinophilia

## Introduction

The presence of >10 eosinophils/μL in cerebrospinal fluid (CSF) or at least 10% eosinophils in total CSF leukocyte count confirms eosinophilic meningitis.[1] Normal CSF does not contain eosinophils. Hence, their presence suggests various etiologies – infectious and noninfectious. These include parasites such as *Angiostrongylus cantonensis* and *Gnathostoma spinigerum*, fungal infections of the brain, drugs such as intraventricular antimicrobials, and myeloproliferative diseases such as hypereosinophilic syndrome, Hodgkin's disease, leukemia, and lymphoma.[2] Parasitic infestation should be suspected in the presence of peripheral eosinophilia >1500 cells/mm$^3$.[3] In India, eosinophilic meningitis has been reported in two case series[4,5] and two case reports.[6,7]

We present three patients with eosinophilic meningitis from the same locality with peripheral eosinophilia.

## Case Reports

### Case 1

A 30-year-old, otherwise healthy male presented with complaints of acute-onset occipital headache. It was associated with nonprojectile vomiting and blurring of the right eye vision.

**Address for correspondence:** Dr. Muhammed Jasim Abdul Jalal, Department of Family Medicine, VPS Lakeshore Hospital, NH-47 Bypass, Maradu, Nettoor, Kochi - 682 040, Kerala, India.
E-mail: jasimabduljalal@yahoo.com



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**Table 1: Cerebrospinal fluid findings**

| | Case 1 | Case 2 | Case 3 |
|---|---|---|---|
| CSF protein | 38 | 82 | 90 |
| CSF glucose | 64 | 40 | 45 |
| CSF total count (cells/mm$^3$) | 153 | 180 | 326 |
| CSF differential count (%) | | | |
| Lymphocyte | 63 | 70 | 41 |
| Neutrophils | 3 | 12 | 3 |
| Eosinophils | 34 | 18 | 55 |

CSF=Cerebrospinal fluid

**Table 2: Peripheral eosinophil count**

| | Case 1 | Case 2 | Case 3 |
|---|---|---|---|
| Peripheral eosinophil count before albendazole therapy (on the day of admission) | 21.9% | 30.6% | 20.6% |
| Peripheral eosinophil count 3 weeks after albendazole therapy (during review) | 1.9% | 1.6% | 3% |
| Total WBC count before albendazole therapy (on the day of admission) | 12,990/μL | 8810/μL | 8220/μL |
| Total WBC count 3 weeks after albendazole therapy (during review) | 9930/μL | 7770/μL | 6000/μL |

WBC=White blood cell

There was no history of photophobia or phonophobia.

On examination, the patient was afebrile and hemodynamically stable with blood pressure of 130/80 mmHg and heart rate of 80/min. He was conscious and oriented. Cranial nerve examination was normal. Pupils were equal and reactive to the light. Fundus examination showed bilateral peripapillary hemorrhages. There was evidence of papilledema bilaterally. He had bilateral flexor plantar response. There were no other focal neurological deficits. All other systems were within normal limits.

Magnetic resonance imaging (MRI) showed rounded hyperintense area in the right subcortical frontal region with faint enhancement on contrast [Figure 1]. CSF study [Table 1] showed pleocytosis (153 cells/mm$^3$) with predominant eosinophils (34%). Gram-stain, acid-fast bacilli stain, and fluid culture were all negative. Moreover, CSF polymerase chain reaction was negative for all bacterial, viral, and parasitic infections. Peripheral eosinophil count was 21.9% [Table 2].

On further questioning, the patient reported that he and his family traveled to a rural hilly area 2 weeks before the onset of symptoms. He gave a history of consumption of raw cabbage during the trip. He denied swimming in pool/pond and exposure to seafood/molluscs. The patient also denied any other animal contact/insect bites. There was no history of any exposure to rats. There was no history of any medications – antibiotics/nonsteroidal anti-inflammatory drugs (NSAIDs).

## Case 2

A 60-year-old female (mother of case 1) also had similar semiology of illness. She had holocranial headache

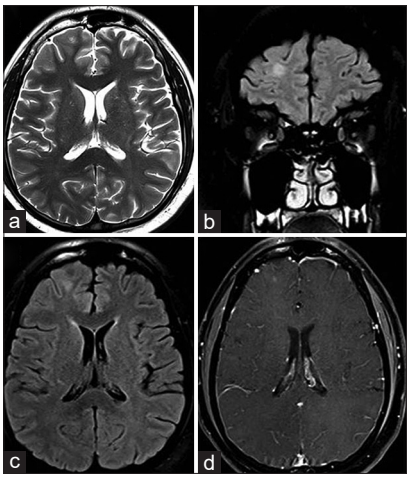


**Figure 1:** Axial T2 (a), coronal (b), and axial fluid-attenuated inversion recovery (c) magnetic resonance images showing rounded hyperintense area in the right subcortical frontal region. Postcontrast magnetic resonance T1-weighted images (d) showing faint enhancement of the lesion

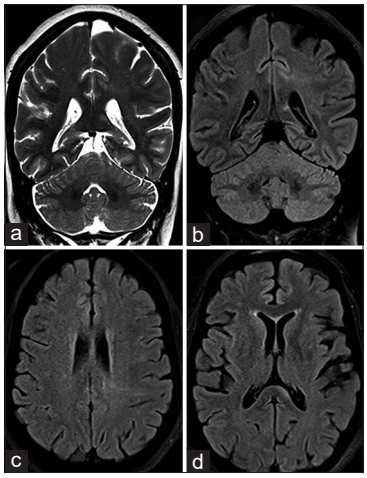


**Figure 2:** Coronal T2 (a) and coronal fluid-attenuated inversion recovery (b) and axial fluid-attenuated inversion recovery (c and d) images showing T2-hyperintense area in the splenium of the corpus callosum to the left side. Arrowhead shows linear hyperintensity in the left periventricular white matter region

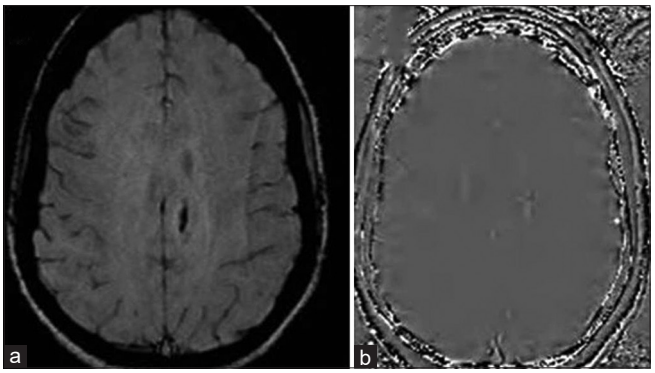


**Figure 3:** Susceptibility weighted imaging - Maximum intensity projection (a) and Phase images (b) showing linear hypo-intense sub-cortical area in the left parietal cortex which could represent micro-hemorrhage in the parasite migratory tract

associated with neck pain. General and systemic examination was within normal limits apart from papilledema with bilateral peripapillary hemorrhages on fundus examination. Brain imaging did not show any focal lesions/extra-axial collections.

MRI showed T2-hyperintense area in the splenium of corpus callosum to the left side [Figure 2]. Susceptibility-weighted imaging, maximum intensity projection, and phase images showed linear hypointense subcortical area in the left parietal cortex which could represent microhemorrhage in the parasite migratory tract [Figure 3]. CSF study [Table 1] showed pleocytosis with eosinophilic predominance (18%). Peripheral eosinophil count was 30.6% [Table 2].

On further questioning, the patient reported that she and her family traveled to a rural hilly area 2 weeks before the onset of symptoms. She gave a history of consumption of raw cabbage during the trip. She denied swimming in pool/pond and exposure to seafood/molluscs. The patient also denied any other animal contact/insect bites. There was no history of any exposure to rats. There was no history of any medications – antibiotics/NSAIDs.

### Case 3

A 32-year-old, otherwise healthy male presented with complaints of acute-onset holocranial headache. He had associated vomiting. General and systemic examination was within normal limits.

Computed tomography brain did not show any focal lesions/extra-axial collections. MRI brain was not done due to financial constraints. CSF study [Table 1] showed pleocytosis with eosinophilic predominance [55%]. Peripheral eosinophil count was 20.6% [Table 2].

The patient was from the same rural hilly area. He also gave a history of consumption of raw carrot. He denied swimming in pool/pond and exposure to seafood/molluscs. The patient also denied any other animal contact/insect bites. There was no history of any exposure to rats. There was no history of any medications – antibiotics/NSAIDs.

Antibiotic (ceftriaxone) was started empirically in all the three patients on the day of admission itself. Antihelminthic drug (albendazole) was started in regard to peripheral eosinophilia and eosinophilic predominance in CSF. Peripheral eosinophilia improved 3 weeks after the initiation of albendazole therapy [Table 2].

## Discussion

Eosinophilic meningitis is diagnosed when eosinophils are identified in CSF of a patient with clinical features of meningitis. Eosinophil counts may be >10 eosinophils per ml or 10% of total cell count in CSF.[8]

Severe intermittent and intractable occipital/temporal headache is the most common symptom of eosinophilic meningitis. The patient may present with hyperesthesia, paresthesia, weakness, and flaccid paralysis.

Parasitic infestation is the most common cause of eosinophilic meningitis. *Angiostrongylus cantonensis*, *Gnathostoma spinigerum*, and *Baylisascaris procyonis* are the common parasites attributing to eosinophilic meningitis. Third-stage larvae are carried to the central nervous system by the bloodstream and cause infection in humans as they ingest these larvae. Molluscs, slugs, snail slime, and other paratenic hosts are sources of human infection. They contaminate foods and infect humans when they take uncooked or improperly cooked foods. Stool and urine studies may be performed for identification of the parasites.[9] Serological tests for parasites by immunoblotting techniques have been found to be specific and reliable. Other parasites such as *Paragonimus*, *Schistosoma*, and *Taenia* are also known to cause eosinophilic meningitis.

Coccidioidomycosis is the most common fungal infection causing eosinophilic meningitis. Systemic mycosis occurs through inhalation where after dissemination results in eosinophilic meningitis.

Eosinophilic meningitis is also known as bacterial and viral infections such as streptococci, coxsackie viral meningitis, lymphocytic choriomeningitis virus, rickettsia, Rocky Mountain spotted fever, neurosyphilis, and tubercular meningitis.[10,11]

Eosinophilic meningitis is one of the manifestations of CNS invasion by maggots and flies and their larval stages. Although thiabendazole and other antihelminthics have been used, they usually have a limited role, and surgical removal is usually needed.[12]

Drug-induced eosinophilic meningitis is caused by ibuprofen, antibiotics such as ciprofloxacin, gentamycin, and vancomycin, or catheters impregnated with rifampin and minocycline. Malfunctioning plastic implants, nonorganic implants, contrast media, and iodized oils are also known to cause eosinophilic meningitis. Illicit drug users, especially those with HIV, frequently present with eosinophilic arachnoiditis.[8] Hodgkin's lymphoma is the most commonly associated neoplasm to cause eosinophilic meningitis.

Identification of the organism in host tissues is confirmatory for the diagnosis of both angiostrongyliasis and gnathostomiasis. Isolation of the organism from central nervous system has low sensitivity.[13] Serological tests can be done, but are not widely available. Diagnosis is most often clinical, history-based, and with CSF and peripheral eosinophilia.

We could effectively rule out malignancies, drugs, and viral and bacterial meningitis as the causes based on history, nonresponse to antibacterial therapy, and response to anthelmintic therapy. Since there was no history of exposure to pond water or pruritic skin lesions in all our patients, *Schistosoma* was unlikely.

Our patients did not give any history of consumption of crustaceans, and they did not have pulmonary involvement. Hence, paragonimiasis was ruled out. Neurocysticercosis was not considered since there were no cystic lesions on neuroimaging in any of our patients.

Only one case was reported from India, in which microbiologic diagnosis of *Angiostrongylus* was proved as the worm was retrieved from the eye in this case.[6] Ingestion of monitor lizards as an aphrodisiac is another cause for eosinophilic meningitis.[4,5] In our cases, the possible etiology could be *Angiostrongylus* since it is most often associated with the raw vegetables such as carrot and cabbage.

## Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

## Conflicts of interest

There are no conflicts of interest.

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