Sir,

Eosinophilic meningitis is a rarely encountered condition in India and is characterized by the presence of 10 or more eosinophils per microliter in the cerebrospinal fluid (CSF) or eosinophilia of at least 10 percent of the total CSF leukocyte count. The usual etiology is the invasion of the central nervous system by helminthic parasites, the most common being Angiostrongylus cantonensis, but other infectious and noninfectious causes have also been implicated.\[1\] Angiostrongylus eosinophilic meningitis is endemically seen in Southeast Asia and the Pacific Basin, beyond which it is a rare entity. In India, it has only been documented in a few case reports and case series.\[2‑6\] We report an interesting case of eosinophilic meningitis in three members of a family living in the same household in Kerala.

A 48-year-old lady (Patient A), a known case of diabetes mellitus, presented with complaints of fever, numbness of left thigh radiating to the leg since 1 week, followed by severe headache, multiple episodes of vomiting, and altered sensorium since 4 days. On examination, she was conscious, irritable, moving all four limbs equally, and had neck stiffness. Magnetic resonance imaging (MRI) brain showed multiple discrete foci of T2 fluid-attenuated inversion recovery (FLAIR) hyperintensities in the bilateral frontal subcortical white matter without post-contrast enhancement [Figure 1]. Magnetic resonance (MR) venogram was normal. CSF study showed an elevated total count (450) with lymphocytic predominance and 20 percent eosinophils. CSF meningoencephalitis panel (real-time polymerase chain reaction which was tested for Streptococcus pneumoniae, Neisseria meningitidis, Haemophilus species, herpes simplex virus, cytomegalovirus, enterovirus, Mycobacterium tuberculosis complex, and varicella-zoster virus) was negative. There was no peripheral blood eosinophilia. Electroencephalogram (EEG) showed mild diffuse electrophysiological dysfunction without any epileptiform abnormalities. As the patient had significant eosinophilia in the CSF, a possibility of eosinophilic meningitis was considered. Ophthalmology evaluation did not show any evidence of larva migrans or infestation. On further retaking the history, the patient had a history of significant exposure to snails as her house was near a marshy area and was infested with them. They were seen predominantly in the bathrooms and pipes in her home and up to 150 snails were found at a time. A dead snail was also recovered from her vomitus. On further probing, it was learnt that her mother (Patient B) and husband (Patient C) who were living in the same house had a history of continuous headaches in the past week. Her husband also had paresthesia over his right hand and left thigh. They were also admitted for evaluation and both of them also had significant eosinophilia in the CSF and blood [Table 1]. The computerized tomography (CT) head of patient B was normal (she was uncooperative for MRI) and the MRI brain of patient C showed T2 hyperintensities over bilateral frontoparietal white matter [Figure 2].

| Table 1: Laboratory findings |
|-----------------------------|
| Patient A   | Patient B   | Patient C   |
| Peripheral eosinophilia (%) | 1           | 16          | 27          |
| CSF* cells/mm\(^3\)         | 450         | 510         | 310         |
| CSF differential count (%)  | N02 L51 E47 | N01 L77 E22 |             |
| CSF protein (mg/dl)\(^2\)   | 40.9        | 49          | 173         |
| CSF sugar (mg/dl)\(^3\)     | 116         | 86          | 60          |

\(^{*}\)Cerebrospinal fluid, \(^{1}\)N – Neutrophils, \(^{2}\)L – Lymphocytes, \(^{3}\)E – Eosinophils.
All three patients were treated with iv steroids (dexamethasone 16 mg/day), iv antibiotics (ceftriaxone 4 g/day), antihelminthic (albendazole 800 mg/day in two divided doses), and supportive care for 14 days. Patient A had some worsening initially in hospital with disorientation and hallucinations, which was followed by dramatic improvement. Her mother and husband had steady improvement after treatment initiation. At discharge, they were conscious, oriented, and with no focal neurological deficits. The three of them were also asymptomatic on follow-up after 1 month.

As three members of the same family had eosinophilic meningitis with a common exposure to snails which is an intermediate host of A. catonensis, it can be presumed to be the causative agent. Rats are the definitive hosts of A. catonensis worms. The eggs are laid in the pulmonary arteries of rats which hatch into larvae that reach the feces via the trachea and gastrointestinal tract. Snails and slugs that feed on rodent feces become intermediate hosts and allow the larvae to molt into infective third-stage forms. Humans become infected when they consume raw snails, vegetables or water contaminated with mollusk slime, and crabs or freshwater shrimp that have eaten infected mollusks.[1]

In South India, the more commonly reported cause of developing eosinophilic meningitis is the consumption of raw meat of monitor lizard (Varanus bengalensis) which is believed to have rejuvenating and aphrodisiac properties.[3] Snail exposure as a probable cause has been rarely reported in this region,[2] however it is seen frequently in endemic areas. Serological testing is not commonly available except in endemic areas and the larvae can be seen in the CSF or eye in only less than 10 percent of cases.[2]

The symptoms may be mild or severe (as seen in our three patients who had different degrees of severity), comprising of headache, altered sensorium, neck stiffness, nausea, vomiting, paresthesias/hyperaesthesias, and rarely extraocular and facial nerve paralysis. Paresthesias are a symptom rarely reported in bacterial, viral, and fungal meningitis, and hence the features of acute meningitis associated with the same can also provide a clinical clue to the diagnosis. The cause of paresthesias is hypothesized to be due to neuron root invasion by A. catonensis or the immune reaction towards it.[7]

MRI findings are usually nonspecific, ranging from normal to hyperintense lesions on T2-weighted images, high signal intensities on T1-weighted images over the globus pallidus and cerebral peduncle, ventricular enlargement, and leptomeningeal enhancement.[9]

The disease is usually self-limited and most patients recover completely. Treatment options include analgesics, corticosteroids, and periodic removal of CSF to alleviate symptoms due to raised intracranial pressure. Patients who received corticosteroid therapy for 2 weeks were found to be less likely to have persistent headaches and had a reduced need for repeat lumbar puncture.[9] Antihelminthic treatment alone may lead to clinical exacerbation of symptoms due to increased inflammatory response to the dying organisms. Most patients are usually treated with a combination of antihelminthics and steroids in common practice; however, it was shown in a randomized trial that combination therapy did not show any advantage over steroids alone for headache relief.[10]

Ours is a unique case where multiple members in the same household developed eosinophilic meningitis probably due to contamination of water or food with snails. This adds to the existing literature on eosinophilic meningitis in India and shows how snails, which are usually considered harmless, can create an outbreak of eosinophilic meningitis and cause a potential public health concern.

Poornima N. Nambiar, Javed Ahamed TP, Sarika Sasidharan, Lovely Catherine Mathew, K. A. Salam
Department of Neurosciences, Meitra Hospital, Calicut, Kerala, India

Address for correspondence: Dr. Poornima N. Nambiar, Department of Neurology, Meitra Hospital, Edakkad PO, Calicut - 673 004, Kerala, India.
E-mail: poornima.narayanan@gmail.com

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Letters to the Editor

Submitted: 28-Jan-2022 Revised: 12-Mar-2022 Accepted: 13-Mar-2022
Published: 05-May-2022

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DOI: 10.4103/aian.aian_95_22