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Understanding Eosinophilic Meningitis: A Comprehensive Overview

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Eosinophilic meningitis (EM) presents a diagnostic challenge due to its varied etiologies, encompassing both infectious and non-infectious causes. Typically, cerebrospinal fluid (CSF) devoid of eosinophils serves as the norm, with their presence indicating a potential underlying condition such as parasitic infections or other systemic disorders.

Eosinophilic meningitis is defined as presence of 10 or more eosinophils per microliter in CSF or eosinophils constituting at least 10% of total CSF leucocyte count [1]. Infection with helminthic parasites is considered as the most common source. Other infectious agents include fungi, bacteria, rickettsiae and viruses. Non-infectious causes of EM include malignancy like Hodgkins lymphoma, Non-Hodgkins lymphoma, eosinophilic leukemia, medications like Ciprofloxacin, Ibuprofen, Vancomycin and systemic diseases like sarcoidosis, and hypereosinophilic syndrome.

Predominant infectious agents include Angiostrongyliasis, Gnathostomiasis, Neurocysticercosis, cerebral Toxocariasis and Schistosomiasis. Among these Angiostrongyliasis stands out as the most prevalent cause, notably affecting regions like South-East Asia, the Pacific basin, and the Caribbean [2]. Human infection typically stems from the ingestion of raw intermediate hosts such as snails or crustaceans harboring larvae.

3 types of presentation include headache, encephalitis, ocular manifestations like retinal detachment which occurs when larvae migrates to the eyes.

Clinical features develop 2-35 days after ingestion of larvae with the most common symptom being headache [3]. Fever maybe present only in less than 50% of cases. Associated neurological findings include radiculopathies, cranial nerve palsies and cognitive dysfunction. Neck stiffness is seen in 50% of cases. A high clinical suspicion along with CSF eosinophilia should strongly suggest the possibility of eosinophilic meningitis (EM). At least 2/3rd of patients have peripheral blood eosinophilia [4]. Specific immunodiagnosis maybe done by Western Blot analysis.

A. cantonenensis meningitis is differentiated from other helminthic infections of the central nervous system by the characteristic absence of focal lesions on brain imaging [2]. Treatment is usually supportive. Anti helminthic treatment can often worsen symptoms due to inflammatory reaction to antigens [4]. A short course of steroids is found to relieve headache [5]. Symptoms usually resolve

completely by 3-6 weeks.

EM due to Gnathostomiasis is more fulminant [6]. It reaches CNS by migrating along nerve roots. As with the case of Angiostrongyliasis, this parasite is also endemic throughout Southeast Asia [7]. The larvae enter humans by consumption of raw fish or poultry. Radiculitis is the most distinctive clinical manifestation of *G. spinigerum* infection [6]. Other clinical manifestations include cranial nerve palsies, myelitis or intermittent subcutaneous migratory swelling. CSF shows xanthochromia and brain imaging shows scattered multiple focal lesions. Similar to Angiostrongyliasis, treatment here is also supportive.

Fungal eosinophilic meningitis by *Coccidioides Immitis* is one of the most common causes of EM in the US [6]. Meningitis is seen in 50% of patients with disseminated disease. Similar to parasitic infection, CSF eosinophilia is a prominent finding in coccidioidal meningitis [8]. Rarely, eosinophilic meningitis has been reported in bacterial infections like Tuberculous meningitis [1] and neurosyphilis.

Various hematological malignancies have been associated with CSF eosinophilia, most common being Hodgkin's disease [9]. Other malignancies associated with EM include acute Lymphoblastic leukemia, T-lineage non Hodgkin's lymphoma, chronic eosinophilic leukemia, and disseminated glioblastoma.

CSF eosinophilia is also associated with use of certain drugs like ibuprofen, ciprofloxacin, intraventricular vancomycin [10] and gentamycin.

In this issue of the *Journal*, Karadan U et al has presented an interesting case of eosinophilic meningitis presenting as atypical lumbosacral meningitis. In addition, the patient also had severe and persistent headache with features of raised intracranial pressure [11].

In conclusion, while EM remains a rare entity, its diverse etiologies necessitate a high index of suspicion among clinicians encountering patients with unexplained neurological symptoms. Vigilance in recognizing CSF eosinophilia coupled with a comprehensive understanding of potential underlying causes is paramount in achieving timely diagnosis and effective management of this challenging condition.

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