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Eosinophilic Meningits: A Review

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Introduction

Clinical features of meningitis (that include headache, nausea, emesis, and stiffness of the neck and back, paresthesias, hyperaesthesia, weakness and flaccid paralysis) and identification of eosinophils in cerebrospinal fluid indicate eosinophilic meningitis. Aetiology includes infective cause's especially parasitic infections, fungal infections, and non-infective causes.

Discussion

Eosinophilic meningitis is diagnosed when the patient is found to have clinical features of meningitis and there is identification of eosinophils in cerebrospinal fluid (CSF). Other authors have considered the patient to have eosinophilic meningitis when the counts are more than 10 erythrocytes per ml or 10% of total cell count in CSF [1]. Consumption of raw intermediate or paratenic host in an area that is endemic to the parasite and absence of other diseases form the basis of epidemiological criteria for the eosinophilic meningitis syndrome [2].

The most common symptom reported with eosinophilic meningitis is a severe headache that can be intermittent, intractable. The headache may be located in the occipital region or temporal region. The headache continues throughout the episode. There is associated nausea, emesis, and stiffness of the neck and back. Paresthesias, hyperaesthesia, weakness and flaccid paralysis are occasionally seen. Fever and cranial nerve involvement is rare [2,3]. Case of myeloradiculopathy have also been reported [4].

This content of pre-formed cytokines, with diverse potential biologic activities, provides eosinophils with capabilities distinct from most other leukocytes. Stimulation for release of specific cytokines, such as IL-4, leads to a regulated signal transduction cascade, which is dependent on the formation of leukotriene C_4 within eosinophils where it acts as an intracrine mediator. IL-4 release occurs selectively and is by means of vesicular transport. The capabilities of eosinophils not only to rapidly release pre-formed cytokines but also to differentially regulate which cytokines are released endow eosinophils with distinct abilities in innate and acquired immunity [1,2].

Eosinophils are known to participate in inflammatory processes that accompany allergies, helminthic infections, and proliferative diseases. They contain many chemokines and cytokines within specific granules and cytoplasmic vesicles. Rapid release of preformed cytokines and differential release of specific cytokines release like IL-4 lead to the eosinophils abilities in innate and acquired immunity [5]. They cause exocytic degradation of parasites due to the cellular granules that are extruded on exposure to parasitic infections. They also have a role in the neoplastic diseases of epithelial origin. Regeneration and remodelling of tissues also needs the eosinophilic activity. Neural damage e.g. in cerebellum and white matter result from several processes such as loss of Purkinje cells and spongy changes [1,2].

In another study, CSF levels for IL-5, IL-10, and IL-3 were also found to be higher in these patients. IL-2, IL-4, IFN-γ and TNF-α were not found to be significantly raised and did not show a correlation to the eosinophil levels. T helper 1 and 2 cytokines response has been noted and may help diagnose parasite associated meningitis when detected in CSF [6].

Vascular endothelial growth factor (VEGF) can be detected in patients with eosinophilic meningitis. It's associated with raised CSF protein, WBC count and eosinophili counts. Blood brain barrier damage may result into an increase leucocyte count and CSF protein levels. VEGF results in separation of endothelial tight junctions, and increase vescicle transport. Disruption of blood brain barrier may occur due to interaction between eosinophils and mediations such as IL-4, IL-5, independent of VEGF [7].

One of the commonest cause is parasitic infection specially angiostrongyliasis, gnathostomiasis, and baylisascariasis. Other parasites like paragonimus, schistosoma and taenia are also known to have caused eosinophilic meningitis. Humans are infected when they ingest third stage larvae that are carried to the central nervous system by the blood stream. An inflammatory response occurs in response to the larvae burrowed into the neural tissue. Uncooked or improperly cooked foods contaminated with molluscs, slugs, snail slime and other paratenic hosts are sources of human infection. Stool and urine studies may be performed for identification of the parasites. Serological tests for parasites by immunoblotting techniques have been found to be specific and reliable. In a study on eosinophilic meningitis patients secondary to Angiostrongylus cantonesis, 4-3-3β proteins were found in CSF. These levels tend to decline over 2-3 weeks suggesting that the protein level rises in association with the neuronal injury [2,8,9].

Fungal infections, most commonly coccidiomycosis, also cause eosinophilic meningitis. Inhalational route leads to systemic mycosis where after dissemination can result into the eosinophilic meningitis. Other causes include cryptococcosis, candidiasis, aspergillosis, histoplasmosis, blastomycosis, and mucormycosis. Predominant lymphocytosis and modest pleocytosis with elevated protein (>150 mg/dl) and reduced sugars are hall mark of CSF in fungal infections. Cultures of CSF are seldom positive and may be falsely negative. IgG and IgM antibodies may be detected. In lack of treatment, they can be fatal. Treatment options like fluconazole, itraconazole, voriconazole, and amphotericin B are available.

Eosinophilic meningitis is also known in bacterial and viral infections like streptococci, coxsackie viral meningitis, lymphocytic choriomeningitis virus, rickettsia, Rocky mountain spotted fever, neurosyphilis, tubercular meningitis [1,2].

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Myiasis is the invasion of tissues by maggots and flies and their larval stages. Facial lesions, bone erosions and CNS invasion by the larvae are known. Eosinophilic meningitis is one of the manifestations of CNS invasion. Though thialbendazole and other antihelminthics have been used, they usually have limited role and surgical removal is usually needed [1].

Antibiotics like ciprofloxacin, gentamycin and vancomycin or catheters impregnated with rifampin and minocycline are also known to cause eosinophilic meningitis. Other medicines like ibuprofen and treatment modalities like malfunctioning plastic implants, non organic implants, contrast media and iodized oils are implicated. Illicit drug users especially those with HIV frequently present with eosinophilic arachnoiditis [1,2,10].

Eosinophilia of unknown origin lasting more than 6 months (idiopathic hypereosinophilic syndrome) with absolute eosinphil count >1500 µL can cause systemic damage and dysfunction due to release of cytokines including neural system. Amongst neoplasms, Hodgkin's Lymphoma is the most commonly associated disorder associated with eosinophilic pleocytosis. However, eosinophilic pleocytosis is rare in Hodgkin's lymphoma. Other conditions which are known to be associated with eosinophilic meningitis include non-Hodgkin's lymphoma, disseminated glioblastoma, ALL and carcinomatosis. Bone marrow evaluation with molecular genetics and cytogenetics for the diagnosis of primary haematological disorders and flow cytometry for detecting lymphoid subsets for lymphoid malignancies may be used [1,2].

Idiopathic eosinophilic meningitis have been known where any cause could not be identified [1].

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