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Case Report

Eosinophilic Meningitis Caused by Herpes Simplex Infection

Sarah Kabbani, Mustafa Abugideiri, Rachel Bartash, Chemeka Watson, Yelena Burklin, and Nadine Rouphael*

Department of Medicine, Emory University, USA

Abstract

Eosinophilic meningitis is rare and is mainly caused by parasitic infections. Here we described the first case of HSV eosinophilic meningitis.

***Corresponding author**

Nadine G Rouphael, The Hope Clinic of the Emory Vaccine Center, Emory University, 500 Irvin Court, Suite 200, Decatur, GA 30030, USA, Tel: 404-712-1435; Fax: 404-499-9728; Email: nroupha@emory.edu

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INTRODUCTION

Eosinophilic meningitis is a rare diagnosis and is defined as the presence of more than 10 eosinophils/μL in the CSF and/or pleocytosis with eosinophils accounting for more than 10% of the total CSF leukocytes. Parasitic infections are the main cause, although other infectious and non-infectious etiologies have been described. Here we report the first case of HSV associated eosinophilic meningitis.

CASE PRESENTATION

A 42-year-old African American male with HIV/AIDS, diagnosed in 1999, presented with a four-day history of severe bilateral headache. He denied any photophobia, phonophobia, neck stiffness or fever. He has a history of CNS toxoplasmosis in 2003 and was on suppressive treatment with pyrimethamine and sulfadiazine. He was adherent to his antiretroviral therapy of Zidovudine, Lamivudine, Abacavir, Ritonavir and Darunavir with a CD4 T cell count of 179 cells/μl and viral load of 30 copies/ml at presentation. He had no history of central nervous system (CNS) manipulation or surgery, no recent medication initiation (other than a vitamin D supplement), no recent travel and no exposure to animals.

On physical exam, his temperature was 36.3°C and blood pressure was 133/79, with a heart rate of 88 beats per minute and respiration rate of 16 per minute. He was in mild distress but without nuchal rigidity and negative Kernig's and Brudzinski's signs. He had no palpable lymphadenopathy and no hepatosplenomegaly. His neurologic exam was unrevealing except for a known mild resting tremor in the right hand that was a sequela of CNS toxoplasmosis. His genital exam was unremarkable.

Laboratory tests showed a white blood cell count (WBC) of 6,200 cells/μL (range, 4,200 to 9,010), with a normal differential (52% neutrophils, 39% lymphocytes, 8% monocytes and 1% eosinophils). Creatinine level was 1,16 mg/dl and alanine, aspartate aminotransferase levels were 11 and 17 units/L respectively. Two lumbar punctures revealed a protein level 137mg/dl (range 15-45) and glucose level of 41mg/dl (range 40-70) with a CSF/blood ratio of 0.46, numerous red blood cells between 2-4,880 cells/μL and WBC varying from 43 to 365 cells/μL with an eosinophil count between 10 to 40 cells//μL (11-20%) (Table 1). The opening pressure was 14 cm H_2O, The cerebrospinal fluid (CSF) gram stains and culture were negative for parasites, bacteria, mycobacteria and fungi with cytology negative for malignant cells. VDRL titer was negative. HSV PCR in the CSF was positive (while EBV PCR was negative). MRI of the brain revealed an interval resolution of previously seen left basal ganglia lesion with residual sequelae of bilateral basal ganglia involvement (with T2 hyperintensity).

The patient was treated with high dose acyclovir (10mg/Kg IV every 8 hours) for 7 days with improvement of his symptoms after 5 days of treatment, he was switched to Valacyclovir 1g every 8 hours at discharge to complete a 14 day antiviral course and continued to do well at follow up.

DISCUSSION

Eosinophilic meningitis (EM) is defined as the presence of more than 10 eosinophils/μL in the CSF and/or pleocytosis with eosinophils accounting for more than 10% of the total CSF leukocytes [1]. Our patient with confirmed HSV meningitis, lack of travel history and an alternative exposure, was diagnosed with EM secondary to HSV. This rare entity is caused by different infectious (parasitic and non-parasitic) and non-infectious etiologies, however parasitic infections are the main cause [1].

Parasitic infections seen in endemic countries, typically outside North America and Europe, result commonly in EM through helminthic larval migration into the nervous system of *Baylisascaris procyonis, Ganthostoma spinigerum* and *Angiostrongylus cantonensis* [2]. A detailed travel, dietary and animal exposure histories are an essential part of medical history

Table 1:

	WBC in tube 1 (per µL)	WBC in tube 4 (per µL)	RBC (per µL)	Protein (mg/dL)	Glucose (mg/dL)
First lumbar puncture	43 (2%N, 78%L, 10%E, 10% M)	50 (1%N, 66%L, 13%M, 20%E)	4880 (tube 1) 270 (tube 4)	137	41
Second lumbar puncture	225 (72% L, 4% M, 24% E)	365 (7% N, 70% L, 12% M, 11% E)	10 (tube 1) 2 (tube 4)	ND	ND

Abbreviations: N: Neutrophils; L: Lymphocytes; M: Macrophages; E: Eosinophils; ND: Not Done

taking in a patient presenting with EM. Peripheral eosinophilia is also typically found. CSF examination usually does not reveal the presence of the larvae. Treatment remains mostly supportive, antihelminthic drugs are avoided as they could worsen the local inflammatory response due to dying organisms, and worsen neurological symptoms. Corticosteroids have shown benefit by decreasing the inflammatory response, and thus decreasing headaches and the need for repeat lumbar punctures in patients with *A. cantonensis* [3]. Other parasitic infections that can localize to the CNS and cause paragonimiasis, schistosomiasis, *Echinococcus*, fascioliasis, toxocariasis and *cysticercosis.*

In the US, the infectious etiologies of EM are typically not parasitic. In AIDS patients in particular, coccidioidal meningitis (30% of which are EM) [4] and rarely *Cryptococcus* have been identified as causing EM. Our patient had an acute presentation, a normal opening pressure, a negative CSF crytococcal antigen, and a negative CSF fungal culture making cryptococcal meningitis highly unlikely. In addition, he had no history of travel to coccidioidomycosis endemic areas (in the US southern Arizona, the southern and central valleys of California, southwestern New Mexico, and west Texas) [5], which argues against coccidioidal meningitis. EM can have other infectious etiologies such as bacterial (tuberculosis, syphilis, group B streptococcus), rickettsial [6] and viral causes (coxsackie [7], chronic lymphocytic choriomeningitis virus) [8] though, to our knowledge, this is the first report in the medical literature of HSV causing EM. The absence of herpetic oral and genital mucosal lesions on exam does not exclude the diagnosis. Elevated protein level, normal glucose level and lymphocytic predominance seen in CSF all support the diagnosis of viral meningoencephalitis. The confirmatory test for herpetic meningitis is a positive HSV PCR in the CSF [9]. The treatment of the non-parasitic infectious EM usually relies on targeting the etiologic agent.

Non-infectious etiologies described are mostly related to neoplastic disease (commonly lymphoma and leukemia) [10], sarcoidosis, and rheumatologic disorders. EM can be associated with ventriculo peritoneal shunt [11], and it is usually an indication of shunt infection, but could be an inflammatory response to the hardware [12]. It has also been described as an adverse reaction to dye or medications given intrathecally or systemically (such as NSAIDs [13], quinolones [14], cotrimoxazole [15]), or idiopathic. Treatment of the noninfectious causes usually target the underlying conditions, revision of the shunt if needed, and removal of the offending agent once identified can improve eosinophilic counts in CSF and relieve symptoms.

While eosinophilic meningitis is a rare entity, presence of eosinophils in the CSF should always be considered pathologic. Ruling out an infectious process is the first step in approaching a patient and requires a careful evaluation and a detailed travel and exposure history. Understanding the different etiologies that can cause EM is critical in diagnosis and initiation of appropriate therapy. Although viruses are considered unlikely, with this case and improved molecular diagnostics, viral etiologies should be considered as part of a comprehensive diagnostic workup for EM.

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