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Headache in an adolescent

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Abbreviations: ANA: Antinuclear Antibodies; CNS: Central Nervous System; CBC: Complete Blood Count; CK: Creatine Kinase; CMP: Complete Metabolic Profile; CT: Computed Tomography; DNA: Deoxyribonucleic Acid; EBV: Epstein-Barr Virus; ESR: Erythrocyte Sedimentation Rate; HIV: Human Immunodeficiency Virus; ICP: Intracranial Pressure; IgM: Immunoglobulin M; MRA: Magnetic Resonance Angiography; MRI: Magnetic Resonance Imaging; MRV: Magnetic Resonance Venography

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Case presentation

A 17-year-old previously healthy female presented to the emergency department from her ophthalmology clinic for seven days of sharp, throbbing, frontal headaches and new onset papilledema. She endorsed six weeks of fatigue with weakness, seven days of chills and sore throat, three days of rhinorrhea, neck pain, right neck swelling and bilateral eyelid swelling. She reported having headaches responsive to over the counter pain medications in the past. Three days prior to presentation, she presented at an outside hospital's emergency room for her headaches. A limited laboratory work-up at that time was normal, head CT was negative for any intracranial pathology, and she was discharged with a diagnosis of viral upper respiratory infection.

On admission, she had a blood pressure of 105/70 mmHg, heart rate of 74 beats per minute, respiratory rate of 14 and 98% oxygen saturation, bilateral papilledema and right sided tender cervical lymphadenopathy. Her neurological exam, including strength, sensation and cranial nerves exam was unremarkable. Laboratories were notable for leukocytosis, transaminitis and elevated Erythrocyte Sedimentation Rate (ESR). Complete Blood Count (CBC), Complete Metabolic Profile (CMP), coagulation studies, urinalysis and respiratory viral panel were all otherwise unremarkable. Further evaluation included testing for Tuberculosis, Human Immunodeficiency Virus (HIV) and Antinuclear Antibodies (ANA) in addition to an elevated Creatine Kinase (CK), all of which were negative. EBV Immunoglobulin M (IgM) was positive. Magnetic Resonance Angiography (MRA) of the brain and orbits were unremarkable. Magnetic Resonance Venography (MRV) demonstrated hypoplastic appearance of the distal right transverse sinus and Magnetic Resonance Imaging (MRI) showed maxillary sinus disease (Figures 1A and 1B). Lumbar puncture revealed an elevated opening pressure of 55cmH20.

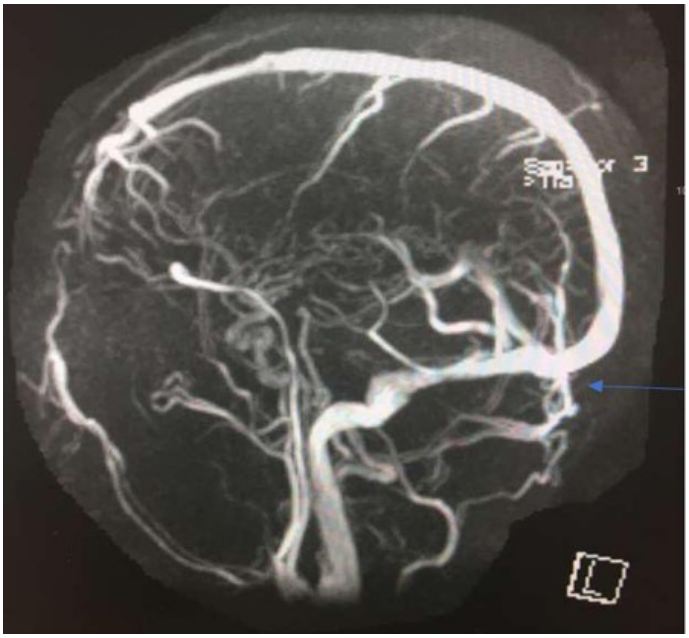

Figure 1a: Magnetic Resonance Venography (MRV) with hypoplastic appearance of the distal right transverse sinus

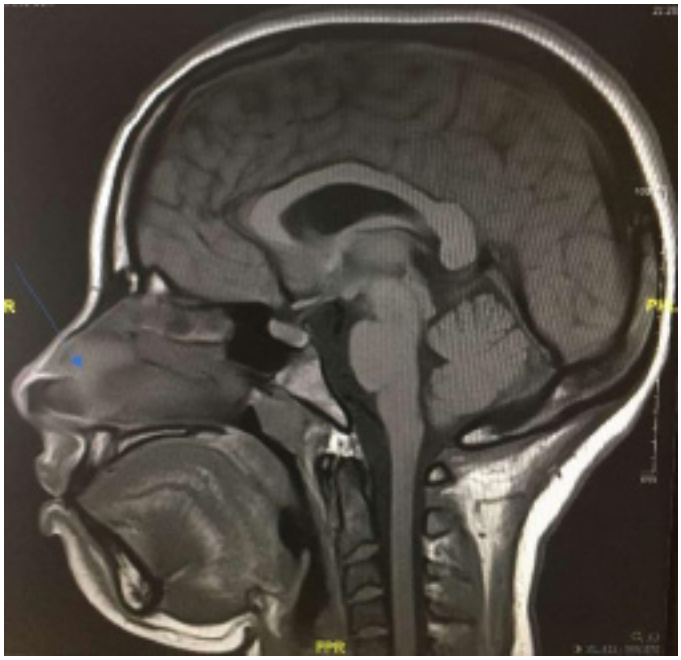

Figure 1b: Magnetic Resonance Imaging (MRI) with maxillary sinus disease

Table 1: Differential for Increased ICP

Trauma
Brain Abscess
Hydrocephalus
Pseudotumor Cerebri
Lead encephalopathy
Reyes Syndrome
Hyperthermia
Acidosis
Anesthetic agents (esp. Halothane)
Hypertension
Pulmonary Insufficiency -> CO2 retention
Guillain-Barre syndrome
Certain Spinal Cord Tumors
Venous Sinus Thrombosis
Obstructive Sleep Apnea
Anemia
Drug-related
Renal Failure
Endocrinopathies, ie: Cushing's, Addison's
Meningitis

Diagnosis

Increased Intracranial Pressure (ICP) due to EBV infection.

Clinical Course

The patient's headache markedly improved following lumbar puncture. Due to her positive EBV IgM, correlating clinical symptoms and improvement of headache with CSF removal, a diagnosis of increased ICP due to EBV infection was made. The patient was started on acetazolamide and discharged home on hospital day 1. At her follow-up appointment three weeks af-

ter discharge, her headaches and papilledema had nearly resolved.

Discussion

EBV is a highly prevalent infection throughout the world, with greater than 90% of adults estimated to have serological evidence suggesting a previous EBV infection [1]. The individual response of a body's immune system determines the ultimate manifestation of an EBV infection in a particular patient. The symptoms of EBV in infectious mononucleosis are well known and include sore throat, cervical lymphadenopathy, fatigue, malaise, fever and hepatosplenomegaly, some of which were identified in this patient. Neurological sequelae may also ensue, the most common being headache in approximately 50% of EBV cases [2]. More dramatic neurological sequelae of EBV are rare, such as encephalitis, meningitis, acute demyelinating encephalitis, cranial nerve palsy, cerebellitis, and myelitis [3]. Increased ICP is an extremely uncommon complication in EBV infection and has been most commonly reported in immunocompromised patients. However, there are also limited case reports of EBV associated increased ICP in immunocompetent patients as well, suggesting the importance of considering EBV infection when generating a differential for increased ICP and headaches.

EBV is a gama-herpes virus with double stranded DNA, which occurs in 2 similar yet distinct strains, including EBV1 and EBV2 with EBV1 being more common worldwide [4]. It is typically transmitted via oral route through saliva, infecting the intraoral epithelial cells and further replicating in order to infect and continue the viral lifecycle in the cells of other nearby tissues. Its primary hosts are the B-lymphocytes within the blood stream and the entire lymph system including lymph fluid, the liver and spleen [2]. The aforementioned symptoms of acute EBV infection as manifested in the infectious mononucleosis disease state are due to cytokine release by activated T-cells. The intensity of symptoms is thought to depend on the viral load that a given patient has been exposed to. However, differing age groups demonstrate varying symptom intensities. For instance, those exposed at a younger age tend to have more subclinical symptomatology as compared with older exposures, thus clinically affecting the adolescent populations more dramatically [1]. In developed countries, EBV infections more often affect adolescents, whereas in developing countries, children ages 3-4 already show serologic signs of exposure [5]. Regardless, EBV is typically transmitted via saliva; however, it may also be transmitted through sexual contact via genital secretions [1].

There are very few known cases in the literature which suggest this occurrence of EBV causing increased ICP. Cases have been precipitated by both acute and chronic EBV infections, in patients with ages ranging from school age through teenage years and have demonstrated typical symptoms such as headache, abdominal pain, nausea, vomiting, fevers and malaise [6, 3]. The only obvious neurological sign in these cases was papilledema. Both chronic and acute EBV infections causing increased ICP have resulted in elevated CSF pressures. Interestingly, only one case was found through literature search that demonstrated evidence of EVB infection in the CSF itself. Specifically, antiviral capsid antigen Immunoglobulin G and Immunoglobulin M were found in the CSF of an 8-year-old with increased ICP due to chronic EBV. This report could not identify a specific pathophysiological model that caused increased ICP; however, it was proposed that there may have been some degree of auto-immune or autoreactive inflammatory process present, resulting in such elevated pressures [3].

Although the exact mechanism in which EBV results in neurological sequelae is unclear, it is likely that virions cross the blood brain barrier to infect the central nervous system (CNS), though there is often no CSF pleocytosis noted in these cases. Inflammation resulting from EBV itself may weaken the defenses at the BBB, further facilitating the viral infection. Typically, neurological symptoms of EBV occur around the same time as systemic symptoms, as was seen in the case above [7]. Interestingly, it was the headache rather than the more common symptoms of EBV infection that brought the patient in for primary evaluation. Our patient did not have evidence of infection in the CNS. Other reported cases of increased ICP resulting from EBV infection also lack evidence of an infection present in the CNS, thus bringing into question the mechanism that leads to this phenomenon. In the limited available case reports related to this topic, it is difficult to determine a clearly suggested mechanism for an EBV infection's ability to cause increased ICP.

The primary management for both EBV and increased ICP revolves around supportive care. In this patient's case, our management included relief of pressure via therapeutic lumbar puncture, which was successful. Acetazolamide may be beneficial in patients with increased ICP and papilledema. Acetazolamide acts as a carbonic anhydrase inhibitor in the choroid plexus of the brain, thereby decreasing the quantity and movement of cerebral spinal fluid [8]. Therefore, given her symptoms were overall controlled and appropriate for her ultimate diagnosis without any known negative sequelae, it is felt that this patient underwent the proper management. Our case highlights the importance of recognizing the atypical manifestations of EBV virus to improve time to diagnosis and proper management of increased ICP.

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