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Case Report

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Concomitant herpes genitalis (HSV-2) and herpes meningitis (HSV-2)

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Abstract

Concomitant herpes simplex virus 2 (HSV-2) genitalis and meningitis is an uncommon presentation. In this case, a female patient in her twenties, with a recent history of herpes genitalis (HSV-2) lesions, presented with fever, headache, neck pain, and photosensitivity. Complete Blood Count (CBC), Comprehensive Metabolic Panel (CMP), and head Computed Tomography (CT) were unremarkable. Lumbar puncture was obtained due to concern for meningitis. Cerebrospinal Fluid (CSF) analysis was notable for an elevated lymphocyte count and positive HSV-2 PCR. She was administered intravenous acyclovir and admitted to inpatient care. Her headache was managed with oral acetaminophen and intravenous ketorolac as needed. After 24 hours, her symptoms resolved. She was transitioned to oral valacyclovir and discharged home on her third day of hospitalization with a 10-day course of antiviral therapy. This case emphasizes the need to consider genitourinary infections as a possible etiology and precipitant for acute meningitis.

Keywords: Herpes simplex meningitis; HSV-2; Headache; Lumbar puncture; Meningoencephalitis.

Background

The etiology of acute meningitis is not always clearly identified. Diagnostic considerations include viral, bacterial, fungal, and noninfectious causes (e.g., aseptic and chemical meningitis). Accurate diagnosis is paramount given the risks of delayed treatment. This case report presents a rare occurrence of acute meningitis as a complication of genital herpes (HSV-2). When evaluating patients with active genital herpes outbreak, it is important for clinicians to recognize the potential for concomitant infections, such as meningitis. This case report describes genitourinary infections, specifically herpes simplex virus, as a potential etiology for meningitis [1].

Case report

Emergency department course

A female patient in her twenties presented to the Emergency Department (ED) with symptoms of fever, headache, neck pain, and photosensitivity. The patient reported chronic headaches in the form of migraine. The presenting headache, however, differed from her chronic migraines (typically frontal and left temporal migraines). For this presentation, the patient's headache consisted of thirty-six hours of generalized intractable headache, with a "helmet like" distribution that was unrelieved by over-the-counter medications. She also reported neck stiffness and nausea associated with a few episodes of vomiting. During the course of their interview (review of systems), the patient reported a recent outbreak of genital lesions. These were confirmed as herpes simplex virus 2 (HSV-2) via outpatient swab, and the patient was subsequently initiated on oral antiviral therapy (valacyclovir, five days prior to ED presentation).

On exam in the ED, vital signs were as follows:

Temperature: 36.4°C (97.6°F).
Blood pressure: 127/66 mm Hg.
Pulse: 82 bpm.
Respiratory rate: 18 breaths per minute.
Pulse oximetry: 99% on room air.

Physical exam revealed an ill-appearing female patient lying in bed with legs curled to chest. There were no focal neurologic deficits noted, though photosensitivity and pain with range of motion of the neck were observed. The patient exhibited a positive Brudzinski sign (passive flexion of neck results in involuntary hip and knee flexion). The patient additionally manifested a positive jolt accentuation test (headache exacerbated by rapid horizontal rotation of head). Given the constellation of findings, the ED team initiated therapy with intravenous fluids, compazine, diphenhydramine, magnesium, and acetaminophen (commonly referred to as a "migraine cocktail").

Laboratory diagnostics

Laboratory tests included serum studies: Complete Blood Count (CBC), Comprehensive Metabolic Panel (CMP), Lactic Acid (LA) (0.5 mmol/L), and procalcitonin (0.05 ng/mL) were all found to be unremarkable, with C-reactive protein (CRP) elevated at 2.24 mg/dL. A urinalysis was obtained and found to be negative for infection. CT scan of the brain was obtained and interpreted as normal without acute pathology (see detailed report below). A lumbar puncture was performed. The opening pressure (OP) was 17 cm H_2O. No organisms were identified on gram stain.

The cerebrospinal fluid cell count showed:

Total Nucleated Cells, CSF: 345 cells/µL.
Red Blood Cells, CSF: 7 cells/µL.
Neutrophils, CSF: 1 cell/µL.
Lymphocytes, CSF: 94 cells/µL.
Monocyte/Histiocyte, CSF: 5 cells/µL.
Clarity, CSF: Cloudy.
Pathology interpretation: Chronic inflammation.
Enterovirus CSF PCR quant: Not detected.
HSV-1 PCR: Not detected.
HSV-2 PCR: Detected.

A noncontrast CT of the head and CT venogram of the head performed in the emergency department were normal without cerebral parenchymal abnormalities, hemorrhage, herniation, hydrocephalus, or cerebral venous thrombosis. There was no abnormal enhancement on postcontrast CTV images. Meningitis is a clinical and laboratory diagnosis. In the setting of infectious meningitis, CT is most often normal and best used for the detection of complications such as hydrocephalus, infectious subdural collections (empyema), or infarct. Occasionally hyperdense or enhancing inflammatory debris-exudate can be seen in the basal cisterns or sulci. Herpes simplex viral encephalitis has a propensity to involve the limbic system, notably the temporal lobe and insula, though imaging in this case did not support parenchymal involvement at the time of evaluation.

Diagnosis and disposition

Given the elevated white blood cell (WBC) count in the CSF with lymphocytic predominance, as well as positive HSV-2 PCR, the diagnosis of HSV-2 meningitis was determined. The patient was initiated on intravenous acyclovir (10 mg/kg) and admitted to the hospital for multidisciplinary care.

Treatment

Following admission, the patient continued to receive intravenous acyclovir 10 mg/kg every eight hours, as well as continuous intravenous fluids to minimize risk of renal injury. Her headache had significantly improved by the time of admission, and she received further symptomatic treatment with scheduled acetaminophen and as needed intravenous ketorolac (Toradol). About 24 hours after presentation to the emergency department, her headache and other symptoms had completely resolved. Infectious Disease (ID) was closely involved during her hospitalization and obtained hepatitis C, hepatitis B, and human immunodeficiency virus (HIV) serologies, all of which were negative. She remained hemodynamically and neurologically stable during the course of her admission. She was transitioned to valacyclovir 1 gram by mouth three times daily and discharged home on the third day of hospital admission to complete a ten-day course of antiviral therapy.

Discussion

Herpes Simplex Virus-2 (HSV-2) is a well-documented cause of acute and subacute viral meningitis, though is relatively uncommon to be recorded as a complication of new primary infection or active outbreak. The incidence of HSV-2 meningitis is

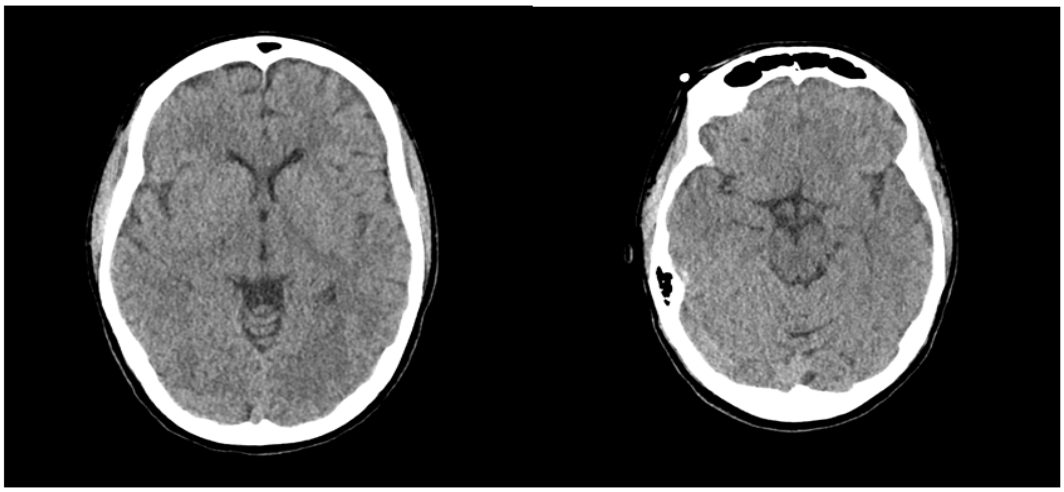

Figure 1: Noncontrast CT and CT venogram of the head.

reported as 0.7/100,000 patients per year with a median age at diagnosis of 35 [5]. PCR technology has increased the rate of detection. Women are affected more frequently, comprising 76% of cases. Cardinal symptoms are headache, photophobia or phonophobia, and neck stiffness. During the initial emergency department or primary care evaluation, it is paramount to rule out life threatening causes of meningitis, such as fulminant bacterial etiologies including *Streptococcus pneumoniae*, *Neisseria meningitidis*, and *Listeria monocytogenes*. If HSV-2 can be detected on CSF PCR during the initial evaluation, this may prevent unnecessary imaging or other preemptive medical intervention. In addition, early diagnosis can expedite the time to appropriate treatment. After primary infection, HSV maintains latency in neurons and commonly reactivates in immunocompromised individuals [2]. It is well known that reactivation is much less common in immunocompetent individuals, such as our patient. Impaired T cell function predisposes a person to complications of this infection, however both innate and cell mediated immunity remain important [3]. Antimicrobial delivery and stewardship are two of the primary challenges in such cases. Arguably, during the clinical phase in which meningitis is considered likely but lumbar puncture has not yet been performed, broad spectrum antimicrobials may be given. In essentially all cases, antiviral therapy is initiated (e.g., IV acyclovir). The clinical challenge is whether empiric antibiotics and steroids should also be given prior to lumbar puncture. Understandably, if the pretest probability of meningitis is high, many providers' practice includes empiric antibiotic and steroid treatment (in addition to antiviral therapy). If the subsequent lumbar puncture identifies a viral meningitis, these empiric antibiotics are not indicated, and in hindsight would have preferably been held. However, many Emergency Medicine providers are uncomfortable withholding empiric antibiotics until CSF results return as the interval between presentation and results may be many hours. In the setting of bacterial meningitis, this delay may increase morbidity and mortality for the patient. Thus, patients may receive empiric antibiotics when in reality the evolving viral pathology will not respond to this treatment. What results and workup can help address this challenge? In this case, the combination of normal serum WBC, normal lactic acid, normal procalcitonin, non-toxic (though ill) appearing patient, and high clinical suspicion aided the providers in the decision to withhold antibiotics [4].

Conclusion

Though an uncommon clinical scenario, herpes simplex virus (HSV) meningitis should be considered in patients with active genital lesions presenting with headache, altered mental status, meningismus, fever. Appropriate workup and treatment include diagnostic labs, blood culture, CT imaging, lumbar puncture including HSV PCR (HSV-1 and HSV-2), symptomatic management, intravenous hydration, and initiation of antiviral therapy. Though classically associated with encephalitis and altered mental status, HSV can cause primary meningitis without acute mental status changes. Though this manifestation results in a more subtle presentation, the urgency of the diagnosis remains high. Early identification, diagnosis, and treatment of HSV meningitis is critical to optimal care of these patients [5].

Take-home points

- Genitourinary infections such as HSV-2 should be considered as a possible, although uncommon, precipitant and source of acute meningitis, especially in sexually active patients.
- Diagnosis of HSV-2 meningitis can be aided by observation of active genital lesions on physical exam accompanied by signs and symptoms of CNS involvement. Diagnosis is confirmed via CSF analysis and PCR.
- Rapid diagnosis of HSV-2 meningitis can prevent unnecessary investigations and treatment.

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