Excellent Abstract

PRIMARY GENITAL HERPES INFECTIONS

Student Case Presentation

Case Report:

CC: Copious vaginal discharge and vaginal discomfort.
HPI: This 17 y.o. BF, LMP 5/13/92, was in good health until 1 week prior to her visit to the clinic when she noticed an increase in vaginal discharge, white in color, and without any associated itching or odor. The patient notes dysuria and increased frequency for 3 days. The day before her visit she described her genitals as "swelling up" and she has felt a burning pain in this area when walking since then. She denies any abdominal pain or fever. Menstrual hx. is unremarkable with first menses at 12 y.o., cycles every 26 days, lasting 4 days. She has been sexually active since age 14. She is currently with one partner, they have been together for 1 year and their last unprotected sexual activity was approximately 2 weeks ago. He denies any symptoms. The pt. has had chlamydia one time a year ago which was treated at that time, and has had one urinary tract infection at age 15.
PMH: Unremarkable, except as noted in HPI. The pt. has NKDA, and is on no chronic medications.
FH: Both parents are alive and well. No hx. of diabetes, HTN or heart disease.
SH: Pt. lives at home in N.E. Washington D.C. with her mother, uncle and maternal grandmother. school ? pregnancy ?
ROS: Unremarkable, except as noted in HPI.

PHYSICAL EXAMINATION
General: WNWD BF, walking slowly and obviously in moderate discomfort.
Vitals: T=37.4C; P=68; R=16; BP=126/70; Wt.=164lbs.
Skin: Clear without lesions
HEENT: Eyes: PERRLA, EOMI. Ears: TM's clear bilaterally. Nose: Clear. Pharynx: Clear.
Nodes: tender firm 1 cm mobile inguinal nodes bilaterally.
Neck: FROM. without masses or thyromegally.
Pulm: CTA bilat.
CV: RRR, nl S1, S2. No murmurs, rubs or gallops
Back: No CVAT. No scoliosis.
Abd: Soft, NT, ND. +BS. No HSM or masses. No guarding or rebound.
GU: Tanner stage 5, external genitalia normal and without lesions. Some clear whitish discharge was noted. When labia minora were spread, shallow ulcerations were visualized extending most of the introitus bilaterally with several small satellite vesicles. These lesions were extremely painful and neither a speculum exam nor a bimanual exam could be performed.
Ext: FROM. Without lesions or abnormalities.
Neuro: Alert and oriented. CN II-XII grossly intact; sensory and motor intact. Reflexes 2+ and symmetrical. No clonus.

Labs:
Wet prep: Many WBC's seen. No hyphae, Trichomonas or clue cells.
UA: SG=1.008, pH=7.0, 15-20 WBC, ORBC, few bact, no glu, ket, bld, nitr, bil.
RPR: NR

Assessment and Plan This is a 17 y.o. BF with a clinical history and presentation of a primary genital herpes infection. She has a history of unprotected sex, but cannot be cultured for gonorrhea or chlamydia at this time. Therapeutic plan: Ceftriaxone 250mg IM now, E-mycin 333mg P.O. T.I.D. x10 days and Acyclovir 200mg p.o. 5 times a day for 2 weeks. Pt. is to return to clinic in 2 weeks to be examined again, including a speculum exam, cervical and vaginal cultures. normals for abx?

DISCUSSION

Herpes (Greek "to creep") has been described in the medical literature as early as 100 A.D. The etiological agent in genital herpes infections is the double stranded DNA herpes simplex virus (HSV). HSV can be divided by neutralization into two antigenic types: HSV-1 and HSV-2. 5-15% of all first episodes of genital herpes are caused by antigenic type HSV-1, with the majority attributable to HSV-2.

Clinical differentiation between genital HSV infections and other infectious or noninfectious causes of genital ulcerations may be difficult. In the United States, most genital ulcerations in heterosexual persons are caused by HSV. It is common, however, for patients who have one venereal disease to have a second, and so finding an etiology for a genital lesion doesn't preclude the presence of a second disease.

Epidemiology: Prevalence of genital HSV has increased markedly between the early 1960s and 1980s and a concomitant increase in neonatal HSV infection has been seen.[1,2] In the general U.S. population, HSV-2 antibody prevalence rates range from 20-60%, increasing with age and correlating with sexual activity.[3] As many as two-thirds of serologically positive individuals may have no history of symptomatic disease making antibody screening a more reliable method of detecting true prevalence.[4,5,6]

Pathogenesis: Transmission occurs when there is close contact with a person who is shedding virus. Infection occurs by inoculation of HSV onto susceptible mucosal surfaces or through small cracks in the skin. Average incubation is 3 to 7 days. Viral replication causes production of mononucleated giant cells and eosinophilic intranuclear inclusions. Concomitant with initial infection, HSV ascends peripheral sensory nerves and enters sensory or autonomic nerve root ganglions where latency is established.[7]

Clinical Features: Pain, itching, dysuria, vaginal or urethral discharge and tender inguinal adenopathy are the predominant local symptoms of disease. First episodes of genital herpes are often

associated with systemic symptoms. Fever, headache, malaise and myalgias are reported in nearly 40% of men and 70% of women with primary HSV-2 infections. HSV characteristically presents as grouped vesicles with a red areola. Multiple small lesions may coalesce into areas of ulceration. Lesions are painful in over 97% of patients. Lesions persist for 4-15 days until crusting or reepithelization occurs.

Complications: Complications include local extension (cervicitis, urethritis, rare incidents of pelvic inflammatory disease), and spread of virus to extragenital sites. Aseptic meningitis, transverse myelitis, or the sacral radiculopathy syndrome have been seen in primary HSV-2 infections. Symptoms typically appear 3-12 days after onset of genital lesions. Disseminated infection occurs rarely, but may be predisposed to in pregnancy and in immunocompromised patients. Herpes neonatorum is an additional complication, although recurrent HSV infections only have a 0-8% risk of transmission to the newborn, while primary genital HSV infections carry a 50% risk of transmission.[8]

Laboratory Features: Viral isolation in tissue culture is the preferred method for laboratory confirmation. Alternatively, rapid detection methods such as cytological examination (Tzanck smear) or immunofluorescence may be used. HSV can also be confirmed by DNA hybridization and ELIZA techniques, which are better at differentiating the antigenic type.

Therapy: Treatment is Acyclovir 200mg p.o. 5 times a day for 10-14 days. Acyclovir is an acyclic nucleoside analogue that is a substrate for HSV-specified thymidine kinase. It is phosphorylated and then incorporated into the growing viral DNA chain and causes chain termination. While treatment with Acyclovir has been shown to decrease the course and severity of primary herpes infections, it has no effect on the long-term natural history of reoccurrences.[9] Patients need to be counseled about the clinical manifestations and high rate of recurrence.

Summary: The first episode of genital herpes simplex virus infection is a disease of both systemic and local manifestations. Over half the patients with primary genital herpes suffer from constitutional complaints, and one-third complain of headache, stiff neck and mild photophobia during the first week of disease. Although duration of viral shedding from external genital lesions is similar in males and females with primary genital herpes, the duration of symptoms and frequency of complications are greater in women.[10] Treatment with oral Acyclovir has been shown to markedly decrease the course and severity of the first infection. Patients with genital herpes, especially HSV-2, are at a high risk for recurrence and must be counseled as to what they should expect and how to prevent spread of the disease by avoiding unprotected sexual contact until all lesions have completely crusted over and reepithelialized.

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