Dr. Gallagher, Oral and Maxillofacial Surgeon, presents

Practical Practice Pearls

For Dental and Medical Professionals

This newsletter is published monthly and contains useful information about current pharmacology and therapeutics, pathology, techniques, and procedures used for the management of diseases and conditions of the hard and soft tissues of the face and mouth. Please contact us to be added or removed from our fax list, and/or with your comments and suggestions for "Pearl Topics". 12210 Pecan Street, Austin, Texas 78727 phone: 512 258-1636; fax: 512 258-6352; email: dgallagher@jawpain.com

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Managing Oral Herpes Simplex Virus Infections

The majority of adults have been exposed to herpes simplex virus (HSV), but most people are not aware of it because most infections are asymptomatic. The HSV resides in the nerve ganglion where it is sheltered from antiviral medications and the immune system. The immune system keeps the virus within the ganglion, but during periods of immune deficiency the virus follows the peripheral nerves to the surface of the skin or mucosa. Acute immune deficiency may occur in the healthy person following generalized emotional and/or physical stress. Sunburn, prolonged insomnia, anxiety, trauma, and a poor diet are examples of conditions that may contribute to HSV infections. HSV infections may become particularly serious in immuno-compromised persons, particularly people with immune deficiency diseases, such as AIDS.

Antiviral Treatment for Oral Herpes Simplex Infections

Oral

Famciclovir (Famvir) 250mg PO ASAP, then 125mg bid x 5 days (our favorite)
or, Acyclovir (Zovirax) 400mg PO tid x 5 days
or, Valcyclovir (Valtrex), 500mg PO qd x 3days

Topical

Acyclovir (Zovirax) 5% ointment, 3gm or 15gm, apply 6x's/day or q3h
or, Docosanol (Abreva) 10% cream, 2gm, apply 5x's/day
or, Penciclovir (Denavir) 1% cream, 1.5gm, apply q2h during the day

Primary HSV appears as painful, diffuse erythematous gingivitis. Mucosal lesions begin as pinhead ulcerations that cluster and enlarge over several days. Fever, malaise, and lymphadenopathy also occur. Whereas primary herpetic ulcerative stomatitis commonly occurs in children, secondary herpetic lesions usually appear in adolescents and older patients. Secondary HSV are commonly preceded by prodromal symptoms of warmth, itching, and tingles of the localized skin or mucosal surfaces where the lesions will appear. Shortly thereafter, often within hours, pinpoint vesicles appear usually on keratinized mucosa of the lip and/or the skin. The vesicles coalesce into a blister filled with serous fluid. This lesion contains viral particles, and as it enlarges it dissects the epithelium from its basement membrane. The denuded ulcer is painful, and has a typical "halo erythema" pattern, where the borders of the ulcer are a brighter red than the center of the lesion.

Regardless of treatment, herpetic lesions will heal without scarring in about two weeks in a healthy person. However, timely palliative therapy, as well as direct treatment with antiviral medications can substantially reduce the severity and the duration of the infection.

Antiviral medications inhibit synthesis and hence, replication, of herpes viral DNA. They have antiviral activity against herpes simplex virus 1 (HSV-1), 2 (HSV-2), and varicella-zoster virus (VZV). The three antiviral medications most useful for treating HSV (and also for treating VZV) are acyclovir (Zovirax), valacyclovir (Valtrex), and famciclovir (Famvir). After oral administration, valacyclovir is rapidly converted to acyclovir. Acyclovir has a plasma half life of about 3 hours. Famciclovir undergoes rapid biotransformation to the active antiviral compound penciclovir, with an intracellular HSV half life of about 10 hours.

Treatment is directed at minimizing viral activity, limiting cellular damage, and enhancing conditions for wound healing. Prodromal symptoms indicate that HSV is causing biochemical changes within epithelial cells near the peripheral nerve endings. Antiviral medications commencing at this time will have the most beneficial effect in limiting the quantity and size of the lesions, as well as limiting the duration of the acute disease process.

When vesicles appear use the tip of a sterile needle to lance through the dead epithelium to permit escape of the serous fluid. Evacuation of the vesicular fluid removes a lot of the virus from the wound, limits expansion of the blister (less blister dissection) and opens a channel into the lesion. After the fluid has been expressed from the blister use a cotton swab soaked in a volatile solvent (we prefer ethyl chloride, diethyl ether, or acetone) to cleanse the lesion. The blister is not denuded because there is only a hole in the epithelium. The volatile agents kill the HSV virus upon contact. Then cover the lesion with antiviral ointment. Avoid using alcohol because it hurts. At the very least, lance the lesion and cover it with the antiviral ointment.

Symptomatic medical management with a nonsteroidal anti-inflammatory analgesic (ibuprofen 800mg tid, Vioxx 50mg qd, *etc*.), vitamin C (1gm qd), plenty of fluid intake, and rest are always helpful. For intraoral lesions prescribe chlorhexidine (Periogard, Peridex) oral rinses twice a day. Antibiotic therapy is usually not necessary for secondary herpetic lesions, but may be useful in preventing bacterial infections in primary herpes infections. Ask every patient to use a lip balm (Blistex, Carmex) to prevent desiccation and cracking of the skin until the lesion is fully healed.

Counsel patients about the management of future lesions: Apply antiviral ointment as soon as prodromal symptoms occur, and ask the patient to call you about commencing other therapy. HSV lesions that are kindled in some patients by dental treatment (particularly injections) may be prevented by premedication with antiviral medications.

Words of caution: When diagnosing HSV and VZV infections, differentiate from other benign ulcerative lesions such as aphthous stomatitis, traumatic ulcers, lichen planus, and acute ulcerative gingivitis (ANUG).