Herpes Zoster (Shingles)

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This infection is caused by reactivation of varicella-zoster virus (VZV), which may remain latent in the dorsal root and cranial nerve ganglia for decades. Reactivation often occurs for no apparent reason, although stress and immunosuppression may increase the risk.

Herpes zoster may begin with a systemic constellation of mild symptoms that include fever, anorexia, and lassitude. Unilateral dysesthesia and pain along the site of the future eruption usually precede the rash by 1 to 3 days. The rash consists of clusters of grouped papules, papulovesicles, vesicles (A), or erosions on an erythematous base, or urticaria-like plaques (B) or erosions (C). Sometimes the eruption manifests as a plaque of crusted pustules in a dermatome (D). Elderly patients, who are relatively immunosuppressed, may have a prolonged course of herpes zoster and are more susceptible to secondary infections.

Herpes zoster remains infectious until all the vesicles and pustules have evolved into crusted plaques; they resolve over the course of 2 to 3 weeks. The differential diagnosis includes insect bites, urticaria, herpes simplex virus infection, and cellulitis.Diagnostic testing is sometimes useful. Options include Tzanck preparation, viral culture, direct fluorescence antibody (DFA) testing, and skin biopsy. DFA testing is more sensitive than conventional viral cultures because of the lability of VZV. Variations of herpes zoster include scattered vesicles (satellite lesions) that occur outside 1 or 2 dermatomes. A minority of patients may experience dysesthesia without urticarial plaques or vesicles (zoster sine herpeticum).

Treatment consists of a 7-day course of acyclovir, valacyclovir, or famciclovir. A typical regimen for elderly persons is valacyclovir, 1 g tid. Antiviral agents may reduce the duration of postherpetic neuralgia.1,2 This condition, which leads to chronic pain that is often refractory, is particularly frequent and severe in elderly persons. Therapeutic options include lidocaine patches, opiates, and a variety of anticonvulsant, antidepressant, and antipsychotic agents. A live zoster vaccine has been approved for prevention of postherpetic neuralgia in patients aged 60 years or older. Some subtypes of herpes zoster require intravenous antiviral therapy. Ophthalmic herpes zoster, which involves the first division of the fifth cranial nerve, may cause scarring of the cornea and secondary panophthalmitis, with subsequent loss of vision. Characteristic signs include vesicles on the tip of the nose and an ocular foreign-body sensation.

Ramsay Hunt syndrome is herpes zoster infection of the geniculate ganglion, which lies at the genu of the seventh nerve. It is characterized by ipsilateral facial palsy similar to Bell palsy. Vesicles develop in the external auditory meatus, on the pinna, and sometimes in the soft palate and can cause deafness. Glossopharyngeal and vagal zoster affects the jugular and petrosal ganglia, which are adjacent and often involved simultaneously, although individual ganglial involvement can occur. A painful vesicular rash typically affects the palate, posterior tongue, epiglottis, tonsillar pillars and, occasionally, the external ear. The unilateral distribution helps distinguish this variation of herpes zoster from herpes simplex virus infection and herpangina.

Disseminated herpes zoster involves more than 3 dermatomes or has more than 20 lesions outside a dermatome; it particularly affects patients with non-Hodgkin lymphoma or HIV. It manifests as generalized vesicles, papulovesicles, or erosions (E). Disseminated herpes zoster may involve internal organs; it presents as hepatitis, pneumonitis, meningoencephalitis, myelitis, or motor radiculopathy. Ophthalmic herpes zoster, Ramsay Hunt syndrome, and disseminated herpes zoster are treated with IV acyclovir, 10 mg/kg every 8 hours, with renal adjustment of dosing as necessary.
References:

REFERENCES:

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