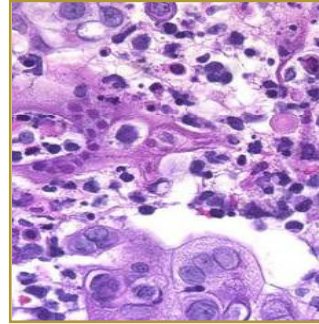




HERPES ZOSTER



EPIDEMIOLOGY: 95% of adults in the United States have antibodies to the varicella-zoster virus.
Occurs annually in 300,000-500,000 individuals

ETIOLOGY: Unknown; however, decreased cellular immunity is a causative factor

PATHOGENESIS: Manifests as a vesicular rash, usually in a single dermatome

CLINICAL: Rash in a unilateral dermatomal distribution; the rash may be erythematous, vesicular, pustular, or crusting, depending on the stage of disease

HISTOLOGY: Intraepidermal vesicle produced by profound degeneration of epidermal cells, resulting in marked acantholysis

VARICELLA-ZOSTER VIRUS INFECTION initially produces chickenpox. The virus will lay dormant in a patient until focal reactivation along a ganglion's distribution results in herpes zoster (shingles). An average of about 20 percent of people who have had chicken pox will develop the zoster virus. The symptoms may include a burning sensation, pruritus, tingling, or extreme sensitivity in the affected area, it is usually limited to one side of the body. Patients may also complain of headaches or fever. The symptoms may be present for anywhere between one to three days prior to a rash that develops. The rash converts into blisters that last for around two to three weeks. The blisters start out clear but then look yellow or bloody before they crust over (scab) and disappear. Initial therapy may include nonsteroidal anti-inflammatory drugs (NSAIDs). In many cases, narcotic analgesia is necessary. Wet dressings with tap water or 5% aluminum acetate (Burow solution) and apply to affected skin for 30-60 minutes 4-6 times per day. Lotions (ie, Calamine) may help relieve discomfort.

BIBLIOGRAPHY

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