

# SHINGLES CASE STUDY

## Acyclovir Therapy in Acute Thoracic Herpes Zoster: a case study

**Entry from: JT Carpenter-Kling**  
Brooklyn

Herpes Zoster is a localized disease characterized by unilateral radicular pain and a vesicular eruption limited to the dermatome innervated by a single spinal or cranial sensory ganglion.

The major goals of therapy in patients with herpes zoster are:

1. to limit the extent, duration and severity of disease in the primary dermatome;
2. to prevent disease elsewhere; and
3. to prevent post-herpetic neuralgia

This case study reports on a patient with thoracic herpes zoster and the clinical response to oral acyclovir therapy.

### Case Report

A 13-year-old male patient presented with grouped vesicles on an erythematous base in T10-distribution, 68 hours after the appearance of the first vesicles. He experienced slight tenderness in the involved dermatome two days prior to the eruption. There were no prodromal constitutional symptoms such as headache, fever or malaise.

When the vesicles started erupting, he experienced severe pain in the T10-distribution. This constant, burning pain was so severe, it kept him awake at night.

There was a positive history of varicella at the age of four years. No oral or topical treatment had been used.

On examination the rash consisted of tense grouped vesicles unilaterally in the T10-distribution, not crossing the midline. (Fig. 1 and 2.) These lesions were hyperesthetic to light touch. Regional lymph nodes were enlarged and tender. The patient was otherwise well.

His immune status was evaluated and he was found to be immuno-competent. His WBC, lymphocyte count and immunoglobulins were within normal limits and his HIV test was negative (Eliza test).

Other tests done include:

S-Glucose normal

LFT and U+E were normal

ESR was 0 mm/h

Vesicle fluid taken for electron microscopy was positive for Herpes Zoster virus (Fig 3).

The patient was started on oral Zovirax 800 mg 5x/day for seven days. No other oral or topical medication was given. Within two days after the initiation of Zovirax therapy, the pain had reduced significantly and there was no new vesicle formation.

The patient was seen one week later and he was not complaining of pain, although the area was still slightly tender to light touch. Most of the vesicles had formed crusts and the erythema had subsided. (Fig 4.)

At the second week follow-up, there was no pain and no tenderness to touch. There were a few crusts left, but most of them had healed, leaving slight post-inflammatory hypopigmentation. (Fig. 5).

The patient suffered no side-effects due to the Zovirax treatment.

At the six week follow-up, the lesions had healed completely, and there was no post-herpetic neuralgia.

### Discussion

The incidence of herpes zoster is determined by factors which influence the host-parasite relationship. One of these is age. More than two thirds of reported cases occur in individuals over 50 years of age and less than 10% of cases occur under the age of 20 years. The incidence of herpes zoster in immunosuppressed patients is increased 20 to 100 times, as well as the severity of the disease. The higher incidence and severity of herpes zoster in older individuals, as well as in individuals of any age who are immunosuppressed, is associated with deficient cell-mediated immune response to varicella-zoster virus antigens.

The varicella-zoster virus causes both herpes zoster and varicella. In contrast to varicella, which follows primary exogenous varicella-zoster virus infection, herpes zoster appears to represent reactivation of an endogenous infection that has persisted in latent form following an earlier attack of varicella. The relationship of herpes zoster to varicella was first noted by Von Bokay in 1988, who observed that susceptible children acquired varicella after contact with individuals with herpes zoster.

In the ganglia a latent infection is established in the sensory neurons and the virus then persists silently and does not multiply, but it retains the capacity to revert to full infectiousness. The mechanisms involved in the activation of the varicella-zoster virus are unclear. When host resistance falls below a critical level, the reactivated virus can no longer be contained and the reversion is successful. The virus then multiplies and spreads within the ganglion causing neuronal necrosis and intense inflammation, which is accompanied by severe neuralgia. The infectious varicella-zoster virus is then released around sensory nerve endings in the skin where it produces the characteristic cluster of vesicles.

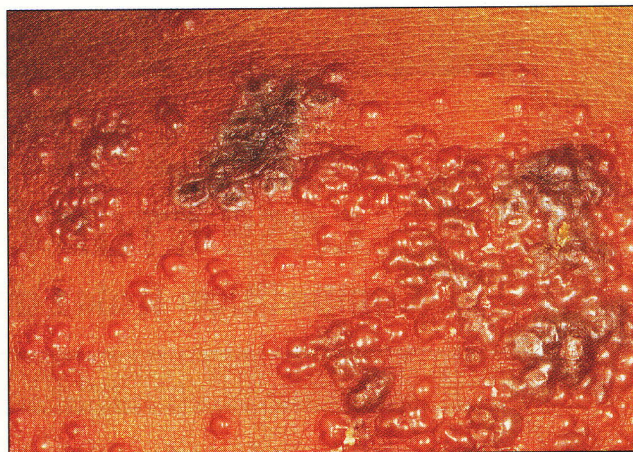
Vesicles form within 12-24 hours and evolve into pustules by the third day. These dry up and crust in 7-10 days. Crusts generally persist for two to three weeks. In normal individuals new lesions continue to appear for one to four days. (Occasionally for as long as seven days.)

In this case study of a 13-year-old immunocompetent male, the oral acyclovir at a dosage of 800 mg 5x/day for seven days prevented progression of the lesions, decreased pain in the acute stage and shortened the healing time, if compared to the normal clinical pattern observed if left untreated.

### Bibliography:

1. Oxman MN: Varicella and herpes zoster, in Fitzpatrick TB et al: Dermatology in general medicine, ed 3, New York, 1987, McGraw-Hill Book Co.
2. Arnold HL et al: Andrews diseases of the skin, ed 8, 1990, Saunders Co.
3. Bean B et al: Acyclovir therapy for acute herpes zoster. The Lancet, July 1982, p.118.





Figures 1 and 2. Rash of tense grouped vesicles unilaterally in the T10-distribution, not crossing the midline.

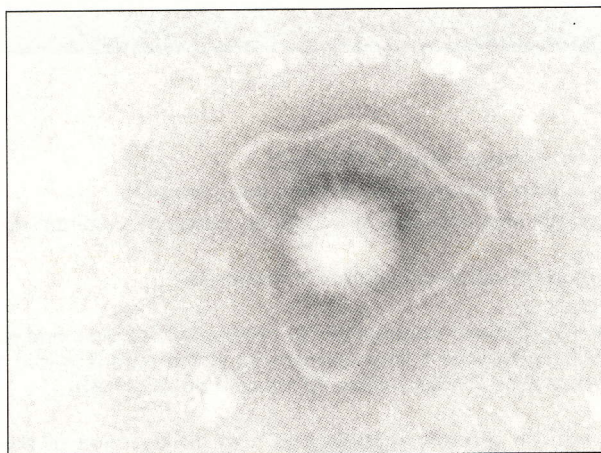


Figure 3. Vesicle fluid taken for electron microscopy was positive for Herpes Zoster virus.

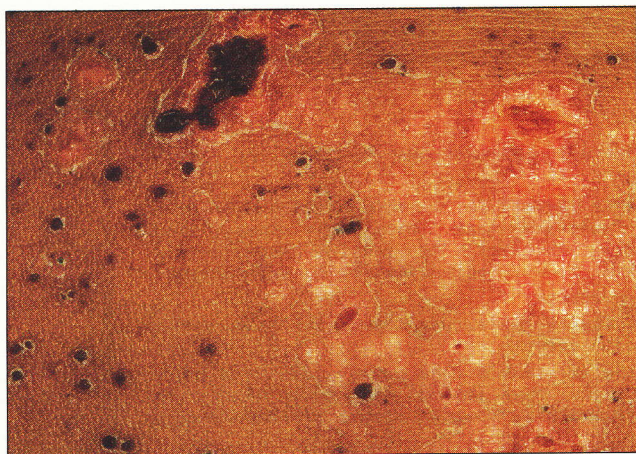


Figure 4. Most of the vesicles had formed crusts and the erythema had subsided.

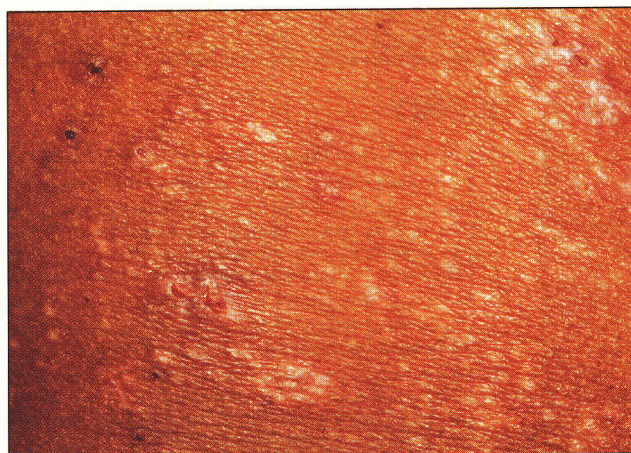


Figure 5. A slight post-inflammatory hypopigmentation at the second week follow-up.

# Shingles Case Study / Gordelroos Siekteverslag



Wellcome

FIND A BETTER WAY.

Wellcome (Pty) Ltd, 68 Rigger Road, Spartan, Kempton Park, 1619. Tel (011) 975-1146. Co. Reg. No. 66/00480/07.  
 Wellcome (Edms) Bpk, Riggerweg 68, Spartan, Kempton Park, 1619. Tel (011) 975-1146. Mpy. Reg. Nr. 66/00480/07.