

# Shingles Case Study Competition

## Herpes Zoster Generalisatus: Report of a case successfully treated with Acyclovir

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### Introduction

Herpes zoster (shingles) is caused by the varicella-zoster virus. An attack is due to reactivation, usually for no apparent reason, of virus which has remained dormant in a sensory root ganglion since an earlier episode of varicella (chickenpox). The incidence of herpes zoster (HZ) is highest in old age, and in immunocompromised patients such as those with haematological malignancies, advanced HIV-disease and those receiving immunosuppressive drugs. Attacks usually start with burning pain, followed by erythema and grouped, sometimes blood-filled, vesicles scattered over one or more dermatomes. The clear vesicles quickly become purulent, and, over the space of a few days, rupture and become crusted. Crusts usually separate in 2-3 weeks, sometimes leaving depressed depigmented scars.

Zoster is characteristically unilateral and may affect one or more adjacent dermatomes. The thoracic segment and the ophthalmic division of the trigeminal nerve are involved more commonly. It is not uncommon for a few varicelliform lesions to be found outside the main segment, or segments, of involvement, but a more widespread varicelliform eruption accompanying segmental zoster, herpes zoster generalisatus (HZG), should raise suspicion of an underlying immunocompromised state or malignancy, particularly if the lesions are unusually hemorrhagic or necrotic.

The introduction of the synthetic acyclic purine nucleoside analogue acyclovir in 1982 marked a new generation in antiviral chemotherapy. Acyclovir is highly effective against many herpes viruses while maintaining a highly favourable therapeutic index. The safety and efficacy of acyclovir (oral and intravenous) in decreasing the morbidity associated with both first-episode and recurrent genital herpes has been demonstrated in placebo-controlled clinical trials. In addition, the effectiveness of chronic daily therapy to suppress recurrent episodes in normal and immunocompromised individuals has been established. Recent studies demonstrate effectiveness against varicella zoster virus in treating HZ and varicella.

### Case Report

A 64-year-old man presented with a two day history of an extremely painful skin rash involving the left arm and hand. Additionally, he stated that similar lesions had appeared on the face, trunk, genitals and lower limbs during the preceding day.

Previous medical history included a below knee

amputation for intractable ulceration due to atherosclerosis, bilateral total hip replacements for debilitating osteoarthritis, seronegative spondyloarthropathy treated with nonsteroidal anti-inflammatory drugs and diffuse obstructive airways disease. The patient was not treated with immunosuppressive drugs.

On examination, the patient was afebrile with normal temperature, pulse and blood pressure. Dermatological examination revealed grouped vesicles and pustules on an erythematous background involving the C4-C6 dermatomes of the left arm and hand (Figures 1 and 2). Numerous outlying varicelliform vesicles were noted on the eyelids, the upper lip, scalp, genitals, trunk and lower limbs. These clinical features are consistent with a diagnosis of HZG. Complications such as secondary bacterial infection, motor nerve and eye involvement were not detected.

A Tzanck smear was prepared from scrapings obtained from the floor or a vesicle. Employing a Giemsa stain, multinucleated cells with faceted nuclei could be demonstrated (Figure 3). A biopsy revealed variable epidermal necrosis, intraepidermal blistering and a dermal cellular infiltrate composed of lymphocytes and some neutrophils. Ballooned keratinocytes contained eosinophilic intranuclear inclusions and giant cells with moulded nuclei were evident. In many keratinocytes, nuclear chromatin was pushed peripherally by steelgrey nucleoplasm. Varicella-zoster IgM was negative but IgM highly positive. The presence of varicella-zoster virus was confirmed by viral culture from aspirated vesicle fluid.

Haematogram, chest X-ray and blood chemistry were normal. Polyclonal elevation of IgG was detected with mild depression of IgA and IgM levels.

The patient was treated with oral acyclovir, 800mg 5 times daily for 7 days. The patient received no other treatment. The patient responded to treatment rapidly. At initial follow-up, one week later, established vesicles in the area of segmental involvement were virtually healed and no new lesions appeared (Figure 4). Outlying vesicles were also healed and formation of new lesions ceased. Pain relief was impressive and complications did not develop. The varicella-zoster IgG value was further increased. Laboratory parameters remained within normal limits. At subsequent follow-up, two months later, the patient complained of mild hyperaesthesia in the area of involvement, but was otherwise well.

### Discussion

An apparently immunocompetent patient with HZG who responded dramatically to treatment with oral acyclovir, 800mg 5 times daily for 7 days, is reported. The

polyclonal elevation of and increased sedimentation rate were ascribed to the seronegative spondyloarthritis. Whether the depressed levels of IgA and IgM contributed to the development of HZG, is speculative.

At our institution, currently accepted indications for treatment of HZ with systemic acyclovir in immunocompetent hosts include: HZG, multisegmental zoster, ophthalmic zoster and zoster associated with severe pain, especially in the older patient. Treatment should be initiated within 48 hours of the onset of the rash. The decision to treat the patient with acyclovir was influenced by his age and the presence of severe pain, multidermatomal involvement, and disseminated infection.

Herpes zoster, including HZG, in the immunocompetent patient is not a listed indication for treatment with systemic acyclovir. However, successful treatment of this infection

has been reported previously and was borne out by the present case.

## Conclusion

Acyclovir treatment should be strongly considered in patients with HZG. A controlled trial to establish whether acyclovir is the therapeutic modality of choice for HZG is certainly warranted. Whether acyclovir treatment will prevent debilitating post herpetic neuralgia remains to be determined. Acyclovir is a costly drug and, furthermore, the development of viral resistance to acyclovir has been reported. Therefore, careful patient selection is crucially important.

## Reference

Liesegang TJ. The varicella-zoster virus: systemic and ocular features. *J Am Acad Dermatol* 1984;11:165-191.



Figure 1: Herpes zoster involving the C4-C6 dermatomes. Plaques are composed of grouped vesicles and pustules on an erythematous background.

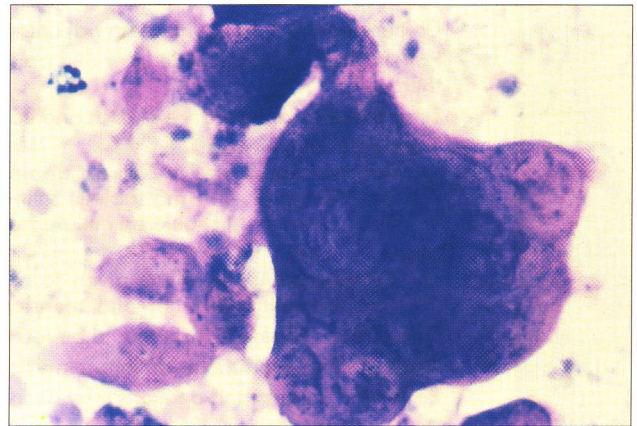


Figure 3: Photomicrograph of a Tzanck smear revealing a multinucleated giant cell (Giemsa x 400).

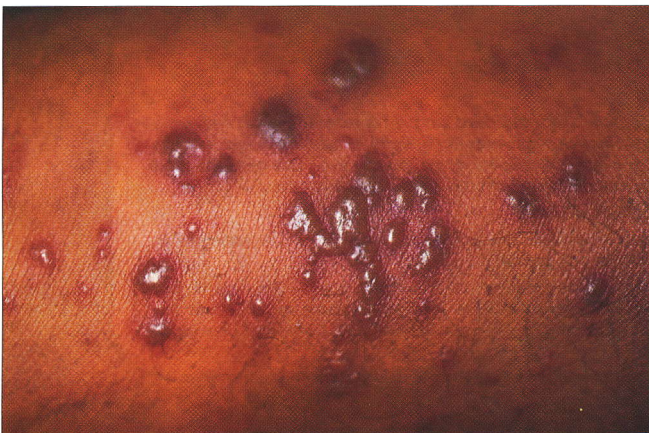


Figure 2: Closer view demonstrating vesicles and pustules. Note that some lesions contain blood.



Figure 4: Scabbed lesions following one week of treatment with oral acyclovir. Note absence of erythema and vesiculation.