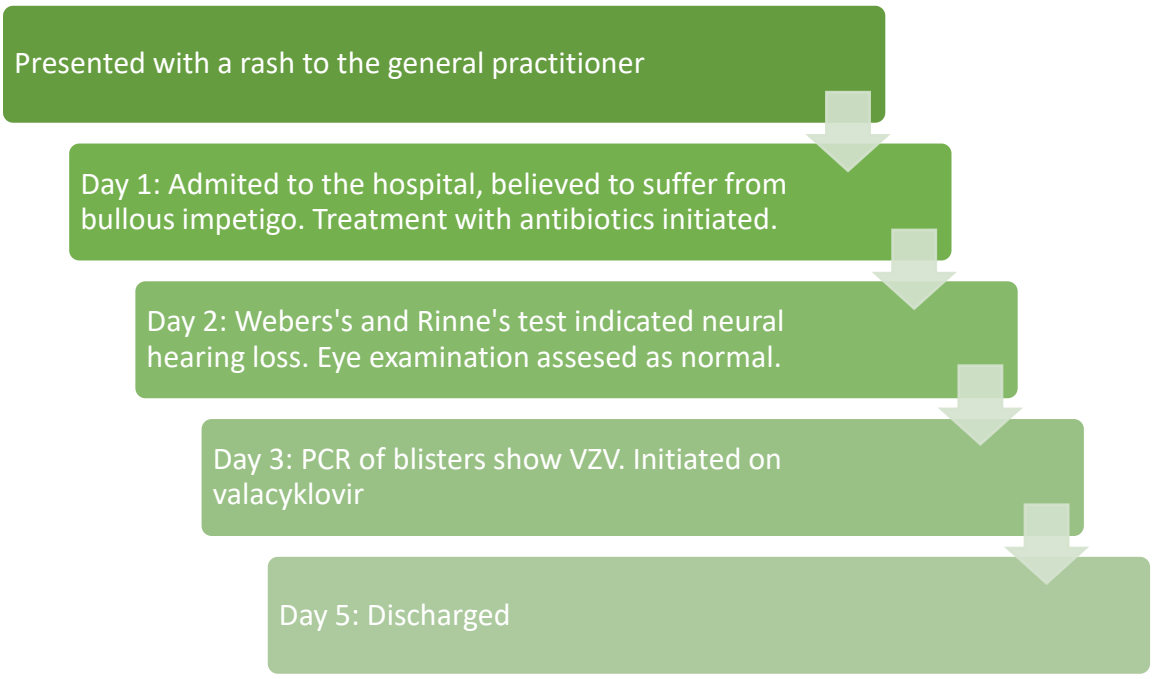
Herpes Zoster in an immunocompetent 18-year-old presenting like an impetigo: A case report

Introduction

Chicken pox is caused by a primary infection with varicella zoster virus (VZV), an alphaherpes virus, and is symptomatically recognized by fever and a diffuse vesicular rash over multiple dermatomes. The virus persists in sensory nerve ganglia after the primary infection (1). Endogenous reactivation of VZV spreads unilaterally usually along a single dermatome causing herpes zoster, known as shingles, which is characterized by a painful localized vesicular rash (2).

Potential risk factors of herpes zoster include old age, female gender and immunosuppressed individuals, primarily cell mediated immunity (3). Here we report a case of an immunocompetent young woman which developed a severe case of herpes zoster initially interpreted as bullous impetigo. We discuss relevant literature on follow-up and prognosis as well as herpes zoster as a marker for an underlying undiagnosed disease.

Timeline



Case presentation

An 18-year-old Caucasian female presented to the general practitioner with fever, general fatigue, an exuding redness that had spread over the right side of the face and headache. Upon initial examination enlarged lymph nodes at the jaw angle were found. Small intact blisters with yellow crust on a palm sized erythematous area on the right cheek were noticed upon inspection of the face. Laboratory analysis revealed a C-reactive protein (CRP) of 8 ng/L and leukocyte concentration of 3 x 10^9/L. Her medical history was scarce and only included a broken arm 5 years prior. She had chicken pox at 8 months of age. She was not on any regular medication including oral contraceptives.

She was referred to a local hospital where further examination revealed a heart rate of 75, with a regular rhythm. Auscultation revealed no audible murmurs. Lung auscultation revealed vesicular breathing sounds bilaterally with no audible wheezes or rales. She had a saturation of 100% on air and a respiratory rate of 16 breaths/min. Surprisingly, her blood pressure was 140/100 mmHg. She had no uveitis, no impairment of eye movements and no signs of light sensitivity. The patient's condition was interpreted as severe bullous impetigo and treatment with cloxacillin 2g x 4 were initiated.

The next day the patient had not improved. The affected area was now swollen thus making eye opening difficult. However, she experienced little to no pain. The patient had developed a subjective decrease of hearing. Weber’s test was lateralized to the affected side and Rinne’s test was negative thus indicating a unilateral neural hearing loss. Eye examination revealed no uveal, corneal nor eyelid involvement.

Initial interpretation of the patient's condition explained as bullous impetigo was disregarded and re-evaluated to herpes zoster affecting dermatomes of the maxillary nerve (CN V2) and Vestibulocochlear nerve (CN V8). Treatment with valacyclovir was initiated. The patient denied drug injections and unprotected sex. Alere determine HIV-1/2 AG/Ab combo test was negative. No further screening was performed. The patient was discharged 2 days later in her habitual state. The patient was remitted to an ear nose and throat specialist for follow up.

Discussion

Potential risk factors for herpes zoster is not completely known and some results and studies are contradicting. It is evident that increasing age, immunosuppressed individuals, HIV patients and patients with cancer have a higher incidence of zoster (3, 4). However, it is not yet fully understood if individuals with herpes zoster should be tested for cancer, HIV or other immunosuppressive diseases.

Two studies comparing herpes zoster incidence in HIV-positive and HIV-negative individuals have shown an 12-17-fold increased risk of developing herpes zoster in HIV-positive individuals (5, 6). Surprisingly in areas of high HIV-prevalence there are reports indicating that Herpes-zoster has a positive predictive value of 85-95% for an underlying HIV infection (7, 8).

Genetic susceptibility to herpes zoster was studied by Haanpää et al which found that 53% of 60 immunocompetent patients with herpes zoster carried the ATA haplotype of the promoter region for interleukin 10, a cytokine which can downregulate cell-mediated T-cell response, compared to only 38% of 400 blood donors (9). This is in coherence with previous knowledge that zoster primarily infects patients with impaired cell mediated immunity.

Currently the viral tropism of VZV is uncertain. (10) It is known that primary infection can be caused by inoculation of the respiratory mucosa, however it is not known how the virus transfers to the skin and sensory ganglia. Pre-clinical studies have shown. That T-cells may be infected and serve as the link between mucosa to skin and ganglia (11, 12). This possibly partly explains why patients with HIV and other cell mediated immunodeficiencies where T-cells are disturbed are at risk for zoster infections.

A meta-analysis done by Schmidt et al identified 46 published papers that studied the association between herpes zoster and cancer, 10 of which looked at all cancer types combined (13). They found an absolute risk of 0.7 - 1.8% for any cancer at one year after presentation with herpes zoster. The study supports an association between herpes zoster and occult cancer however the low absolute risk of cancer limits the clinical implications.

To summarize, here we report a case of herpes zoster presenting as bullous impetigo in a previously healthy immunocompetent female and discuss relevant follow up and if screening is indicated. Conclusively, little studies have been made on follow up and screening programs after herpes zoster, it seems screening is not indicated for cancer, however in areas with high HIV prevalence zoster can imply an underlying HIV infection.

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