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Atypical presentation of herpes zoster in a case with acute myeloblastic leukemia

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ABSTRACT

Herpes zoster (HZ) is often associated with painful erythematous vesicular eruptions of the skin or mucous membranes. Approximately 10% to 30% of the population will suffer from HZ during their lifetime. HZ is infrequent in healthy children. However, diminished cellular immunity seems to increase risk of reactivation because incidence increases with age and in immunocompromised states. We report a 7-year-old girl with acute myeloblastic leukemia HZ infection on the right palmar, elbow and forearm region (C7, C8 and T1 dermatomes). We want to indicate unusual localization of HZ on the acute myeloblastic leukemia child patient.

1. Introduction

Herpes zoster (HZ) virus usually causes chickenpox in childhood. HZ is a common viral infection of the nervous system. HZ occurs in up to 20% of people infected with varicella-zoster virus. HZ rarely occurs in healthy children, but may occur frequently and may take a complicated course in children receiving chemotherapy[1]. The characteristic feature of all herpes viruses is that the virus stays latent or dormant in the sensory root ganglia and then reactivates to produce recurrent diseases[1].

During the latent period, the virus infects both neuronal and satellite neuroglial cells, which are the most abundant glial cells in the peripheral nervous system[1]. The virus travels to the skin transaxonally through the sensory roots causing the characteristic vesicular rash within the corresponding dermatome(s)[2].

The clinical picture can involve any dermatome, but the preferred sites are the thoracic or lumbar nerve segments and the distribution area of the trigeminal nerve[3]. It may also spread to involve the nerve roots adjacent to the dorsal root ganglia causing a plexitis or neuritis.

The ability of this virus to spread and infect various neuronal cells explains the diversity of its clinical manifestations. Viral reactivation may occur reduced cell-mediated immunity to HZ occurs with aging, explaining the increased incidence in the elderly and from other causes such as tumors, human immunodeficiency virus (HIV) and immunosuppressant drugs. Diagnosis is usually clinical from typical unilateral dermatomal pain and rash[4].

We want to indicate unusual localization of HZ on the acute myeloblastic leukemia child patient.

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2. Case report

In this article, we report a case of HZ located on the right palmar, elbow and forearm region (C7, C8 and T1 dermatomes) in a child with acute myeloblastic leukemia (AML) because of unusual localization. A 7-year-old girl followed with the diagnosis of AML M3 for 32 months was admitted with the complaints of swallow, burning sensation, grouped vesicles on an erythematous base, pain on the right palmar, elbow and forearm and fever for 2 days. On personal history, she suffered from chickenpox 3 years ago. She was administered AML M3 AIDA continuous protocol 6-mercaptopurine 90 mg/(m^2·d) and methotrexate 15 mg/(m^2·week). Now, she is on the 94th week before development of vesicular rashes.

On physical examination, vesicular rashes were noted the right on C7, C8 and T1 dermatomes (Figure 1). The reminder findings of physical examination were normal.

Figure 1. Grouped vesicles with erythematous base on the right palmar, elbow and froearm region (C7, C8 and T1 dermatomes).

Laboratory findings were as follows: Hemoglobin 12 g/dL, white blood cell count 2 000/mm^3, platelet count 29 000/mm^3, sedimentation rate 40 mm/h and C-reactive protein 3.2 mg/dL. Anti-HIV and Varicella-Zoster virus (VZV) immunoglobulin M were negative. Unfortunately, VZV immunoglobulin G could not be analyzed. Intravenous acyclovir (15 mg/kg 3 times daily) for 7 days and paracetamol (15 mg/kg 4 times daily per oral) were administrated on the 4th of treatment, her pain relieved and the vesicular lesions were faded. The patient was discharged on the 8th day of hospitalization in good health.

3. Discussion

HZ infection is usually associated with vesicular eruptions of the skin or mucous membranes. From birth to age 9 years, annual incidence is 0.74 cases per 1 000 population in USA[2]. Usually a single, unilateral dermatome is involved[2]. HZ is more likely to occur in situations such as malignancy, steroid therapy, and old age when the immune system is depressed, thus allowing reactivation of the herpes virus[5]. HZ was seen only 2%-3% of patients with leukemia or after bone marrow transplantation[6]. Our patient was a child with acute myeloblastic leukemia.

The characteristic changes in infected cells are 'ballooning degeneration' with the formation of intranuclear inclusion bodies and multinucleated giant cells. Viral particles within neurons and satellite cells in the sensory ganglia or in peripheral sensory nerves were demonstrated early in the disease via electron microscopy or fluorescent antibody staining[7].

During primary infection, the etiological agent has the ability to infect the sensory nerves in mucocutaneous sites and then to become latent in the sensory-nerve ganglia[8]. In most patients, a painful eruption of grouped vesicles on an erythematous base develops within a sensory dermatome, usually on the trunk. In some cases, the face, neck, scalp or an extremity may be involved[8]. The cutaneous eruption typically involves a single dermatome and rarely crosses the midline.

Trunk from T3 to L2 are most frequently affected; the thoracic region alone accounts for more than one-half of all reported cases, and lesions rarely occur distal to the elbows or knees[9]. In immunocompromised patients, particularly in those affected by cellular immunodeficiency, HZ can be clinically more severe, with atypical presentations at the immunosuppressed patient[10]. In accordance with the literature, the patient with AML of HZ, which was localized in C7, C8 and T1 dermatomes, was an immunocompromised patient and she showed atypical presentation.

The diagnosis of HZ is usually made based on unilateral pain in a defined area accompanied by a typical rash in the dermatomal distribution of a segmental nerve[4]. Antiviral agents have been shown to decrease the duration of HZ rash and the severity of pain associated with the rash. Antiviral agents inhibiting DNA polymerase such as acyclovir, valacyclovir and famciclovir should be used in HZ.

However, these agents are effective within 72 h after the onset of the rash if patients have severe or moderate pain or severe or moderate rash or they are immonocompromised or suffer from HZ ophtalmicus[10]. A paracetamol and weak opioid (codeine) compound is effective in many patients. Stronger opioids (dihydrocodeine, tramadol, and morphine) are

sometimes required[4]. In our patient, we initiated parenteral acyclovir, paracetamol and antiseptic solution (mixture with rivanol) 48 h after the appearance of HZ lesions and in the fourth of treatment, her pain relieved.

In conclusion, our patient showed that lesions of HZ might be atypically localized on ulnar nerve in immunocompromised patients and acyclovir therapy was useful to relieve of the symptoms.

Conflict of interest statement

We declare that we have no conflict of interest.

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