

Herpes Zoster Infection of the Vulva

VULVANIN HERPES ZOSTER ENFEKSİYONU

Hakan KARALÖK, MD,^a Ebru A.KARALÖK, MD,^a Cihangir UZUNÇAKMAK, MD,^a Kadir SAVAN, MD^a

^aClinic of Obstetrics and Gynecology, Bakırköy Dr. Sadi Konuk Research Hospital, İSTANBUL

Abstract

Herpes zoster infection is caused by a reactivation of the Varicella zoster virus (VZV) which is characterised by localised, painful and blistering lesions along a dermatomal distribution. After primary infection the VZV lies dormant state in the sensory nervous system. Sacral dermatomal zoster is an extremely rare form of the herpes zoster infection. In the literature, some individual cases of recognised vulvar and anogenital infection with VZV have been described in adults. This case report describes this relatively uncommon presentation of herpes zoster infection which affected a 68 year old female patient and presents a review of the literature. After 7 days, 5x800 mg orally acyclovir treatment, the patient recovered without any complication.

Key Words: Herpes zoster, herpes genitalis

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Özet

Herpes zoster, Varicella zoster virüsünün (VZV) reaktivasyonu ile oluşan, dermatomal dağılımlı, lokalize ve ağrılı veziküler lezyonlarla karakterize bir enfeksiyondur. VZV, primer enfeksiyondan sonra duyu sinirlerinde latent fazda kalır. Herpes zoster enfeksiyonlarında sakral dermatomal tutulum oldukça nadir görülmektedir. Literatürde, erişkinlerdeki vulvar ve anogenital VZV enfeksiyonu ile ilgili az sayıda olgu bildirilmiştir. 68 yaşında bir kadın olguda saptanan ve klinikte nadir görülen yerleşimli genital herpes zoster enfeksiyonu nadir görüldüğü için bu yazıda sunulmuştur. Yedi gün süreli 5 x 800 mg oral asiklovir tedavisi sonrasında olgumuz herhangi bir komplikasyon gelişmeden iyileşmiştir.

Anahtar Kelimeler: Herpes zoster, genital herpes

Varicella zoster Virus (VZV), a neurotropic human herpes virus causes chicken pox and then remains dormant state for decades in the nerve cells.¹ The virus gets reactivated after a variable period of time usually ranging from 5-40 years in 15% patients and causes herpes zoster infection.^{2,3} Herpes zoster is an acute, localized infection with VZV, which causes painful, blistering and dermatomal eruptions. In a study conducted in 2005, the overall incidence of herpes zoster in Europe was found to be 3 per 1000 people per year and more than 10 per 1000 people per

year in those aged > 80 years.⁴ In another study, Fleming et al.⁵ have shown that the incidence of herpes zoster was greater in the females than males. The typical blistering eruptions appear in 2 to 3 days, after the virus has reached the skin. Lesions typically appear along a single dermatome and are unilateral. Thoracic dermatomes are affected in 70% of cases, followed by lumbar with 17% and facial with 2% but lower sacral dermatomal zoster is an extremely rare form of the herpes zoster infection.^{6,7} In the literature, some individual cases of recognised vulvar and anogenital infection with VZV have been described in adults, and cases of genital infection with this agent in children have also been reported.^{8,9} This case report describes this relatively uncommon presentation of herpes zoster infection which affected a 68 year old female patient and presents a review of the literature.

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Yazışma Adresi/Correspondence: Hakan KARALÖK, MD
Bakırköy Dr. Sadi Konuk Research Hospital
Clinic of Obstetrics and Gynecology, İSTANBUL
hakankaralok@yahoo.com

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Case Report

A 68-year-old female presented with pain and a group of papulovesicular lesions along the dermatomes of S2 and S4 of the left side of her vulva. The first symptoms of patient were severe pain and local burning around the vulva and perineal area. On examination, there were numerous vesicular lesions involving the entire of left vulvar region with mainly left sacral distribution without any palpable inguinal lymph node (Figure 1). These lesions appeared to be consistent with herpes zoster. Systemic examination didn't reveal any abnormality: the temperature, vital signs and abdominopelvic ultrasound of the patient were normal. Complete blood counting, urine analysis, fasting blood sugar, liver function tests were all within normal limits. Hepatitis B surface antigen, Anti Hepatitis C virus antibodies, VDRL (Venereal Diseases Reference Laboratory), Anti Human Immunodeficiency virus antibodies and Anti-HSV (Herpes simplex Virus) antibodies were all negative. The clinical appearance and dermatomal distribution of the lesions, clinical course of the patient and the substantial increase in the complement-fixing antibody titer in the convalescent serum samples are highly correlated with herpes zoster. For this diagnosis; the patient had undergone to acyclovir treatment (800 mg orally 5 times a day for a period of 7 days) and symptomatic



Figure 1. Left vulvar genital herpes zoster lesions.

treatment for the relieving local pain. On the dermatologic examination, the lesions remained confined to the involved dermatomes only and no local or systemic complications developed during her treatment period. Within 72 hours, her pain was markedly regressed. After the treatment, the cutaneous lesions started to heal gradually in a period of two weeks and the patient recovered without any complication like postherpetic neuralgia in an observation period of 6 months.

Discussion

Herpes zoster infection is caused by a reactivation of the VZV which is characterised by localised painful and blistering lesions along a dermatomal distribution. Patients usually feel quite unwell with fever and headache. The lymph nodes draining the affected area are often enlarged and tender. Patients are infectious, both from virus in the lesions and in some instances the nose and throat. In uncomplicated cases, recovery is complete in 2-4 weeks. The principal challenge in the management of infection is the rapid resolution of pain and prompt management with antiviral agents. Because of zoster is often a self-limiting illness, antiviral therapy is only used for shortening the duration of the illness and limiting long term sequelae. Valaciclovir and famciclovir are significantly superior to aciclovir in reducing the incidence and duration of complications.¹⁰ The window of time for starting acyclovir-like medications is up to 72 hours after onset of pain, and preferably before the appearance of the lesions.¹¹ In our case, the patient had promptly undergone 5x800 mg/daily acyclovir and symptomatic treatment. We used orally acyclovir treatment because of a possibility for change the medication with intravenous forms in severe cases.

But within 72 hours, our patient's lesions and pain were gradually regressed and at the least in a period of two weeks, she was recovered without any complication like postherpetic neuralgia in an observation period of 6 months.

In clinical practice, some elderly patients need a corticosteroid to reduce the risk of postherpetic

neuralgia which is persistence of pain that may last from months to years following the initial episode. This complication, is thought to be the result of virus-induced damage to the affected sensory nerve ganglion. In a randomized, controlled trial done in the UK, the prevalence of postherpetic neuralgia in patients over age 60 in the placebo arm was 61% at 1 month, 24 % at 3 months and 13% at 6 months. The rate at 6 months for such patients was 35% in another study.¹² Overall, the rate of postherpetic neuralgia is said to be 20%.¹¹ In addition, repeated and disseminated zoster eruptions may indicate an underlying problem with the impaired cellular immunity such as leukemia, Hodgkin's disease and other cancers, chemotherapy or HIV infection.¹³⁻¹⁶ In our case, after all of the clinical and laboratory investigations like abdominopelvic ultrasound, serological markers for malignancies and Anti Human Immunodeficiency virus antibodies test, we didn't find any immunosuppressive reason that could explain genital zoster infection.

Consequently, like our case, diagnosis of herpes zoster infection is often made on the clinical appearance of the lesions. Serologic testing is useful to confirm past infection with VZV.¹⁷ Although, distinction of VZV associated genital infection from that caused by HSV-1 or HSV-2 is important in clinical practice. Because VZV genital infections are not as common as those associated with HSV infection, and recurrences would not be expected, at least in immunocompetent individuals, their clinical significance and potential for sexual transmission may be diminished.¹⁸ Additionally, when the lesions caused by VZV, higher doses of famciclovir or valaciclovir treatment are required for effective VZV therapy compared to those required for treatment of HSV infection.¹⁷

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