

Oral Herpes Zoster

Daniel Polvino, MD*, Grant Wei, MD*, Christopher Bryczkowski, MD*

*Rutgers Robert Wood Johnson Medical School, Department of Emergency Medicine, New Brunswick, NJ

Correspondence should be addressed to Grant Wei, MD at weigr@rwjms.rutgers.edu

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History of present illness: A 65-year-old man presented to the emergency department with a chief complaint of progressively worsening oral pain for three days. The patient had previously seen his dentist three days prior when the pain began and no lesions or source of pain could be identified. Over the past 24 hours, the patient noticed the development of painful lesions in his mouth. He reported decreased intake of solids and liquids due to pain. He denied a history of past rashes or any other lesions on his body.

Significant findings: Physical exam findings revealed vesicular lesions on the lip, hard and soft palates which did not cross the midline. The lesions appeared in the distribution of the maxillary branch (V2) of the trigeminal nerve, consistent with herpes zoster.

Discussion: Herpes zoster, commonly known as shingles, is a frequent reason for patients to present to the emergency department or clinic. The prodrome typically involves pain and tingling before the eruption of grouped vesicles in a dermatomal distribution and can be accompanied by fever, headache and malaise. It is estimated that 30% of Americans will experience at least one episode of herpes zoster in their lifetime. While the disease is typically not life-threatening, it can cause significant morbidity.¹

Herpes zoster is due to reactivation of the varicella zoster virus, which causes chicken pox and remains dormant in the dorsal root ganglion. The incidence of the disease increases with age, with the majority of cases presenting after the age of 50.² Risk factors include increasing age, hospitalization, co-morbid conditions and cutaneous or mucosal trauma. One study found that those with craniofacial herpes zoster were twenty-five times as likely to have had facial trauma in the week prior compared to controls.³

Diagnosis is often clinical, requiring no additional lab tests or imaging. A Tzanck smear may be performed to identify multinucleated giant cells. The differential diagnosis for oral lesions includes herpes zoster, primary syphilis, aphthous stomatitis and thermal burns.⁴ Further diagnostic testing, such as direct fluorescent antibody testing, polymerase chain reaction (PCR) and viral culture may aid in the diagnosis but are frequently not used in the clinical setting.

The treatment of herpes zoster is antiretrovirals and pain control. If the lesions are identified within 72 hours, strong evidence exists for the use of acyclovir or one of its derivatives to reduce pain, promote healing, and prevent new lesion formation.⁵ Acyclovir requires more frequent dosing due to poor gastrointestinal absorption compared to valacyclovir. Studies have also shown a modest improvement in the resolution of symptoms and prevention of post-herpetic neuralgia with the use of valacyclovir.⁶ Non-steroidal anti-inflammatories are often adequate for pain control. Early studies found a small benefit with the addition of corticosteroids in preventing post-herpetic neuralgia, but a recent meta-analysis found no difference in the occurrence of postherpetic neuralgia in patients receiving corticosteroids.⁷

Transmission is by direct contact with lesions. Patients with oral lesions should be instructed to avoid oral contact with others including kissing and sharing of food or beverage. Patients should be expressly advised to avoid immunocompromised or pregnant contacts. The virus is no longer transmissible once the lesions are crusted over.⁸ Patients should be instructed to follow up with their primary care provider and dermatology if the eruption is severe and to return to the ED if they have any visual changes or spreading of lesions.

The patient was treated with intramuscular ketorolac and given a prescription for valacyclovir. The patient was referred to the dermatology clinic but on review of records did not seek follow up care.

Topics: Oral lesions, herpes zoster, shingles.

References:

1. Yawn BP, Saddier P, Wollan PC, St Sauver JL, Kurland MJ, Sy LS. A population-based study of the incidence and complication rates of herpes zoster before zoster vaccine introduction. *Mayo Clin Proc.* 2007;82(11):1341-1349.
2. Schmader K. Herpes zoster in older adults. *Clin Infect Dis.* 2001;32(10):1481-1486.
3. Zhang JX, Joesoef RM, Bialek S, Wang C, Harpaz R. Association of physical trauma with risk of herpes zoster among medicare beneficiaries in the United States. *J Infect Dis.* 2013;207(6):1007-1011. doi: 10.1093/infdis/jis937.
4. Hairston BR, Bruce AJ, Rogers RS III. Viral diseases of the oral mucosa. *Dermatol Clin.* 2003;21(1):17-32.
5. Wood MJ, Kay R, Dworkin RH, Soong SJ, Whitley RJ. Oral acyclovir therapy accelerates pain resolution in patients with herpes zoster: a meta-analysis of placebo-controlled trials. *Clin Infect Dis.* 1996;22(2):341-347.

6. Beutner KR, Friedman DJ, Forszpaniak C, Andersen PL, Wood MJ. Valaciclovir compared with acyclovir for improved therapy for herpes zoster in immunocompetent adults. *Antimicrob Agents Chemother.* 1995;39(7):1546-1553.
7. Han Y, Zhang J, Chen J, He L, Zhou M, Zhu C. Corticosteroids for preventing postherpetic neuralgia. *Cochrane Database Syst Rev.* 2013;(3):CD005582. doi: 10.1002/14651858.CD005582.pub4.
8. Centers for Disease Control and Prevention. Shingles (Herpes Zoster). CDC.gov. <https://www.cdc.gov/shingles/about/transmission.html>. Accessed 24 July 2018.