CLINICAL VIGNETTE

Herpes Zoster Infection Following a Cosmetic Facial Procedure

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Introduction

Varicella zoster virus (VZV) infection is associated with two clinical entities: Primary varicella infection, also known as chickenpox, typically occurs in childhood and is associated with a vesicular rash primarily involving the face and trunk. Subsequent reactivation of latent VZV results in a dermatomal vesicular rash, known as herpes zoster or shingles.[1] Age is the most important risk factor for herpes zoster infection, with the majority of cases occurring in patients over 50 years of age.[2] In addition, immunocompromised patients, including patients with hematopoietic stem cell and solid organ transplants, are at increased risk.[1] Some studies report physical trauma associated with herpes zoster infection. One age-matched, case-control study using Medicare data found herpes zoster patients were 3.4 times more likely to have experienced physical trauma in the week prior, compared to controls.[3] Herpes zoster has also been reported following invasive medical procedures.[4,5] However, the relationship between herpes zoster and noninvasive medical procedures remains unclear.

We present a case of herpes zoster infection following a recent cosmetic facial procedure in a young, immunocompetent patient.

Case

A 32-year-old woman with no significant medical history presented to the emergency room with progressive right facial rash. One week earlier, she had undergone a cosmetic facial procedure which included massage and microcurrent therapy. Over subsequent days, she developed erythematous, tender papules on the right cheek which progressed to purulent vesicles, associated with a burning sensation. She had initially presented to urgent care where she was diagnosed with a hypersensitivity reaction and prescribed corticosteroids. However, the rash progressed despite this treatment. She had no chronic illness and was not taking medications. Vitals were unremarkable. Skin examination was notable for numerous grouped vesicles and bullae with purulent drainage in the distribution of the right maxillary nerve. Laboratory studies included a white blood cell count of 8.74 $x10^3$ cells/uL, hemoglobin of 13.8 $x10^3$ cells/uL, platelet count of 235 $x10^3$ cells/uL. Bacterial cultures of the vesicular fluid demonstrated few Propionibacterium acnes and rare Propionibacterium granulosum. Polymerase chain reaction of the vesicular fluid detected VZV. The patient was initiated on oral antiviral therapy and antibiotic therapy. At one-month follow-up, she reported resolution of the facial lesions but continued to experience localized neuropathic pain.

Discussion

This young immunocompetent patient developed herpes zoster infection following a cosmetic facial procedure. The majority of herpes zoster cases occur in older patients, with less than 20 percent of cases occurred in patients less than 40 years of age.[2] In younger, immunocompetent patients, herpes zoster infection may be misdiagnosed and mistreated, as occurred in our patient, who was initially diagnosed with a hypersensitivity reaction. Although molecular testing including PCR assays may be available to identify VZV in fluid or tissue samples, herpes zoster is ultimately a clinical diagnosis.

The association between herpes zoster infection and physical trauma has been established in observational studies.[3,5,6] However, these studies evaluated the association of herpes zoster infection with serious inadvertent physical trauma resulting in bruising or musculoskeletal injury. Herpes zoster infection has been reported following invasive cosmetic procedures, such as rhinoplasty or liposuction.[5,7] However, there are few reports of herpes zoster infection following elective, noninvasive procedures, in our patient. The pathogenesis of VZV reactivation in the setting of physical trauma remains unclear, although it has been hypothesized that physical manipulation of regional sensory ganglia may result in transient release of VZV to the sensory nerves.[3] In addition, physical trauma or local inflammation may disrupt cell-mediated immunity in the direct vicinity of the trauma, enabling propagation of the VZV reactivation.[3]

Oral antiviral therapy, such as acyclovir and valacyclovir, is the mainstay of treatment for herpes zoster infection. Treatment reduces duration of cutaneous lesions and pain, as well as reduced incidence of post-herpetic neuralgia at 3 to 6 months.[4,8] Treatment is most effective when initiated within 72 hours of onset of the rash. Thus, timely recognition and initiation of empiric treatment is important. The addition of corticosteroids to reduce inflammation and pain associated with herpes zoster infection is controversial, particularly in the postsurgical

patients. Our patient used corticosteroids prior to her emergency room presentation which did not improve her symptoms and may have contributed to the progression of her rash.[9]

Conclusion

Risk factors for herpes zoster infection include older age and immunocompromised status. However, severe cases may occur in young, immunocompetent patients. Clinicians should maintain a high index of suspicion in patients presenting with clinical features of herpes zoster and consider sending VZV PCR analysis of lesions to confirm the diagnosis. The association between physical trauma and herpes zoster has also been previously established. Our patient developed herpes zoster following an elective, noninvasive cosmetic procedure. Treatment with oral antiviral therapy facilities resolution of the zoster infection and mitigates risk of post-herpetic neuralgia.

REFERENCES

1. **Kennedy PGE, Gershon AA**. Clinical Features of Varicella-Zoster Virus Infection. *Viruses*. 2018 Nov 2;10(11):609. doi: 10.3390/v10110609. PMID: 30400213; PMCID: PMC6266119.
2. **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 Nov;82(11):1341-9. doi: 10.4065/82.11.1341. Erratum in: Mayo Clin Proc. 2008 Feb;83(2):255. PMID: 17976353.
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 Mar 15;207(6):1007-11. doi: 10.1093/infdis/jis937. Epub 2013 Jan 10. PMID: 23307932.
4. **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 Feb;22(2):341-7. doi: 10.1093/clinids/22.2.341. PMID: 8838194.
5. **Celik H, Hanci M, Karacaoglan N**. Facial Herpes Zoster Following Rhinoplasty: A Rare Complication. *Aesthet Surg J*. 2019 Jan 1;39(1):NP1-NP3. doi: 10.1093/asj/sjx225. PMID: 29340617.
6. **Thomas SL, Wheeler JG, Hall AJ**. Case-control study of the effect of mechanical trauma on the risk of herpes zoster. *BMJ*. 2004 Feb 21;328(7437):439. doi: 10.1136/bmj.37991.511829.F7. Epub 2004 Jan 23. PMID: 14742350; PMCID: PMC344263.
7. **Andrews TR, Perdikis G, Shack RB**. Herpes zoster as a rare complication of liposuction. *Plast Reconstr Surg*. 2004 May;113(6):1838-40. doi: 10.1097/01.prs.0000117663.06099.a0. PMID: 15114155.
8. **Jackson JL, Gibbons R, Meyer G, Inouye L**. The effect of treating herpes zoster with oral acyclovir in preventing postherpetic neuralgia. A meta-analysis. *Arch Intern Med*. 1997 Apr 28;157(8):909-12. PMID: 9129551.
9. **Gan EY, Tian EA, Tey HL**. Management of herpes zoster and post-herpetic neuralgia. *Am J Clin Dermatol*. 2013 Apr;14(2):77-85. doi: 10.1007/s40257-013-0011-2. PMID: 23456596.