Case Report

Reactivation of herpes simplex labialis following adult spine deformity correction surgery

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ABSTRACT

Background: A depressed host defense is a major contributor to the oral shedding of herpes simplex virus (HSV) type 1. Here, we present an instance in which herpes simplex labialis was reactivated following major spinal deformity surgery.

Case Description: A 59-year-old female underwent spinal deformity correction for lumbar degenerative scoliosis. On postoperative days 2–3, she presented with pyrexia (38°C) and tachycardia (94/min); by day 5 she had multiple ulcers around her lips and was HSV IgG positive. She had a remote history of herpes simplex I infection 7 years previously. Once started on oral acyclovir, the lesions improved, and by day 15 postoperative, her pyrexia and all lesions completely resolved.

Conclusion: HSV-1 should be suspected in patients with a previous history of HSV and postoperative pyrexia. Adequate prophylactic administration of acyclovir should result in resolution of these outbreaks, in this case, attributed to overly extensive spinal deformity surgery.

Keywords: Herpes labialis, Herpes simplex virus, Spine deformity surgery

INTRODUCTION

Herpes simplex virus (HSV) type 1 is a latent infection of the dorsal root ganglia. The seroprevalence of HSV is high, occurring in 73% of individuals aged 12 years and older (i.e., demonstrating antibodies to at least one type of HSV). In HSV, the T-cell immune function plays a major role in maintaining the virus in its latent state. Notably, the major risk for VZV or HSV reactivation is immunosuppression. Here, we present a 59-year-old female, whose transient immunosuppression following overly extensive lumbar deformity surgery developed a postoperative outbreak of oral HSV warranting treatment with acyclovir.

CASE REPORT

A 59-year-old female underwent lumbar surgery for degenerative scoliosis. Stage I involved L1-L2, L2-L3, L3-L4, and L4-L5 oblique lateral interbody fusions and a L5-S1 anterior lumbar interbody fusion. Stage II included posterior fixation from L1 to the pelvis [Figure 1].

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Two to three days following these overly extensive procedures (i.e., Stages I and II), she presented with pyrexia (38°C) and tachycardia (94/min); on the 5th postoperative day, she developed multiple ulcers around the lips. As she had a history of herpes simplex I infection 7 years ago, and the antigen tests were positive for HSV IgG, she received a 15-day course of oral acyclovir. During this period, her pyrexia gradually resolved, and her oral lesions improved [Figure 2].

DISCUSSION

Reactivation of herpes simplex labialis type 1 (HSV) after major spine deformity surgery is rare. In this case, it was likely attributed to transient immunosuppression secondary to overly extensive spinal surgery. Stage I was four-level oblique lumbar interbody fusions (L1-L5) and L5-S1 anterior lumbar interbody fusion. Stage II was posterior pedicle screw instrumentation (L1-S2) and sacroiliac joint fusion using bedrock technique.

Etiology of HSV-1 postoperative spine infection

The massive stress associated with such overly extensive spinal deformity procedures contribute to her HSV-1 outbreak. This was associated with oral ulceration and symptoms/signs of neural hyperactivity characterized by anxiety, tachycardia, shallow breathing, and hypoxia. It is critical to identify and treat HSV-1 infections in postoperative patients, as this case, as HSV-1 may cause necrotizing encephalitis [2] [Table 1].

Early antiviral therapy with acyclovir for HSV-1

Early antiviral therapy with acyclovir significantly reduces morbidity and mortality due to HSV encephalitis. To prevent relapse, a typical course of treatment lasts 21 days. In our patient, the treatment was stopped after 15 days when the symptoms resolved. Porteous et al. [5] report on the reactivation of HSV-1 in 41% of the 44 of the critically ill postsurgical patients in his series. Although the HSV oral shedding was not related to the patients’ surgical outcomes, they these patients exhibited higher mortality rates due to suppressed humoral/cell mediated immunity.

Consideration of prophylaxis with acyclovir in patients with histories of oral HSV-1

Given the strong correlation between surgical procedures and HSV infections, prophylactic use of acyclovir in patients...
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with a history of HSV orolabial ulcers should be considered routinely.\(^4\)

**CONCLUSION**

Patients undergoing major spinal surgery (i.e., some overly extensive) may become transiently immunosuppressed, resulting reactivation of HSV-1. For patients, with documented postoperative HSV-1, a course of acyclovir should be administered; only select patients considered at increased risk for postoperative HSV-1 should prophylactically receive this therapy.

**Declaration of patient consent**

Patient’s consent not required as patients identity is not disclosed or compromised.

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**Conflicts of interest**

There are no conflicts of interest.

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**Table 1: Epidemiology, clinical manifestations, diagnosis, and treatment of HSV type 1.**

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| **Epidemiology** | Infection occurs equally between the sexes and without seasonal variation. The prevalence is highest in low- and middle-income countries. |
| **Transmission** | Oral-oral, oral-genital, or genital-genital contact |
| **Clinical manifestations** | Gingivostomatitis and pharyngitis |
| | Herpes labialis is the most frequent sign of reactivation disease |
| | Genital ulcerations and tender lymphadenopathy |
| | Herpetic whitlow, herpes gladiatorum, erythema multiforme, and eczema herpeticum |
| | Keratitis and acute retinal necrosis |
| | Severe disease (e.g., encephalitis, meningitis, hepatitis, respiratory tract infections, and esophagitis) |
| **Risk factors** | HIV infection, malignancy, organ transplantation, malnutrition, burn and skin disorders, and pregnancy |
| **Diagnosis** | DNA detection through polymerase chain reaction or culture of the virus |
| | Direct fluorescent antibody testing or Tzanck smears are less sensitive and less specific |
| **Treatment** | Antiviral agents for HSV infection include acyclovir, valacyclovir, and famciclovir; metabolites of these nucleoside derivatives interfere with the synthesis of viral DNA and are well tolerated. |

HSV: Herpes simplex virus

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