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CASE REPORT | ESOPHAGUS

Varicella Zoster Virus Esophagitis in an Immunocompetent Patient

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ABSTRACT

The varicella zoster virus is highly infectious, spreading via direct contact or respiratory droplets, and can lead to life-threatening complications. Although disseminated disease tends to occur most commonly in immunocompromised patients, we present a peculiar case of varicella zoster esophagitis in a healthy, immunocompetent adult. To provide prompt treatment, clinicians should be aware that the varicella zoster virus could cause severe esophagitis, even in immunocompetent patients.

INTRODUCTION

The varicella zoster virus (VZV) is highly infectious, spreading via direct contact and respiratory droplets. In rare cases, VZV infections can result in life-threatening complications including hepatitis, pneumonitis, and encephalitis.[1–4] Disseminated disease more commonly occurs in immunocompromised patients; however, we present a peculiar case of varicella esophagitis in a healthy, immunocompetent patient.

CASE PRESENTATION

A previously healthy 43-year-old Hispanic man presented to the emergency department with 4 days of progressively worsening odynophagia. Less than 2 weeks before presentation, he had visited an urgent care for shortness of breath and was prescribed azithromycin and 5 days of steroids for suspected bronchitis. Immediately after completing this treatment course, he began to experience significant odynophagia. He then developed anorexia, sialorrhea, and hematemesis, prompting another visit to the emergency department.

The patient reported that he was born in Argentina, which he had last visited within 6 months, and had traveled to Mexico in the past 3 months. His immunizations were reported to be up to date, and previous infection with chickenpox was unknown. His occupation was as a university professor, and he had notable exposure to cattle in his research. He reported 1 sexual partner in his lifetime (his wife) and denied sex with men or injection drug use and previous incarceration.

On arrival, he was tachypneic and febrile (maximum temperature of 38.1°C). His blood pressure was initially stable, and his oxygen saturation was 91% on ambient air. He later developed hypotension, which responded to fluid resuscitation. He was well-nourished, alert, and oriented, and in mild distress. His skin examination revealed a diffuse rash consisting of erythematous macules, papules, vesicles, and scabs in various stages of development from the forehead to the upper thigh area (Figure 1). Examination of the oral cavity was not completed because of significant trismus and pain with opening the mouth. His lungs had diffuse bilateral crackles with expiratory wheezing, worse on the right side.

Initial investigations showed leukocytosis. Human immunodeficiency virus antibodies were found to be negative. The patient underwent esophagogastroduodenoscopy on the first day of admission, which showed numerous severe ulcerations in the mid and distal esophagus, as well as gastritis and duodenitis (Figure 2). The center and margins of the lesions were biopsied for pathologic review. Hematoxylin and eosin staining demonstrated classic intranuclear inclusion bodies, and immunohistochemistry of the ulcerations in

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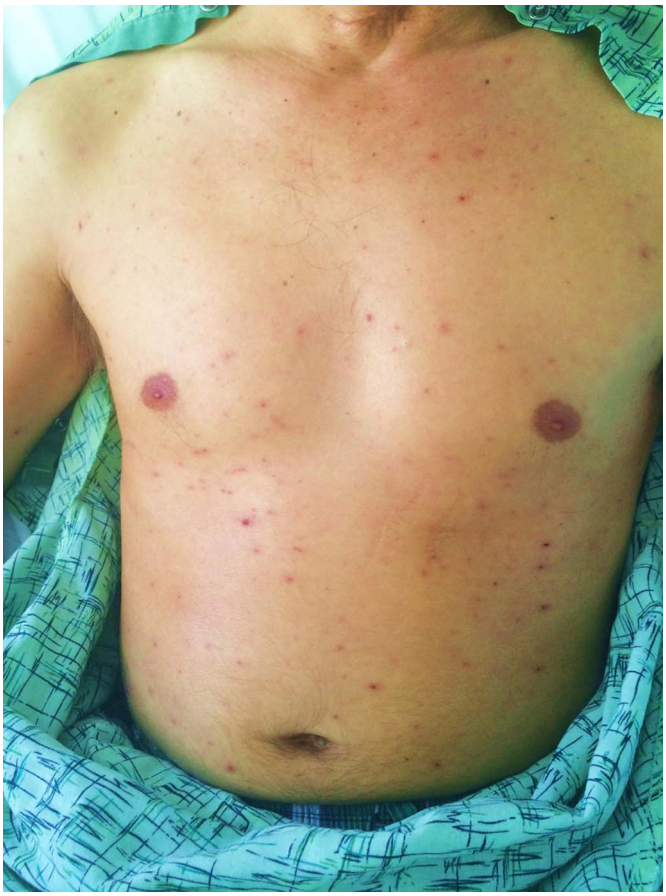

Figure 1. Presence of diffuse vesicular rash in various stages of development.

the esophagus was positive for VZV and negative for herpes simplex virus 1 and herpes simplex virus 2 (Figure 3). Serum varicella IgM levels were elevated, and varicella zoster by PCR was detected in the serum. Given his hypoxia and need for supplemental oxygen, chest computed tomography (CT) was performed, which revealed ground glass opacities in the right lung, worse in the right upper lobe and concerning for an infectious process. Abdominal CT was later performed during the hospital stay, and there was no concerning lymphadenopathy. His total serum IgG was also normal, ruling out an underlying immunocompromised state.

The patient was started on intravenous acyclovir on the first day of admission at 5 mg/kg every 8 hours. The patient was discharged from the hospital on day 15. On discharge, he was switched to oral valacyclovir 1,000 mg 3 times daily, completing a total of 24 days of antiviral therapy. He also completed 10 days of antimicrobial treatment for aspiration pneumonia. Symptomatic treatment was achieved with viscous oral lidocaine and pantoprazole. Chest follow-up CT 3 weeks after hospital discharge showed resolution of pneumonia. Follow-up esophagogastroduodenoscopy was perfomed 3 months after the initial presentation and showed resolution of the vesicular lesions.

DISCUSSION

A review of the literature yielded 15 cases of disseminated varicella zoster in immunocompetent patients. The review illustrated the variability in presenting symptoms of patients with disseminated VZV. Dissemination is defined as more than 20 vesicles outside the area of primary and adjacent dermatomes, but can be multidermatomal.[5,6] Common complications of VZV infection include pneumonitis and aseptic meningitis.[2,3,6–9] However, the rarity of esophagitis as a presenting sign of disseminated VZV must be highlighted because only 4 other cases have been reported.[10–12] Furthermore, only 2 of those cases involved immunocompetent patients, in which the classic VZV rash did not accompany the presentation of esophagitis—but did in this patient's case.[10,11]

The ethnicity of our patient must also be highlighted because there are several cases of disseminated VZV occurring in healthy Hispanic people.[2,10] Disseminated infection may be more prevalent in

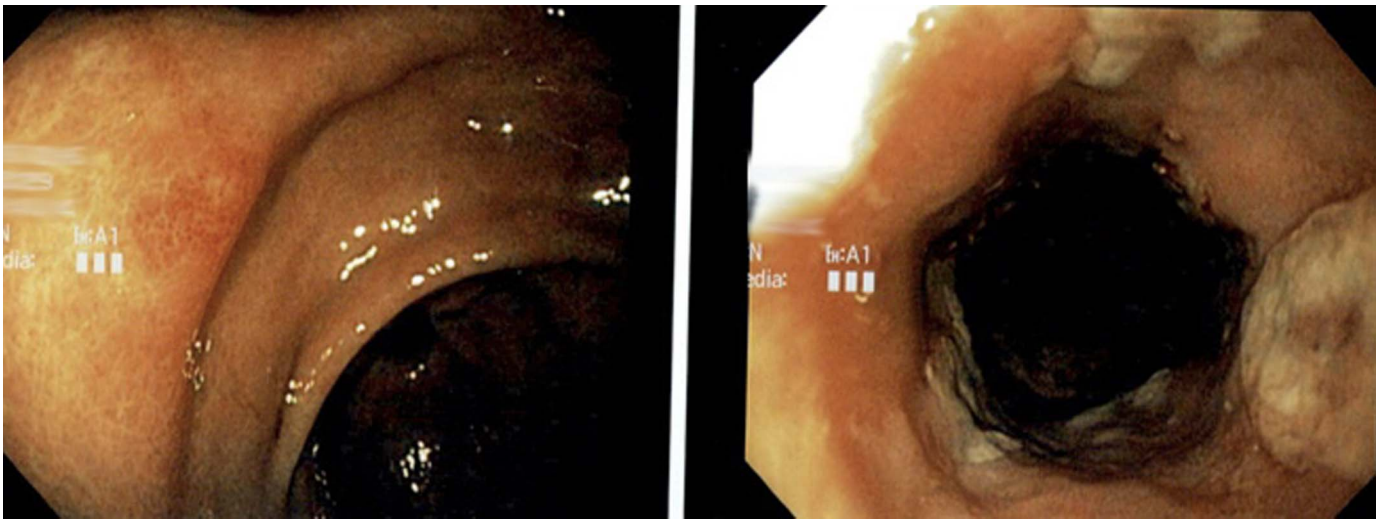

Figure 2. Esophagogastroduodenoscopy showing numerous lesions in the mid to distal esophagus.

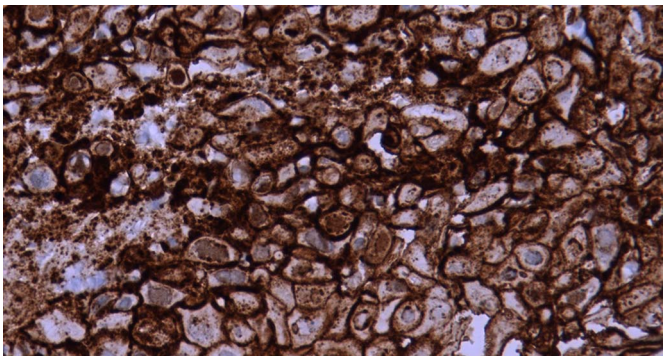

Figure 3. Biopsy of esophageal ulcers showing varicella zoster virus-positive cells (hematoxylin and eosin stain).

patient's immigrating from tropical countries because the virus may not be a common childhood infection in those countries, thus resulting in the infection occurring later in adulthood.[2] Furthermore, in developing countries, there may be no requirement to vaccinate children against VZV like there is in the United States.[13]

Primary VZV is spread via respiratory secretions, and then viral replication occurs in the lymphatic tissue, later becoming dormant in the nerve roots.[14] Any condition that may cause immunity to fade allows reactivation, which may explain why the elderly population, who experience a decline in immune function, tends to experience outbreaks of VZV.[4,5] However, in the case of this patient, neither an underlying immunocompromised state nor advanced age was present. It is also important to be mindful of recent corticosteroid use because there exist some data to support that the administration of steroids may increase the risk of severe or fatal varicella.[15] This emphasizes the complexity of making a prompt diagnosis when there is no apparent evidence of an underlying immunocompromised state.

Failure to initiate appropriate therapy could lead to progression to disseminated intravascular coagulation, acute respiratory distress syndrome, and death.[2,13,16] To provide prompt and appropriate treatment, clinicians should be alert to the VZV as a cause of severe esophagitis, even in immunocompetent patients. A delay in initiating antiviral therapy at the onset of clinical suspicion could lead to life-threatening or even fatal outcomes.[2,13,16]

DISCLOSURES

Author contributions: A. Shallal wrote the manuscript. M. Reaume, T. Tariq, and D. Garg edited the manuscript. P. Patel edited the manuscript and is the article guarantor.

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Informed patient consent was obtained for this case report.

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