Case Report Annals of Dermatology 2022;34(6):475-477 • https://doi.org/10.5021/ad.20.165

Dysphagia with Unilateral Vocal Cord Paralysis in Herpes Zoster: A Case Report

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Received June 17, 2020
Revised January 19, 2021
Accepted February 1, 2021

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Herpes zoster is caused by the varicella-zoster virus, which becomes latent in ganglia after primary infection. When the varicella-zoster virus reactivates on the cranial nerve, the patient can suffer from cranial nerve palsy, pain, and skin lesions on the head and neck area. A 57-year-old immunocompetent male presented with dysphagia lasting 10 days. Computed tomography and other neurological findings were normal. However, laryngoscopy showed right vocal cord paralysis, which might be the reason for dysphagia in this patient. There was a grouped crusted lesion on the right posterior auricular area that appeared 5 days after the dysphagia. After famciclovir and prednisolone combination therapy, the patient was cured with no sequelae. This is a rare case of herpes zoster in an immunocompetent patient who presented with dysphagia. In addition, it was difficult to make an accurate diagnosis because his skin lesion appeared several days after dysphagia.

Keywords: Dysphagia, Herpes zoster, Varicella zoster virus infection

INTRODUCTION

Herpes zoster is caused by the varicella-zoster virus (VZV), which becomes latent in ganglia after primary infection. Old age, psychological stress, malignancy, human immunodeficiency virus (HIV) infection, and other immunocompromised states might be risk factors for herpes zoster[1]. Skin lesions usually appear in one or two adjacent dermatomes, which present as grouped erythematous vesicles and pustules. Thoracic area (50%~60%) is the most common site; however, the cranial nerves are often involved[1]. Facial palsy, dizziness, hearing loss, dysphagia, and other neurological symptoms might occur when the cranial nerves are involved[2]. If a skin lesion appears after a neurological deficit, it is challenging to make an accurate diagnosis.

In this case report, a skin lesion appeared 5 days after the dysphagia, which made it difficult to diagnose. We finally diagnosed herpes zoster by a grouped crusted lesion on the patient's right postauricular area and the result of tissue VZV PCR.

CASE REPORT

A 57-year-old immunocompetent male suffered from dysphagia and hoarseness 5 days before the skin lesion. Before the patient visited our department of dermatology, he visited the gastroenterology and the otorhinolaryngology clinic to evaluate dysphagia and hoarseness. At the gastroenterology clinic, he underwent upper gastrointestinal endoscopy, but they could not find a specific reason for dysphagia. Additional laryngoscopy was performed for further evaluation in the otorhinolaryngology clinic. Right vocal cord paralysis was seen on the laryngoscopy (Fig. 1). On neurologic examination, there were no other abnormal findings. In addition, computed tomography for head and neck was performed, but there was no specific abnormal finding that can explain vocal cord paralysis. The patient finally came to our dermatology clinic because of a painful skin lesion that appeared 5 days after the dysphagia. Localized crusted papules were observed in his right postauricular area (Fig. 2). Skin biopsy and VZV PCR were performed on the skin lesion. Biopsy revealed epidermal necrosis and dense inflammatory cell infiltration. VZV PCR

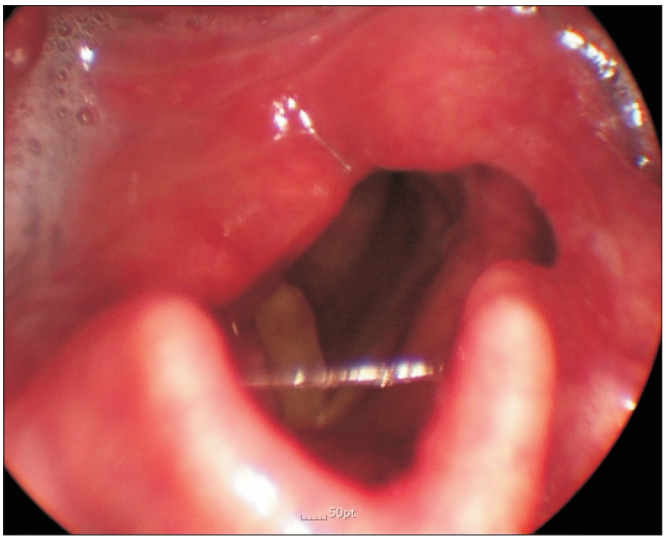

Fig. 1. Right vocal cord paralysis was seen on the laryngoscopy.

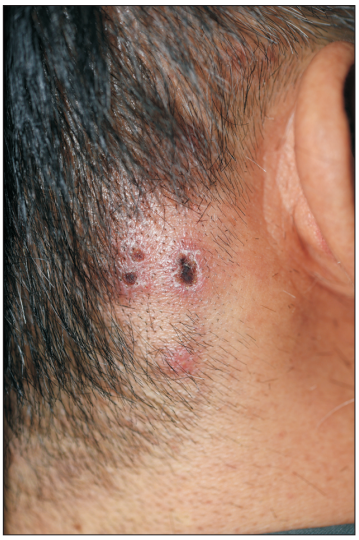

Fig. 2. Localized dark erythematous crusted papules were observed in the right postauricular area.

was positive in the biopsy tissue. Based on the clinical findings and PCR results, we finally diagnosed herpes zoster with vocal cord paralysis. The patient was treated with oral famciclovir (750 mg/day for 7 days) and oral prednisolone (20 mg/day for 3 days and 10 mg/day for another 8 days). After oral antiviral and prednisolone therapy, the skin lesion and dysphagia improved. In addition, on the follow-up laryngoscopy after a week, vocal cord palsy improved without any other sequelae.

We received the patient's consent form about publishing all photographic materials.

DISCUSSION

Herpes zoster is the most common form of VZV reactivation. VZV becomes latent in ganglia after the primary infection and reactivates when the host's immunity decreases[3]. The typical clinical manifestation of herpes zoster is characterized by a skin rash following the dermatome accompanied pain, paresthesia, and neurological deficit. It usually involves the thoracic (50%~60%), the cervical (10%~20%), and the trigeminal (10%~20%) area[1]. The ratio of patients with herpes zoster who develop cranial or peripheral nerve palsies is only 5%[4]. The trigeminal and facial nerves are commonly involved cranial nerves associated with VZV infection; however, lower cranial neuropathy is relatively rare[5].

VZV infection associated with dysphagia is not common[6]. However, dysphagia can occur in various circumstances. One example is Ramsay Hunt syndrome (RHS). RHS is the most common clinical manifestation of cranial neuropathy caused by VZV infection[5]. VZV reactivation occurs in the geniculate ganglion of the facial nerve, which leads to facial palsy, otalgia, and herpetic skin lesions on the auricle and the external auditory canal[2]. Several previous case reports showed that facial palsy and dysphagia develop due to multiple cranial neuropathies in RHS[2,7]. However, in this case, there was no sign of facial palsy, and the skin lesion appeared only in the postauricular area several days after dysphagia. Additionally, some other reports reported that dysphagia could occur with herpes zoster[5,6]. When VZV reactivates at the jugular foramen level, the glossopharyngeal, vagus, and accessory nerve injury can cause dysphagia[5] In addition, VZV infection in the cervical area could cause multiple or single cranial nerve palsy without any cutaneous lesion[6]. In this case, however, there was no other neurological deficit except dysphagia, and the herpetic lesion was limited to the postauricular area.

Unilateral vocal cord paralysis was found on our patient's laryngoscopy. Unilateral vocal cord paralysis is one of the reasons for dysphagia[8]. The vocal cord is regulated by the vagus nerve. When the vagus nerve is injured, unilateral vocal cord paralysis may occur[9]. Thus, VZV reactivation on the vagus nerve could be associated with isolated vocal cord paralysis[10]. If VZV reactivates on the vagus nerve, skin lesions might appear on the auricle and the external ear canal because of the auricular branch of the vagus nerve. In this case, it is interesting to note that the skin lesion was only limited to the post-

auricular area. Although the reason for this unusual clinical presentation is uncertain, there might be some hypotheses. One of the hypotheses is that the posterior auricular branch of the facial nerve might be coinfected with the vagus nerve. However, this cannot explain why the facial palsy did not occur in the patient. Another hypothesis is that VZV reactivates on the vagus nerve and also the spinal nerve (C2-C3). In the postauricular area, greater auricular nerve and lesser occipital nerve are mainly innervating the sensory nerve that arrives from the spinal nerve (C2-C3). The latter is more feasible than the first one in our patient.

In multiple cranial neuropathies due to herpes zoster, combination therapy with antiviral agents and corticosteroids is preferred[11]. Moreover, the prognosis tended to be better in early treatment, especially within 3 days after the symptoms[12]. This patient was treated with the combination therapy 10 days after the first symptom (dysphagia) developed. Although his treatment started late, he was successfully treated with no sequelae. We think that he had no other symptoms to suspect the multiple cranial neuropathies and this is the main reason for successful treatment.

Herein, we report a case of herpes zoster with dysphagia which occurred before the skin lesion and pain. It was hard to diagnose it early because of its unusual presentation, but it was successfully treated with antiviral and corticosteroids combination therapy. Clinicians should always consider that dysphagia and hoarseness might be the only symptom of herpes zoster in the early stage. Therefore, evaluation of VZV infection might be considered if there is no specific reason for dysphagia.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

FUNDING SOURCE

None.

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