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Unusual Cause of Stridor in an 80-Year-old Man

To the Editor:

Bilateral vocal fold immobility is rarely associated with infections. We report an 80-year-old man who presented with sudden-onset stridor after receiving high-dose steroids. He was found to have bilateral vocal fold immobility associated with subglottic ulceration caused by herpes simplex virus (HSV), likely from reactivation through the vagus nerve. There have been 2 cases reported of bilateral vocal fold immobility associated with HSV,[1,2] but our case is the first with histopathologic confirmation.

An 80-year-old man with type 2 diabetes mellitus and remote surgery for colorectal cancer presented to the hospital with a small-bowel obstruction. Because of a reported contrast allergy, he received 2 doses of intravenous steroids before an abdominal computed tomography scan performed to evaluate the etiology of the obstruction. Forty-eight hours after presentation, the patient abruptly developed sore throat and inspiratory stridor in the absence of voice changes. Fiberoptic laryngoscopy revealed bilateral vocal fold immobility in the absence of any glottic lesions. The patient was intubated because of severe respiratory distress. Intravenous high-dose dexamethasone was begun empirically. A computed tomography scan of his head, neck, and chest was negative. After 48 hours of steroids, the patient was taken to the operating room, where extubation during direct suspension laryngoscopy revealed persistent bilateral vocal cord paralysis. A 1.0 × 0.5-cm posterior midline subglottic ulcer was noted, which was biopsied. Tracheostomy was performed at the same time. Histologic examination of the subglottic biopsy showed an ulcer with adjacent squamous metaplasia. Within the epithelium herpes viral cytopathic effect was seen, including multinucleated cells with nuclear molding and margination of the chromatin (**Figure**). An HSV immunostain was also positive (**Figure**). The patient's serum enzyme-linked immunosorbent assay for HSV type 1 (HSV-1) IgG antibodies was positive, but results of serologic testing for Lyme, human immunodeficiency virus, rapid plasma reagin, and HSV type 2 were negative. Taken together, herpes laryngitis due to HSV-1 was considered the cause of the patient's vocal cord paralysis.

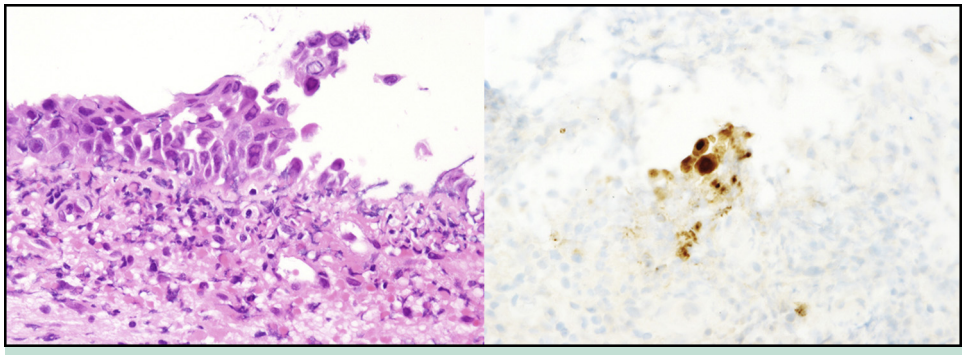

Figure Routine hematoxylin and eosin-stained histologic section of the subglottic biopsy shows a superficial ulcer. (Left) Adjacent herpes viral cytopathic effect is seen within the epithelial cells at the ulcer edge and consists of multinucleated cells with nuclear molding and margination of the chromatin. (Right) Immunohistochemistry for herpes simplex virus is also positive in these cells.

The most common manifestations of HSV-1 include recurrent eruption in the oral region, which is presumed to be from reactivation of the virus from latency in the trigeminal ganglia, esophagitis from reactivation in the vagus nerve, and encephalitis, which is thought to be from retrograde transmission of the virus to the brain. Herpes simplex virus has also

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been indirectly implicated in other neuropathic syndromes, such as facial or Bell's palsy and polyganglionitis.[3] In our patient, we suppose that the bilateral vocal fold immobility associated with HSV is a motor neuropathy from antegrade reactivation of the virus through the vagus nerve.

It is noteworthy that the onset of our patient's symptoms coincided with recent use of intravenous corticosteroids. Although this patient had many medical comorbidities and acute physiologic stress due to a small-bowel obstruction, the corticosteroids that he received could have been contributory, if not the main inciting factor, as has been reported in other cases. It has been suggested that steroids provide a reactivation trigger to latently infected ganglion.[4]

The patient was started on intravenous acyclovir, and within 3 days there was significant improvement at the ulcer site. The patient was discharged on valacyclovir 1000 mg every 8 hours for 21 days. Six weeks later, fiberoptic laryngoscopy revealed complete resolution of ulceration, as well as normal vocal cord function.

This case illustrates that the neurotropic virus HSV-1 can present as bilateral vocal fold immobility and that corticosteroids may contribute to its reactivation. It is important to recognize this syndrome and its association with HSV because antiviral treatment may be beneficial.

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