

Letter to the Editor

Unilateral oropharyngeal mucosal lesions as a clue to the pathogen of encephalitis



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Dear Editor,

Varicella zoster virus (VZV) encephalitis is suspected when encephalitis patients simultaneously present with the characteristic disseminated rash of varicella [1,2]. In contrast, a diagnosis of VZV encephalitis may be delayed in patients without skin rash. Here, we report a patient with VZV encephalitis in whom unilateral oropharyngeal mucosal lesions suggested VZV infection at the initial visit.

A 75-year-old male patient developed a sore throat and fever followed by abnormal behavior and a decreased level of consciousness. He was admitted to our hospital 5 days after symptom onset. His past medical history included angina pectoris, chronic atrial fibrillation, diabetes mellitus, and sleep apnea syndrome. There was no apparent history of chickenpox. He had been taking aspirin (100 mg/day), apixaban (5 mg/day), atorvastatin (5 mg/day), metformin (1500 mg/day), alogliptin (25 mg/day), miglitol (150 mg/day), carvedilol (5 mg/day), and famotidine (20 mg/day). In addition, insulin lispro (8–10.4 unit/day) and insulin glargine (4 unit/day) had been administered.

On admission, he complained of a sore throat. His level of consciousness was mildly decreased. The Glasgow Coma Scale (GCS) was E4V4M6. His vital signs were as follows: blood pressure of 134/92 mmHg, pulse rate of 127 beats per minute and irregular, body temperature of 37.2 °C. There was wet hoarseness. No skin rash was noted on his head, face, ears, trunk, or four limbs. Examination of the oral cavity demonstrated pooling of saliva as well as mucosal lesions in the left posterior palate and left posterior portion of the upper surface of the tongue (Fig. 1A). No paralysis was noted in the face, soft palate, or tongue. There were no other neurological focal signs. Endoscopic examination revealed pooling of saliva and sputum in the pharynx as well as multiple mucosal lesions on the left side of the epiglottis and left side of the pharyngeal wall (Fig. 1B, C). There was no paralysis of the pharyngeal muscles including the vocal cords. We suspected VZV infection based on the characteristic distribution of the mucosal lesions, which were on the sensitization area of the left glossopharyngeal and vagus nerves.

Blood tests showed a white blood cell count of 8070/ μ L, a C-reactive protein level of 12.06 mg/dL (reference range: < 0.14 mg/dL), glucose of 275 mg/dL, and hemoglobin A1c of 7.8%. Testing for human im-

munodeficiency virus was negative. Brain CT demonstrated no abnormalities, while chest CT revealed findings of bronchiolitis in the right middle lobe. A lumbar puncture yielded watery clear cerebrospinal fluid (CSF). The cell count was 265/ μ L, comprising mononuclear cells of 239/ μ L and polymorphonuclear cells of 26/ μ L. Total protein and glucose levels of the CSF were 148 mg/dL (reference range: 8–43 mg/dL) and 140 mg/dL, respectively. The intravenous administration of aciclovir and ampicillin/sulbactam was initiated for the treatment of encephalitis and bronchiolitis, respectively. Thereafter, VZV-DNA and VZV-IgG were found to be positive in the CSF. The titer of VZV-IgG was evaluated using an enzyme immunoassay method, and was 2.14 (reference range: < 0.2). Testing for herpes simplex virus-DNA, Epstein-Barr virus-DNA, cytomegalovirus-DNA, and enterovirus-DNA was negative in the CSF. The diagnosis of VZV encephalitis was established. After the initiation of treatment, his level of consciousness and unilateral oropharyngeal mucosal lesions gradually improved. On CSF examination 21 days after symptom onset, the cell count had decreased to 8/ μ L, and VZV-DNA had become negative.

Our patient simultaneously developed unilateral oropharyngeal mucosal lesions and encephalitis, probably due to VZV reactivation. Sensory innervations of the pharyngeal mucosa are supplied by both the glossopharyngeal and vagus nerves [3]. The pharyngeal mucosa between the level of the Eustachian tube orifice and the level rostral to the epiglottis is sensitized by fibers of the glossopharyngeal nerve, while the pharyngeal mucosa from the level of the epiglottis is innervated by sensory fibers of the vagus nerve [3]. Thus, the distribution of the oropharyngeal mucosal lesions of our patient corresponded to the sensitization area of the left glossopharyngeal and vagus nerves. In contrast, there were no findings suggesting involvement of the motor fibers of the vagus nerve in our patient.

The combination of cranial nerve palsies and unilateral oropharyngeal mucosal lesions has been reported in patients with VZV infection [3]. However, the simultaneous occurrence of encephalitis and unilateral oropharyngeal mucosal lesions in association with VZV infection has not been reported. Unilateral oropharyngeal mucosal lesions in encephalitis patients may provide a clue to the pathogen of encephalitis.

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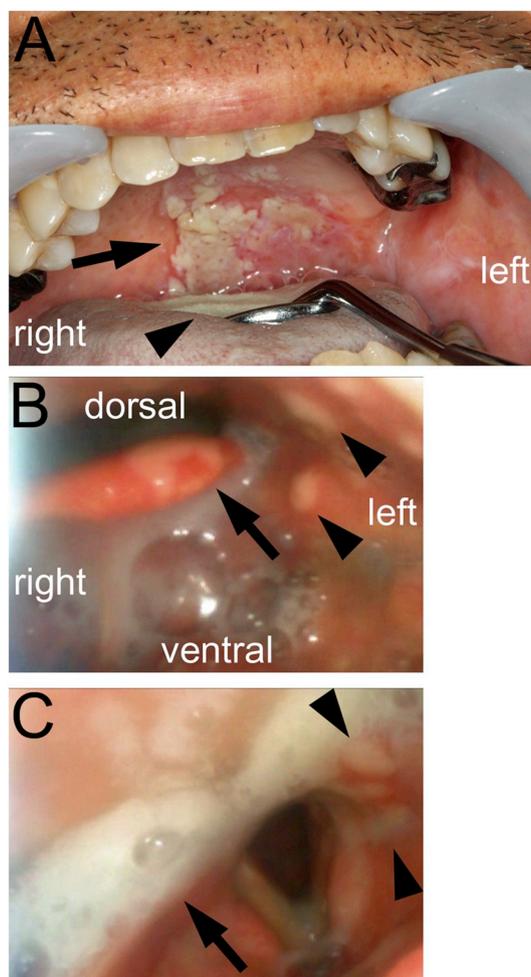


Fig. 1. Examination of the oral cavity demonstrated mucosal erosions showing a mixture of white and red on the left posterior palate (A, arrow). The right margin of this lesion corresponded to the midline of the palate. In addition, there were white lesions on the left posterior portion of the upper surface of the tongue (A, arrowhead). Endoscopic examination revealed pooling of saliva (B) and sputum (C, arrow) in the pharynx as well as mucosal erosions on the left side of the epiglottis (B, arrow) and left side of the pharyngeal wall (B, C, arrowheads). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

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