Case report

Ti.: “High” vagus nerve lesions in varicella Zoster infection☆

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1. Introduction

Sudden onset and complete loss of swallowing ability are rare events and require acute medical intervention.

Acute swallowing disorders can be caused by central causes (e.g., stroke, brainstem disease), or neuromuscular diseases such as myasthenia gravis, botulism, and a rare brachiofacial variant of polyradiculitis [1]. More frequent are chronic progressive swallowing disorders, such as in motor neuron disease, pseudobulbar syndromes, and myopathies [2].

Isolated recurrent laryngeal nerve lesions produce hoarseness and some degree of swallowing impairment, but usually no complete loss of swallowing ability.

This case report illustrates the rare event of a “high” vagus nerve lesion, resulting in a transient complete swallowing loss needing parenteral feeding, and followed by complete reversibility. The anatomical distinction between a “high” and low vagus nerve lesion is crucial and refers to peripheral vagus nerve lesions only [3,4].

2. Case report

A 79-year-old previously healthy woman presented with a reddish, erysipelas like swelling of the left ear and cheek. Local pain radiated into the occipital part of the skull. Within 24 h she was unable to swallow. She was admitted to the hospital and a recurrent laryngeal nerve palsy was noted at the first otolaryngologic examination.

On examination, she had a swollen and reddish-colored edematous left ear and also swelling of the left side of the face. (Fig. 1) She spoke with a hoarse voice. She was unable to swallow and parenteral feeding had to be initiated.

The soft palate was lower on the left side, and elevated only minimally upon phonation. The mucous membranes of the oral cavity showed no inflammation or vesicles. There was no numbness in the oral cavity and taste sensation was normal also in the posterior part of the tongue. The rest of the neurological examination was normal, and there were no reflex abnormalities, and no long tract signs or gait abnormalities that pointed to central nervous system involvement.

Neurological investigation excluded a brainstem lesion and no associated cranial nerve lesions were detected. The clinical syndrome correlated with a “high” vagus nerve lesion. The pain distribution was...
attributed to involvement of the meningeal rami and the auricular nerve.

The otolaryngologic examination confirmed a left vocal cord palsy, which was also evident on MRI (Fig. 4). Swallowing was impossible and there was no evidence of an auditory deficit at the time. An endoscopic examination was not performed.

The dermatological exam suspected erysipelas and grouped, small, and eroded papules on an erythematous base on the left ear (Fig. 1) indicating a Herpes infection. Due to the acute and painful development, antimicrobial therapy with Meropenem and Zovirax was initiated and after 5 days steroids were also added.

Under speech therapy treatment, mild improvement of swallowing occurred after 10 days and the patient subsequently completely recovered. After the regression of the ear swelling, crusts were detected in the auricle. (Fig. 2).

3. Findings

3.1. MRI of the skull and neck

The brain and bony skull were normal, the swelling of the left ear and adjacent tissue were visible, and no abscess formation or tumor was found (Fig. 3). The left pharyngeal fold was paralyzed (Fig. 4).

3.2. Laboratory results

C-reactive protein (CRP) was not elevated. A mild lymphocytopenia (1100/μl) was noted.

Virology was negative for the following: Herpes simplex, tick borne encephalitis (FSME), Coxsackie, Cytomegalovirus, Epstein-Barr, Influenza A, B, measles, adenovirus, ECHO-virus, Mumps, Borrelia IgM and IgG antibodies.

Varicella Zoster IgG was elevated at >4000 MIU/ml (normal high up to 50), and varicella Zoster IgM was 0.351 (index).
which would point to a more distal anatomical site. Several cases of Varicella Zoster IgG. A selective involvement of the vagal nerve was suspected based on skin changes and the high titer of antibodies against VZV. A biopsy showed necrosis of the nerve, and an isolated herpes zoster (HZ) infection of the vagus nerve was confirmed. The patient recovered fully with no sequelae.

The mechanism of the high vagus nerve lesion is presumably neuropraxia of the nerve due to local swelling and inflammation, with preservation of the axons. This would be compatible with the effectiveness of steroids and the rapid reversibility, which could not be explained by regeneration alone. This remains hypothetical as electrophysiological studies are not available for this part of the nervous system.

The identification of a rare “high” vagus nerve lesion is important to consider in an acute swallowing disorder, and needs to be included in the differential diagnostic considerations. The combination of local headache, abrupt swallowing inability, and local swelling of the ear may be a useful clinical guide.

Table 1 summarizes several causes of “high” vagus nerve lesions. The clinical course is mainly distinguished between acute (trauma, infection and vascular) and insidious onset (usually space occupying lesions).

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