Hemigasia: an unusual presentation of multiple sclerosis

Patients with multiple sclerosis (MS) rarely complain of taste disturbances, although electrogustatory examinations often demonstrate dysfunction of the taste pathway in patients with advanced disease, especially in those with prominent brainstem involvement. A 25 year old native American man presented with a two day history of gradually progressive loss of taste on the entire right half of his tongue (hemigasia). One week later he developed numbness of the right inner cheek, double vision, and a tendency to fall to the left. He had clockwise rotary nystagmus, right pupillary nuclear areflexia, left central facial palsy, left hyperreflexia, and intention tremor with the right hand. The right hemigasia was unchanged. The gag reflex was diminished on the right side, and the right palate and the right inner cheek were numb. Routine laboratory tests were all normal. CSF analysis revealed no red cells, five lymphocytes, 53 mg/dL protein, normal glucose, 5-6 mg/mL myelin basic protein (normal range 0-5.1 mg/mL), and three oligoclonal bands without correlates in serum.

Eleven days after the onset of the taste disturbance he developed paroxysms of pain around the right eye, periorbital numbness, and tingling sensation in the right cheek. New findings were a decreased right corneal reflex and hypeaesthesia to pain and temperature in the distribution of the right mandibular division. Cranial nerve examination revealed multiple, bilateral, periventricular areas of increased T2 signal. A similar lesion was found in the right medulla on the floor of the fourth ventricle (fig). In the CSF myelin basic protein was 162 mg/L with 160 oligoclonal bands without serum correlate were identified. The patient was treated with a 10 day course of 1 gm/day intravenous methylprednisolone followed by a two week oral prednisone taper with symptomatic improvement.

Thirteen months after presentation he developed acute left hemiparesis. MRI demonstrated an increase in the number of periventricular lesions of increased T2 signal and a new right sided lesion at the expected location of the internal capsule. Visual evoked potentials and lower extremity somatosensory evoked potentials showed prolonged wave latencies. CSF showed elevated protein (63 mg/dL) and myelin basic protein (14.8 mg/mL), and five oligoclonal bands. The patient was treated with a 10 day course of IV methylprednisolone and his hemiparesis recovered. On follow up he still had the previously described deficits.

Hemigasia involving the entire right half of the tongue is usually explained by a lesion in the ipsilateral nucleus solitarius, where fibres from the left hemisphere (anterior two thirds of the tongue) and fibres from the glossopharyngeal nerve (posterior third) come together. Recent evidence suggests the presence of an accessory taste pathway through the trigeminal nerve. Hypoguesia is found in 5-10% of patients with advanced MS and is frequently associated with sensory involvement of the trigeminal nerve. Taste disturbance as the initial symptom of MS has previously been reported only by Harris. His patient, a 21 year old woman, developed numbness of the right side of her face and right sided hemigasia, that persisted for one year. Four years later she presented with trigeminal neuralgia, but it was not until 10 years later, that multifocal symptoms led to the diagnosis of MS. The remarkable aspect of our case is that the right-sided hemigasia was the sole presenting symptom, although the investigation demonstrated multinuclear central nervous system lesions. Right trigeminal sensory involvement occurred almost two weeks after the hemigasia, and prominent, more widespread brainstem symptomatology developed later only.

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