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Case Report

Facial muscle paralysis: behind the words

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A 67-year old woman presented with right facial muscle paralysis of acute onset of six hours. Her medical history included diabetes mellitus on metformin since several years and an ear infection of viral etiology 4 weeks ago. Clinical examination (using the House-Brackmann scale) revealed pain behind the right ear, inability to close her right eye, unilateral tongue numbness and increased sense of hearing on the right side, while other cranial nerve function was spared.

QUESTION

Based on the patient's history and physical examination, which one of the following is the correct diagnosis?

A. Bell's palsy
B. Diabetes mellitus
C. Ramsay-Hunt syndrome
D. Upper motor neuron lesion
E. Lower motor neuron lesion
F. Stroke
G. Brain tumor
H. Mandible fractures

DISCUSSION

The answer is A: Bell's palsy.

Bell's palsy is a very common neurologic disorder, first described by Sir Charles Bell. Bell's palsy is a form of abrupt, unilateral (95%), peripheral facial paresis or paralysis, resulting from damage to the 7th cranial nerve, resulting from compressive, traumatic, infective, inflammatory or metabolic abnormalities. Symptoms include one-sided facial paralysis, pain in the mastoid region, tearing, drooling, numbness, stiffness, hypersensitivity to sound in the affected ear and impairment of taste. Bell's palsy is a diagnosis of exclusion [1]. Much controversy still exists around etiology, as well as pathophysiology of the palsy, ranging from viral infection, vascular ischemia, to autoimmune inflammation or even heredity [2]. With the aid of PCR polymerase chain reactions, virus (Herpes simplex virus type I) mediated inflammatory immune mechanism seems to be the cause.

Bell's palsy has a generally good prognosis, as it usually self-resolves gradually after 2 weeks in two thirds of the patients and most individuals recover completely after 3 to 6 moths. If bilateral palsy occurs, one can particularly consider Guillain-Barré syndrome or Lyme disease. If palsy recurs, we can consider lymphoma, sarcoidosis and Lyme disease.

Treatment has been widely debated. Treatment modalities and elimination of nerve damage worsening include:

a) Analgesics

b) Early administration of anti-inflammatory agents (prednisolone) [4], in combination with anti-viral agents (aciclovir, valaciclovir, and famciclovir- all nucleotide analogues that interfere with DNA polymerase)

c) Botulinum toxin injections [5], and, lately,

d) Decompression surgery [6].

Early recovery gives a good prognosis and late recovery a bad prognosis. If recovery begins within one week, 88% obtain full recovery.

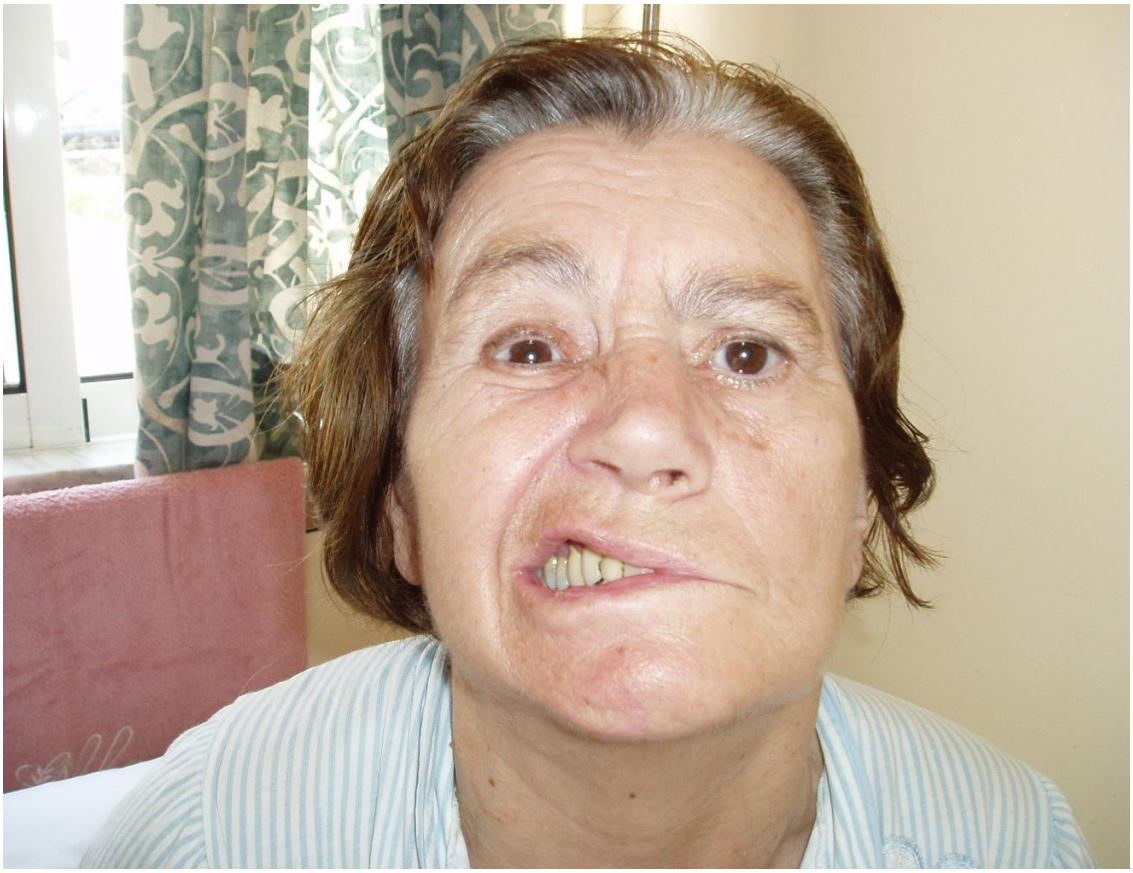
Ramsay-Hunt syndrome is the second most common acute facial paralysis. It is often complicated by vestibulocochlear dysfunction characterized by the concomitant appearance of rush behind the ear, painful rash on the eardrum, tongue or roof of the mouth, on the same side of the weakness, as well as hearing loss and possible vertigo (involvement of zoster oticus-reactivation of varicella–zoster virus). When the rash is absent it is known as zoster sine herpete. Pain is often a prominent feature and vesicles are seen in the

A Table showing all main characteristics of the diseases

Selected Differential Diagnosis of Facial Muscles Paralysis

Condition	***Characteristics***
Bell's palsy	Peripheral type of facial paralysis involving all the affected side's muscles, sense of taste, hearing.
Diabetes Mellitus (DM)	Debated whether to be considered as a focal mononeuropathy of DM.
Ramsay-Hunt syndrome	Auditory symptoms, vertigo and relevant symptoms to Bell's palsy. Distinctive symptom: painful rash over the ear.
Upper motor neuron lesion (UMN)	Weakness of the lower face only, lesion frontalis is spared, normal furrowing of the brow is preserved, and eye closure and blinking are not affected.
Lower motor neuron lesion (LMN)	The patient can't wrinkle their forehead - the final common pathway to the muscles is destroyed
Stroke	Other muscles on one side of the body may also be involved.
Brain Tumor	Usually develops slowly and causes headaches, seizures, or hearing loss.
Mandible fractures	History of injury, tenderness, confirmation with diagnostic radiology.

A picture showing the woman with Bell's Sign



ipsilateral ear, on the hard palate and/or on the anterior two thirds of the tongue. Immunodeficiency is a risk factor.

Antiviral medication in combination with steroids is the preferred treatment.

An upper motor neuron lesion (UMN) is a lesion of the neural pathway above the anterior horn cell or motor nuclei of the cranial nerves. Symptoms include flexor and extensor muscle spasticity, weakness on the lower side of the face, a clasp-knife response and possibly Babinsky sign. Intracranial pathological findings must be ruled out with the use of diagnostic radiology. In an UMN lesion, the upper facial muscles are partially spared because of alternative pathways in the brainstem, i.e. the patient can wrinkle their forehead (unless there is bilateral lesion) and the sagging of the face seen with LMN palsies is not as prominent. There appear to be different pathways for voluntary and emotional movement.

In a lower motor neuron lesion (LMN) the patient can't wrinkle their forehead - the final common pathway to the muscles is destroyed. Lesion must be either in the pons, or outside the brainstem (posterior fossa, bony canal, middle ear or outside skull).

Stroke may cause facial paralysis, too. With a stroke, other muscles on one side of the body may also be other muscles on one side of the body may also be involved.

Facial paralysis that is due to a brain tumor usually develops slowly and causes headaches, seizures, or hearing loss.

Mandibular fractures usually occur in two or more locations because of the bone's U shape and articulations at the temporomandibular joints. Fractures also may occur at a site apart from the site of direct trauma. Mandibular or maxillofacial fractures usually are consistent with a relevant history of injury.

It seems that the development of peripheral facial paralysis is not part of a multifocal neuropathy in the progress of DM [3]. However, there is more small fiber polyneuropathy in the ordinary diabetic people while diabetics with recent onset palsies have more frequent concurrent clinical/subclinical entrapment neuropathies.

Surgical decompression medial to the geniculate ganglion significantly improves the chances of normal or near-normal return of facial function in the group that has a high probability of a poor result. Surgical decompression must be performed within 2 weeks of onset of total paralysis for it to be effective. Lately, transcutaneous electrical stimulation treatment may have a positive effect on unresolved facial nerve paralysis.[6]

Surgical transmastoid decompression of the facial nerve in severe cases is being investigated but cannot currently be recommended. Where nerve fails to regenerate, cosmetic surgery to elevate the mouth or anastomosis of the hypoglossal nerve to the facial nerve may help.[6]

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