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# Diagnostic Difficulties in Etiology of the Lesion of Peripheral Neuron of the Facial Nerve During the Growth of Sialoma

Trudności diagnostyczne dotyczące etiologii uszkodzenia obwodowego neuronu nerwu twarzowego w rozwoju nowotworu ślinianki

There are varied causes of lesions of the facial nerve: 75% of them is idiopathic paralysis or Bell paralysis (1), then there are injuries, diseases of the ears (otitis media and externa, otic zoster) and carcinomas of neuron VII, cerebellopontine angle tumors, tumors of the pons, of the petrosal bone, the middle ear, leukemias, diabetes, tumors of the salivary gland (2, 3, 4).

Especially careful diagnosing is required for the cases of the lesions of the peripheral neuron n. VII, where, despite conventional treatment, there is no distinct improvement or where it is necessary to determine the right etiopatogenic factor among several possible ones.

The patient under observation merits particular attention because it is necessary to determine the underlying cause of the lesion of the peripheral neuron of the facial nerve.

## CASE DESCRIPTION

A patient G. Z., aged 65, admitted to Neurology Clinic of the Medical Academy in Lublin because a months-long outpatient treatment of the lesion of the peripheral part of the facial nerve proved ineffective.

In January 1987 the patient noticed a hard, immovable and painful tuberculum near the right angle of the mandible. In February 1987 he was treated for Lerich syndrome. Diabetes was then found and a diabetol treatment was started. Months later, after an intense pain lasting several days in the vicinity of the right parotid gland, there were suddenly symptoms of the lesion of the peripheral neuron of the right facial nerve. Polyneuropathy and fatty liver symptoms were then reported. Despite intensive treatment the heightened weakness of the orbicular muscle of the right eye was so considerable that there was a threat of corneal ulceration, which was an indication for a palpebral raphe.

As the function of the nerve did not improve and the infiltration in the right retromandibular region enlarged, the patient was admitted to the Clinic. The examination conducted on the day of admission revealed: projection of the right retromandibular region with a palpably tender hard tuberculum of 1 × 2 cm, fused with the base and merging with the mastoid bone, the smoothing of the forehead skin, asymmetry of palpebral fissures, the smoothing of the right nasal-labial fold with a lower position of the right angle of the mouth. The insufficient function of the mimic muscles of the right facial part consisted in the absence of wrinkling of the forehead skin, the occurrence of the Bell signs, defective closure of the right palpabral fissure 5 mm wide and drawing out of the mouth to the left while trying to expose the teeth.

A preliminary comparative examination of hearing acuity indicated a slight impairment on the right. The patient reported that this deficiency appeared simultaneously with the insufficiency of the facial nerve and was not preceded by hyperacusis.

The examination of taste revealed a loss of sensibility of all basic kinds of taste on two thirds of the anterior rightside part of the tongue. In all limbs there was diminished muscle mass, the thinning of the skin, its pallor and delicate peeling off in the outer parts of the limbs. The muscle power of the outer limb parts was slightly diminished, the lower right limb positioned itself distinctly below the left limb. Radial reflexes, elbow reflexes and the right Achilles tendon reflex could not be produced. The right knee reflex was weakened and there was only a trace of the left Achilles tendon reflex. All kinds of superficial dysesthesia were of glove character. Other irregularities worth noting were the protrusion of the liver 5 cm from under the right costal arch and pulselessness in the large arteries of both lower limbs despite lack of distinct traits of limb ischemia.

The results of lab tests that need to be noted were: varying but generally correct glucose levels in the blood and trace glucose content in the urine during insulin treatment at high daily doses, impairment of the liver function manifested by the elevated thymol turbidity test up to 9.9 units M. L., heightened transaminase activity (ASPAT 216 units, ALAT 323 units) and gamma glutamyltranspeptidase (16.3 units). The activity of HBS antigen was reported in the blood.

An ultrasonographic examination of the abdominal cavity confirmed the earlier finding of fatty liver symptoms. The audiogram was estimated as within the age norm. The roentgenogram of the pyramids showed no deviations from the norm. A tomographic examination of the right mandibular angle revealed a solid oval formation 2 × 1 cm in the lower pole of the parotid gland. This tumor showed minor contrast augmentation. The tumor did not exceed the gland boundary. Nor was lymphadenopathy of the parapharyngeal space reported. The course of vessels in this area was not altered.

After a laryngologic consultation the patient was transferred to the Otolaryngologic Clinic of the Medical Academy in Lublin (head: Prof. B. Semczuk, M.D.)

where he was operated on. During the operation it turned out that the tumor was not solid and grew into the adjacent tissues. The extent of neoplastic infiltration or precisely two infiltrating tumors with cancerous texture was the reason why not only the whole parotid gland with a part of the facial nerve was removed but also the adjacent parts of the masseter muscle and the sternocleidomastoid muscle, the anterior osteocartilaginous wall of the external auditory meatus and the apex of the mastoid bone. The external carotid artery was also ligated and 2 upper lymph nodes of the neck were removed. The operation went well beyond macroscopic changes, suggesting a large safety margin (Prof. W. Gołąbek, M.D.).

## DISCUSSION

The 65-year-old patient with a lesion of the peripheral neuron of the facial nerve was at the same time suffering from other diseases: insulin-dependent diabetes, diabetic polyneuropathy, fatty liver (possibly following symptomless viral hepatitis) and Lerich syndrome. Although each of those pathological processes might cause the lesion of the peripheral neuron of the facial nerve, we regarded the neoplastic process developing in the parotid gland as the main cause of the nerve lesion.

While investigating the etiology of lesion of the peripheral neuron of the facial nerve before a tomographic examination of the salivary gland, we concentrated our attention primarily on localizing the lesion starting from the nucleus of this nerve. The absence of signs of lesion of the long tracts and infiltration of the adjacent nuclei of cranial nerves permitted to exclude the trunk location of the disease. The regular width and shape of the auditory foramen and the absence of typical audiometric changes permitted to exclude a process developing in the cerebellopontine angle and in the petrous pyramid.

The absence of a morbid condition of the middle ear made very improbable a lesion of the facial nerve in its course in the vicinity of the tympanum. An examination of the skin of the ear concha and its adjacent region as well as the interview on the earliest period of the illness gave no grounds for assuming past otic zoster or Ramsay-Hunt syndrome.

To attribute the lesion of the peripheral neuron of the facial nerve to a neoplastic process developing in the parotid gland can be difficult in the initial evolution of the process (2, 4) and in patients in whom other diseases that certainly contributed to the pathogeny of the facial nerve dysfunction developed before the clinical manifestation of sialoma. However, to establish the right cause of the lesion of the peripheral neuron of the facial nerve dysfunction developed before the clinical manifestation of sialoma. However, to establish the right cause of the lesion of the peripheral neuron of the facial nerve is essential for the choice of the most effective treatment.

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## STRESZCZENIE

Badania przeprowadzono u 65-letniego chorego z uszkodzeniem obwodowego neuronu nerwu twarzowego. Cierpiał on wcześniej z powodu cukrzycy, stłuszczenia wątroby, zaawansowanej miażdżycy naczyń kończyn dolnych, a następnie wskutek guza naciekającego ślinianke przyuszną. W związku z tym rozważano kilka czynników etiopatogenetycznych, łącznie z miejscowym uciskiem nerwu twarzowego spowodowanym przez proces nowotworowy. Badanie tomograficzne i zabieg operacyjny potwierdziły patogenetyczną rolę nowotworu ślinianki w uszkodzeniu nerwu twarzowego.