Facial nerve palsy – central or peripheral? Diagnosis is not always obvious

Ośrodkowe czy obwodowe porażenie nerwu twarzowego? Diagnoza nie zawsze jest oczywista

Joanna Marszał, Anna Bartochowska, Wojciech Gawęcki

Department of Otolaryngology, Head and Neck Surgery, Poznan University of Medical Science, Poznan, Poland

Abstract

We herein report a case of a 63-year-old man with facial nerve palsy and unusual medical history. He reported to ENT outpatient department due to the progression of facial nerve paresis. In the past he experienced a subarachnoid hemorrhage complicated by mouth drooping on the left side. He had also a history of external malignant otitis and exudative otitis media treated with grommet placement. Determining the reason of the progression of facial nerve paresis was not easy. Finally, it turned out that it was caused by cholesteatoma. Presented case shows that symptoms of central and peripheral facial nerve palsy may overlap thus making the diagnosis not obvious.

Key words: *facial nerve palsy, subarachnoid hemorrhage, cholesteatoma.*

Streszczenie

W artykule przedstawiamy przypadek 63-letniego mężczyzny z porażeniem nerwu twarzowego i nietypową historią choroby. Pacjent zgłosił się do przyklinicznej poradni laryngologicznej z powodu nasilenia niedowładu nerwu twarzowego. W przeszłości doznał krwotoku podpajęczynówkowego powikłanego opadaniem kącika ust po lewej stronie. Był również leczony z powodu złośliwego zapalenia ucha zewnętrznego oraz wysiękowego zapalenia ucha środkowego. Ustalenie przyczyny progresji niedowładu nerwu twarzowego nie było łatwe. Ostatecznie okazało się, że była ona związana z perlakiem. Przedstawiony przypadek pokazuje, że objawy porażenia ośrodkowego i obwodowego nerwu twarzowego mogą się na siebie nakładać, co powoduje, iż postawienie właściwej diagnozy może nie być oczywiste.

Słowa kluczowe: *porażenie nerwu twarzowego, krwawienie podpajęczynówkowe, perlak.*

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Case report

A 63-year-old man reported to the ENT outpatient department complaining of progression of facial nerve palsy for 2 weeks. Clinical examination revealed drooping of the brow, incomplete lid closure and drooping of the corner of the mouth on the left side (grade V according to House-Brackmann (HB) scale). Both external auditory canals were intact. The right tympanic membrane was normal, while the left one was retracted, with the scars in inferior quadrants, small marginal perforation in the posterior superior quadrant and watery discharge visible during the Valsalva maneuver. High resolution computed tomography (HRCT) showed a soft tissue mass in the left tympanic cavity and mastoid spaces and increased mastoid sclerosis (Figure 1). In pure-tone audiometry, mixed hypoacusis on the left side was found.

The man suffered from diabetes and had a medical history of subarachnoid hemorrhage (SAH) 3 months earlier. One of the first symptoms was mouth drooping on the left side followed by slurred speech and weakness on the right side of the body. Common carotid arteries and vertebral arteries arteriography showed no vascular abnormalities. Almost complete recovery was obtained by intensive-care treatment and rehabilitation. Only a slight weakness of the marginal branch of the left facial nerve was noticed.

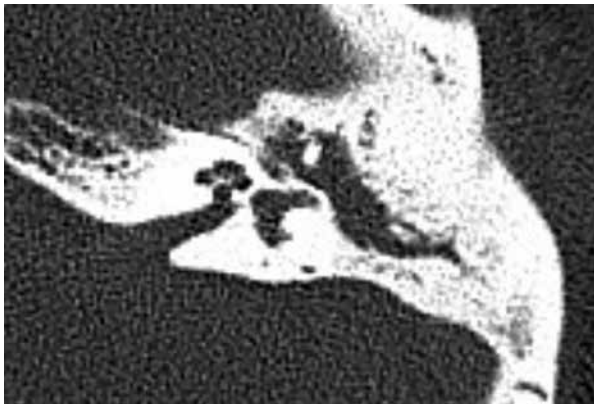

Figure 1. Computed tomography scan of the left temporal bone, axial section

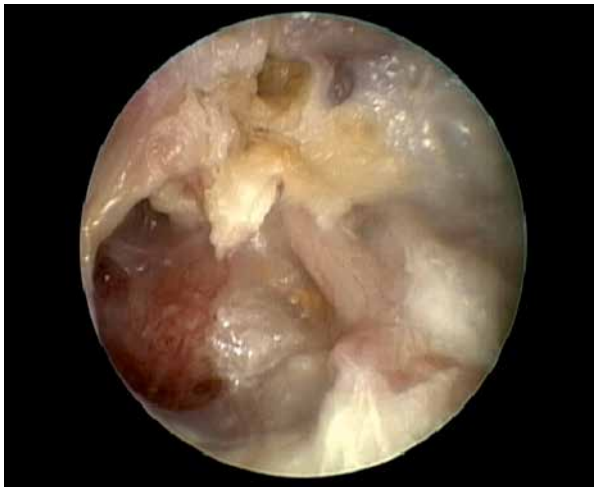

Figure 2. Intraoperative view of the left ear — deficiency of the bone overlying the facial nerve canal in the tympanic segment

The man had also a history of external malignant otitis 7 years prior to SAH. At that time he had complained of headache and left ear discharge. Facial nerve function was normal. Ear swab was positive for Pseudomonas aeruginosa. Medical history also revealed a grommet placement into the left eardrum due to hearing loss and tinnitus in another department 2 years prior to facial nerve palsy.

Questions

What was the cause of facial nerve paresis? What kind of treatment should have been administered?

Replies

The patient presented with peripheral facial nerve paresis. Besides, he did not report any alarming neurological symptoms. It made the diagnosis of the stroke or SAH unlikely. However, in the literature we can find cases of the isolated peripheral facial nerve palsy in the course of the pontine stroke [1] so we should always consider it in differential diagnosis. There was no ear pain, no granulation tissue and oedema in the external meatus, which are typical for external malignant otitis. Therefore, the most probable site of inflammation was the middle ear.

The patient was qualified for middle ear surgery. The canal wall-up technique was used and revealed a presence of inflammatory granulation tissue and cholesteatoma in the tympanic cavity and mastoid. Deficiency of the bone overlying the facial nerve in the tympanic segment was revealed (Figure 2). Cholesteatoma was removed and the facial nerve was decompressed. After the procedure, the facial nerve function improved within 4 weeks to grade II according to HB, i.e. to the level obtained after SAH rehabilitation.

Facial nerve palsy is the second most common complication of chronic otitis media [2]. Its etiology is usually related to the impairment in blood supply following compression by cholesteatoma or granulation tissue or direct intoxication of the nerve by pathogens [3, 4]. Rapid surgical treatment is necessary to prevent permanent palsy [5]. In our case, pathogenesis of paresis could have been complex (including central component in the past), while cholesteatoma could have been iatrogenic after grommet placement. Removal of the pathological tissue together with facial nerve decompression enabled almost complete recovery of its function.

In summary, this case shows that symptoms of central and peripheral facial nerve palsy may overlap thus making the diagnosis not obvious. Progression of facial nerve palsy requires extended diagnostics and may suggest exacerbation of the disease with the development of further complications.

Conflict of interest

The authors declare no conflict of interest.

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Address for correspondence:

Joanna Marszał

Department of Otolaryngology, Head and Neck Surgery, Poznan University of Medical Science, Poznan, Poland

e-mail: joan.marszal@gmail.com