Limitations of Magnetic Resonance Imaging in the Evaluation of Perineural Tumor Spread Causing Facial Nerve Paralysis

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Objective: To present and discuss the clinical presentation and treatment in patients with long-duration unilateral facial paralysis and normal magnetic resonance imaging (MRI) findings.

Design: Case series.

Setting: Ear, nose, and throat department of the University of Cologne, Cologne, Germany.

Patients: A total of 486 patients with unilateral facial paralysis who were treated from 1986 to 1998. Besides the usual diagnostic workup, a complete electrophysiological evaluation, including investigations such as needle electromyography and neuromyography (also known as electroneurography), of the facial nerve was performed at repeated intervals. In 19 patients, a malignant tumor was delineated with ultrasonography or MRI. In 8 of these patients, the initially performed MRI did not detect any parotid gland lesion causing the paralysis, whereas long duration of the paralysis and electroneurography indicated malignancy.

Results: Exploration surgery was performed as total parotidectomy in these 8 patients and malignant parotid gland tumors were proved in all 8 patients.

Conclusions: Individuals with facial nerve paralysis without any signs of regeneration 6 months after the onset of paralysis and/or persistent electrophysiological evidence of ongoing neuronal degeneration should undergo surgical exploration of the parotid gland and facial nerve, even if MRI studies show no tumoral lesion.


ELL PALSY, by definition, is a diagnosis of exclusion. Eighty percent of all peripheral facial paralyses are labeled as idiopathic or Bell palsy and a complete recovery occurs in 70% of all patients.1 Conversely, approximately 20% of these facial palsies can be demonstrated to have a specific cause. All patients exhibiting facial paralysis thus should undergo a thorough neurotologic evaluation to identify the underlying abnormality.

Facial paralysis of neoplastic origin is uncommon. It is estimated to represent the etiology in approximately 5% of all cases.2 Neoplastic involvement may be by neurogenic primary lesions of the seventh cranial nerve or by secondary, extrinsic neoplasms.

Parotid gland disease must be acknowledged as a significant clinicopathologic entity in the pathogenesis of facial paralysis. The presentation of facial paralysis in the presence of parotid gland lesion is generally considered an ominous sign, almost uniformly heralding malignant disease. Usually, tumors are palpable and detectable using ultrasonography, computed tomography, or magnetic resonance imaging (MRI).3 Nevertheless, it is known that computed tomography and ultrasonography may fail to depict small parotid gland lesions, especially if they are located in the deep, retromandibular portion of the parotid gland or close to the stylomastoid foramen.4,5 Magnetic resonance imaging has higher accuracy in identifying soft tissue lesions. With its high soft tissue definition, even very small lesions of the parotid gland and the facial nerve become identifiable, as well as the deep portion of the parotid gland and the intramastoid, tympanic, and labyrinthine sections of the facial nerve.6-9

The purpose of this article was to present a series of cases of facial paralyses caused by malignant parotid tumors, which had not been identified by gadolinium-enhanced MRI.
PATIENTS AND METHODS

From 1986 to 1998, we treated in our outpatient clinic 486 patients with peripheral, unilateral facial paralysis. In addition to the usual neurotologic investigations (standard pure tone and speech audiometry, impedance audiometry, modified Short Increment Sensitivity Index, Carhart tests, brainstem audiometry in cases of equivocal findings, Schirmer tear test, stapedial reflex testing, and taste evaluation), we performed ultrasound of the parotid gland. A complete electrophysiological workup including needle electromyography and neuromyography (also known as electroneurography) was performed at repeated intervals. As a routine, all patients were submitted to these investigations at the time of first contact with our department, then follow-up visits were scheduled 2, 6, and 12 weeks after onset of paralysis. In cases of persistent paralysis with or without electrophysiological signs of neuronal degeneration (ie, positive sharp waves in needle electromyography) follow-up was continued at 4-week intervals. In 344 patients (76%) any specific cause of the paralysis was ruled out and the paralysis was labeled as idiopathic. The other patients showed specific lesions as described in Table 1. In 27 patients (6%) a unilateral facial paralysis was the first symptom of a malignant tumor of the parotid gland. Eight of these patients had been referred to our service for facial reanimation surgery because of abnormally long duration of complete facial nerve paralysis (Table 2). In all 8 patients, previously performed gadopentetate (Gd-DTPA)–enhanced MRI of the parotid gland and of the pontine angle region had not shown any pathological change, destruction, or lesion. In these 8 patients we performed, in addition to the neurotologic examination and electromyography, another Gd-DTPA–enhanced MRI and exploration surgery.

RESULTS

Our subgroup of 8 patients showed no electrophysiological regeneration after more than 6 months of paralysis (range, 6–36 months; mean, 17.6 months; Table 2). After electromyographic diagnosis indicated neoplastic invasion, another Gd-DTPA–enhanced MRI of the cerebellopontine angle and the parotid gland was performed in patients in whom MRI studies did not cover the whole parotid gland or in whom MRI studies had low diagnostic quality. Electromyographic criteria for neoplastic invasion were pathological spontaneous activities without interference activity pattern or intermediate activity pattern, but sometimes with transitory single oscillations in patients with clinically complete facial paralysis.

In 2 patients a parotid gland tumor was diagnosed immediately (Figure 1). The formerly performed MRI studies had simply not covered the parotid gland lesion, but only the cerebellopontine angle region for acoustic neuroma exclusion and the upper parts of the parotid gland. In 1 patient, tumoral invasion of the deep portion of the parotid gland was demonstrated using a head and neck coil and high-resolution MRI reconstruction. In 5 patients, the repeated Gd-DTPA–enhanced MRI did not depict any pathologic alteration.

All 8 patients underwent total parotidectomy for diagnostic and therapeutic reasons. Histopathological investigations revealed malignant tumors of the parotid gland in all 8 patients: adenoid cystic carcinoma with perineural spread in 4 patients, basal cell adenocarcinoma in 2 patients, mucoepidermoid carcinoma in 1 patient, and acinic cell carcinoma in 1 patient.

The MRI study did not detect the lesion in all patients with adenoid cystic carcinoma and in 1 patient with mucoepidermoid carcinoma. Histopathological studies revealed predominant perineural, intraneural, and perivascular spread of the cancer without a real circumscript tumor (Figure 2).

In 1 patient with basal cell adenocarcinoma, the initially performed MRI did only depict an accompanying mastoiditis, but not the tumor itself. A second high-resolution Gd-DTPA–enhanced MRI scan hinted at the parotid gland tumor; perhaps the histological results proved very low mitotic activity and subsequently the slow growth of the tumor may have affected the documented MRI signal intensity similarly (Figure 3). In the remaining 2 patients, the initial MRI studies simply did not cover the parotid gland lesion. In these patients the MRI studies were performed primarily to exclude a cerebellopontine angle tumor, but later served as guarantee for safe tumor exclusion.

COMMENT

Delineation of circumscribed tumoral lesions of the parotid gland today is the domain of ultrasonography, and, for the deeper portions of the parotid gland, of MRI. Especially for the evaluation of tumors of the stylomastoid foramen and the retromandibular part of the parotid gland, MRI is the method of choice.6–10

Magnetic resonance imaging allows excellent visualization of the facial nerve throughout its entire course.

Table 1. Diagnoses and Nerve Function in Patients With Unilateral Facial Paralysis

<table>
<thead>
<tr>
<th>No. (%) of Patients (N = 486)</th>
<th>Diagnosis</th>
<th>Facial Nerve Function After 6 mo*</th>
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<tbody>
<tr>
<td>344 (71) Idiopathic (“Bell palsy”)</td>
<td>Recovery</td>
<td></td>
</tr>
<tr>
<td>22 (4) Herpes zoster (IgM)</td>
<td>Recovery</td>
<td></td>
</tr>
<tr>
<td>37 (8) Pontine angle tumor</td>
<td>Mixed, degeneration and regeneration</td>
<td></td>
</tr>
<tr>
<td>11 (2) Any central dysfunction†</td>
<td>No recovery, denervation potentials</td>
<td></td>
</tr>
<tr>
<td>27 (6) Malignant parotid gland tumor</td>
<td>No recovery, denervation potentials</td>
<td></td>
</tr>
<tr>
<td>22 (4) Basal cell adenocarcinoma</td>
<td>No paralysis</td>
<td></td>
</tr>
<tr>
<td>11 (2) Herpes zoster (IgG)</td>
<td>Recovery</td>
<td></td>
</tr>
<tr>
<td>1 (0.2) Any central dysfunction‡</td>
<td>No recovery, denervation potentials</td>
<td></td>
</tr>
<tr>
<td>0 Benign parotid gland tumor</td>
<td>No paralysis</td>
<td></td>
</tr>
<tr>
<td>45 (9) Trauma</td>
<td>Depending on extent of trauma</td>
<td></td>
</tr>
</tbody>
</table>

*Electromyographic findings.
†Central dysfunction includes apoplectic insult, hemorrhage, and other central nervous system disorders proved unequivocally by computed tomography or magnetic resonance imaging.

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Figure 1. Left, Magnetic resonance imaging (MRI) study of a 23-year-old woman with complete left facial paralysis of 30 months’ duration (September 1998). T1-weighted, T2-weighted, and gadopentetate (Gd-DTPA)–enhanced transversal MRI of the parotid gland depicts a large tumor of the lower deep portion of the left parotid gland (arrow). The tumor is fairly visible in native T1 image, but disappears after Gd-DTPA enhancement (repetition time, 600/2000 milliseconds; echo time, 20/80 milliseconds; field of view, 240 mm; slice thickness, 4 mm). Right, MRI study of the same patient in January 1997. No tumor is visible on the 2 contiguous lowest slices. The scout does not cover the tumor region. The results were interpreted as exclusion of tumor, idiopathic facial nerve paralysis.

Table 2. Details of 8 Patients With False-Negative MRI Findings*

<table>
<thead>
<tr>
<th>Patient Age, y/Sex</th>
<th>Time Since Paralysis Onset, mo</th>
<th>No. of MRI Studies</th>
<th>Presumed Cause for False-Negative MRI</th>
<th>Histologic Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>40/M</td>
<td>18</td>
<td>1</td>
<td>Perineural spread? size?</td>
<td>ACC</td>
</tr>
<tr>
<td>52/M</td>
<td>24</td>
<td>3</td>
<td>Perineural spread? size?</td>
<td>ACC</td>
</tr>
<tr>
<td>67/M</td>
<td>9</td>
<td>1</td>
<td>Tumor not covered</td>
<td>Acinic cell carcinoma</td>
</tr>
<tr>
<td>42/M</td>
<td>6</td>
<td>1</td>
<td>Similar signal intensity</td>
<td>BCC</td>
</tr>
<tr>
<td>23/F</td>
<td>30</td>
<td>3</td>
<td>Tumor not covered</td>
<td>BCC</td>
</tr>
<tr>
<td>57/F</td>
<td>24</td>
<td>2</td>
<td>Perineural spread? size?</td>
<td>ACC</td>
</tr>
<tr>
<td>55/M</td>
<td>14</td>
<td>1</td>
<td>Perineural spread? size?</td>
<td>ACC</td>
</tr>
<tr>
<td>63/M</td>
<td>16</td>
<td>1</td>
<td>Size?</td>
<td>Mucoperidermoid carcinoma</td>
</tr>
</tbody>
</table>

*MRI indicates magnetic resonance imaging; ACC, adenoid cystic carcinoma; and BCC, basal cell adenocarcinoma.
from the brainstem to the stylomastoid foramen, and contrast-enhanced Gd-DTPA MRI has become an essential part of the evaluation of patients with facial paralysis, even if the data remain somewhat inconclusive.11,12 Although Bell palsy has been considered a diagnosis largely made by exclusion, several reports of gadolinium-enhanced MRI for facial paralysis suggest that the diagnosis of Bell palsy in some instances can be confirmed by MRI rather than by deduction from the absence of abnormal findings.13-17 While MRI has become a valuable tool in evaluating patients with facial paralysis, its limitations should be recognized and must be interpreted in conjunction with the clinical presentation of facial paralysis.11

We present 8 patients with unilateral facial paralysis, all of them initially diagnosed as having Bell palsy. Initial and subsequent MRI showed no evidence of tumorous lesion. Although the history of each patient was not consistent with Bell palsy in regard to the long duration of the unilateral facial paralysis (>6 months without signs of regeneration), once the diagnosis was suggested by the negative results of the MRI studies, it was accepted in each case. No electrophysiological evaluation was ordered, and the patients were not appropriately followed up clinically by their physicians.

Unilateral facial paralysis that progresses beyond 3 weeks strongly suggests neoplastic involvement.1 An incomplete facial paralysis progressive to complete dys-
edged as exceptions but should demonstrate the need for neurotologic screening of every case of facial paralysis whether immediate or slowly progressive in onset.

Worse than gradual progression of facial paralysis is the remarkable persistence of a facial paralysis. Absence of facial tone or of any resumption of clinical function 6 months following paralysis suggests neoplasm and the patient must be treated accordingly.

Our patient group demonstrates the need to perform close electrophysiological monitoring in patients with atypical facial paralysis. The diagnosis of Bell palsy on the basis of MRI findings in some situations may result in a false sense of security in the management of patients with unilateral facial paralysis. Jackson et al wrote in 1980, Despite the technological sophistication of the various diagnostic modalities (CAT scan), they remain limited in their powers of resolution. False-negative data are not uncommon. It is therefore not unusual that diagnosis and therapeutic direction depend on the very direct approaches of surgery. In these circumstances, surgery serves as a diagnostic and therapeutic agent.

Nineteen years later we still suggest surgery as diagnostic and therapeutic agent in patients with peripheral facial paralysis and without signs of regeneration 6 to 8 months after paralysis onset. Individuals experiencing a facial nerve paralysis without any signs of regeneration 6 months after onset of paralysis and/or persistent electrophysiologic evidence of ongoing neuronal degeneration should undergo surgical exploration of the parotid gland and facial nerve, even if MRI studies show no tumorous lesion.

CONCLUSIONS

Magnetic resonance imaging studies may prove but never can exclude neoplastic lesions. Bell palsy is a diagnosis of exclusion. Electrophysiological findings and regular follow-up visits continue to be the most important aspects in the management of patients with unilateral, peripheral facial paralysis. Although MRI with and without Gd-DTPA enhancement of the parotid gland, the stylomastoid foramen, and the intratemporal segment of the facial nerve provides useful information, negative radiographic studies should not be taken as the final word indicating absence of neoplasms. In patients with long-duration palsies and no signs of regeneration, surgical exploration of the parotid gland and the facial nerve is compelling even with normal MRI findings.

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REFERENCES