Characteristics and Prognosis of Malignant External Otitis With Facial Paralysis

Ethan Soudry, MD; Ben Zion Joshua, MD; Jaqueline Sulkes, PhD; Ben I. Nageris, MD

Objective: To compare the characteristics and prognosis of patients with malignant (necrotizing) external otitis (MEO) with and without facial nerve palsy in today’s era of third-generation antibiotics.

Design: Comparative retrospective case series.

Setting: Department of Otolaryngology–Head and Neck Surgery, Rabin Medical Center, a tertiary care medical center.

Patients: Forty-eight patients with MEO diagnosed and treated from 1990 to 2004. Eight had facial paralysis and 40 had normal facial nerve function.

Main Outcome Measures: Clinical, laboratory, and imaging findings and survival.

Results: There was no statistically significant difference between patients with and without facial nerve involvement in terms of age, comorbidities, duration of complaints, physical findings, erythrocyte sedimentation rate, and bone scan findings. Computed tomography indicated a more progressive disease in patients with facial nerve involvement. However, no statistically significant between-group difference was found in overall survival.

Conclusion: Although facial nerve involvement is a sign of progression of MEO, it does not, by itself, worsen prognosis.

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T O OUR KNOWLEDGE, THE first comprehensive description of malignant (necrotizing) external otitis (MEO) was provided by Chandler1 in 1968. Thereafter, studies showed that involvement of the cranial nerves, most often the facial nerve, is associated with advanced infection and a mortality rate of up to 50%.2 The most common pathogen in MEO is Pseudomonas aeruginosa.

The treatment of MEO has evolved from surgery to aminoglycosides and semisynthetic penicillins to quinolones and third-generation cephalosporins. However, to our knowledge there are no studies of the effect of this change on the prognosis of MEO with facial paralysis. The purpose of this study was to compare the characteristics and prognosis of patients with MEO with and without facial paralysis.

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Involvement (Table 1). Facial paralysis was detected in 7 patients at presentation and developed during hospitalization in 1 patient (F+ group). The remaining patients (n = 40) were categorized as F−. Average age at diagnosis was 78 years in the F+ group and 71 years in the F− group. The most common comorbidity was diabetes (Table 1). Older age was related to a higher occurrence of diabetes and chronic renal failure in both groups (P = .02 and P = .05, respectively). The duration of patient complaints prior to hospitalization was 65 days in the F+ group and 53 days in the F− group. The difference was not significant (P = .79).

Clinical examination revealed edema of the ear canal in 7 patients (88%) in the F+ group and 29 patients (73%) in the F− group, and granulation tissue in 6 patients (75%) and 32 patients (80%), respectively. Average erythrocyte sedimentation rate was 77 mm/h in the F+ group and 70 mm/h in the F− group.

Bone scan findings were positive in all patients with facial nerve involvement and in 34 patients with normal facial nerve function. There was a positive correlation between presence of ear canal edema and a positive bone scan finding (P = .01).

Computed tomography was performed in 7 patients in the F+ group and 22 patients in the F− group. Evidence of canal bone destruction and of mastoid, temporomandibular joint, parapharyngeal, or nasopharyngeal involvement was found in several patients (Table 2). However, only mastoid involvement was significantly more common in the patients with facial paralysis (P = .03). Computed tomography findings of nasopharyngeal involvement (P = .004), bone involvement (P = .04), and temporomandibular joint involvement (P = .07) were correlated with a higher erythrocyte sedimentation rate. Bone involvement was also significantly correlated with a finding of granulation tissue (P = .02).

In-hospital management included local and systemic antibiotics. Cefazidime was given intravenously to 6 patients in the F+ group and 29 in the F− group. The other patients were treated parenterally with quinolones (except for 1 patient in the F+ group who was treated with intravenous voriconazole and 2 patients in the F− group who were treated with intravenous vancomycin) on the basis of the tissue culture results. All patients were treated with antibiotics for at least 6 weeks. When clinical findings demonstrated recovery, treatment was stopped, and a gallium scan was performed a week later. Negative findings on the gallium scan proved complete recovery. For patients who did not improve after 3 weeks of intravenous antibiotic treatment, surgery was considered.

Conservative treatment did not provide adequate response in 13 patients, and so they underwent surgical debridement. This included 4 patients in the F+ group and 9 in the F− group. In addition, 2 of 4 F+ patients and 3 of 9 F− patients underwent canal wall down mastoidectomy.

Median follow-up time was 10 months (range, 3.6-179 months) in the F+ group and 25 months (range, 0.6-144 months) in the F− group. During follow-up, 1 patient in the F+ group (13%) and 18 patients in the F− group (43%) died. However, comparison of the survival curves yielded no statistically significant difference between the 2 groups. However, power analysis yielded a value of 60% for this study sample. Factors found to be correlated with low survival were presence of ischemic heart disease (P = .07), history of cerebrovascular accident (P = .06), high erythrocyte sedimentation rate (P = .05), and nasopharyngeal involvement on computed tomography scan (P = .04).

Malignant external otitis is a severe infection of the external ear canal that spreads through the canal into neighboring tissues. Cartilage, bone, nerves, blood vessels, and adjacent organs may be affected. The most serious complication is skull base osteomyelitis with involvement of

Table 1. Characteristics of Patients With Malignant External Otitisa

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>With Facial Paralysis (n = 8)</th>
<th>Without Facial Paralysis (n = 40)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>Male</td>
<td>7</td>
<td>24</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>1</td>
<td>16</td>
</tr>
<tr>
<td>Side</td>
<td>Right</td>
<td>4</td>
<td>17</td>
</tr>
<tr>
<td></td>
<td>Left</td>
<td>4</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>Bilateral</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Bilateral</td>
<td>5 (62)</td>
<td>33 (83)</td>
</tr>
<tr>
<td>Ischemic heart disease</td>
<td></td>
<td>5 (62)</td>
<td>15 (38)</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td></td>
<td>3 (38)</td>
<td>9 (23)</td>
</tr>
</tbody>
</table>

a Unless otherwise indicated, data are reported as number or number (percentage) of patients. No significant differences were found for any comparison.

Table 2. Computed Tomography Findings in Patients With Malignant External Otitisa

<table>
<thead>
<tr>
<th>Finding</th>
<th>With Facial Paralysis (n = 8)</th>
<th>Without Facial Paralysis (n = 40)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bone destruction</td>
<td>4 (57)</td>
<td>6 (27)</td>
<td>.37</td>
</tr>
<tr>
<td>Mastoid involvement</td>
<td>6 (75)</td>
<td>8 (20)</td>
<td>.03</td>
</tr>
<tr>
<td>Parapharyngeal and temporomandibular joint involvement</td>
<td>4 (57)</td>
<td>7 (22)</td>
<td>.39</td>
</tr>
<tr>
<td>Nasopharyngeal involvement</td>
<td>2 (28)</td>
<td>3 (13)</td>
<td>.56</td>
</tr>
</tbody>
</table>

a Unless otherwise indicated, data are reported as number (percentage) of patients.
vital structures. Progression of the infection has been associated with multiple cranial nerve neuropathies, predominantly facial nerve neuropathy. The inflammatory process apparently interferes with the conductive abilities of the facial nerve, and in severe cases, the integrity of the nerve itself may be completely disrupted, with replacement by granulation tissue. Therefore, facial nerve decompression will not restore normal facial nerve function. Facial nerve involvement is reportedly associated with a mortality rate of up to 50%.

In 1985, Corey et al reviewed the findings of 83 patients with MEO reported in the literature up to that time. Fifty-eight had facial paralysis (70%). The paralysis presented relatively late in the course of the disease (after 2 months) and indicated advancement of the disease process. However, it did not clearly worsen outcome.

In the present series of patients with MEO, we did not find a statistically significant difference between patients with and without facial nerve involvement in terms of age, comorbidities, duration of complaints, physical findings, erythrocyte sedimentation rate, or bone scan findings. In the group as a whole, however, older age was correlated with diabetes and chronic renal failure but, surprisingly, not significantly with survival. Ischemic heart disease and a history of cerebrovascular accident were correlated with lower survival rates. Elevated erythrocyte sedimentation rate was correlated with advanced disease findings on computed tomography (bone, nasopharyngeal, and temporomandibular joint involvement) and poorer survival.

Computed tomography in the patients with facial paralysis showed significantly more frequent involvement of the mastoid in addition to relatively more frequent involvement of the parapharyngeal space and nasopharynx. These findings provide an anatomic confirmation that facial nerve involvement is indeed a sign of disease progression, and they might explain its underlying mechanism, via either the mastoid or an extracranial route. This progression, and they might explain its underlying mechanism, via either the mastoid or an extracranial route. This progression, and they might explain its underlying mechanism, via either the mastoid or an extracranial route. When overall survival rates were compared between the groups, no statistically significant difference was found. This suggests that although facial nerve involvement is a sign of disease progression, current antibiotic therapy is apparently sufficient for good recovery, similar to that in patients with MEO and normal facial nerve function. Facial paralysis by itself does not seem to be an adverse prognostic factor in MEO.

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Author Contributions: Dr Nageris had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: Soudry and Nageris. Acquisition of data: Soudry and Joshua. Analysis and interpretation of data: Soudry, Joshua, Sulkes, and Nageris. Drafting of the manuscript: Soudry, Joshua, Sulkes, and Nageris. Critical revision of the manuscript for intellectual content: Soudry and Nageris. Statistical analysis: Sulkes. Administrative, technical, and material support: Soudry. Study supervision: Nageris.

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REFERENCES