The Journal of Laryngology and Otology
March 1987. Vol. **101.** pp. 205–210

Malignant external otitis*

by

A. Babiatzki and J. Sadé (Tel Aviv, Israel)

Malignant external otitis (MEO) has been described as a clinical entity relatively recently (Malzer and Kelemen, 1959; Chandler, 1968, 1972, 1977). This is a serious pseudomonas infection of the external ear which, when untreated, may extend towards the base of the skull. MEO occurs shortly in diabetics. The purpose of this study is to describe in detail the characteristics of this important disease, as found by us in a relatively large number of patients, as a supplement to the information accumulated so far by others.

Our definition of MEO is the following:

1. A clinically severe external otitis which does not improve after 8 days of conservative treatment.
2. Pain, especially at night, which worsens after this period.
3. The presence of granulation tissue, mostly over the base of the external ear canal, mandatorily.
4. A culture of pseudomonas pyoceaneus, usually.†
5. A positive temporal bone scan, sooner or later.
6. Diabetes, in most patients.

Material

A. *The patients and the course of their disease:*

During the years 1973–1985, 50 patients with a diagnosis of MEO came under our care; 36 of the 50 (72 per cent) were males, 14 or (28 per cent) were females. One of our patients was an Arab, but all the rest were Jews, of various ethnic origins: Ashkenazi as well as Sfardic, Eastern, North African and Yemenites, no one group showing a preponderance over any other. The relevance of these numbers can be appreciated in relation to the general composition of our patients which is approximately 75 per cent Jews and 25 per cent Arabs. Our youngest patient was 32 years old, the oldest 92—the mean age being 67.2; most patients, i.e. 96 per cent, were at least 50 years old.

B. *The season:*

31, i.e. 62 per cent of our patients had their first symptoms during the months of July, August, September and October—which is the season when external otitis is most rampant in Israel. The distribution of MEO amongst our patients during the years 1973–1985 was unpredictable—some years showing more patients, some less—some years none at all.

C. *The History:*

The central symptom of this interesting

* This study is part of Dr. Babiazki's study which is required by the ENT board in Israel.

† As will be seen below, we have had one case which did not grow pseudomonas—yet, as all the other features fitted the clinical picture, we considered it as MEO.

https://doi.org/10.1017/S0022215100101549 Published online by Cambridge University Press

disease is severe earache, which is mostly distressing at night. However, only five patients presented themselves after one week of such pain. Most patients (30, or 60 per cent) came after one to four weeks of such suffering and 15 patients (30 per cent) after having been sick for over one month. None of our patients had suffered previously from an ear infection; however, three patients had had recent ear insults—one had had a ventilating tube placed in his ear drum, and two had had their external ears irrigated to remove wax.

D. *Physical examination:*

All patients presented a swollen, tender external ear canal as well as some granulation tissue. In 68 per cent the granulation tissue was found mainly at some place along the floor of the external canal.

E. *Bacteriology:*

In 49 patients a heavy culture of pseudomonas aeruginosa was grown from the exudate. Three were accompanied by staphylococus aureus, three by proteus vulgaris and another two by Klebsiella. One patient with a classical clinical picture of malignant external otitis persistently grew only staphylococus aureus as well as staphylococus albus.

F. *Diabetes:*

Thirty-four (or 68 per cent) of our 50 patients had diabetes, in 31 of whom the diabetes had previously been known, while in three it was discovered only when the patient was hospitalized. Sixteen (32 per cent) had no diabetes and the glucose tolerance test was also within normal limits. Of the 34 diabetic patients, 21 (62 per cent) were treated with insulin, and 13 (38 per cent) required only a special diet or oral treatment.

Altogether we had 42 per cent severe diabetics, 26 per cent light diabetics and 32 per cent with no diabetes at all.

G. *General blood counts and blood chemistry:*

Altogether no specific features were detected so far as the blood tests were concerned. The red and white blood counts did not fluctuate with the course of the disease, usually being within normal limits. The patients in whom the disease progressed or in whom its control was difficult or impossible, showed very high sedimentation rates. Blood proteins and gamma globulins were found to be within normal limits. The percentage of patients with kidney damage was 20 per cent, when high values of urea and creatinine were detected.

H. *X-ray and technetium scan:*

Bone destruction of the pyramid tip was discovered on base of skull X-ray films in two patients—both of whom succumbed to their disease. It is not impossible that CAT scanning might reveal a higher incidence of bone destruction. Of our 39 patients who were scanned with technetium, 30 (77 per cent) showed an increased or positive uptake but nine (23 per cent) did not show any such increase. In view of the fact that such an uptake may sometimes remain 'positive' for as long as two years, no post-treatment follow-up of the uptake was undertaken.

I. *Pathology:*

1. 12 patients presented involvement of the middle ear and mastoid region during the course of their disease. In one case, an extension from one ear to the other through the base of the skull occurred; this was evidenced not only by the clinical course but also by involvement of the contralateral pyramid in the destructive process as seen by X-rays of the base of the skull. The extension to the other ear manifested itself first by pain, later by middle ear effusion; the pyramid was seen to be involved on X-rays.
2. In 12 patients, the infection extended anteriorly, breaking through the tympanic plate into the parotid region.
3. Five patients showed cranial nerve

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involvement of the VIIth through XIIth and three of these five died of their disease.

J. *Treatment:*

Over the years our treatment has evolved, like that of others (Chandler, 1968, 1972, 1977; Doroghazi, 1981; Meyerhoff *et al.*, 1977; Lucente *et al.*, 1982, 1983; Zaky and Bentley, 1976; Strauss and Aber, 1980; Mendez and Zuenzer, 1979; Ostfeld *et al.*, 1981; Parisier *et al.*, 1981; Dinapoli and Thomas, 1971; Schwartz *et al.*, 1971), from extensive surgery and diverse antibiotic treatment towards minimal surgery, amounting to debridement, supplemented by specific long-term antibiotics. Time has taught us that total surgical eradication is impractical because of deep penetration into the skull base as well as lack of demarcation lines in this diffuse pathological process; furthermore, we have at times had the impression that a non-involved middle ear (or non-involved mastoid cavity) has become infected after extensive surgery. The antibiotic treatment was bewildering to us until 1978, when the antibiotics to which pseudomonas aeruginosa was specifically sensitive began to be available to us. A big change also occurred when it became clear that, in most cases, large doses of some of the semi-synthetic penicillins in combination with appropriate aminoglycosides, if given for 6 to 8 weeks, will subdue the infection, which otherwise pursues a malignant course. Semi-synthetic penicillins or aminoglycosides, given alone, may produce resistant bacteria rather swiftly. The nephrotoxicity of some of the antibiotics has led us to treat these patients in co-ordination with the nephrologic department, and creatinine and urea are monitored twice a week.

K. *The outcome:*

The outcome for patients with malignant external otitis is a function of their age, their general vascular status and probably their immunological state—a situation about which very little is known even today. During our 'first period', i.e., until appropriate antibiotic treatment was available, two out of our first 10 patients died of their disease in great agony. Since 1978, out of 40 patients only three have died, and in one of the cases the death was due to myocardial infarction. The overall mortality rate is 10 per cent but this increases to 60 per cent in cases with cranial nerve involvement.

The follow-up of our cases included 38 patients; 7 patients could not be traced. Of the 38 patients whom we were able to trace, 11 were found to have died, over the years, of various ailments associated with old age, such as cardiovascular diseases or cancer.

Discussion

In 1959, Meltzer and Kelemen described a diabetic patient who died in great agony from external otitis which penetrated deep into the skull base. This was the first MEO described. The disease itself has become better recognised only in the last decade or so, possibly because it involves elderly individuals whose life span has become prolonged to expose them to this disease when their resistance is low. Its lethal character in untreated patients brought Chandler (1968) to call it 'Malignant External Otitis'. In 1977, Chandler summarized his personal experience, presenting 72 patients. Sixty-eight of these were elderly diabetics but neither Chandler nor we found any correlation between the severity of the disease and the severity of the diabetes; yet those of our patients who did have diabetes had had it for a long time, as seen from the character of their vascular involvement when their fundus was examined. Chandler's observation (1968, 1972, 1977) that MEO is due to pseudomonas in diabetics was found by us to be true; but with one exception as one of our patients (whose symptoms were exactly the same as all the others) grew only staphylococcus aureus and staphylococcus albus in repeated cultures. However, this patient should be viewed as an exception.

Since Chandler's classical reports, several reviews describing sporadic cases of MEO have appeared (Doroghazi, 1981; Meyerhoff *et al.*, 1977; Lucente *et al.*, 1982, 1983; Zaky and Bentley, 1976; Strauss and Aber, 1980; Mendez and Zuenzer, 1979; Ostfeld *et al.*,

https://doi.org/10.1017/S0022215100101549 Published online by Cambridge University Press

1981; Parisier *et al.*, 1981; Dinapoli and Thomas, 1971; Schwartz *et al.*, 1971; Raines and Schindler, 1980; Mader and Love, 1982), all stressing that the disease originates in the external ear canal and can spread to the soft tissues beneath the temporal bone. This spread leads to osteomyelitis of the skull base and potentially to paralysis of the facial and other cranial nerves.

Later, other intracranial complications, such as sigmoid sinus thrombosis, will occur if the disease continues unchecked, and will eventually lead to death.

One exception is Badrawy *et al's.*, (1975) review which sees MEO as originating in the mastoid—spreading from 'inside out' into the external ear and base of skull.

We believe that the evidence at hand really points to the external ear as the port of entry of MEO, as evidenced by the early symptomatology of external otitis as well as its highest seasonal incidence in the summer, when external otitis is rife. It is, therefore, not surprising that MEO has been reported mostly from humid, warm places and is found much less in cold climates. Israel presents some summers which are excessively hot and humid, and during those summers external otitis has been more frequent than usual, as also has MEO. Local insult to the external ear has been observed by us in about 8 per cent of cases. Extension of the inflammation and its prognosis depend on prompt diagnosis and correct treatment. Mendez and Zuenzer (1979) reported some correlation between the clinical and radiographic findings in nine patients, and they reported that in the early stage of the disease the only radiographic finding was clouding of the mastoid air cells; later they found bone destruction, which could extend to the middle ear cavity, temporomandibular joint and base of skull—yet our own findings show mastoid involvement in only 6 per cent.

Ostfeld *et al.* (1981) described three cases of malignant external otitis and they were the first to stress the diagnostic value of bone scanning with TC 99; they found a good correlation between positive findings in the bone scan and the clinical course of malignant external otitis. These findings were later confirmed by Parisier, *et al.* (1981), who demonstrated positive bone uptake in 18 patients. These authors also pointed out that Gallium-67 scanning had helped them to evaluate the effectiveness of antibiotic therapy and to determine when antibiotic therapy could be discontinued. Similar statements have been made by Strauss and Aber (1980), who reported six patients. Clinically the diagnosis is quite simple if one should be on the alert with elderly patients, especially diabetics, who have a pseudomonas external otitis which fails to improve markedly with local treatment within about a week. When treatment fails, the prognosis is grave and the end is very painful; in our five patients who succumbed to the disease, the pain became resistant even to morphine. Chandler (1977) reported an overall 32 per cent mortality rate, increasing up to 50 per cent once facial nerve paralysis has appeared. Myerhoff *et al.* who in 1977 summarized all the cases of progressive necrotizing pseudomonas infections published in the English Literature, found an overall mortality rate of 35 per cent, rising to 51 per cent in cases with facial nerve paralysis, and to 75 per cent in cases with multiple cranial nerve paralyses. However, today, with appropriate antibiotics most patients can be cured before the disease progresses. The treatment should include a combination of a semi-synthetic penicillin with an appropriate aminoglycoside, for a period of at least three weeks up to twelve weeks. Some effective cephalosporins are also available today.

Doroghazi (1981) reported 21 cases, and he also stressed the need for a combination (bi-drug) antibiotic therapy. All his 13 patients in whom therapy was successful had received such a combination of aminoglycoside and semi-synthetic penicillin, whereas all six patients with recurrent disease occurred when only one antibiotic was used. There are several other reports which also stress the bi-drug antibiotic therapy (Chandler, 1968; Doroghazi, 1981; Meyerhoff *et al.*, 1977; Lucente *et al.*, 1983; Zaky and Bentley, 1976; Strauss and Aber, 1980). Of our last 40 patients who received bi-drug therapy 37 were cured of the infection (one died from acute myocardial infarction). It is very encouraging

https://doi.org/10.1017/S0022215100101549 Published online by Cambridge University Press

to see how, within a few days of correct treatment, the patient's intense earache, especially night pain, disappears. Antibiotic treatment has its medical and economic problems; it is costly, as it should be administered diligently for 6–8 weeks. One should also take into account the state of the patient's kidneys, to which aminoglycosydes are often toxic, i.e. elevated creatinine and urea as well as sodium retention should be watched for. The reason why two types of antibiotics need to be administered simultaneously is that otherwise the pseudomonas micro-organism becomes swiftly resistant. Short-term antibiotic treatment often leads to exacerbations which are difficult to control; hence, despite all the inconvenience involved in prolonged treatment, this should be continued even though the patient's symptoms have completely disappeared. Indeed, the nephrologist should become an active partner once the kidneys show the slightest signs of failure. The semi-synthetic penicillins, which should be administered intravenously, can sometimes be given through a subclavian route. In most cases, extensive surgery is anything but useful, though regular conservative debridement should be the rule; granulation tissue and necrotic cartilage are removed and this can often be done under local anaesthesia. At the same time, 24 per cent of our patients presented middle ear involvement, and in 24 per cent a break through anteriorly towards the parotid region was observed—needing wider debridement. More aggressive treatment was advocated by Raines and Schindler (1980), who reported three cases in whom a subtotal temporal bone resection was performed 'in order to gain access to the primary focus of the infection and provide adequate drainage', their patients having demonstrated an abscess cavity in the soft tissues at the base of the skull. However, this drastic treatment is not advocated by most authors, and we think that the encouraging results of our conservative treatment do not justify it. Another modality of treatment has been discussed by Mader and Love (1982), who advocated adjunctive hyperbaric oxygen therapy in addition to conservative antibiotic therapy.

It is obvious that MEO is not a uniform entity; it can appear in a mild or a severe form, with a broad spectrum between the two extremes. This may depend on the virulence of the bacterium, the resistance of the patient, the length of time between the onset of the external otitis and the time when the patient arrives for treatment, and possibly previous treatment. Our patients arrived relatively early, some after being sick for about one week, but others after as long as two months. The early arrival of our patients for treatment may be related to our social medical system in Israel, which encourages early visits to the physician. The question obviously arises whether all MEO patients should receive the same long-term treatment. Should a patient with a two-week history of MEO receive the same treatment as an eight-week patient, especially if the second has a facial nerve paralysis?

The answer is that, most probably, the first one should receive shorter treatment; the other longer. Yet, until clinical trials have led us to the correct answer, we are bound to adopt some arbitrary clinical policy. We must try to be practical and, while our average patient is treated for six weeks, those with neurological complication are treated for a longer time. The care of MEO patients is very time-consuming and needs all that a physician can give. Our thanks go to all the associates and physicians in our department who contributed to the treatment of the patients described in this study, and especially to Dr Joseph Weinberg.

Summary

During the years 1972–1985, 50 patients with malignant external otitis (MEO) were seen in our department. All our patients complained of severe earache; they presented initially with an apparently simple external otitis, but failed to improve when the usual measures were adopted. They all presented with granulation tissue in the external ear canal, and five of our patients had multiple cranial nerve involvement. MEO is in effect a severe external otitis which, if untreated, proceeds towards an osteomyelitis of the skull base. MEO is more prevalent in the summer,

https://doi.org/10.1017/S0022215100101549 Published online by Cambridge University Press

when external otitis is rampant. In some years, a relatively large number of these patients appear; in others there are none. The reason for this is unknown. In Israel, the disease is more prevalent in Jews than in Arabs. Diabetes was present in 68 per cent of our patients—severe diabetes in 42 per cent, mild diabetes in 26 per cent but 32 per cent of our patients were diabetes-free. The only otological past history in our patients was of a recent traumatic insult to the external ear canal; this was the case in about 8 per cent of them. Today, the treatment of choice of this important disease is local debridement supplemented by appropriate antibiotic treatment for 6–8 weeks. This should include some semi-synthetic penicillin to which pseudomonas aeruginosa is sensitive, combined with an appropriate aminoglycoside. During the earlier years of our encounter with MEO, two of our 10 patients died of it; later on, when we learned better how to treat it, the mortality rate decreased. While the prognosis from the disease itself is now good when properly treated, these patients are at high risk, a large percentage of them succumbing within a few years to cardiovascular and allied ailments.

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Address for correspondence:
Professor Jacob Sadé,
Department of Otolaryngology,
Meir Hospital,
Kfar Saba,
Israel 44 281.

https://doi.org/10.1017/S0022215100101549 Published online by Cambridge University Press