*Int. Adv. Otol. 2010; 6:(2) 274-276*

SHORT REPORT

# Malignant External Otitis; Changing Faces

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The introduction of new antibiotic, oral quinolones, seems to have led to the belief that this form of treatment suffices to 'cure' Malignant External Otitis. Indeed, the high mortality rate has decreased significantly ever since.

We argue, however, that the measure of 'cure' has not been clearly determined. In our view, it is paramount that we need to consider the patient who is faced with the possibility of lasting residual damage to the facial nerve and its function.

Although facial nerve paralysis has traditionally been designated to be a poor prognostic factor, we would now be well advised to recognize it as the most important indicator of residual morbidity.

*Submitted : 20 January 2010* *Accepted : 05 February 2010*

Malignant external otitis (MEO) is a devastating disease that poses a difficult therapeutic challenge. MEO tends to affect elderly patients with diabetes. It presents with severe otalgia, purulent otorhoea and granulations in the ear canal at the bone-cartilage junction. Spread of infection occurs through the fissures of Santorini and the tympanomastoid suture, leading to involvement of the stylomastoid and jugular foramina. Cranial nerve palsies may arise, and the mortality rate was high (about 50 per cent in the presence of facial nerve palsies in older series), hence the name malignant despite its infectious etiology. The most common causative organism is Pseudomonas Aeruginosa.[1-4] The treatment of MEO has evolved from primarily surgical to one in which prolonged medical therapy of the underlying osteomyelitis with limited surgical debridement leads to 'cure'.[5]

Recently, we read that surgical intervention is no longer indicated for MEO. "Surgical debridement, once the mainstay of treatment, has been superseded by systemic antibiotic therapy". [4] This, because of the case of one patient suffering from a facial paralysis that did not improve despite decompression of the facial nerve. Incidentally, of the other 5 patients in that study who did not undergo surgery, not a single one experienced a complete recovery either.[4] Furthermore, the conclusion was also based on an article in an authoritative journal. [6] "In this article: Rubin Grandis et al. assert that there is no role for surgical management in MEO other than diagnostic biopsies". Ever since the introduction of the newer types of antibiotics, however, not even one proper study has been published with the results of surgical intervention, extensive netoyage/ debridement of the infected bone and other tissue, in combination with these antibiotics.

Statements such as: "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 decompression will not restore facial nerve function", are in our view, based on results from a too distant past.[2] On the other hand, the following statement remains to be the real problem, especially considering the fact that the facial nerve is involved in a quarter of all patients.[7] It may be that nerve involvement occurs because of infection or bacterial toxins directly inhibiting neurotransmission. Therefore, the longer the length of time that the nerve is exposed to toxins, the less likely it is to recover. Because the seventh nerve is usually the first to be affected because of its proximity to the external ear canal, it is liable to be affected for a longer period of time.[4]

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The authors declare that they have no conflict of interest.

***What is 'cure'?***

Although the introduction of these new types of antibiotics has indeed led to a decline in the high mortality rate from this disease, we believe that the notion that surgical intervention is no longer indicated deserves a critical annotation. First, in most studies there appears to be little or no recovery of facial nerve function once this disease has affected the nerve. Secondly, P. Aeruginosa has developed an increasing resistance against many types of antibiotics.[1,3]

Third, antibiotics have to be administered for an extended period of time with possibly serious consequences for both the patient and the continued increase in resistance to antibiotics.

In the fourth place we have to question whether the, admittedly, meager results of surgical intervention in the past, even when performed by excellent surgeons, would not have been better if one would have been able to make use of the currently available antibiotics.

Fifth, we need to ask ourselves whether the lack of surgical intervention actually amounts to under treatment (under treatment has been called the major factor for recurrence)?[8]

Finally, as a rule, antibiotics cannot adequately penetrate pus and necrotic tissue, which is why this should be surgically removed. It has been known for centuries, and therefore has become a medical adage that "where there is pus it should be evacuated." (ubi pus, ibi evacua). In our opinion the same applies to necrotic tissue.

Of course surgical intervention should not be the only (mono) form of treatment. Antibiotics will remain to be the mainstay. The base of the skull area is an anatomically complex area. Which means there often is a significant risk of causing damage during surgery, without any real chance at the complete removal of the infection.

However, the question remains to be whether a combination therapy of surgical intervention, (debridement, mastoidectomy, subtotal pretrosectomy, facial nerve decompression) by an experienced surgeon, together with an appropriate course of antibiotics (two different types perhaps?) could speed up the process of recovery from infection, shorten the period of medicinal therapy and most importantly, improve the functioning of the facial nerve.

The existing literature does not provide an answer to that question. Apart from the fact, that in recent years there have not been any studies focussing on combination therapy, the functioning of the N. facialis has been completely underexposed. Even in studies with an emphasis on the group of patients with cranial nerve palsies the focus remains to be on decreasing the mortality alone.

Besides, treatment has oftentimes been ceased when "clinical findings demonstrated recovery". In this case, it is also implied that there are no further signs of infection -and thus no reason for treatment-, because the Gallium-scan has become negative.[1,2]

In most studies there is no description of a reliable measure for the severity of the paralysis, neither at the onset of paralysis, nor in the follow-up. (Incidentally, a more reliable grading scale describing the severity of paralysis than the currently used House Brackmann scale would be preferable.) What's more, whenever in the aftermath the functioning of the facial nerve is described, it often turns out that a large percentage of patients did not enjoy a complete recovery of facial nerve function.[9,10]

We are of the opinion that even though Lancet Infectious Diseases, of course, is a renowned journal, one should not simply adopt all published conclusions. The article by Rubin Grandis et al. does not address the recovery of the facial nerve function either. In our view, an optimal form of treatment has not been found, especially not with regard to the morbidity.[1]

Like others before us, we still have a strong preference for combination therapy.[11] Or as Benuck and Traisman wrote: "surgical intervention is warranted if there is radiographic evidence of osteomyelitis, mastoiditis, cranial nerve palsies, or lateral sinus trombosis. In such instances canaluloplasty, cartilaginous or bony debridement, mastoidectomy, and cranial nerve decompression is indicated. Surgical exploration may be necessary if medical management fails to demonstrate clinical improvement."[12]

## Conclusion

Although facial nerve paralysis has traditionally been designated to be a poor prognostic factor, we would now be well advised to recognize it as the most important indicator of residual morbidity. [4,13] As long as the functional recovery of the nerve remains limited, we simply cannot afford to lean back and accept the conclusion that antibiotics alone will cure the patient.

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