Necrotizing otitis externa

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NECROTIZING otitis externa, first described by Meltzen and Kelemen in 1959, is now well documented as a clinical entity consisting of Pseudomonas infection of the external auditory meatus in diabetic subjects characterized by severe pain and a potentially fatal outcome. Persistent granulation tissue in the floor of the external canal, at the junction of its bony and cartilagenous parts, is an important pointer to the diagnosis of this condition.

The purpose of this paper is to describe three further cases and to suggest a relationship between the progress of the disease and the degree of pneumatization of the temporal bone.

Case 1. 60-year-old diabetic female who presented with severe left-sided earache. She had gross perichondritis with abscess formation in the region of the concha. This was incised under general anaesthesia and the meatus dilated sufficiently to allow a speculum to be introduced. A large polypoid swelling was seen to arise from the floor of the meatus and was removed. The tympanic membrane was normal. Pseudomonas was cultured from the pus while histology of the polyp showed granulation tissue. The patient was relieved of the pain but within a week the abscess had reformed together with a number of small satellite abscesses. It was felt that total excision of the pinna was indicated but as the patient refused this, wide excision of necrotic cartilage and overlying skin was performed under Carbenicillin cover. The ear healed well, leaving a deformed nodular skin tag for an auricle. X-rays at the time of presentation showed a dense sclerotic mastoid.

Case 2. 58-year-old male diabetic presented with excruciating pain of some weeks' duration during which he had been treated with all sorts of local and systemic antibiotics. Examination showed gross perichondritis with closure of the external meatus and a greenish purulent discharge. Pseudomonas was cultured from the aural swab. X-rays showed well pneumatized mastoids. The cells on the affected side were cloudy and the cell outlines blurred. Exploration of the mastoid was carried out under Carbenicillin cover. There was marked thickening of the periosteum over the mastoid process. The bone was necrotic, the cell-lining grossly oedematous. The inflammatory process extended into the tip of the mastoid but the antrum and middle ear appeared healthy. Wide excision of diseased tissue was performed retaining a functioning middle ear. Relief from pain was immediate. Two months post-operatively he has a scanty discharge from the open mastoid cavity but is otherwise well and free from pain.

Case 3. 50-year-old female diabetic presented with severe pain, swelling and discharge from her right ear. X-rays showed markedly cellular mastoids. The apparent cloudiness of the cells was ascribed to oedema of the overlying tissues. A diagnosis of otitis externa was made and the patient started on Ampicillin pending the results of swab culture. With persistence of pain and the development of pre- and post-auricular swellings it was decided to explore the mastoid.

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At operation the mastoid cells were necrotic and full of oedomatous mucosa in the lower half of the mastoid process. The cells nearer the antrum appeared healthy as was the middle ear. A sinus track was found leading from the floor of the external meatus to a parotid abscess. A modified radical mastoidectomy was performed and the parotid abscess drained through a separate incision. The patient was well for some weeks when the pain returned and she developed facial palsy. The ear was re-explored, more necrotic bone removed and a radical mastoidectomy completed. The facial nerve was intact in its canal. The patient's condition continued to deteriorate and some time later she developed paralysis of the IX, X, XI cranial nerves with a retro-pharyngeal abscess which drained spontaneously. X-rays showed rarefaction and bone erosion in the region of the petrous pyramid suggestive of osteomyelitis (Fig. 1). She died within a week from meningitis and pneumonia.

Discussion

Otitis externa is a common condition in ENT practice. It responds readily to treatment and heals completely, usually without complications.

Extension beyond the confines of the ear is prevented by an intact perichondrium. If the perichondrium is damaged, perichondritis with or without abscess formation results.

Infection will spread into the surrounding tissues if the causative organism is (1) particularly virulent, or (2) unresponsive to the antibiotics normally used, e.g. Pseudomonas, or (3) the patient's resistance to infection is diminished, as in diabetes. Spread of infection is facilitated by performed pathways such as the fissures of Santorini.

Necrotizing Otitis Externa appears to be such an infection, the cellulitis spreading into the soft tissues at the base of the skull in the roof of the parapharyngeal gutter. Its spread is inexorable unless treated—but slow. Bone is only secondarily involved. Whether the temporal bone is included in the inflammatory process or not depends, I believe, on the degree of its pneumatization. The more sclerotic the bone the more resistant is it to infection.

Mention of the degree of pneumatization of the mastoid is made in only 4 of the 13 cases presented by Chandler (1968). Of these one was sclerotic and the disease spread away from the temporal bone, though it was severe enough to need total parotidectomy, removal of the ascending ramus and condyle of the mandible, and the entire tympanic plate and styloid process. The other three cases had a cellular mastoid and were extensively involved in the disease process. Likewise the two cases described by Evans and Richards (1973), and the first case of Meltzer and Keleman (1959).

It would appear that the cellulitis readily breaks through the thin cortical bone of a cellular or diploetic mastoid. Further spread is facilitated by the nature of the terrain. Coalescence of air cells due to hyperaemic osteoporosis and pressure necrosis of bony cell walls takes place as in acute mastoiditis secondary to acute otitis media—only in this case the spread of infection is inwards from the mastoid process towards the antrum. Haematogenous spread is likewise facilitated by the vascular marrow spaces.

When the cellulitis reaches a sclerotic mastoid, both the thick cortical bone and the absence of marrow spaces and air-cells militate against spread. This is a

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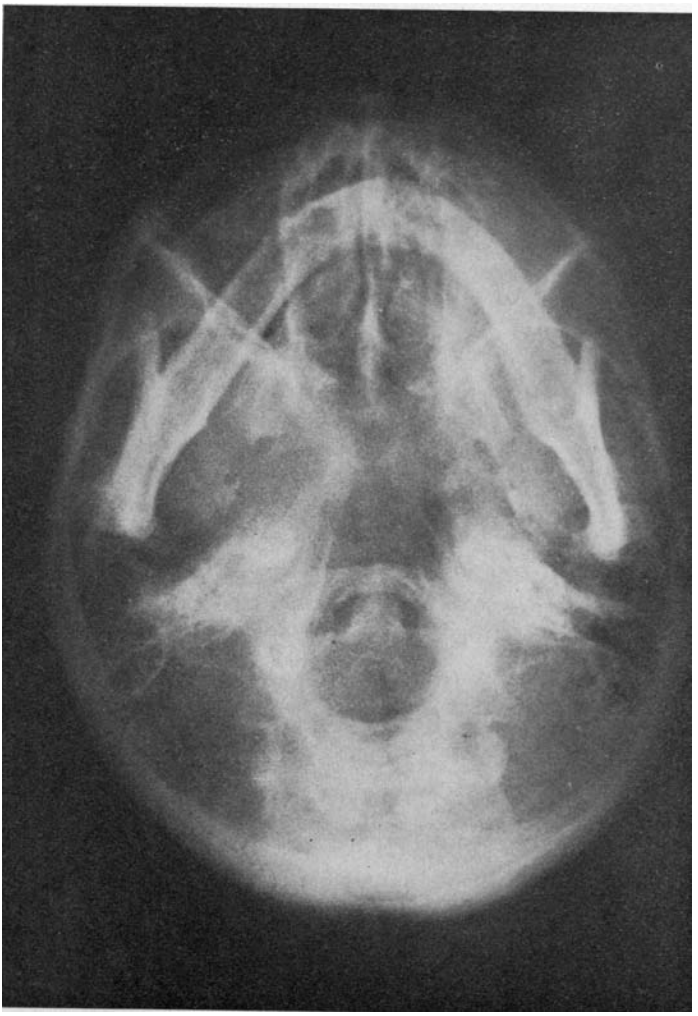


FIG. 1.
Showing rarefaction and bone destruction in the region of the right petrous apex.

similar state of affairs to that obtaining in chronic mastoiditis where slow penetration of acellular bone by granulation tissue accompanied by osteoporosis is the rule.

If it is indeed the case that progress of the disease varies with the degree of pneumatization of the mastoid, mastoid X-rays at the time of presentation will divide these cases into two groups:

1. Those with sclerotic mastoids who will do well with appropriate antibiotic treatment and control of diabetes, and possibly require soft tissue surgery.

2. Those with cellular mastoids. In these cases X-rays should be repeated at weekly intervals during the course of the disease and any sign of bone involvement taken as an immediate indication for mastoid surgery.

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Summary

Three cases of Necrotizing Otitis Externa are described and a suggestion made of a possible relation between the severity and progress of the disease and the degree of pneumatization of the mastoid.

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