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Clinical records

Middle ear tuberculosis

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IN the early 18th century Jean Louis Petit gave an accurate clinical description of tuberculosis. Almost 100 years later (1882) Robert Koch discovered the tubercule bacillus and just one year later Eschle demonstrated it in a lesion of the ear.

Although otologists have been aware of the existence of this disease in the middle ear for 96 years it continues to be a challenge and the diagnosis of the disease is usually missed preoperatively. In 1915 Logan Turner reported that 50 per cent of cases of otitis media in the first year of life were due to tuberculosis, the percentage dropping to 2 per cent by adolescence.

Cases reported in the literature nowadays are rather scanty. The amazing thing about middle ear tuberculosis is the change that has occurred in its clinical behaviour. The description given by various authors some 30 years ago differs considerably from the one found today.

The incidence of tuberculosis may vary from country to country, depending on the socio-economic situation and on the effort put into the prevention and cure of this disease. In West-Germany the registered cases of pulmonary tuberculosis for December 1977 were 311,277 or 131·9 per 100,000 of the population. The number of extrapulmonary cases registered was 13,530 or 22 per 100,000 of the population.

Material and method

Between 1967 and July 1979 approximately 18,000 ears were operated on in the university E.N.T. clinic of Tübingen, West-Germany. In 4,000 cases biopsy specimens from the middle ear were examined, 14 of which were proved to be cases of middle ear tuberculosis. The oldest patient was 69 years old and the youngest 10 months. Out of these 14 cases a correct preoperative diagnosis was made in only one case, and this was mainly because the patient was suffering from active pulmonary tuberculosis. He had a unilateral odourless discharge, and the tympanic membrane appeared granular and friable. The clinical findings of these cases varied considerably, and it is perhaps this variability which makes it difficult to diagnose the disease. However, there are certain important clinical facts which, if kept in mind, should lead the clinician always to suspect this disease. Nine patients had a central perforation. Out of these, 5 had granulation tissue on the tympanic membrane; 2 had large polypi protruding from central perforations (Figs. 1 and 2) and two had dry perforations. In 5 patients the tympanic membrane was intact, and one was diagnosed as having otosclerosis. Two cases had a bulging drum: one of these had a vascular bulge

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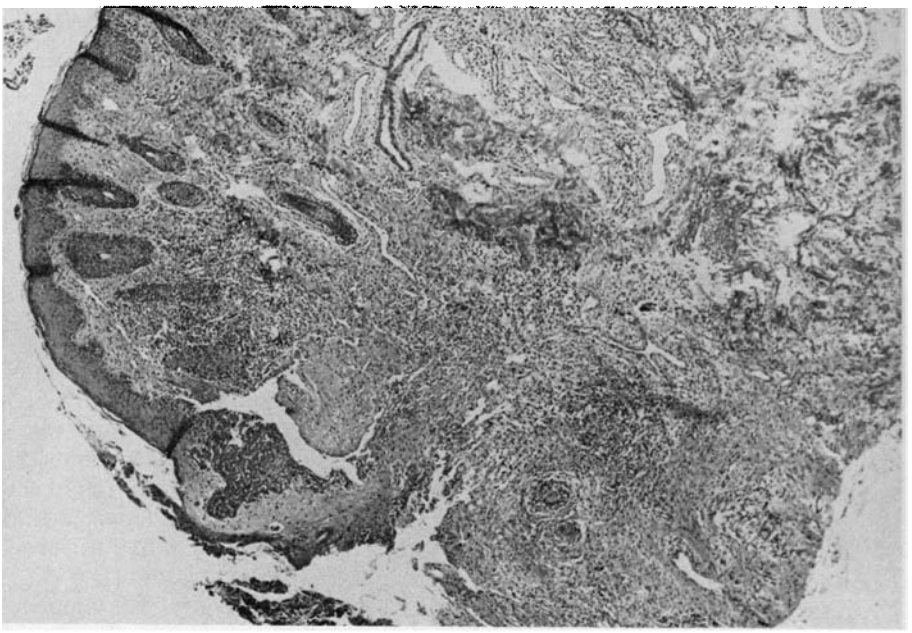


FIG. 1

Photo-micrograph of polyp covered with squamous epithelium. Note multiple giant cells, central necrosis with epithelioid cells. Haematoxylin-Eosin. (Magnification $\times 62.5$).

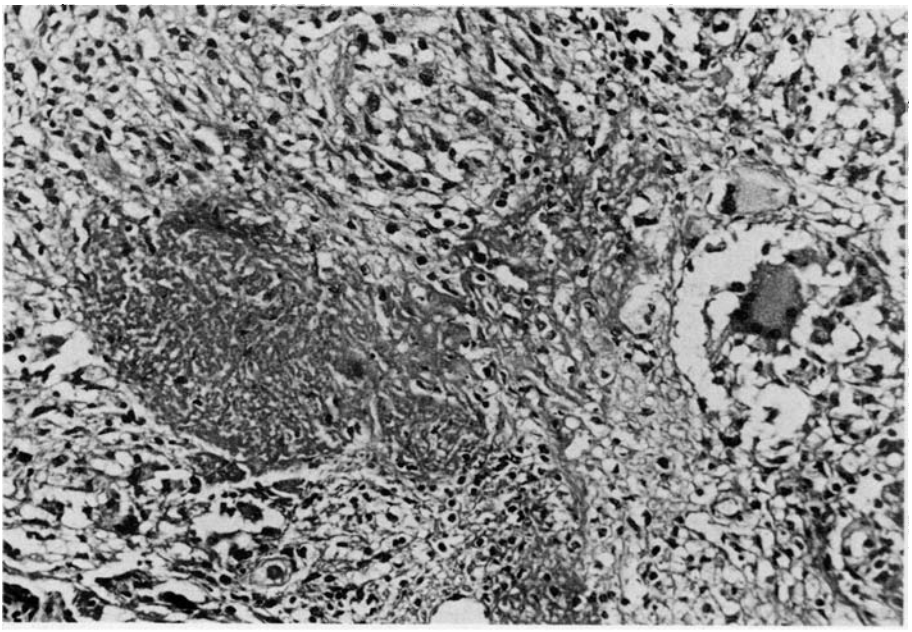


FIG. 2

Higher magnification of polyp showing typical tubercle with central caseation, epithelioid cells and Langhans giant cells. Haematoxylin-Eosin. (Magnification $\times 390$).

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which was thought to be a glomus tumor; the other had a granular mass in the middle ear. The last two cases, without perforation, showed the classical appearance of serous otitis media. These cases were explored and were found to have a whitish oedematous mucosa on the promontory which histologically turned out to be tuberculous.

The symptoms varied considerably, the two most common symptoms being deafness and otorrhoea. Eight patients had unilateral and 1 had bilateral otorrhoea. The duration of discharge also varied: in one 43-year-old patient the discharge had been present since childhood; in two others it had been present for 5 and 3 years; but in the remaining 6 cases it had been present for less than one year, the minimum period being 2 weeks. There was one patient who had facial paralysis, and on inquiry 8 out of 14 patients complained of pain. The only single case who volunteered this symptom was the one who had facial paralysis. The pain was of a deep-seated, dull, aching type which is a characteristic feature. It is because of the mild nature of the pain that patients often do not volunteer this information.

The hearing loss varied in degree but was present in 13 cases. Eight patients had a moderate conductive hearing loss and 3 had a mixed loss. One case had a profound sensorineural deafness. In the case of the child the hearing loss could not be confirmed. Six patients had mild tinnitus and one had severe tinnitus with attacks of vertigo (Table I). The mastoid X-rays did not show any characteristic changes other than those found in non-specific middle ear infection. Additional investigations included chest X-rays, smear cultures and animal inoculations, in one case preoperatively and in 8 during surgery. In all cases the diagnosis was finally confirmed histologically. Two patients were found to have open pulmonary tuberculosis and 4 had healed pulmonary lesions. One gave a history of tuberculous meningitis in his childhood. No acid-fast bacilli were found in the 9 smears examined. The culture and animal inoculation test were positive in 4 out of 9 cases.

The following 3 cases described in greater detail show the variety of problems faced in diagnosing this disease.

Case 1

A 38-year-old female attended the outpatient department because of a hearing loss of 3 years' duration. She complained of tinnitus in both ears. There was no history of giddiness or otorrhoea, but she had had acute otitis media 12 years earlier. There was no history of any major illness in the past.

On examination the tympanic membrane was found to be greyish-white in colour on the left side and scarred on the right side. Her mastoid X-rays were normal and the audiogram showed a bilateral 50 dB conductive loss. She was diagnosed as having otosclerosis and a stapedectomy was done successfully.

Two years later she attended the outpatient department to get an appointment for a stapedectomy on the right side. During this operation she was found to have an unusually thick mucosa on the promontory. There was no sign of any infection. The stapedectomy was performed in the usual manner and the mucosa was sent for histological examination. This showed tuberculosis.

Case 2

A 41-year-old female was referred for a pulsatile tinnitus of 4 months' duration in her left ear. The tinnitus had disappeared 4 weeks prior to attending

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TABLE I

ANALYSIS OF SIGNS AND SYMPTOMS.
THE LAST CASE WAS THE ONLY CASE WITH FACIAL PARALYSIS

			Otorrhoea		Deafness		Tinnitus			Tympanic membrane	
Age	Sex	Pain	R	L	R	L	R	L	Giddiness	R	L
43	F	Mild	40 yrs		Profound		Severe		Occasional	Grey tissue	
57	F	Mild			Moderate conductive		Mild			Bulging	
37	M	Mild		3 yrs		Mixed					Central perf.
10 mth	M		6 mth	6 mth	Not confirmed	Not confirmed				Grey tissue	Central perf.
44	M	Mild	8 mth		Moderate conductive					Grey tissue	
15	M	Mild	5 mth		Moderate conductive					Central perf.	
69	M	Mild		2 wks		Mixed			Occasional		Central perf.
38	F				Moderate conductive	Moderate conductive	Mild	Mild		Scarred	Grey
22	F				Mixed		Mild			Grey	
41	F							Mild	Occasional	Bulging	
26	M					Moderate conductive					Grey
31	M	Mild	4 mth		Moderate conductive		Mild			Central perf.	
41	M	Mild	5 mth		Moderate conductive		Mild			Central perf.	
32	M	Severe	5 yrs		Moderate conductive					Central perf.	

the outpatient department. There was no history of otorrhoea or giddiness, and the hearing was normal. On examination her left tympanic membrane was found to be red and bulging.

Her mastoid X-rays were normal. There was no bony erosion. She was diagnosed as a suspected case of intratympanic glomus tumor. She was explored under general anaesthesia and a smooth granulomatous mass, 1 cm in diameter, adherent to the tympanic membrane and the promontory, was removed. The biopsy proved it to be tuberculous. Subsequently it was found that her father had died of tuberculosis.

Case 3

An adult male complained of chronic otorrhoea of 5 years' duration and sudden facial paralysis with pain for 3 days. On examination he was found to have a central perforation with granulations in the middle ear. The patient had a complete facial paralysis with a complete reaction of degeneration.

The ear was explored. The mastoid and middle ear were found to be lined with an oedematous whitish mucosa. The chorda tympani was engulfed by granulation tissue. The chorda was further explored up to its branching from the facial nerve. Granulation tissue was present in the chorda-facial angle where it had damaged the facial nerve; this was removed. The histology confirmed the diagnosis. The ear healed without any complications and the facial paralysis recovered fully within 6 months.

Discussion

Though this lesion can almost always be suspected on the operating table, looking at all these variables in the clinical picture one wonders whether it would ever be possible to diagnose or even suspect tuberculous otitis media clinically in patients who do not have other obvious tubercular lesions. Nowadays it is unfortunately not common practice to enquire about tuberculosis in each and every case of chronic middle ear disease, because the number of cases of pulmonary lesions has declined considerably over the last few years. The onset of suppurative otitis media in a patient with pulmonary tuberculosis should lead the doctor to suspect tuberculous otitis media (Schuknecht, 1974), but it should not only be suspected when a patient with pulmonary tuberculosis gets otorrheoa.

The text books describe many so-called characteristic clinical findings in middle ear tuberculosis. These are a profuse discharge, a profound hearing loss, facial paralysis and multiple perforations (Shambaugh, 1967; Mawson, 1963; Wallner, 1953). Theissing (1955, 1966) is of the opinion that facial paralysis is common in children. We only had one child in this series who had no facial paralysis; out of 14, only one patient had facial paralysis. Midholm and Pedersen (1971) described inner ear deafness only in cases of long-standing disease with labyrinthine involvement. The case with severe hearing loss in our series had had a discharge since childhood and it is thought that the progressive disease had led to cochlear damage.

The characteristic sign of multiple perforations is hardly seen any more. Jeans and Friedmann (1960) report that, if perforations are present, 20–30 per cent have multiple perforations. None of our 9 cases had more than one per-

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foration. Perhaps a coalescence of perforations occurs early in the disease-process, and it is very rare to see a patient at this stage (Stone, 1967).

We feel that the symptom of pain is of great importance in diagnosing these cases. If the tuberculous granulation tissue involves the mastoid cavity it appears to be under pressure. This fact is especially observed during surgery. When the mastoid cells are opened the granulation tissue bulges out of the cells as if released from pressure. We feel that the tension or the mild pressure exerted by this granulation tissue causes a mild ache. The visual appearance of granulations under tension is also characteristic of tuberculosis.

The case of a female who had otoscerlosis is of great clinical interest. She might have developed tuberculosis of the right ear after she was operated on on her left ear, or she might already have had the disease in the right ear. The fact that she developed normal hearing after the stapedectomy, in which the vestibule was opened in the presence of subacute middle ear tuberculosis, is remarkable. A possible explanation could be that propagation of the disease to the inner ear is very slow (Midholm and Pedersen, 1971) and that in this particular case the antitubercular treatment was started within four days after the surgical intervention, thus preventing inner ear damage.

The second case, with an initial diagnosis of a glomus jugulare tumor, was similar to the one reported by Palva *et al.* (1971). The granuloma in our case was on the medial side of the tympanic membrane, touching the promontory. Cases have been reported in which the granuloma was originally thought to be a tumor. Hiranandani (1967) reported a case of petrous apex tuberculosis with VI nerve palsy. Grabschied (1937) reported a case of tuberculoma mimicking a cerebello-pontine angle tumor. A tuberculous mass in the petrous apex involving the jugular foramen and the foramen magnum was found to be the cause of the cranial nerve palsies.

The third case, with a facial paralysis, is the only case in our series with facial nerve involvement. This patient complained of pain. Legent and Baron (1975) have described three similar cases and emphasize the importance of early decompression. In patients who have facial paralysis in chronic middle ear disease without cholesteatoma, tuberculosis must be the prime suspect. The interesting feature in our own case was the path of spread of the tuberculous granulation tissue, which was via the chorda tympani. The complete recovery following surgery with decompression is consistent with the findings of Legent and Baron (1975).

The outcome of surgical treatment in our 14 cases was very encouraging. Harbert and Riordan (1964) say that the surgical intervention in their cases was probably the worst thing that could have been done. In none of our own cases, however, did we have to repeat surgery. Palva *et al.* (1973), in their series, found a severe hearing impairment after surgery. We did not observe postoperative deterioration of the hearing in any of our cases. All the ears healed by primary intention. Of course, in all these cases, antitubercular chemotherapy was instituted immediately after the diagnosis was confirmed.

Friedmann says: "The otologist is concerned with patients who present themselves primarily with symptoms of otitis media. There may or may not be evidence of tuberculosis in other sites, but if it is present it is usually discovered only accidentally". The statement is so very true as regards the diagnosis of tuberculous otitis media. We feel that immediate postoperative antitubercular

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therapy will prevent any adverse effect on the ear due to the surgical procedure. If the otologist keeps in mind the dull ache in the ear without obvious inflammatory signs, the whitish grey appearance of the mucosa, and the bulging of granulation tissue from the mastoid cells during surgery, we are sure that a greater number of cases of tuberculous otitis media would be diagnosed at an earlier stage.

Summary

Fourteen cases of tuberculous otitis media are reported. With the declining incidence of pulmonary tuberculosis, the number of reported cases of tuberculous otitis media has become scanty. Nowadays the so-called classical signs of the lesion such as a profuse discharge, profound hearing loss, facial paralysis and multiple perforations, are hardly ever seen. Surgical intervention when done under cover of anti-tubercular drugs has no deleterious effect on the result of surgery. In order not to miss tuberculous otitis media, whenever the otologist is dealing with chronic middle ear disease, the following points should always be kept in mind:

(a) A dull ache in the ear without obvious inflammatory signs.
(b) A whitish-grey appearance of the mucosa.
(c) Bulging of granulation tissue from the mastoid cells during surgery.

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