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Clinical Records

Tuberculous mastoiditis

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Introduction

In 1980 Plester *et al.* wrote: 'One wonders whether it would ever be possible to diagnose or even suspect tuberculous otitis media clinically in patients who do not have other tubercular lesions'. It is undoubtedly true to say that tuberculous otitis media and mastoiditis have become so rare in the last 40 years in the so-called developed countries that many otologists have never seen a case.

A delay in diagnosis of some four to five years is not uncommon. Various authors have described series of cases none of which were correctly diagnosed before operation (Harbert and Riordan, 1964; Sahn and Davidson, 1974; Emmett *et al.*, 1977; Lucente *et al.*, 1978). However Palva *et al.* (1973) diagnosed six out of their 14 cases pre-operatively.

The purpose of this paper is once again to remind otologists of the possibility of tubercular ear disease, even though the incidence is now probably below 0.1% of all cases of suppurative otitis media attending hospital (Jeanes and Friedmann, 1960). In his book Pathology of the Ear (1974), Friedmann reported a series of 17 cases confirmed histologically. Although the incidence is low there is considerable variation between authors from different countries. For instance Palva *et al.* (1973) had 14 tuberculous ears among some 1,600 chronic ears which were operated on whereas Plester *et al.* (1980) found only the same number among approximately 18,000 operations.

It is possible that more cases will occur among immigrants from Asia, Africa and the West Indies where pulmonary and extra-pulmonary tuberculosis is still rife (Brutoco and Spencer, 1980). We propose to discuss those symptoms and signs, which in their various combinations should alert one to the possibility of tuberculous disease and to stress the need for appropriate bacteriological and histological investigation.

Case reports

Case 1

A 53-year-old lecturer in engineering had been seen by several eminent otolaryngologists because of painless right otorrhoea which had started at the age of 20. In 1975 he had a flare-up of infection with associated vertigo which resulted in total loss of hearing in his right ear and a temporary right facial weakness. For a year or so previously he had been aware of a loud clicking noise in the region of the right temporo-mandibular joint when eating.

He first presented to GB in October 1978 with right otorrhoea and a recurrence of slight facial weakness. On questioning, it emerged that the ear first discharged in 1945 while he was serving in the RAF and undergoing sanatorium treatment for a tuberculous pleural effusion. Regrettably this information had not been given to otologists who had previously seen him. He did not receive any chemotherapy at this time. Ten years later the pulmonary lesion was thought to be unstable and he was treated with PAS and INAH for five months which was clearly inadequate by present standards.

Examination of the right ear disclosed profuse purulent discharge with no recognisable tympanic membrane or ossicles, pale granulations and a large area of unhealthy bone in the tympanum. There was marked antero-posterior movement of the right mandibular condyle visible within the meatus when he opened and closed his mouth. No abnormality was found in the left external or middle ear. Audiometric tests confirmed a dead right ear and a moderate high frequency sensori-neural hearing loss in the left ear. The remainder of the ENT examination was unremarkable.

Mastoid tomograms (Fig. 1) and CT scan (Fig. 2) disclosed blurring of the cell margins with a possible sequestrum and erosion into the temporo-mandibular joint. Chest x-ray showed scattered calcified pleural shadows and obliteration of the left costophrenic angle. Large numbers of acid-fast bacilli were demonstrated on Ziehl-Neelsen staining (Fig. 3) of material draining from the right ear. A course of treatment with Rifampicin, Ethambutol and Isoniazid was begun under the supervision of a Chest Physician. Culture confirmed Mycobacterium tuberculosis, fully sensitive to all anti-tuberculous drugs. Sputum cultures were negative for tuberculosis. After six weeks of therapy a negative ear swab was obtained.

Operation. Three months after diagnosis a radical mastoidectomy was carried out. The mastoid air cells contained much carious bone and dense granulations (Figs. 4 and 5). A large sequestrum measuring 2 cm x 1 cm (Fig. 6) was removed from close to the facial nerve. A sinus led into the temporo-mandibular joint. Post-operatively the mastoid cavity was packed with Bismuth Iodoform Paraffin Paste which was left in for many weeks to prevent subluxation of the mandibular condyle. He retained excellent function of the temporo-mandibular joint and the condyle stabilized in the normal position.

There was gradual improvement and almost complete recovery of facial nerve function (Fig. 7). Ethambutol was discontinued after five months' therapy but the remaining drugs were continued for two years. He has returned to living a busy and very energetic life and has a dry mastoid cavity.

Comment

There was a delay of 25 years in diagnosis because the history of pulmonary tuberculosis had not been elicited previously.

The diagnosis in this case rested on:

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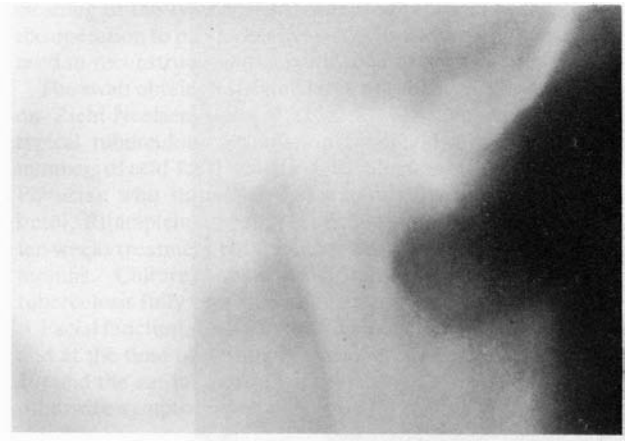


FIG. 1

Case 1. Lateral tomogram showing bone destruction and sequestrum formation.



FIG. 2

Case 1. CT scan clearly demonstrates sequestrum and erosion of TM joint (arrowed).



FIG. 3

ZN stain of pus from right ear showing vast numbers of acid-fast bacilli (Case 1).

1. Painless profuse otorrhoea for many years.
2. Total destruction of tympanic membrane and ossicles.
3. Clinically obvious sequestrum.
4. Temporo-mandibular joint involvement.
5. A dead ear, presumably due to tuberculous labyrinthitis three years previously.
6. Two episodes of facial paresis.
7. The finding of profuse acid-fast bacilli.
8. Pre-existing pulmonary tuberculosis.

Excellent healing of the mastoid cavity was achieved after chemotherapy and surgery. Operation was delayed for three months, firstly to allow the sequestrum to become better defined, secondly to allow chemotherapy to have maximum effect, and finally to preserve temporo-mandibular joint function.

Case 2

A 63-year-old English lady was seen by an ENT surgeon in June 1983 with a six-month history of painless left otorrhoea and deafness. The notes record that the eardrum was intact but thickened and indrawn. Her hearing loss was reported as betweeen 60 and 70db in the affected ear. A routine ear swab grew diphtheroids only and she was treated with antiseptic ear drops. Shortly afterwards the patient developed an intestinal obstruction and was found to have a carcinoma of the ascending colon, for which a right hemicolectomy was performed. She also had a series of small haemoptyses at this tin for which no cause was found. The chest x-ray showed no a normality.

She was first seen by EHR on 24 Januar 1984, having developed a left facial paralysis with some i balance. The clinical findings were otorrhoea, left-sided sever leafness and

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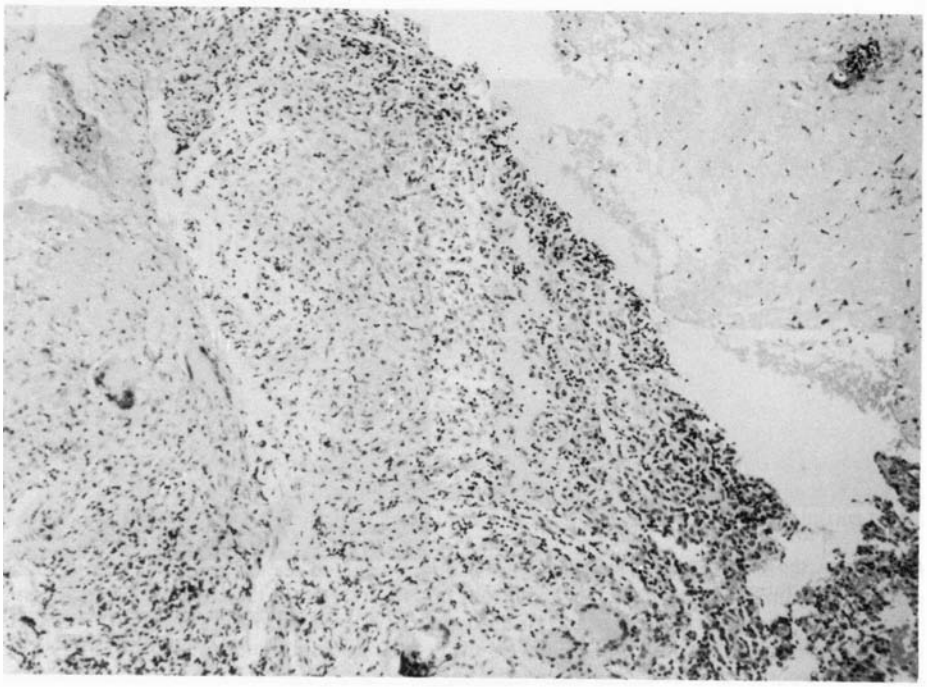


FIG. 4

Tuberculous granulation from the middle ear of Case 1. Langhans-type giant cells are present together with epitheliod cells and some lymphocytes. Caseous material is present in the upper right corner (H & E x 100).

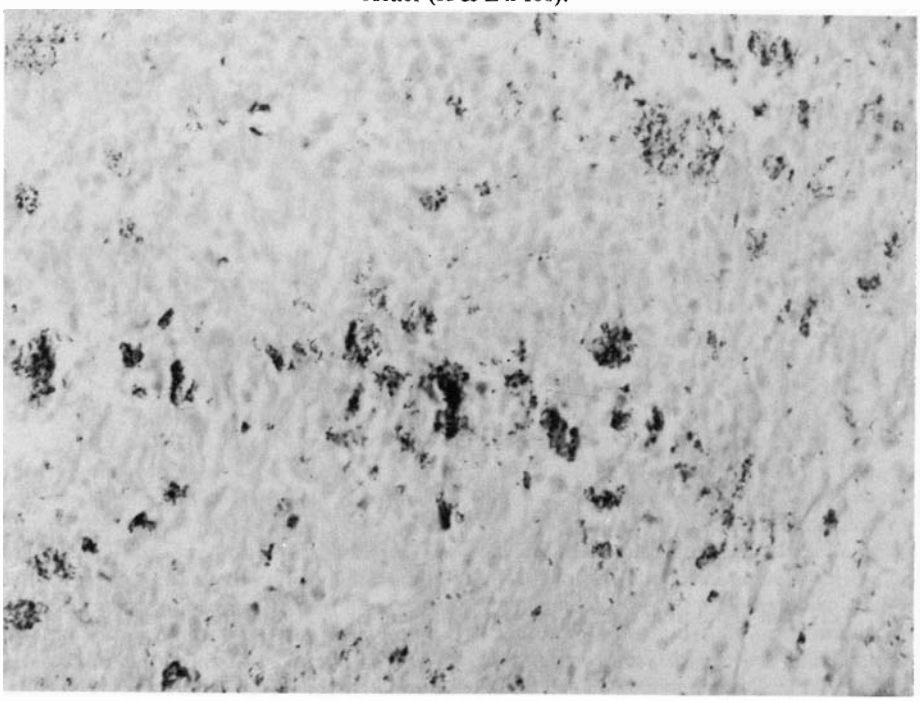


FIG. 5

Necrotic tissue from middle ear of Case 1 stained for acid-fast bacilli. Vast numbers of acid fast bacilli are present, often in clumps (ZN x 400).

complete lower motor neurone facial paralysis confirmed by nerve conduction tests and electromyography. The tympanic membrane was destroyed and the malleus handle and incus were seen to be embedded in pale granulations which filled the middle ear, the appearances raising the suspicion of tuberculous otitis. Some granulations were removed in the clinic and sent for histology and culture for mycobacterium tuberculosis. Mastoid x-rays and tomograms showed clouding of mastoid air cells on the left, but no evidence of bone erosion.

Operation. A left mastoidectomy was performed the following day. The mastoid was moderately cellular and the superficial cells were found to be clear. However the periantral cells, aditus and antrum were filled with pink granulations, while the mesotympanum and epitympanum were full of pale amorphous debris. The ossicles were intact, although the lenticular process had separated from the stapes head. There was dehiscence of the facial nerve at the second genu. All infected material was carefully dissected from around the ossicles and

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window niches and the lenticular process was replaced on the stapes head. The bony meatal wall was removed to facilitate cleaning of the tympano-mastoid, and replaced at the end of the operation to produce a closed cavity. Temporalis fascia was used to reconstruct the tympanic membrane.

The swab obtained showed large numbers of acid-fast bacilli on Ziehl-Neelsen staining (Fig. 8). The histology showed typical tuberculous granulation tissue, also containing vast numbers of acid-fast bacilli (Fig. 9). She was referred to a Chest Physician who started antituberculous therapy with Ethambutol, Rifampicin and INAH. Ethambutol was stopped after ten weeks treatment but the other drugs were continued for six months. Culture subsequently confirmed Mycobacterium tuberculosis fully sensitive to all antituberculous drugs.

Facial function started to return eight months after operation and at the time of writing is almost completely restored (Fig. 10) and the ear is dry. Unfortunately she has a dead ear but is otherwise symptom-free.

Comment

There was a one-year delay in making a correct diagnosis from the onset of otorrhoea and six months from her first attendance at an ENT clinic. Pointers to diagnosis were:

1. Painless, profuse otorrhoea, not responding to routine treatment.
2. Total destruction of the eardrum but not of the ossicles.
3. A middle ear full of pale granulations and caseous material.
4. Severe deafness.
5. Facial paralysis.
6. The finding of profuse acid-fast bacilli (Ziehl-Neelsen film, culture and histology).

In this case no proof was ever obtained of any active or past pulmonary lesion, in spite of repeated small haemoptyses. Moreover, careful history taking and contact tracing failed to reveal any source of infection. Satisfactory healing and recovery of facial nerve function occurred in this as in numerous other reported cases following surgery and chemotherapy. The dead ear was presumed to be due to a tuberculous labyrinthitis which occurred about the time of her facial nerve involvement.

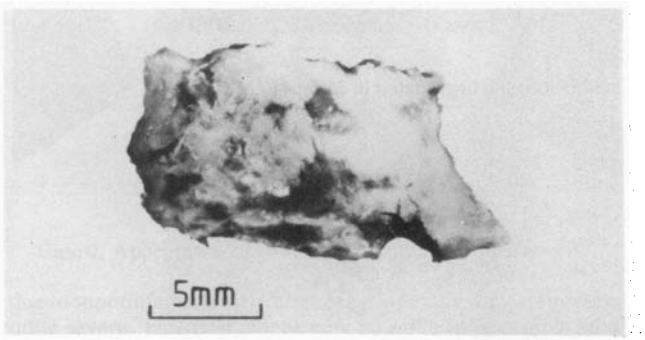


FIG. 6

Sequestrum removed at operation (Case 1).

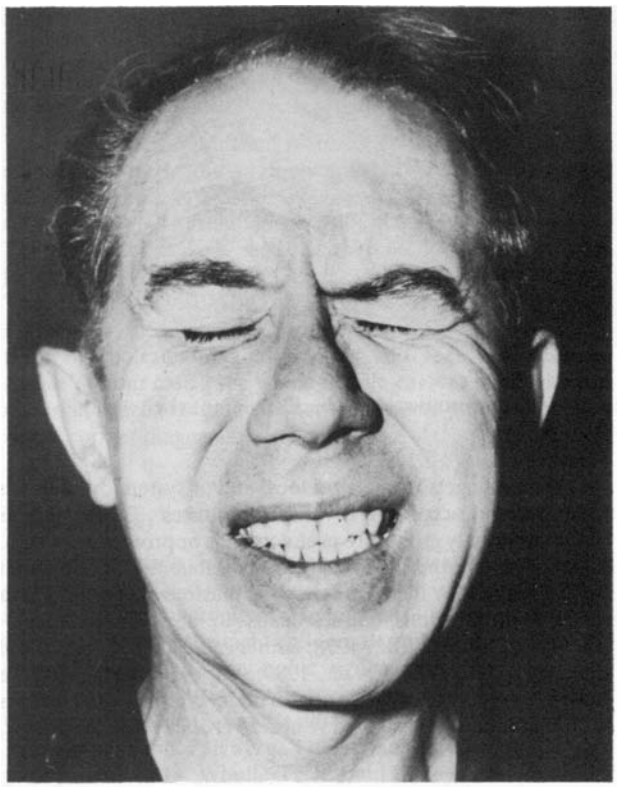


FIG. 7

Case 1. Appearance post-operatively.

Discussion

Tuberculous otitis media was a common disease in the early part of the 20th century. According to Turner and Fraser (1915) 50% of infants with otitis media were found to be tuberculous, while by adolescence this figure had dropped to 2%.

Between 1950 and 1959 Jeanes and Friedmann (1960) found only 12 among 23,000 cases of otitis media seen in one hospital, while Plester *et al.* (1980) found histological proof of tuberculosis in 14 out of 4,000 (0.35%) biopsies from the middle ear.

Most so-called developed countries will show a similar low incidence, due, no doubt, to better hygiene, tuberculin testing of dairy herds, easier access to medical care, radiological and laboratory facilities, BCG vaccination, ready availability of treatment with a variety of specific drugs, notification of cases and contact tracing.

Low incidence inevitably leads to lack of suspicion. There is a need for constant vigilance as sporadic cases continue to crop up here and there. Prolonged ill health, persistent otorrhoea and complications such as profound and irreversible deafness, labyrinthitis, meningitis and facial paralysis are all preventable if diagnosis is made early enough.

Histological diagnosis generally presents little difficulty and all 17 cases summarized by Friedmann (1974) exhibited typical tuberculous granulation tissue. On the other hand bacteriological confirmation of the diagnosis may be extremely difficult and was achieved in only about 50 per cent of Jeanes and Friedmann's (1960) series. Normally acid-fast bacilli are scanty and attempts to culture the organism may fail whereas our two cases were unusual in presenting large numbers of bacilli in the aural discharge.

The classical symptoms and signs still apply, though multiple tympanic perforations are now hardly ever seen. At most they are a temporary phase, rapidly followed by more or less complete destruction of the pars tensa (Palva *et al.*, 1973; Plester *et al.*, 1980; Windle-Taylor and Bailey, 1980). Jeanes and Friedmann (1960) still claimed them to be common, comprising 20–30 per cent of patients in their series.

What, then, is the present position and which clinical features occur with sufficient frequency to lead the oto-rhino-laryngologist to suspect that he may be dealing with a case of tuberculous otitis and to take early steps to confirm the diagnosis bacteriologically and histologically?

Multiple perforations

All recent authors agree that these are uncommon. neither Palva *et al.* (1973), Plester *et al.* (1980) nor Ramages and Gertler (1985) saw a single instance among their cases, while Windle-Taylor and Bailey (1980) found only one case out of 22. More usually one encounters single central or marginal perfor-

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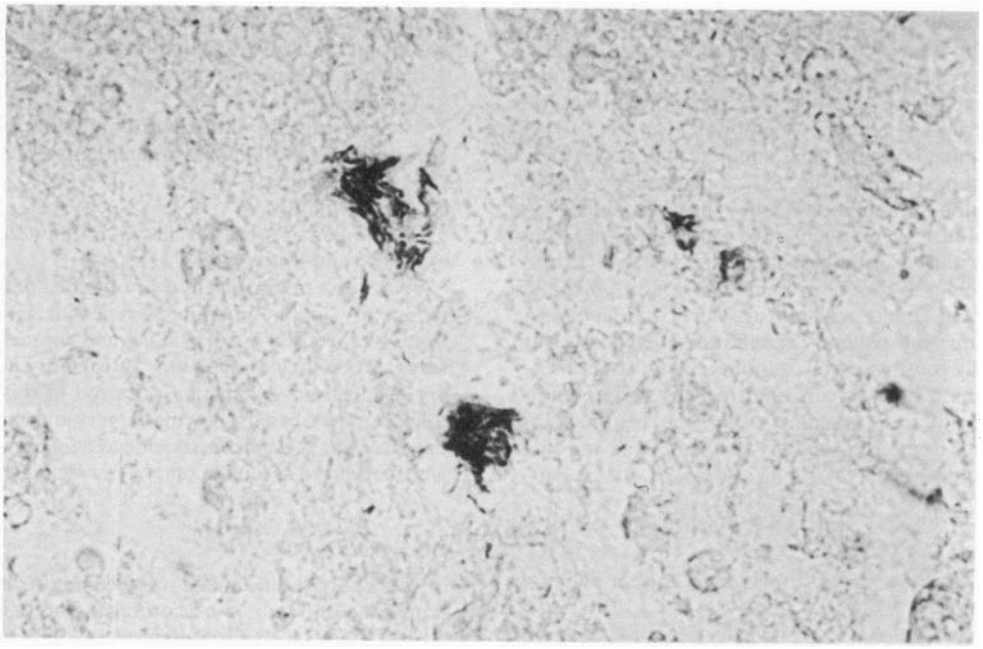


FIG. 8

Case 2. Smear from left ear showing large numbers of acid-fast bacilli aggregated in clumps (ZN x 1250).

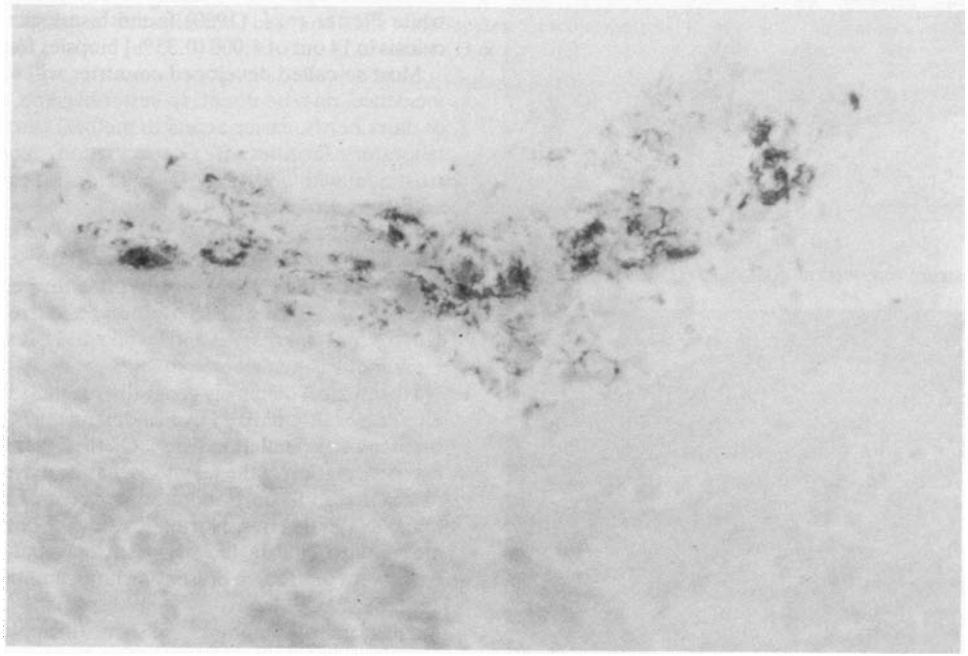


FIG. 9

ZN preparation of granulomatous tissue from middle ear showing numerous acid-fast bacilli tending to be clumped together. Case 2.

ations, total membrane loss, or even no perforation at all (Plester *et al.*, 1980).

Hearing loss

Severe or permanent loss of hearing is a frequent occurrence. Craig (1962), Palva *et al.* (1973; Windle-Taylor and Bailey, 1980; However Ramages and Gertler 1985) did not perform hearing tests routinely, because their patients were either too young or too ill. Both our patients had dead ears.

Profuse otorrhoea

This occurs in a high proportion of cases. In fact, it not uncommonly gets worse after local and/or systemic antibiotics administered according to *in vitro* cultures. The organisms most commonly grown are pseudomonas or proteus sp., staph. aureus and diphtheroids, singly or in combination. The amount of discharge may increase following mastoid surgery before a positive tuberculous culture leads to appropriate chemotherapy (Cawthon *et al.*, 1978; Esmer *et al.*, 1979; Brutoco and Spencer, 1980; Plester *et al.*, 1980). On the other hand there have been reports of patients with intact tympanic membranes and thus no otorrhoea at all (Lucente *et al.*, 1978; Plester *et al.* 1980).

Pain

Occurs not infrequently in tuberculosis of the ear, possibly

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FIG. 10
Case 2. Appearance of patient 18 months post-operatively.

due to superinfection with other organisms and may at times be quite severe. However, there may be none in uncomplicated infections. Appreciable pain was a feature in 11 cases out of 22 in the series of Windle-Taylor and Bailey (1980) and in eight out of 14 in Plester *et al.* (1980). But many patients never complain of pain (Craig, 1962).

Pale mucosa, pale granulations or pale looking drum

These are most significant features, commented on by most authors. Palva *et al.* (1973), for example, found white strands of necrotic material in most of their cases. Ramages and Gertler (1985) found pale granulations to be the most common sign. The finding of caseous material may be confused with cholesteatoma (Palva *et al.*, 1973; Plester *et al.*, 1980; Windle-Taylor and Bailey, 1980). See also our case 2.

However, the material lacks the characteristic glistening features of cholesteatoma and looks and feels like 'cottage cheese'. In our case 2 the whole middle ear was filled with this material, which together with her VIIth nerve paralysis pointed to the correct diagnosis.

Facial paralysis

Young children who develop facial paralysis during the course of middle ear infection should be considered to be suffering from tuberculous disease unless proved otherwise. However, none of the eight children described by Craig (1962) appear to have had facial nerve involvement.

Ramages and Gertler (1985) had six cases out of 25, only one of them in an adult. Though less frequent in adults it is still not a rare complication. Palva *et al.* (1973) had three cases with facial paralysis out of 14. Windle-Taylor and Bailey (1980) had four among 22 whereas Plester *et al.* (1980) encountered only one case among their series of 14. Both our patients developed facial paralysis which eventually recovered.

Vertigo and CNS disturbance

Both our patients had some disturbance of balance at some time during their illness. Palva *et al.* (1973) had five patients with vertigo, which eventually ceased in all. Other intracranial and extracranial complications included VIth nerve involvement, tuberculosis of retina, pons and cerebellum (Birrell, 1973; Jeang and Fletcher, 1983).

Pre-existing pulmonary or extra-pulmonary tuberculosis

Evidence of active or past pulmonary disease, or neck scars from old fistulae in patients with chronic otorrhoea should lead one to consider aural tuberculosis. All six pre-operatively diagnosed patients in Palva *et al.* (1973) series had one or other of these. A family history or known contact with a case of pulmonary tuberculosis should alert one to the possibility that one is dealing with tuberculous otitis. Chest x-ray is obviously helpful, though in one of our cases there were no radiological signs in spite of haemoptysis, while there was a history of past pulmonary infection in the other. Windle-Taylor and Bailey (1980) found positive chest x-rays in half their cases, Ramages and Gertler (1985) smilarly in 13 patients out of 25.

Mastoid x-ray

This may show poor pneumatisation but more often there is normal cellularity with clouding due to granulation tissue, for instance Case 2. There may be bone destruction or sequestration, as in our Case 1. Ramages and Gertler (1985) found visible sequestra at initial otoscopy but do not mention whether they were radiologically apparent.

Race

Aural tuberculosis appears nowadays to be extremely rare among the indiginous population of Western Europe and one is more likely to find this disease in immigrants, especially from the Indian subcontinent, the Caribbean and Africa. It is interesting to note that all 25 cases reported from the Groote Schuur Hospital, Cape Town (Ramages and Gertler, 1985) were either black or coloured. Both our patients were white and one (Case 2) had never been abroad.

Treatment

There is general agreement that anti-tuberculous treatment should precede surgery, unless there are indications for early surgical intervention. In such cases drug treatment should be commenced as soon as diagnosis is confirmed by histology and/or bacteriology. Surgery before chemotherapy in Case 2 was decided upon because of the recent onset of facial paralysis. The choice and dosage of drugs prescribed will be at the instigation and under the supervision of a Chest Physician and includes Rifampicin, Ethambutol and Isoniazid. Both our patients responded well to anti-tuberculous therapy and have dry ears. Vertigo and facial paralysis nearly always recover. Where denervation has occurred the patient will have some degree of permanent weakness and synkinesis.

Conclusions

We believe that it is possible to diagnose tuberculous otitis media and mastoiditis clinically. To do so one has to consider the possibility in cases where there are two or more of the above symptoms and signs, i.e. profuse discharge not responding to conventional medication, moderate-to-severe pain, marked hearing loss, unusually pale granulations, facial paralysis, or vertigo, especially where there is a history of active or past pulmonary or extra-pulmonary tuberculosis. In both our cases a confident diagnosis of tuberculous mastoiditis was made

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by the authors on initial presentation: in Case 1 on the basis of past lung infection, profuse otorrhoea of many years duration, not responding to previous treatments, facial paresis and total hearing loss; in Case 2 mainly on the basis of facial paralysis and the striking appearance of pale granulations and caseation in the middle ear. It was also helpful to be able to remember what tuberculous otitis looked like 30–40 years ago when it was more common.

We wish to stress the importance of asking the laboratory to search for acid-fast bacilli in each instance of otitis media presenting unusual features. Likewise every piece of tissue removed before or during surgery should be sent for histological examination with the possibility of tuberculosis in mind (this to be clearly stated on the request form). Normally acid-fast bacilli are difficult to identify and a careful search has to be made for them. The finding of large numbers of acid-fast bacilli in both our cases on the initial Ziehl-Neelsen stained swab, as well as in the histological sections, was most unusual. It is difficult to envisage any other organ in the body showing such massive colonisation without the patient being more seriously ill.

Summary

Two cases of aural tuberculosis in Caucasian adults are described. Stress is laid on the need to consider tuberculosis in cases of chronic otitis presenting unusual features. Lack of suspicion of tuberculosis frequently leads to long delays in diagnosis and subsequent needless disability.

Acknowledgements

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