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Role of surgery in tuberculous mastoiditis

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Abstract

A study was undertaken in 43 patients to determine the role of surgery in tuberculous mastoiditis. Cortical mastoidectomy was performed on five patients (Group I). Incision and drainage of a post-auricular abscess, removal of sequestrum and meatoplasty in eight (Group II).

Thirty patients had no ear surgery (Group III). Of the 17 patients with facial palsy, three were in Group I, two in Group II, 12 in Group III. The patients in all three groups were treated with anti-tuberculous drugs for a period of no less than six months.

The average time taken for the otorrhoea to subside and granulation tissue to resolve completely was two months in all three groups. The facial nerve recovery in the non-operated ears (Group III) was 92 per cent and in the operated ears (Group I and II) 80 per cent.

The conclusion is that chemotherapy is the management of choice in tuberculous mastoiditis. The only role of surgery is incision and drainage of a post-auricular abscess and removal of sequestrum if present.

Introduction

Tuberculosis of the middle ear cleft was a common disease in the early part of the twentieth century. According to Turner and Fraser (1915), 50 per cent of the infants with otitis media were found to be tuberculous, whilst by adolescence, this figure had dropped to 2 per cent.

Over the years, the incidence of tuberculous otitis media has decreased. Between 1950 and 1959, Jeanes and Friedmann (1960) found only 12 amongst 23,000 cases of otitis media, in one single hospital. Palva *et al.* (1973), during the period 1964 to 1971, operated on 1,638 chronic discharging ears and found 14 (0.9 per cent) to be of tuberculous origin. Plester *et al.* (1980) found histological proof of tuberculosis in 14 of 4,000 (0.35 per cent) biopsies from the middle ear. This decreased incidence in the developed countries is largely due to the improved health services and hygiene, readily available medical care, BCG vaccination and pasteurization of milk.

TABLE I
INCIDENCE OF TUBERCULOUS MASTOIDITIS

Age in (years)	No. with TB mastoiditis	Total no. with CSOM	% with TB mastoiditis
<1	1	353	0.28
1–2	18	2288	0.79
3–5	7	1500	0.46
6–10	6	2280	0.26
11–15	3	1620	0.18
16–20	1	1770	0.06
21–30	4	2950	0.14
31–60	3	2360	0.12
>60	nil	240	–
Total	43	15361	0.28

In South Africa, more especially in the rural regions of the province of Natal, where poverty, malnutrition, overcrowding, poor hygiene and inadequate medical services exist, tuberculosis is very prevalent.

Over a six-year period, at King Edward VIII Hospital alone, 7,443 patients were found to have pulmonary and 4,058 extrapulmonary tuberculosis; of these, 43 had tuberculous mastoiditis.

Materials and methods

Over a six-year period from January, 1985 to December, 1990, tuberculous mastoiditis was diagnosed in 43 patients. There were 26 males and 17 females. Their ages ranged from eight months to 51 years. The number of patients in the various age groups is shown in Table I. All the patients had a history of otorrhoea which varied from one week to four years. In 80 per cent of the patients, the otorrhoea was of less than six months' duration. It was bilateral in 11 and unilateral in 32 patients. Two patients had multiple perforations and 41 had a single perforation. Thirty-nine patients had pale granulation tissue in the external ear canal, whilst four had hyperaemic and friable granulation tissue. Mastoiditis

TABLE II
FACIAL PALSY IN THE DIFFERENT AGE GROUPS

Age group	No. with facial palsy	% of total
1–2	9	53
3–5	6	35
6–10	1	6
11–15	–	–
16–20	–	–
21–30	1	6
Total	17	100

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TABLE III
INTRACRANIAL COMPLICATIONS OF TUBERCULOUS MASTOIDITIS

Intracranial complications	No. of patients
Tuberculous meningitis	6
Tuberculomata	3
Hydrocephalus	3
Multiple cranial nerve palsies	1
(1) left VI, VII and VIII	
(2) bilateral IX and X	
(3) right XII	
Hemiparesis	1
Monoparesis	2

was complicated by a post-auricular abscess in 13 patients, unilateral facial palsy in 16, bilateral facial palsy in one and dizziness with erosion of the lateral semicircular canal in one patient. The duration of the facial palsy varied from one day to three months. The ages of the patients with facial palsy is shown in Table II. Intracranial complications, as illustrated in Table III, were observed in six patients. The presenting symptoms included neck stiffness in six, left-sided convulsions in one, right-sided upper and lower limb weakness in one, unilateral lower limb weakness in two, and diplopia in one patient.

In 40 patients, the chest radiograph displayed features suggestive of pulmonary tuberculosis. These are illustrated in Table IV. In the remaining three patients, the chest radiograph was completely normal.

Periotic lymphadenopathy (preauricular in one and upper cervical in 10) were noted in 11 patients. Active tuberculosis in another site was noted in one patient. This was a 14-year-old boy who, besides having pulmonary tuberculosis and tuberculous mastoiditis, had active tuberculosis involving the left kidney.

The diagnosis of tuberculous mastoiditis was made clinically on all patients. Confirmatory laboratory investigations included bacteriology with culture of acid-fast bacilli from the aural discharge in one and histology of the granulation tissue, in 42 patients. The histology revealed a caseating granuloma with acid-fast bacilli in 30 patients and a tuberculoid granuloma with multinucleated giant cells in twelve. In 29 patients, the tissue for histological examination was obtained from the external ear canal. The biopsy was performed with a microcup forceps in the outpatient clinic. On those patients in whom the granulation tissue was deep in the external ear canal, a microscope with a 200 mm lens was used to take the biopsies. In this group of patients, an urgent request was made to the pathologist for early reporting and thus the results were obtained within 24 hours. In the other 13 patients, the tissue for histological examination was obtained from the mastoid cavity. This was taken at mastoidectomy in eight, and at incision and drainage of the postaural abscess in five.

The diagnosis of tuberculous meningitis was made on biochemical and microscopic analysis of the cerebrospinal fluid, whilst the diagnosis of tuberculomata and hydrocephalus was made on computed tomography of the brain.

With regard to treatment, the patients were divided into three groups. There were eight patients in Group I, five in Group II and 30 in Group III. Of the 17 patients with a facial palsy, there were three in Group I, two in Group II and 12 in Group III. The 13 patients with subperiosteal postauricular abscesses were confined to Groups I and II.

Surgery was performed on Group I and II patients only and it entailed a cortical mastoidectomy in the former, and incision and drainage of the postauricular abscess, removal of sequestrated bone and biopsy of the granulation tissue from the mastoid cavity and a meatoplasty in the latter. No bone drilling was required as the disease itself had created a radical cavity.

The facial nerve was neither explored nor decompressed in any one of these patients.

The operative findings are summarized in Table V.

No surgical procedure was performed in Group III patients, except for a biopsy of the granulation tissue from the external ear canal.

All patients had their ears dry-mopped and an antibiotic ear drop (Otosporin®) applied, twice daily.

Antituberculous chemotherapy was given to all patients. It consisted of four drugs, namely rifampicin, isoniazid, pyrazinamide and ethambutol in patients older than six, and three drugs, rifampicin, isoniazid and pyrazinamide, in children six years and younger. The treatment was given for a period of six months but in those patients, in whom the tuberculous mastoiditis was complicated by meningitis and tuberculomata, the treatment period was extended to one year in four, and 18 months in the other two.

In addition to the above, oral steroid (dexamethasone) was given to patients with tuberculomata of the brain. Three patients, who developed hydrocephalus secondary to tuberculous meningitis, required ventriculo-peritoneal shunt surgery.

One patient died three weeks after admission. This was a 21-year-old female with pulmonary tuberculosis, tuberculous mastoiditis and meningitis and intracranial tuberculoma with left VI, VII and VIII, bilateral IX and X, and right XII cranial nerve palsies. The following three patients are described in greater detail to demonstrate that tuberculous mastoiditis is just part of a disseminated disease.

TABLE IV
CHEST RADIOGRAPH FEATURES

		In combination with			
	Alone	Hilar Lymphadenopathy	Paratracheal Lymphadenopathy	Hilar & Paratracheal Lymphadenopathy	Total
Upper lobe consolidation	9	11			20
Middle lobe consolidation	1			1	2
Lower lobe consolidation	2	1			3
Bilateral patchy bronchopneumonia	6	2	1	1	10
Hilar lympadenopathy	5				5
					40

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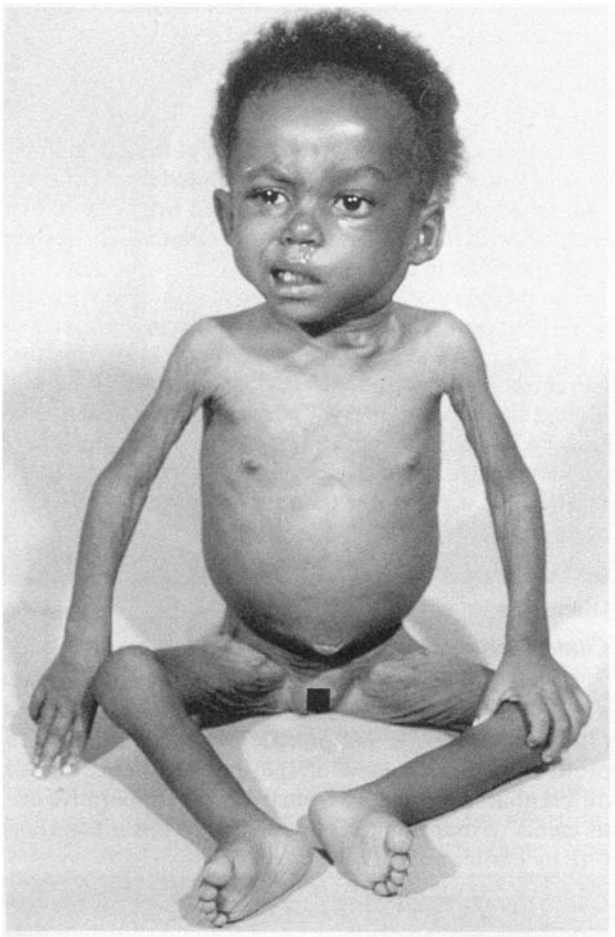

FIG. 1

Marasmic-Kwashiorkor child with a left-sided facial palsy and bilateral cervical lymphadenopathy.

Case 1

Female, aged two years. This child displayed all the typical features of a patient with tuberculous mastoiditis. She came from the rural area of Natal. The presenting symptoms were otorrhoea for two weeks, facial palsy for three months, loss of appetite and weight. There had been tuberculous contact (child's aunt). Examination revealed a malnourished (marasmic-kwashiorkor) child with left-sided otorrhoea, granulation tissue in the external ear canal, ipsilateral facial palsy, submandibular and cervical lymphadenopathy, (Fig. 1). The chest radiograph revealed features typical of pulmonary tuberculosis, patchy and confluent shadowing in right upper zone and perihilar lymphadenopathy (Fig. 2).

Case 2

Male, aged seven years. He was referred from the rural hospital with the problem of bilateral otorrhoea, right-sided facial palsy, neck swelling and unsteady gait. Examination revealed bilateral, axillary and cervical lymphadenopathy (Fig. 3), hepatosplenomegaly with ascites, bilateral otitis media, right facial palsy (Fig. 4), and a right hemiparesis, with greater involvement of the lower than the upper limb. Chest radiograph revealed a right-sided consolidation with paratracheal lymphadenopathy (Fig. 5).

Computed tomography of the brain demonstrated two isodense lesions (tuberculomata), one lying in the left frontal lobe (Fig. 6) and the other in the left superior parietal region (Fig. 7).

The diagnosis of tuberculosis was confirmed histologically from the axillary lymph node and granulation tissue of the right external ear canal. He was managed on anti-tuberculous drugs and dexamethasone. A six-month follow-up photograph (Fig. 8) showed almost complete recovery of the facial palsy and resolution of the cervical lymphadenopathy.

TABLE V

OPERATIVE FINDINGS

Findings	No. of patients
Pale granulation tissue	12
Sequestrated bone	5
Erosion of bony E.A.C., sinus and dural plates	5
Erosion of the bony lateral semicircular canal	1
Caseous material	2

(E.A.C. external ear canal).

Case 3

Male, 14 years old. He was referred from one of the peripheral hospitals, with a three-month history of cough, haematuria, loss of appetite and weight. His father had active pulmonary tuberculosis. Examination revealed bilateral otitis media with multiple perforations, hepatosplenomegaly, swelling of the left leg, and hypertension with a blood pressure recording of 140/100. Doppler studies of the lower limb revealed lower segment deep vein thrombosis. Chest radiograph (Fig. 9) displayed features of pulmonary tuberculosis (extensive bilateral patchy shadowing and left hilar lymphadenopathy).

Acid-fast bacilli were cultured from the urine specimen and aural discharge. An intravenous pyelogram (Fig. 10) revealed hydronephroureter on the right and a non-functioning kidney on the left. A left nephrectomy was performed and renal tuberculosis was confirmed histologically.

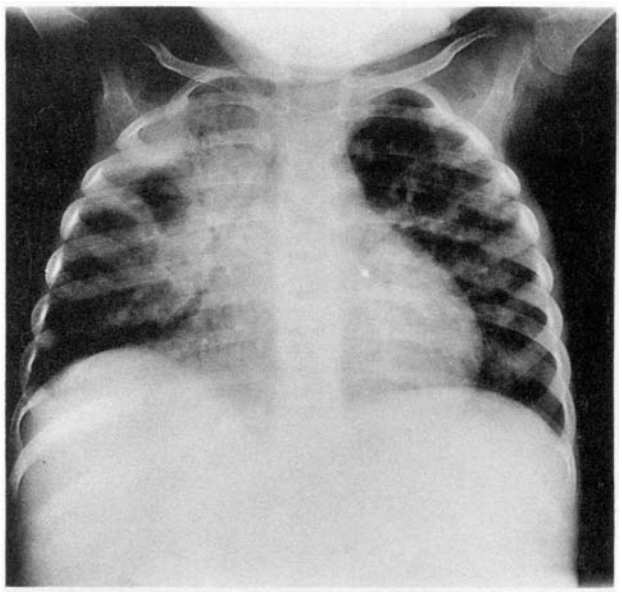

FIG. 2

Chest radiograph showing typical features of pulmonary tuberculosis, patchy and confluent shadowing in the right upper zone and perihilar lymphadenopathy.

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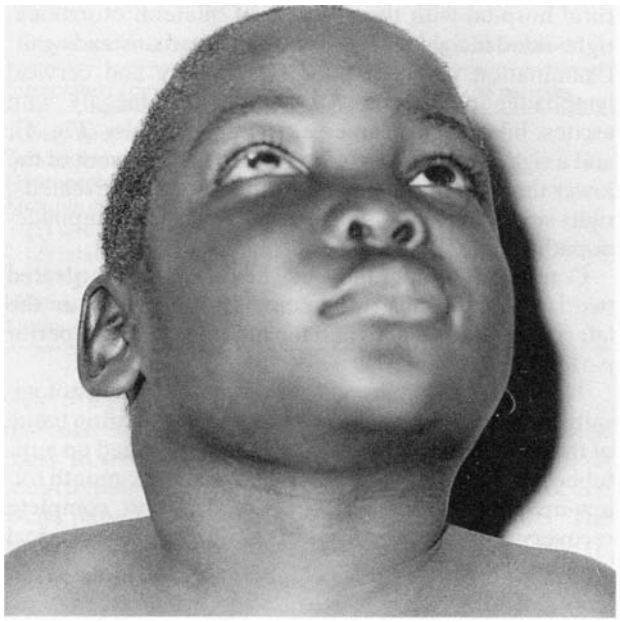

FIG. 3
Bilateral cervical lymphadenopathy.

The deep vein thrombosis settled on conservative treatment. The high blood pressure was controlled initially with methyldopa but after nephrectomy, the blood pressure returned to normal. The aural discharge subsided after four months of anti-tuberculous therapy, but the multiple perforations persisted.

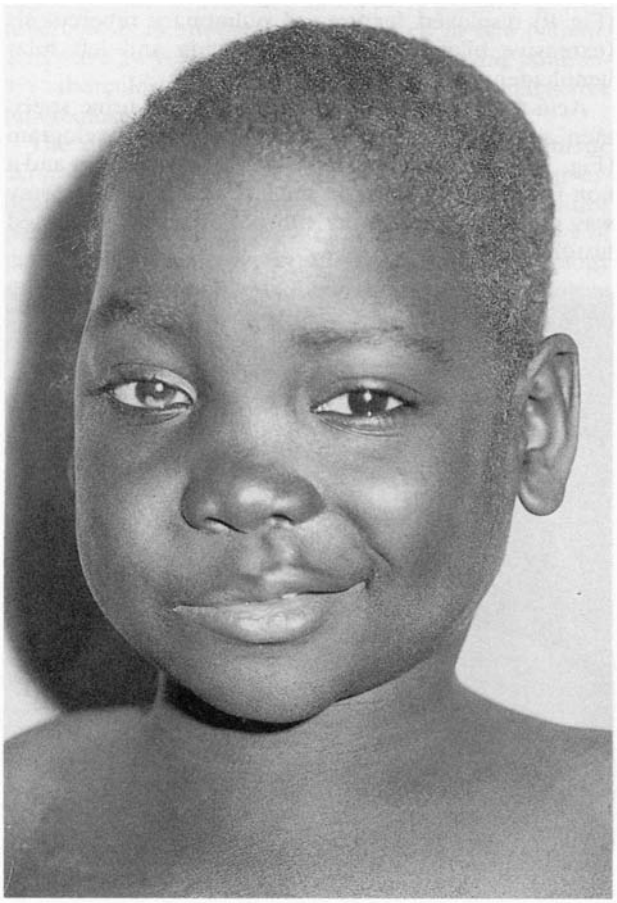

FIG. 4
Complete right lower motor neuron facial palsy.

Results

Dryness of the ear, with resolution of the granulation tissue in the external ear canal, was noted in all patients. The time taken to achieve this ranged from one to five months, with the average being two months. The results were the same in all three groups.

The recovery of the facial palsy in non-operated ears was 91 per cent (10 of 11 patients), whilst in the operated ears (Groups I and II), it was 80 per cent (four of the five patients). The speed and degree of recovery was directly related to the time interval between the onset of palsy and the commencement of treatment. When this interval was less than five days, complete recovery resulted, but when it was greater than two months, no recovery occured (Table VII).

Discussion

Clinical features

The clinical features associated with tuberculous mastoiditis can be categorized into three different groups (Table VI). Painless and profuse otorrhoea is a feature peculiar to tuberculosis of the ear and helps one to differentiate tuberculous from pyogenic suppurative otitis media. Great emphasis was placed on this clinical feature by earlier authors.

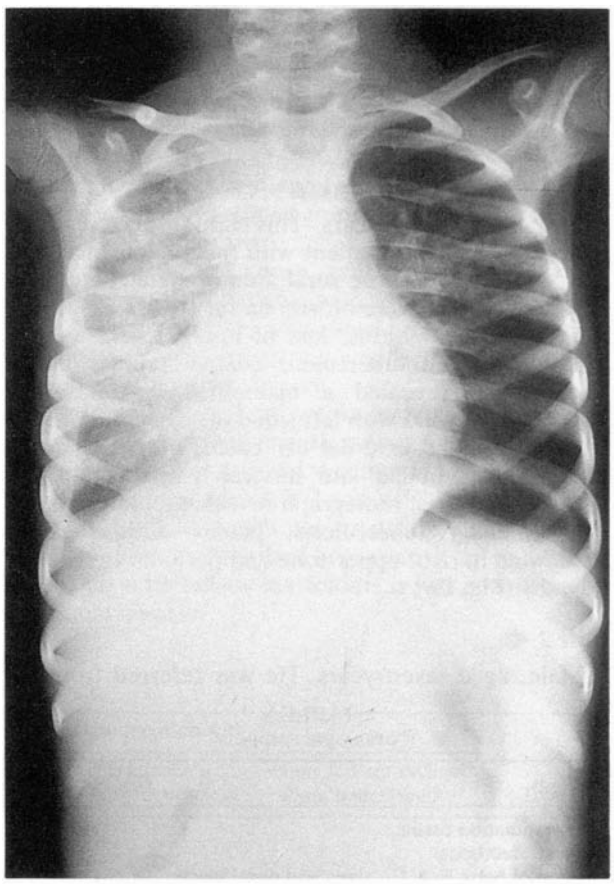

FIG. 5
Chest radiograph displaying a right-sided consolidation with paratracheal lymphadenopathy.

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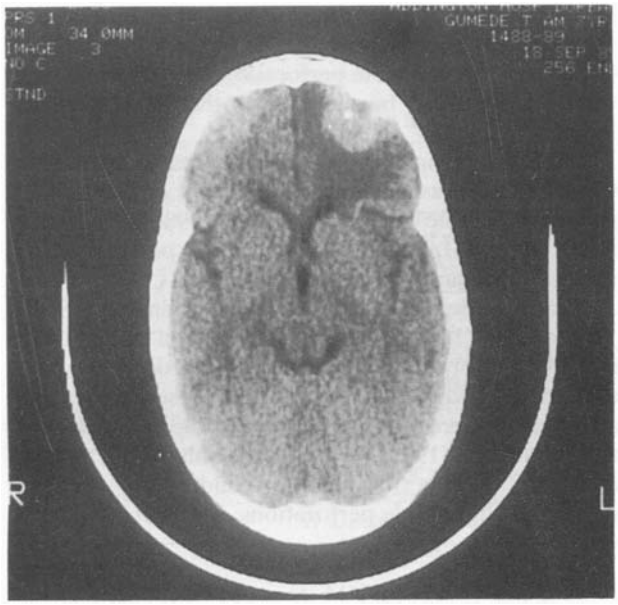

FIG. 6
CT scan showing tuberculoma in the frontal lobe.

Turner and Fraser (1915) reported that the onset of tuberculous mastoiditis is as a rule 'quiescent in character'. In their series of 60 patients, 92 per cent had no history of apparent pain, restlessness or fever, either preceding or accompanying the onset of ear infection. Similar high incidence of painless otorrhoea was reported by Guthrie (1920) 90 per cent and Craig (1962) 100 per cent. Slightly lower incidence was reported by M'Cart (1925), 54 per cent; Windle Taylor and Bailey (1984) 50 per cent; Jeanes and Friedmann (1960) 42 per cent and Ormerod (1931) 35 per cent.

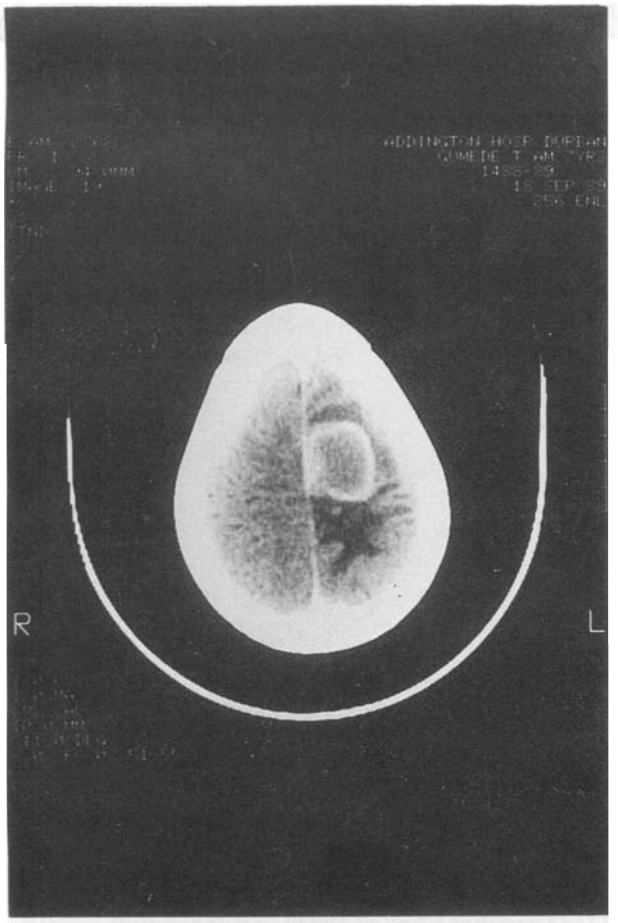

FIG. 7
CT scan showing tuberculoma in the superior parietal region.

In the present series, a history of painless otorrhoea could not be accurately determined because the patients presented late and on average, there was a three month interval between the onset of otorrhoea and the presentation to hospital.

With regard to profuse otorrhoea this is not only peculiar to tuberculous mastoiditis but can also occur in severe pyogenic, suppurative otitis media. However, what is important is when the profuse otorrhoea continues despite mastoidectomy and intravenous antibiotic therapy; in these patients the diagnosis of tuberculosis should be suspected. In the present series, there was one such patient (case 4). He was a six-year-old boy who presented with a postauricular subperiosteal abscess and otorrhoea. He had no clinical features of tuberculosis. At surgery, sequestrated bone and granulation tissue was found in the mastoid cavity. This was removed and sent for histology. No drilling was performed as the disease itself had created a radical cavity. A meatoplasty was performed and the radical cavity packed with quarter inch ribbon gauze, impregnated with BIPP (Bismuth Iodoform Paraffin Paste). Intravenous ampicillin and metronidazole was given for five days. Histology of both

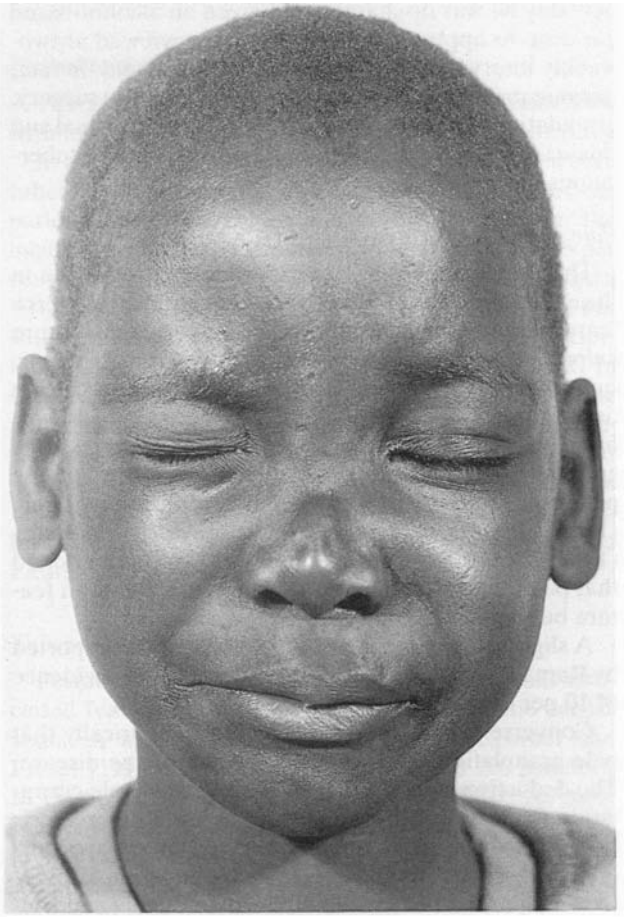

FIG. 8
A six-month follow-up photograph showing almost complete recovery of the right facial palsy and resolution of the cervical lymphadenopathy.

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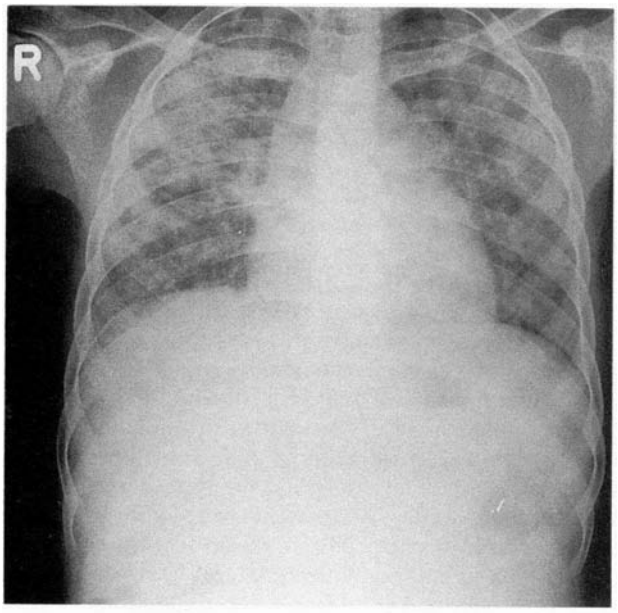

FIG. 9

Chest radiograph displaying features of pulmonary tuberculosis, extensive bilateral patchy shadowing and left hilar lymphadenopathy.

the sequestrated bone and granulation tissue revealed no evidence of tuberculosis. On the fourteenth post-operative day, the pack from the ear was removed. The next day he was discharged and given an alcohol-based ear drop to apply twice daily. He was reviewed at two-weekly intervals. The otorrhoea continued and, in fact, became profuse with time. Ten weeks after the surgery, granulation tissue appeared in the external ear canal and this was biopsied. The histology confirmed it to be tuberculous in origin.

Pale granulation tissue

The presence of pale, avascular granulation tissue in the external ear canal, middle ear and mastoid cavity is a feature unique to tuberculosis of the ear. This feature helps one to differentiate tuberculous from pyogenic suppurative otitis media. In the latter, the granulation tissue is very hyperaemic and friable and bleeds profusely with the slightest touch. In the present series, pale granulation tissue was present in 92 per cent of the patients. Similar high incidence was reported by Craig (1962) 100 per cent, and Windle Taylor and Bailey (1980) 73 per cent. Turner and Fraser (1915) mentioned that pale, flabby, granulation tissue was a common feature but did not quote figures.

A slightly lower incidence of 52 per cent was reported by Ramages and Gertler (1985) and a very low incidence of 10 per cent was reported by M'Cart (1925).

Conversely Ormerod (1931) stated categorically that pale granulation tissue is not a feature of the disease. The deduction from the present series is that pale granulation tissue is definitely a feature of aural tuberculosis but in the absence of overwhelming secondary pyogenic infection. If the secondary pyogenic infection is overwhelming, then the granulation is hyperaemic and friable. Such was the case in four (8 per cent) of the patients.

Multiple perforations

Multiple tympanic membrane perforations is definitely a feature of aural tuberculosis but its incidence is very low. In the present series, there were two patients (5 per cent) with double perforations in both ears. One was a 12-year-old boy and the other a 21-year-old female.

This low incidence of 5 per cent compares favourably with the findings of Leegaard (1921), 4 per cent (1/25 patients), Windle Taylor and Bailey (1980), 5 per cent (1/22 patients) and M'Cart (1925), 9 per cent (5/55 patients), but not with those of Ormerod (1931), 14 per cent (8/58 patients) and Jeanes and Friedmann (1960), 17 per cent (1/12 patients). The second point about multiple perforations is that it tends to occur in older children and adults, as noted in the present series and also by Leegaard (1921), Ormerod (1931) and Jeanes and Friedmann (1960). M'Cart is the only author to have noted multiple perforations in children; of the five patients with multiple perforations, four were children.

Facial palsy

Facial palsy is a feature of tuberculous mastoiditis that is confined mainly to children. The incidence of facial palsy increases with decreasing age.

For some unknown reason, it tends to be unilateral although the patient may have bilateral disease. In the present series, there were 17 (40 per cent) patients with facial palsy and 16 (94 per cent) were children below the age of 10 years, and nine (53 per cent) were in the one to two year age group (Table II). One child had bilateral facial palsy.

This 40 per cent incidence compares favourably with those reported by Turner and Fraser (1915) 45 per cent;

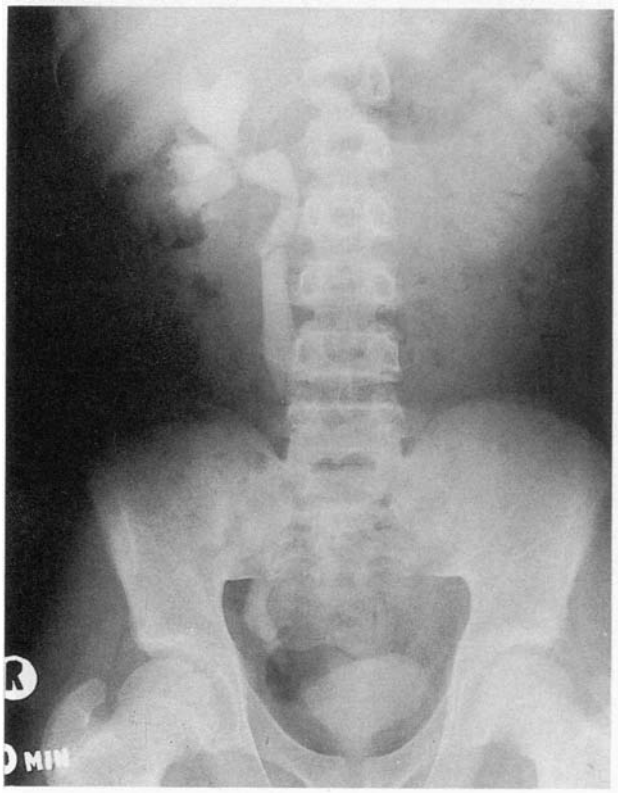

FIG. 10

Intravenous pyelogram showing hydronephroureter on the right and a non-functioning kidney on the left.

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TABLE VI
CLINICAL FEATURES THAT ARE HIGHLY SUGGESTIVE OF TUBERCULOUS MASTOIDITIS

	Otological features alone	Otological features (otorrhoea or M.E. effusion) together with
Pre-operative features	1. Painless otorrhoea 2. Profuse otorrhoea 3. Multiple perforations 4. Presence of pale granulation tissue in the E.A.C., or M.E. 5. Disproportionate hearing loss	1. Facial palsy 2. Pulmonary tuberculosis 3. Periotic lymphadenopathy 4. Active tuberculosis in other sites 1. TB meningitis 2. Renal TB 3. Spinal TB
Intraoperative features	1. Pale granulation tissue within the mastoid cavity. 2. Bone necrosis. 3. Sequestra formation. 4. Presence of caseous material.	
Postoperative features	1. Failure of otorrhoea to subside following mastoidectomy and intravenous antibiotic therapy. 2. Discharging post-auricular sinus.	

Guthrie (1920) 54 per cent; M'Cart (1925) 39 per cent, and Samuel and Fernandes (1986) 39 per cent, but not with those reported by Palva *et al.* (1973) 21 per cent, Windle Taylor and Bailey (1980) 17 per cent, Plester *et al.* (1980) 7 per cent and Ramages and Gertler (1985) 24 per cent.

To date, with the exception of one reported in the present series, there has been no report of bilateral facial palsy occurring in a patient, despite them having bilateral, tuberculous mastoiditis.

Turner and Fraser (1915) even mentioned 'In none of those which the disease affected both ears, was a bilateral facial palsy observed.'

The patient with bilateral facial palsy was a two-year-old male child, (Case 5), who presented with a two-week history of bilateral otorrhoea and neck swelling.

On examination, he was found to be pyrexial with a temperature of 39°C. He had bilateral cervical lymphadenopathy, bilateral otitis media with bilateral facial palsy, and neck stiffness. Chest radiograph displayed features of tuberculosis (nodular shadowing in right upper lobe and perihilar lymphadenopathy). The microscopic and biochemical anlaysis of the cerebrospinal fluid revealed features that were highly suggestive of tuberculous meningitis. Computed tomography of the brain revealed two low, dense lesions (tuberculomata), one in the left parietal region and the other in the right posterior fossa. Bilateral tuberculous mastoiditis was diagnosed histologically from the granulation tissue that was removed from the right and left external ear canals.

Intracranial complications

Intracranial complications of tuberculous mastoiditis are extremely rare. The reason for this is, as stated by Proctor and Lindsay (1942), that the dura mater is very resistant to intracranial extention of tuberculosis from the temporal bone. If intracranial complications do occur, it is usually secondary to haematogenous spread. In the present series, there were six (14 per cent) patients with intracranial lesions (as illustrated in Table III), and all of them had features of disseminated disease, thus supporting Proctor and Lindsay's (1942) theory of haematogenous spread.

Tuberculous meningitis has been a rare complication of tuberculous mastoiditis, as only 13 cases have been reported (Turner and Fraser, 1915; Guthrie, 1920; M'Cart, 1925; Samuel and Fernandes, 1986). Likewise intracranial tuberculomata have been an extremely rare complication of tuberculous mastoiditis and there are only 10 reported cases in the literature (Garland and Armitage, 1933; Bramwell, 1934; Stewart, 1934; Grabscheid, 1937; Mayers *et al.* 1978; Jeang and Fletcher, 1983; O'Brien *et al.*, 1988).

Pulmonary tuberculosis

The incidence of pulmonary tuberculosis associated with mastoiditis varies in different parts of the world.

In a developing country such as South Africa, where tuberculosis is endemic and a large percentage of the patients with tuberculous mastoiditis are chldren, the incidence is high. In the present series there was a 95 per cent incidence. Samuel and Fernandes (1986) reported a 70% (16 of 23 patients) incidence. A slightly lower but still high incidence was reported by Ramages and Gertler (1985) 52 per cent and Craig (1962) 50 per cent. In the developed countries, where a large percentage of the patients are adults, the incidence of pulmonary tuberculosis associated with tuberculous mastoiditis is low. This is shown in reports by Jeanes and Friedmann (1960) who reported a 25 per cent incidence, Windle Taylor and Bailey (1980) 23 per cent, Palva *et al.* (1973) 14 per cent and Plester *et al.* (1980) 14 per cent.

Tuberculous lymphadenitis

Periotic lymphadenopathy was a very prominent associated feature of tuberculous mastoiditis in the earlier years. A high incidence was reported by Turner and Fraser (1915) 95 per cent, Guthrie (1920) 100 per cent, M'Cart (1925) 94 per cent and Craig (1962) 100 per cent. In fact it was because of this high incidence that Craig (1962) stated 'Enlargement of the preauricular gland is so common that I think that it can be regarded as pathognomic of tuberculous mastoiditis.'

Presently, the incidence of periotic lymphadenopathy has declined. In the present series, there was a 23 per cent incidence. A slightly lower incidence of 9 per cent was reported by Samuel and Fernandes (1986).

TABLE VII

RELATIONSHIP BETWEEN THE INTERVAL OF FACIAL PALSY AND THE COMMENCEMENT OF TREATMENT TO THE RECOVERY TIME AND THE DEGREE OF RECOVERY

Interval between onset of facial palsy and commencement of treatment	No. of patients in each group			Average time for recovery	Degree of recovery
	Group I	Group II	Group III		
2 days	1	1	7	1 month	complete
3 days	1	1		2 months	complete
4 days			1	4 months	complete
5 days			1	6 months	complete
2 weeks			1	9 months	incomplete
2 months	1				nil
3 months			1		nil

Other sites of active tuberculosis

The rule is that any patient who has a discharging ear and evidence of active tuberculosis in any other site in the body, then that patient has tuberculosis of the ear until proved otherwise. In the present series, there was one such patient (Case 3). A similar observation was made by Jeanes and Friedmann (1960), who reported that a 27-year-old male patient, who had active tuberculosis of the spine and otorrhoea, was found to have tuberculous mastoiditis on histology of the bone chips removed at mastoidectomy.

Intra-operative features of tuberculous mastoiditis

The typical intra-operative findings in tuberculous mastoiditis are the presence of pale granulation tissue within the mastoid cavity, middle ear and external ear canal, extensive bony destruction with evidence of bone necrosis and sequestra formation. Caseous material may or may not be present.

The operative findings, as shown in Table V, are consistent with those reported by Turner and Fraser (1915), M'Cart (1925), Jeanes and Friedmann (1960), Craig (1962), Palva *et al.* (1973), Windle Taylor and Bailey (1980) and Ramages and Gertler (1985).

Post-operative features of tuberculous mastoiditis

The development of a discharging post auricular sinus and the stubbornness of that otorrhoea to subside, following mastoidectomy and the use of conventional antibiotics readily distinguishes tuberculous from pyogenic, suppurative otitis media, as demonstrated in Case 4. Similar findings were reported by Jeanes and Friedmann (1960), Hiranandani (1967), Palva *et al.* (1973), Emmett *et al.* (1977), Windle Taylor and Bailey (1980).

Diagnosis

The outstanding clinical features of aural tuberculosis makes the diagnosis to be established on clinical grounds a rule. However, in order for the diagnosis of tuberculosis not to be missed, one must acquaint oneself with all the clinical features of aural tuberculosis, which are illustrated in Table VI.

The role of bacteriology and histology, as it has always been in medicine, is to confirm one's clinical suspicion. If one relies on these investigations for diagnosis, then tuberculous mastoiditis will go unnoticed in a large percentage of patients. In the present series, there were four patients in whom the diagnosis of tuberculosis could not be confirmed on the initial biopsy specimen. Two of these patients required repeat and the other two, a third biopsy specimen before the diagnosis of tuberculosis could be confirmed. Had tuberculosis not been suspected clinically in these patients, then the diagnosis would have been missed. These findings have been observed by other authors as well.

Emmett *et al.* (1977) reported that an 18-year-old female had four surgical procedures before the diagnosis of tuberculosis was established. The biopsy specimen taken at the second surgical procedure did not display any features of tuberculosis.

Palva *et al.* (1973) reported that tuberculosis was missed on the initial biopsy in an 18-year-old male patient with otitis media.

M'Cart (1925) reported that in 26 (72 per cent) instances, the diagnosis of tuberculous otitis media was definitely proven on microscopic examination of either the granulation tissue or bone. In the remaining 10 cases, no microscopic examination was done and the diagnosis of tuberculosis was made clinically or at surgery.

Jeanes and Friedmann (1960) reported on a five-year-old boy who presented with a left-sided otorrhoea of 18-months' duration. Mastoid exploration was performed at another hospital six months previously. Histology of the granulation tissue at that time showed no evidence of tuberculosis. However, tuberculosis was confirmed on histology of the granulation tissue removed from the external ear canal, at the second visit.

The tissue for histological analysis to confirm your clinical diagnosis is readily available from the external ear canal and mastoidectomy for this purpose is totally unnecessary. In the present series, tissue for histological examination was obtained from the external ear canal in 29 patients.

Results

The prognosis of facial palsy is not dependent on decompression as stated by Legent and Baron (1975), Lucente *et al.* (1978), Plester *et al.* (1980), Windle Taylor and Bailey (1980), but rather on early diagnosis and treatment with antituberculous drugs. The speed and degree of facial recovery is dependent on the time interval between the onset of facial palsy and the commencement of treatment. If this time interval is less than five days, then complete facial nerve recovery can be expected, but if it is greater than two months, then there will not be any recovery, as shown in Table VII.

Conclusion

Surgery plays a minor role in both the diagnosis and treatment of tuberculous mastoiditis.

The outstanding clinical features of tuberculous mastoiditis enables the diagnosis to be established on clinical grounds. The role of surgery in this regard is to obtain tissue for histological confirmation and it merely involves biopsy of either the polyp or the granulation tissue from the external ear canal. Mastoidectomy for this purpose is totally unnecessary.

With regard to treatment, antituberculous chemotherapy is the management of choice. Surgery is only indicated when tuberculous mastoiditis is complicated by a subperiosteal abscess and it usually entails incision and drainage and removal of sequestrum if present.

Mastoidectomy for the purpose of eradicating the disease is not indicated as complete resolution of the granulated tissue and otorrhoea occurs on antituberculous therapy alone. Decompression of the facial nerve in patients with facial palsy is totally unnecessary as recovery is dependent on early diagnosis and treatment with antituberculous drugs.

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