A SERIES

OF

LARYNGEAL AND AURAL TUBERCULOSIS

L. J. RAMAGES

MB.,ChB.(UCT). FRCS (Edin). FRCS (Glasg). FCS (S.A.) L et O.

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CONTENTS

I. SURVEY OF STUDY DATA

The following study surveys a series of Laryngeal Tuberculosis and a series of Aural Tuberculosis. The Laryngeal Tuberculosis study includes only those patients seen during 1983. The series of Aural Tuberculosis is comprised of cases diagnosed and treated at Groote Schuur Hospital, Cape Town during the years 1972-1983. This study is a sequel to the series of 1967-1971 as reported by Sellars and Seid (1973), and formed the basis of a further publication by Ramages and Gertler (1985).

II. LARYNGEAL TUBERCULOSIS

1. INTRODUCTION

In 1924 Sir StClair Thompson cited laryngeal tuberculosis as the most common specific disease of the larynx (Thompson, 1924a). It was usually concomitant with cavitatory pulmonary tuberculosis and over 70 per cent of these patients suffered a fatal outcome (Thompson, 1924b).

In an autopsy survey of 811 patients who died of tuberculosis, Auerbach (1946) reported that laryngeal involvement was found in 304 of 811 cases (37.5%). The typical pathological features described were severe ulceration and destruction of mucosa and submucosa and even extension into muscles and cartilages of the larynx.

Treatment prior to the era of specific chemotherapeutic agents for tuberculosis comprised the general measures of bed rest, adequate dietary intake, voice rest and sunshine; while the specific treatment consisted of cauterization of laryngeal granulomata via indirect laryngoscopy and 'lung collapse' by artificially-induced pneumothorax. The latter was in keeping with the concept of 'rest' and designed to decrease the expectoration of infected sputum that would coat the laryngeal surfaces.

Since the advent of Streptomycin (late 1940s) and INH (early 1950s) the incidence of pulmonary and laryngeal tuberculosis has declined and so has the clinical awareness of laryngeal tuberculosis.

In 1982 Waldman stated that, with the decrease in the general incidence of tuberculosis, and Head and Neck involvement consequently being rare, the clinician was not "sensitized" to the disease as part of his differential diagnosis (Waldman, 1982). Usually direct laryngoscopy and biopsy are sufficient to diagnose the disease. Tuberculosis should not be overlooked in patients presenting with hoarseness, odynophagia, otorrhoea, pharyngitis, or mucosal membrane lesions.

While there have been sporadic reports of laryngeal tuberculosis based on cases usually accumulated over a number of years, there is a distinct impression gained from recent clinical observation - nine patients within a one-year period - that tuberculous laryngitis is on the increase. By publishing this clinical experience, I hope to re-awaken the awareness and "sensitize" my colleagues to this long known yet frequently overlooked disease.

2. CASE MATERIAL

Table I gives a comparative study of the nine patients diagnosed or seen in the Otolaryngology department during 1983. Eight of these patients were of recent onset and one old patient dating back to 1980. Four of the nine patients were known previously to have suffered from pulmonary tuberculosis; the other five patients presented as primary laryngeal problems. All the patients were referred to the appropriate Tuberculosis Hospitals or Centres for their continued treatment. Severely ill patients who required close ENT supervision because of severe upper airway problems were admitted to our unit initially and their anti-tuberculosis chemotherapeutic regimen dictated from the nearby Tuberculosis Hospital. These patients were then transferred to the Tuberculosis Hospital as soon as it was deemed safe to do so. The treatment regimens of all our patients were one or other of the accepted regimens as prescribed in the Department of Health booklet on Tuberculosis Treatment Regimens (Department of Health and Welfare, Pretoria RSA, 1982). Seven of our eight recent patients made a full recovery from their laryngeal involvement. Two patients (Nos. 3 and 9) had a moderate-to-severe laryngeal deformity as a result of tuberculosis in which specific treatment did not prevent the noxious effects of the disease as so often occurred in days gone by.

Patient No. 3 underscores the danger of resistance developing when the patient is continued on monotherapy rather than on combined chemotherapy. Eighteen months prior to presentation this patient was diagnosed as having pulmonary tuberculosis. A regimen of combined chemotherapy was instituted for six months and, thereafter, monotherapy (INH) was continued for a further twelve months. Whilst on INH therapy the patient was referred by her phthisiologist to the otolaryngology out-patient department because of hoarseness. The patient was followed-up for six consecutive months observing diffuse subglottic oedema and crusting of the vocal cords in a fibrosed and stenosed larynx. Finally her clinical condition necessitated admission, direct laryngoscopy and biopsy which revealed the presence of Acid-fast Bacilli on histological section. It was concluded that the disease had become INH-resistant and this was borne out by the clinical response to an appropriately modified chemotherapeutic regimen.

Patient No. 2 needed a tracheostomy because of florid extensive granulomatous disease virtually obliterating the rima glottidis. In this patient four consecutive sputum examinations were negative for Acid-fast Bacilli while the histology showed caseating tuberculoid granulomata. The patient improved dramatically over a three to four week period while on specific anti-tuberculosis treatment such that his tracheostomy tube could be safely removed.

Patient No. 5 displayed laryngeal disease with concomitant pharyngeal, palatal and tonsillar involvement. This patient had cavitatory pulmonary tuberculosis on chest X-ray and a positive sputum for Acid-fast Bacilli. The response to combined chemotherapy in the initial few weeks of therapy was dramatic.

Patient No. 9 at no time had x-ray changes of tuberculous involvement of the lungs. The diagnosis was made on laryngeal biopsy and response to specific combined chemotherapy. However, residual fibrotic changes left the patient with a grossly distorted and functionally compromised larynx.

All our patients responded dramatically to adequate chemotherapy with responses noted as early as two weeks after commencement of therapy.

TABLE I

Pat. no.	Sex	Race	Age	Duration of hoarseness	Odyno-phagia	Findings at laryn-goscopy	Sputum for AFBS	CXR	Previous diagnosed PTB	Histo.
1.	M	B	56	2 weeks	No	Bilateral granulo-matous lesions of true cords. Left true cord ulcerated.	NEG	POS	1978	Not done
2.	M	C	32	+2 weeks	Yes	Extensive granulo-matous lesions of glottis and supra glottis. Virtually oblitera-ting the rima glottidis.	NEG	POS	No	Caseating tubercu-loid granulom-ata
3.	F	C	33	6 months	No	Crusted true cords. Fibrosed and sten-osed larynx. Severe swelling of entire sub glottis.	Not known	POS	1981	AFB on section

(to continue)

TABLE I (continued)

Pat. no.	Sex	Race	Age	Duration of hoarseness	Odyno-phagia	Findings at laryn-goscopy	Sputum for AFBs	CXR	Previous diagnosed PTB	Histo.
4.	M	W	41	6 months	No	Oedematous reddened granulo-mata of true left cord.	No spu tum	POS	No	AFB on section
5.	F	B	43	3 months	Yes	Oedematous true cords with red granulomata. Ulcers and granulomata of right tonsil and soft palate.	POS	POS	No	Not done
6.	F	B	30	12 months	No	Scarring of vocal cords. No granulo-mata	POS	POS	1971	Not done
7.	M	B	25	3 weeks	No	Oedematous true cords and left false cord granu-lomata.	NEG	POS	No	Not done
8.	M	C	63	12 months	No	Thickened, reddened granulom-atous right true and false cord. Paralysed, left hemi-larynx.	POS	POS	Retro-spective 1980. Not treated	Not done
9.	F	B	47	4 months	No	Fibrosed, stenosed, distorted larynx. No active ulcers or granulom-ata.	Not known	NEG	1980	1980 compat-ible with TB

3. DISCUSSION

(a) INCIDENCE

In recent English publications on the subject of tuberculous laryngitis, a persistent incidence of pulmonary tuberculosis with concomitant tuberculous laryngitis exists, though the impression is that of a decreasing incidence. There have been reports from developed countries such as the U.S.A., England and Germany of rare cases of patients suffering from tuberculous laryngitis without having clinical or radiological evidence of pulmonary tuberculosis. We have documented one such case (Patient No. 9).

Prior to specific anti-tuberculosis chemotherapy, laryngeal tuberculosis was a disease of young people with advanced cavitatory pulmonary tuberculosis and the peak incidence was in the third and fourth decades of life. More recently, the peak incidence is cited as being in the fifth and sixth decades. In our series the mean age was 41 years with most of the patients falling in the third and fourth decades. This expression of 'past history' age incidence of the disease may be due to the fact that many blacks and coloureds in this age group did not get routine BCG innoculation during infancy or later. Added to this is the socio-economic problem of overcrowding, unsatisfactory housing, less health care awareness, and greater exposure to cases of tuberculosis in an already unsatisfactory environment.

Based on the 1983 figures for the otolaryngology department of Groote Schuur Hospital there has been an alarming increase in the number of patients with laryngeal tuberculosis. This appears to be accounted for by the absolute increase in the incidence of pulmonary tuberculosis in the Cape Town precincts.

(b) SITE AND APPEARANCES OF LESIONS

In the 19th century and in the first-half of this century, the classical laryngeal tuberculous lesion was said to occur at the posterior commissure of the larynx and in its posterior half. This specific siting was thought to be due to large amounts of sputum heavily infected with Acid-fast Bacilli accumulating in this part of the larynx in the very ill patient bedridden in the recumbent position. However, in spite of the above description, Auerbach (1946) summarized post-mortem examinations of patients who died from tuberculosis during a five-and-a-half-year period (1940-45) and found that the commonest site of laryngeal lesions was in fact the true vocal cord (47 per cent) and in reducing order of frequency: in the false cord, epiglottis, subglottis and posterior commissure area. Other authors (Parrish, 1910; Wilson, 1943) concur with these findings. Our cases appear to conform to this site frequency.

In the past, appearance of the laryngeal lesion was described as a gross and obvious exophytic lesion with severe ulceration of laryngeal mucosa and destruction of muscle and cartilage. Nowadays the lesion is more confined showing either a local tumour-like appearance or that of chronic non-specific laryngitis without any immediate threat to the airway. In this series, with the exception of Patient No. 2, lesions conformed to the latter. The lesions seen nowadays are obviously just the early manifestations of the same disease process. Recent deaths from neglected cases of laryngeal tuberculosis at John Hopkins Hospital (Boitnott, 1967) and the Massachusetts General Hospital (Castleman and McNealy, 1968) revealed lesions no different from the earlier descriptions.

A recent publication on laryngeal tuberculosis (Hunter et al., 1981) stresses the fact of the "changing behaviour of laryngeal tuberculosis since the advent of specific chemotherapy" - namely:

* Site of attack now true cord or true and false cord rather than posterior commissure and posterior half of the larynx.

* Disease more confined showing a tumour-like or chronic non-specific laryngitis appearance.

* More patients with laryngeal tuberculosis with minimal or no pulmonary tuberculosis changes.

I feel that nothing has materially changed in the character of the Mycobacterium tuberculosis or in its mode of infection but that the changing manifestations may be explained by the following:

* Early detection of pulmonary tuberculosis by mass screening methods and family contact screening.

* Early laryngeal detection because of suspicion of malignant tumour.

* Early vigorous specific treatment.

* Late laryngeal involvement in pulmonary tuberculosis cases treated with combination therapy then converted to monotherapy with subsequent resistant mutant development. We have recorded one such case (patient No. 3).

* Disease manifestation in a previously immunized subject but now having reduced immunity. Though detected late the lesion may have a less florid appearance.

Laryngeal tuberculosis may mimic a tumour or chronic non-specific laryngitis. Table II is a resume of recent publications addressing this problem.

TABLE II

Author:	TB Larynx mimicking: Chronic non-specific laryngitis	Exophytic tumour-like lesion
Anderson and Stevens (1981)	4	1
Bull (1966)	3	7
Maran and Stewart (1971)	1	2
Rohwedder (1974)	7	4
Travis et.al. (1976)	12	1
Hunter et. al. (1981)	2	8
Yarnal et. al. (1981)	0	1
Espinosa et. al. (1981)	0	2
Bailey and Windle-Taylor (1981)	14	23

In cases where the tumour appearance is not typical of exophytic carcinoma, tuberculous laryngitis should always be considered and sputum examination and culture, chest X-rays and biopsies should be carefully reviewed and correlated.

The detailed pathogenesis, macroscopic and microscopic appearances of tuberculous laryngitis which were so accurately described by Auerbach (1946) help us to understand the different pleomorphic appearances in these patients. Tuberculous foci in the submucosa may be variable distances from the mucous membrane which may remain intact or become hypertrophic due to squamous metaplasia. The surface mucosa will ulcerate when the submucosal tubercles expand and exert pressure on the under surface of the mucosa or by caseation necrosis of these tubercles which in turn erode the mucosa. The mycobacteria penetrate the mucosal surface of the larynx via ducts of the submucosal glands or through microlacerations in the mucosa due to forceful coughing in these patients with pulmonary tuberculosis. The haematogenous route remains another possibility.

(c) DIAGNOSIS

Clinical suspicion usually precedes clinical diagnosis and the former comes only in the wake of clinical awareness. The chest X-ray when positive, as in eight of our nine patients, is helpful but does not clinch the diagnosis. Remember that carcinoma of the larynx and pulmonary tuberculosis may co-exist in the same patient. However, if the lesion in such a case shows rapid response to anti-tuberculosis treatment, as noted in all our eight recent patients, then biopsy can be withheld. Initial response to treatment may be fairly rapid and early improvement may be noted as early as two weeks after commencement of therapy.

Positive sputum for Acid-fast Bacilli carries the same diagnostic index as the positive chest X-ray. Positive sputum examinations were obtained only in three patients out of the eight who had positive chest X-rays for tuberculosis.

Histology of the local biopsy is helpful in making the diagnosis and particularly so if the odd Acid-fast Bacillus is seen in the histological sections. Acid-fast Bacilli were seen in two of our four histological confirmations of the diagnosis. It must be accepted, however, that granulomas, as often noted, are non-specific and form part of the differential diagnosis that includes lethal midline granuloma, Wegener's granulomata, sarcoid, leprosy, and fungal disease.

The diagnosis is certain if biopsies submitted for Kirschner culture or guinea pig inoculation yield positive results. In cases where the diagnosis is not suspected at the time of taking biopsies, this important investigation is omitted. A negative Mantoux test does not exclude tuberculosis as a diagnosis. The negative result may be as a consequence of poor technique-where the injection has not been intradermal. Or the patient may be in a state of anergy. The simultaneous performance of a candida skin test is helpful in clarifying the latter. We have not resorted to doing a Mantoux test in these cases as the diagnosis was quite evident from other diagnostic parameters and, in the face of a positive chest X-ray, one would expect a positive Mantoux test and a negative result would have to be explained.

4. TREATMENT

One of the objectives of treatment regimens for both ambulatory and hospitalized patients is that administration of chemotherapeutic agents should be seen to be taken. For this reason it is expedient that the total daily amount of medication be administered as a single dose unless the patient obviously cannot tolerate it. It has been strongly recommended that treatment regimens should be directed towards Rifampicin-containing regimens for all patients and domiciliary treatment for the majority of patients where feasible. If tolerated by the patient, INH should be included in every treatment regimen for new cases, even when laboratory tests indicated resistance of the infecting organism (i.e. primary resistance), since a lesser colony of sensitive strains may well go by undetected (Department of Health and Welfare, Pretoria, RSA, 1982). In cases previously treated with INH and where laboratory tests show resistance to INH (secondary resistance) this chemotherapeutic agent may be omitted from the treatment regimen.

The use of Streptomycin should preferably be avoided in the elderly, the deaf or in those patients with impaired renal function. Some dramatic improvement is noted during the first two or three weeks under the influence of specific anti-tuberculosis chemotherapy - ulcers, red florid granulomata and massive oedema resolve, but residual oedema may linger on for a few months. Notwithstanding the initial rapid response the continued healing process is usually gradual. The patient must be followed up to ensure that resolution is progressing and that a relapse does not occur when anti-tuberculosis treatment is tapered from combined chemotherapeutics to lesser or monodrug therapy. Monodrug therapy is not recommended in any of the modern regimens. Chronic sequellae of laryngeal tuberculosis, such as fibrosis and stenosis of the larynx, cartilage involvement with airway impairment and poor voice may result in spite of adequate anti-tuberculosis treatment. Such was the end-result in patient No. 9. In extreme forms of this unfortunate complication, where the laryngeal sphincter mechanism is incompetent and the patient suffers with repeated aspirations and ineffective cough with an already grossly reduced respiratory reserve, a total laryngectomy may be needed.

III AURAL TUBERCULOSIS:

1. INTRODUCTION:

The overall incidence of tuberculosis affecting the middle ear cleft has decreased dramatically over the past forty to fifty years. This may be ascribed to the recent availability of specific bactericidal anti-tuberculous drugs, improvement in the public health services, massive innoculation of cattle with virtual eradication of Bovine-strain tuberculosis, improvement in housing and widespread B.C.G. innoculation compaigns directed at babies and children of school age. Nonetheless, there remains a persistent and even recent increase in the incidence of tuberculosis in the Cape Town district.

The incidence of aural tuberculosis at the beginning of this century was 3-5 per cent of all cases of chronic otitis media, as described by Jeanes and Friedmann (1960). Today tuberculosis of the middle ear cleft is rare, Jeanes and Friedmann (1960) documented twelve patients with aural tuberculosis out of a total of 23,000 patients (0.05 per cent) with chronic otitis media treated during a ten-year period (1950-1959) in London. Similarly, Palva et al. (1973) recorded fourteen patients with aural tuberculosis out of a total of 1638 operations for chronic disease during the years 1964-1971. Because of this low incidence of aural tuberculosis nowadays, the diagnosis may be overlooked, resulting in patients suffering unduly from a medically curable cause of chronic suppurative otitis media with its likely serious complications.

Four new patients with aural tuberculosis were diagnosed and treated at Groote Schuur Hospital in 1983. This was the motivating factor to review the data on previously diagnosed patients with the same diagnosis dating back from 1972 to 1983 as a sequel to the survey of Sellars and Seid (1973) which described aural tuberculosis in childhood presenting at Groote Schuur Hospital during the years 1967 to 1971.

2. CASE REPORTS:

During the twelve-year period from 1972 to 1983 thirty ears in twenty-five patients afflicted with tuberculous infection of the middle ear cleft were diagnosed and treated at Groote Schuur Hospital. In five patients both ears were affected simultaneously. The youngest patient was five months old while the oldest was 47 years of age. Thirteen patients were under five years of age, eight patients were in the five year-12 year group, while only four patients were adults. Racially, this series consisted of seventeen coloured and eight black patients. There were fifteen male patients and ten female patients. The analysis of these data is shown in Table IIIA, Table IIIB and Table IIIC

TABLE IIIA

PATIENT	RACE	SEX	AGE	EAR EXAMINATION	SURGERY
1	B	M	1	Granulations	Rad. mastoidectomy
2A	C	F	2	Post-auricular fistula Granulations	Modified rad. mastoidectomy
B	C	F	2	Granulations	
3	C	M	7	Sequestra. Granulations. Facial palsy	Rad. mastoidectomy
4	C	F	11	Pale granulations	EUA Biopsy
5A	C	F	4	Post-auricular abscess	Cortical mastoid.
B	C	F	5	Glue ear	Myringotomy & tube
6	C	M	33	Dry central perforation	Tympanoplasty and ossiculoplasty
7	C	F	2 1/2	Granulations	Cervical LN Biopsy
8	C	M	8	Post-auricular fistula. Polyp	Rad. mastoidectomy
9A	B	M	1	Polyp. Granulations	Cortical mastoidect.
B	B	M	1	Polyp. Granulations	EUA Biopsy
10	B	M	12	Granulations	Cortical mastoidect.
11	C	M	2	Facial Palsy	Cortical mastoidect.

(to continue)

TABLE IIIA (continued)

PATIENT	RACE	SEX	AGE	EAR EXAMINATION	SURGERY
12	B	M	6	Post-auricular abscess	Cortical mastoidect.
13	B	F	5/12	Post-auricular abscess	Cortical mastoidect.
14	C	M	12	Post-auricular abscess	Rad. mastoidectomy
15	C	M	3	Facial nerve palsy Granulations	Rad. mastoidectomy
16A	C	M	2	Granulations	EUA Biopsy
B	C	M	2	Granulations	EUA and Biopsy
17	B	F	8	Granulations	EUA and Biopsy
18	C	F	2	Facial nerve palsy Granulations	Rad. mastoidectomy
19	C	M	3	Post-auricular abscess	Rad. mastoidectomy
20A	C	F	8/12	Facial nerve palsy	Rad. mastoidectomy
B	C	F	8/12	Subtotal perforations	
21	C	M	14/12	Post-auricular fistula	Cortical mastoid
22	C	F	40	Facial nerve palsy	Cortical mastoid.
23	B	F	8	Post-auricular palsy	EUA and biopsy
24	B	M	43	Denuded promontory	Biopsy
25	C	M	27	Polypoid middle ear mucosa	Biopsy and Tympano-plasty

TABLE IIIB

PATIENT	PREVIOUS Hx TB	CXR	BACTERIOLOGY	HISTOLOGY
1	Yes	Pos		Pos
2A	Yes	Pos	Pos	
3	Yes	Neg		Pos
4	No		Pos	Pos
5A+B	No		Pos	Pos
6	Yes	Pos	Pos	
7	No		Pos	Pos
8	No	Neg	Pos	Pos
9A+B	No	Pos	Neg	Pos
10	No	Neg		Pos
11	No			Pos
12	No		Neg	Pos
13	No	Pos	Pos	Pos
14	Yes	Pos	Pos	
15	Yes	Pos		Pos
16A+B	No		Neg	Pos
17	No			Pos
18	Yes	Pos		Pos
19	No	Pos	Pos	Pos
20A+B	Yes	Pos		Pos
21	No	Pos	Neg	Pos
22	No	Neg		Pos
23	No	Neg	Pos	Pos
24	Yes	Pos		Neg
25	No	Pos	Pos	Pos

TABLE IIIC

DATA ANALYSIS OF 25 PATIENTS IN PRESENT SERIES

		No. of Patients
CHEST X-RAY :	Positive	13
	Negative	5
	Not recorded	7
BACTERIOLOGY:	Positive	11
	Negative	4
	Not done	10
HISTOLOGY :	Positive	21
	Negative	1
	Not done	3
History of previous Tuberculosis		9
Household family history of active Tuberculosis		2
Presenting with Facial Palsy		6
Presenting with Post-auricular abscess		5
Presenting with Post-auricular fistula		4
Both ears affected		5

3. THE DIAGNOSIS OF AURAL TUBERCULOSIS:

This is usually confirmed by the combination of clinical signs and symptoms, chest and mastoid X-rays, Ziehl Neelsen stains from swabs of aural discharge, surgical exploration and typical operative findings, histological examination to demonstrated classical histo-pathology and acid-fast bacilli, and finally positive Kirschner culture from biopsy material or aural discharge.

The clinical picture in all our patients was that of suppurative otitis media with persistent otorrhoea present for weeks, months or years. The commonest feature on otoscopy was of pale granulations of the middle ear mucosa seen through a central perforation of the tympanic membrane and granulations in the deep part of the external auditory canal. The perforations were usually central or subtotal. The small multiple perforations said to be typical of tuberculous otitis media were not seen. Visible sequestra were noted in three of our patients on initial otoscopic examination. Bone necrosis and sequestration was a common finding at surgical exploration.

Six patients presented at their first visit with chronic suppurative otitis media and a complete ipsi-lateral lower motor neurone facial nerve palsy. All of these patients but one were children, aged eight months, two years, two years, three years, seven years and 40 years.

A post-auricular swelling representing a post-auricular abscess, by extension of mastoid disease through the mastoid cortex, was present in five patients. Spontaneous breakdown of the skin over the mastoid, creating a discharging fistula surrounded by granulations, was seen in a further four patients.

Most patients had extensive destruction of the ossicular chain with severe conductive hearing loss demonstrated in those who had audiometry performed. Audiometry was not performed routinely since many of these patients were small children and/or seriously ill requiring urgent admission and surgery.

Bacteriology was positive for Tuberculosis in 11 patients but only six of these patients had positive findings from aural discharge (Ziehl Neelsen Stains and Kirschner culture). In the remainder a positive result was obtained by culture of middle ear or mastoid mucosa harvested during surgery. This positive yield was high compared to the earlier series of Sellars and Seid (1973) in which only one out of 11 ears yielded postive acid-fast Bacilli on examination of the aural discharge. In six of our cases secondary bacterial infection by proteus alone (four ears) or in combination with alpha-haemolytic streptococcus (two ears) and Klebsiella (one ear) was recorded. The secondary invaders are identical to those recorded in the earlier series by Sellars and Seid (1973).

Previous History of Tuberculosis

Nine patients were known sufferers of pulmonary tuberculosis while two patients had a positive family history of active pulmonary tuberculosis. In the other 14 patients the diagnosis of tuberculosis was made only after admission to our wards, using the aforementioned diagnostic measures. Chest X-rays positive for or suggestive of pulmonary tuberculosis were found in thirteen of the twenty five patients. Five patients had a clear chest X-ray. In seven patients the chest X-ray results were not documented.

4. TREATMENT:

(a) In patients known to have tuberculosis.

Specific anti-tuberculosis treatment, as prescribed by a nearby hospital, and local ear toilette was instituted. In addition, general supportive treatment for anaemia, malnutrition or kwashiorkor, in order to achieve maximal improvement of healing of the aural tuberculosis without having to perform an operation more extensive than would be required, was administered. Seven of the nine patients known to have pulmonary tuberculosis and with a tentative or confirmed diagnosis of aural tuberculosis had ear surgery because of persistent active ear disease or otorrhoea, granulations and sequestration of involved bone; post-auricular abscess or fistula and/or facial nerve palsy. One patient had reconstructive surgery in the convalescent phase. Details of the preoperative findings and surgical procedures are detailed in Table IV. The one patient who has not had an operation to date (Patient No. 24) will require tympanoplasty and ossiculoplasty when the tuberculous disease has been cured.

TABLE IV

TREATMENT OF PATIENTS WITH KNOWN PULMONARY TUBERCULOSIS

PATIENT No.	PRE-OPERATIVE FINDINGS	SURGICAL PROCEDURE
1	Persistent granulations	Radical mastoidectomy
2A	Post-auricular fistula Granulations	Modified radical mastoidectomy
3	Facial nerve palsy. Granulations. Sequestra	Radical mastoidectomy
14	Post-auricular abscess. Granulations	Radical mastoidectomy
15	Facial nerve palsy. Granulations	Radical mastoidectomy
18	Facial nerve palsy. Granulations Aural polyp	Radical mastoidectomy
20	Facial nerve palsy. Granulations	Radical mastoidectomy
6	Dry central perforation	Tympanoplasty and ossiculoplasty
24	Subtotal perforation. Denuded promontory	Nil

(b) In patients not previously known to have tuberculosis

In these sixteen patients the diagnosis was confirmed by examination of tissue harvested at operation. In six patients (Nos. 4, 9B, 16, 17, 23, 25) examination under anaesthesia and biopsy was sufficient to confirm the diagnosis and sufficed as the total surgical procedure, except for patient No. 25 in whom a tympanoplasty was performed as well. In one patient (No. 7) the diagnosis was made on cervical lymph node biopsy. Eight patients (Nos. 5A, 9A, 10, 11, 12, 13, 21 and 22) had cortical mastoidectomies before the diagnosis was established or as part of the treatment because of complications:- three patients with post-auricular abscesses, one with post-auricular fistula, two patients with facial nerve palsies, one with an obstructing polyp in the external ear canal, and one patient with non-responding chronic suppurative otitis media with granulations (patient No. 10). Two patients (Nos. 8 and 19) underwent radical mastoidectomy. One of the patients had a post-auricular abscess as a result of mastoiditis with a concomitant cholesteatoma which eroded the dome of the lateral semicircular canal and the facial nerve canal in the horizontal segment. The other radical mastoidectomy was performed in a patient with a post-auricular abscess where the tuberculosis had destroyed much of the bony structure of the mastoid, leaving the middle cranial fossa dura and the sigmoid sinus devoid of bone and covered with granulations only.

In eight of these sixteen patients the diagnosis was confirmed both by positive bacteriological and histological study. The other eight patients had positive histology only.

Pre-operative findings in the surgical procedures effected in these sixteen patients are detailed in Table V.

TABLE V

TREATMENT OF PATIENTS NOT PREVIOUSLY KNOWN TO HAVE TUBERCULOSIS

PATIENT No.	PRE-OPERATIVE FINDINGS	SURGICAL PROCEDURE
7	Persistent otorrhoea. Cervical adenopathy	Cervical lymph node biopsy
4	Persistent otorrhoea. Granulations	EUA and biopsy
16A	Persistent otorrhoea. Granulations	EUA and biopsy
B	Persistent otorrhoea. Granulations	EUA and biopsy
17	Persistent otorrhoea. Granulations	EUA and biopsy
23	Post-auricular fistula	EUA and biopsy
25	Perforation. Polypoid middle-ear mucosa	Biopsy and tympanoplasty
9B	Granulation Polyps	EUA and biopsy
A	Granulation Polyps	Cortical mastoidectomy
10	Persistent otorrhoea Granulations	Cortical mastoidectomy
11	Facial nerve palsy	Cortical mastoidectomy
12	Post-auricular abscess	Cortical mastoidectomy
13	Post-auricular abscess	Cortical mastoidectomy
21	Post-auricular fistula	Cortical mastoidectomy
22	Facial nerve palsy	Cortical mastoidectomy
5A	Post-auricular abscess	Cortical mastoidectomy
8	Post-auricular fistula. Cholesteatoma.	Radical mastoidectomy
19	Post-auricular abscess	Radical mastoidectomy

Specific anti-tuberculous treatment was commenced as soon as the diagnosis was confirmed.

(c) Results of Treatment

I. The group of patients with known tuberculosis (Table IV) all improved after their surgery, specific anti-tuberculous therapy and supportive measures.

II. The group of patients who underwent a surgical procedure without previously having been diagnosed as having tuberculosis (Table V) responded as follows:

(a) Those patients where the diagnosis of tuberculous otitis media was confirmed on examination under anaesthesia and biopsy or cervical lymph node biopsy recovered uneventfully on specific anti-tuberculous treatment and local ear toilette, except patient No.9. After confirmation of the diagnosis this patient was referred to a local tuberculosis treatment centre for therapy. He returned 12 days later with a post-auricular abscess and acute mastoiditis. A cortical mastoidectomy was performed.

(b) The patients who underwent cortical and radical mastoidectomies to secure a histological diagnosis and to clear stubborn foci of disease, responded well to specific anti-tuberculous therapy.

5. DISCUSSION:

The typical features of tuberculosis otitis media, namely painless otorrhoea with multiple perforations coalescing into a subtotal perforation, pale granulations in the middle ear cleft mucosa, early severe conductive or mixed hearing loss, bone necrosis and sequestration may be altered by a superadded infection such as Proteus, Streptococcus, Klebsiella and Pseudomonas. The peforations seen were usually central or subtotal. Small multiple perforations were not seen in our series, probably because this represents an early transient phase in the disease process which is altered by coalescence into large, central, subtotal or total perforations.

The incidence of facial nerve palsy in our series was six out of thirty ears (20%). This is in keeping with four out of twenty two ears (18%) in the series of Windle-Taylor and Bailey (1980). This is a much higher incidence of facial nerve palsy than is found in non-tuberculosis otitis media. An ipsilateral facial nerve palsy in a child with a suppurative otitis media should alert the paediatrician or otolaryngologist to the high probability of tuberculosis being the cause.

A past or present history of tuberculosis in a patient is suggestive that the ear problem may be tuberculosis as well. A positive family history or strong positive neighbourly contact increases the suspicion of tuberculosis of the middle ear in the case of chronic middle ear suppuration. In our series, eleven out of twenty five patients had a positive past or family history of tuberculosis. In the series of Windle-Taylor and Bailey (1980) six patients out of twenty two patients were in this category.

A negative chest X-ray for pulmonary tuberculosis does not exclude the diagnosis of aural tuberculosis. In our series, twelve out of twenty five patients did not have pulmonary tuberculosis. This compares well with the 50% negative chest X-rays in the series of Windle-Taylor and Bailey (1980).

The histology of biopsies establishes the diagnosis when acid-fast Bacilli are identified in histological sections. Failing to demonstrate acid-fast Bacilli, the diagnosis is at best highly suggestive and further confirmation is desirable. Positive Kirschner culture of pus or tissue taken during surgical exploration unequivocally establishes the diagnosis. Since several weeks of incubation is necessary in order to secure positive cultures for confirmation of the diagnosis, it is not seen as a quick diagnostic tool, and delay of treatment whilst awaiting the results is not desirable. Since members of the Otolaryngology Department at Groote Schuur Hospital are alert to the possibility of tuberculosis in the ear, nose and throat systems, the rate of diagnosis without major operations (usually examination under anaesthesia and biopsy) is high. This we regard as being of great importance, otherwise extensive surgery may be undertaken in order to eradicate all the diseased parts of the temporal bone, which is definitely unnecessary and unduly destructive in nature.

I believe that in the middle ear cleft afflicted with tuberculosis the most limited surgical procedure is needed because even a very progressive, destructive lesion of the temporal bone may be reversed by healing with specific anti-tuberculosis therapy. Failing to recognise this fact, there is a great temptation to be radical because of the extensive destruction found during exploration and to convert such an ear to an extensive radical mastoidectomy.

It is the superimposed acute infection with Proteus, Streptococci, Klebsiella or Pseudomonas which aggravates, or complicates the clinical picture, causing the patient to seek medical treatment: for by itself tuberculosis otitis media causes little pain or constitutional upset. One should then be alert to the clinical presentation of pale granulations, bone necrosis and sequestration and the severity of deafness out of proportion to the apparent degree of pathology as seen on otoscopy (Shambaugh, 1969). Once superinfection has taken place, the usual inoffensive discharge becomes foul in character simulating attico-antral disease. Here the radiological examination of the mastoids usually show a well developed mastoid air cell system with a cloudy appearance rather than the sclerotic contracted mastoid, typically seen with chronic attico-antral disease.

Although an excellent result was achieved (complete air-bone gap closure) with the tympanoplasty performed in the active phase of tuberculous involvement of the middle ear (Patient No. 25) and this appears to concur with the good results described by Munzel (1978), reconstructive surgery in the active phase of the disease appears to be unduly hasty. It would seem more reasonable to delay reconstruction of the middle ear until anti-tuberculosis treatment has been completed and the end-result of the disease in the ear may be more accurately assessed and managed accordingly.

IV. CONCLUSIONS:

With the increase of Pulmonary Tuberculosis within the geographical area served by Groote Schuur Hospital, the anticipated increase in Laryngeal Tuberculosis has been observed and these cases are numerically far greater than the aural manifestations of the same disease. A positive chest X-ray for Pulmonary Tuberculosis was more often noted with Laryngeal Tuberculosis, suggesting that the pathway of spread was via the bronchial tree rather than haematogenous.

Even in this day and age of emphasis on preventative medicine and the era of multiple effective bactericidal agents against Mycobacterium tuberculosis, we have noticed an increase in the incidence of Tuberculous Laryngitis for 1983. Since this result is in keeping with the recently publicized increased incidence of pulmonary tuberculosis in Cape Town precincts, it behoves us to develop an awareness of this possible diagnosis in the patient presenting with hoarseness. Tuberculous Laryngitis may mimic exophytic tumour-like lesions and in these cases, an index of suspicion will lead one to review the personal and family history with regard to Tuberculosis and to consider the chest X-ray as well as the sputum examination as important antecedents to biopsies. The fact that Tuberculous Laryngitis may mimic exophytic tumour-like lesion should in no way blind us to the reality that it may co-exist with exophytic carcinoma.

With the advent of effective specific anti-tuberculosis therapy, the haematogenous extensions of this disease have been markedly curtailed. Although the incidence of Aural Tuberculosis has decreased dramatically over the past fifty years, while tuberculosis still exists, even in highly developed countries such as Great Britain (Windle-Taylor and Bailey, 1980), the United States of America (Glover et al, 1981) and Germany (Munzel, 1978), it should be considered in the apparently non-responding chronic suppurative otitis media in which local ear toilette, topical and/or systemic therapy is being used. Clinical diagnosis usually comes in the wake of clinical awareness. Early diagnosis, conservative surgery and specific anti-tuberculosis therapy make this disease an eminently curable problem with limited destruction and a low complication rate.

An ipsilateral facial nerve palsy in a child with a suppurative otitis media should alert one to the high probability of tuberculosis being the cause.

In Tuberculous otitis media the surgical procedure should be conservative since even an advanced destructive lesion of the temporal bone may be reversed by healing with specific anti-tuberculosis therapy. Failure to recognise this fact leads one to the great temptation to be radical because of the extensive destruction found during exploration and to convert such an ear to an extensive radical mastoidectomy cavity. The surgery required for Tuberculous Laryngitis is usually confined to a biopsy as part of the confirmation of diagnosis. The histology of the biopsy establishes the diagnosis when acid fast Bacilli are identified in histological sections. Failing to demonstrate acid-fast Bacilli, the diagnosis is at best highly suggestive and further confirmation is desirable.

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