OTOMYCOSIS

By H. K. ISMAIL (Ismailia)

Introduction

IT is over a 100 years now that fungus infection of the ear was first described (Andral and Cavarret, 1843; Mayer, 1844).

The disease is known to occur in certain parts of the world; its high incidence in the Suez Canal Zone gave me the chance of carrying on this study.

Incidence

Our survey covers the three cities in the canal zone, namely Port-Said, Suez and Ismailia. During the last 2 years 430 cases of external otitis were seen, among them 176 cases proved to be otomycosis (40 per cent.).

Sex Ratio

Males were more affected, 108 cases, while females were 68; i.e. the ratio is 1·5 : 1.

Age

The highest figures in both sexes were between 25-50 years.

Ear

Both ears were equally affected, the right side in 83, the left in 72, both ears in 21.

Organism

Otomycosis has long been known to be due to *Aspergilli* especially *A. niger* (Tighem, 1867) and *A. fumigatus* (Fresincus, 1856).

Wolf (1947) found *Asp.* responsible for about 90 per cent. of cases.

Gregson *et al.*, Touche (1961) state that *Candida* has been the causative organism in almost half the cases.

Syverton *et al.* (1946) state that fungi can be found in a quarter of the cases of external otitis and that in the rest it is a gram negative organism e.g. *B. pseudomonas*.

Gregson *et al.*, Touche (1961) believe that fungi are secondary invaders and that bacterial ear infections provide a degree of humidity favourable to fungal infections, antibiotic therapy may cause the bacterial infections to subside, so fungi overgrow.

In all our cases the fungus found was *A. niger*. I also do believe that otomycosis is a separate pathological and clinical entity though secondary bacterial infection may take place.

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Aetiology

I think this is best considered under two headings.

(i) *Factors that lower the resistance of the external meatus*

The external auditory canal has its own protective mechanism mainly provided by:

(*a*) The lipide content of its secretion.
(*b*) The slight acidity of its surface (Blank, 1939; Fabricant and Perlestein, 1949).

Peck *et al.* (1939) believe that fatty acids were most efficient as antifungal agents in acid media.

The protective mechanism can be interfered with by:

(1) Elevated temperature and humidity: this is supposed to cause plugging of the sweat and sebaceous glands thus diminishing the lipide flow.
(2) Absence of skin lipides due to the absence of cerumen, frequent washing of ears, swimming, etc.
(3) Trauma to the external ear canal by cotton wool applicators, etc.
(4) Chronic dermatitis of the external meatus whether primary or secondary to a discharging ear.

(ii) *Factors that favour the growth of the organism*

Fungi exist in nature as saprophytes, their normal growth requirements are:

(a) *Optimum temperature.* This differs, *Asp.* on the whole require an elevated temperature, *Asp. fumigatus*, e.g. 45-55C°.
(b) *Moisture.* This varies but on the whole they need a good percentage of moisture; low moisture content usually limits the growth of fungi.
(c) *Metallic elements.* It so luckily happened that *A. niger* has been the favourite organism for experimental studies by botanists; Raulin (1869), Steinberg (1939), and others found that the essential elements for its growth are magnesium, sulphur, iron, zinc and phosphorus in order of importance.
(d) *pH of the medium.* Most fungi grow in media with pH between five or eight (Haldane, 1930), but slightly alkaline media seem to be more favourable. Bunning (1936) stated the internal pH of the cells of *A. niger* is influenced by the pH of the medium if this is less than four, death of cells occurs.

Personal Study

It seems rather a striking phenomenon that although the disease occurs in Egypt, it is most common in the canal zone; its highest incidence is in Port Said especially during the months of July, August and September. In an attempt to explain this increased incidence—as this may help to find out the ætiological factors—I carried out the following study:

(*a*) pH of the drinking water.
(*b*) Variations in the atmospheric temperature.
(*c*) Variations in the relative humidity.

Our study included the three cities of the canal zone, Cairo and England.

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(a) *pH of the drinking water*

The answer I received from the Ministry of Housing and Local Government in London states, "The pH of the drinking water in England and Scotland is generally between 7-8. The supply to the greater London Area is usually about 7·5. In some instances pH values between 6-7 do occur but the number is relatively small. Also some supplies (particularly from moorland gathering grounds) are treated with an alkali e.g. lime to increase the pH value to nine or even higher to prevent corrosion of pipes and plumbo-solvency".

The Official reply from Cairo Municipality states, "The drinking water for the Cairo area ranges between 7-7·3 except during flood time when it reaches 7·5". The information I got from Denmark states that the average pH is 7. In the Canal Zone where the drinking water is the same for the three cities the average pH is 7·6.

I may add that the chemical examination of water for different ions favouring the growth of *A. niger* did not show any appreciable change.

(*b*) Variations in atmospheric temperature (C°)

TABLE I.

Month	Cairo	Port-Said	Suez	Ismailia	London (Kew)
January	12·3	15·3	14·3	14·3	4·4
February	13·5	13·4	11·7	12·5	4·5
March	16·3	16·0	16·3	15·6	6·3
April	20·2	18·8	20·8	20·7	9·0
May	24·2	22·6	24·2	25·4	12·3
June	26·8	25·3	29·1	27·4	14·5
July	27·0	26·7	28·0	29·4	17·7
August	27·6	30·0	27·5	30·0	17·7
September	26·3	25·3	25·6	27·3	14·5
October	22·7	22·8	22·0	23·4	10·7
November	18·7	20·8	18·8	19·9	6·8
December	14·6	17·3	15·1	15·5	5·9

(*c*) Variations in relative humidity (%)

TABLE II.

Month	Cairo	Port-Said	Suez	Ismailia	London (Kew)
January	74	76	68	74	85
February	68	75	66	65	82
March	65	73	63	60	79
April	58	73	59	60	75
May	52	73	60	54	73
June	55	75	61	47	73
July	61	77	62	52	73
August	65	76	65	49	76
September	64	73	67	56	79
October	69	72	64	53	85
November	74	73	60	50	86
December	76	76	65	47	86

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Conclusions

From these investigations I feel justified to think that there are three factors that equally contribute together in the ætiology; they are heat, humidity and alkalinity of water supply. They all act both by promoting the growth of the organism and by interfering with the resistance of the external auditory canal.

Clinical Picture

The disease is diagnosed by:

(1) A triad of symptoms; earache, itching and a sense of a blocked ear. This triad occurs only in otomycosis.
(2) The wet blotting paper like appearance in the external ear.
(3) Laboratory indentification of the organism.

N.B.—The clinical picture may be shadowed by a complication e.g. furunculosis, otitis media.

Treatment

A general review of the literature reveals so many different methods suggested for treatment, the most commonly mentioned are:

(1) After cleaning the ear a full strength metacresylacetate (cresatin) wick is inserted for 24 hours, this may be repeated. The patient is instructed to keep it moist with the following solution:

Oil of cloves	0·2 cc.
Cresatin	8·0 cc.
Olive oil ad	15·0 cc.

this treatment is repeated for 3-4 days (Gill, E.K., 1939).
(2) The bactericidal and fungicidal effect of cresatin is highly augmented by the addition of thymol 1 per cent., but this may produce a burning sensation and hypersensitivity.
(3) Thymol 2 per cent. in 70 per cent. alcohol (McBurney and Searcy, 1936).
(4) Oral administration of potassium iodide beginning with 5 grains three times daily, increasing the dose gradually to 15 grains or more three times daily, this is continued till the ear is clear (Chisolm and Sutton, 1925).
(5) Silver picrate trinitrophenolate, locally used was recommended by Dart (1940).
(6) A powder containing iodine, thymol, tricresol 1 per cent. each in boric acid base for local insufflation in the ear was used by Gill, W. D. (1938).
(7) A powder containing sulfanilamide, sulfathiazol, zinc peroxide in the ratio of 4 : 1 : 1 sprayed over the canal gives high protection against exacerbrations (Senturia and Wolf, 1945).
(8) Salicylic acid in alcohol, this will be considered later.
(9) Gentian violet 1 per cent. solution is used because of its antifungal and antiseptic properties.
(10) Formaldehyde was the first organic fungicide used, its effect being due to its reaction with the aminogroups of the organism.

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Mycostatin

This new drug is produced by *Strep. noursei;* it has been found a fungistatic in vitro against most principal fungi in a concentration of 2·0 units per cc. Most of the unabsorbed mycostatin is passed unchanged in stools Gregson et la Touche (1961) recommended its local insufflation in the ear as a powder in boric acid base, the dose of mycostatin is 100,000 units per gram. They tried it in 83 cases, seven had relapses or prolonged treatment nine cannot be followed and the rest were cured. They gave it the following advantages:

(*a*) A satisfactory result (about 81 per cent.).
(*b*) A painless method.
(*c*) No coloration of the external meatus.

Personal Work

We could not try all these drugs, so we used some and we tried some others.

(1) *Antibiotics and Chemotherapeutics*

Hartlives (1946) noticed that sulfonamides inhibit the growth of *A. niger* but the fungus overcomes this inhibition as the time of incubation is prolonged. This may be because *A. niger* can synthesize para-aminobenzoic acid or some other compounds which reverses the inhibitory action of sulfa.

We tried the effect of all known antibiotics and sulfa drugs in 30 cases. The organism was found completely resistant to all except Gantrisin (Roche) and Badional (Bayer); both are sulfa derivatives. Their effect *in vitro* lasted for 2-3 days. Their clinical trial seem quite valueless.

(2) *Mycostatin*

It has definite antifungal effects both clincally and *in vitro*. It was tried locally in 12 patients, and both locally and systemically in another 14 cases. Its additional systemic use seemed valueless. Cure occurred in 19 cases (about 72 per cent.), yet I believe it is not the ideal treatment because:

(*a*) It is rather an expensive drug.
(*b*) The patient has to attend daily for treatment, for 2-3 weeks.
(*c*) The treatment course is prolonged.

(3) *Cortisone*

It has no effect whether used locally, systemically or both.

(4) *Salicylic Acid*

As it is poorly soluble in water, it is used as 2 per cent. solution in alcohol. Its pharmacological actions are:

(*a*) It is a strong keratolytic agent. It causes hyperkeratinization of the horny layer of the epidermis without much inflammatory reaction to the skin and mucosa.
(*b*) It is both antiseptic and Fungicidal.
(*c*) It aids regeneration of the epithelium.

This drug was tried in 37 patients, cure occurred in 25 cases (about 70 per cent.).

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(5) pH

Bunning's statement about the influence of the pH of the medium on the cells of the fungus (mentioned before), plus the fact that the pH of the external meatus in cases of otomycosis (as tested by litmus paper) is very alkaline; made me feel that the action of salicylic acid can also be due to its acidity, the 2 per cent. solution has a pH of 2·7.

I therefore thought of using stronger salicylic acid solutions; a 3 per cent. and 4 per cent. solutions; the former has a pH of 2·3 while that of the latter is 1·9 per cent. I started to try them *in vitro*; they appeared to have a greater effect; with the 4 per cent. solutions no growth of the organism took place at all even in recurrent and obstinate cases. I then tried it on patients, but fearing the occurence of a chemical external otitis we treated these patients in hospital; luckily this complication never happened.

So our present line of treatment is:—

The patient's ear is syringed with warm saline solution or water, an aluminium acetate wick is put in if there is an associated external otitis. The patient is given salicylic acid 4 per cent. in alcohol as drops three times daily; after 3-4 days he may feel his ear blocked but no itching or pain. The ear is then syringed, and a mass of dead colonies and desquamated epithelium comes out. The drops are then used for few more days. Complete cure is recorded when the external meatus is fully normal.

This method has been used till now in 43 cases with no single failure, nor any complication apart from very slight burning sensation in susceptible individuals.

Therefore I do think this is the best line of treatment for otomycosis, I prefer it to mycostatin because:

(*a*) Better results, no single failure so far.
(*b*) It is a simple method that can be easily carried out by the patient himself.
(*c*) A shorter course of treatment, maximum is one week.
(*d*) A considerably cheaper drug.

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To the Editor

4th August, 1962

Dear Sir,

In their paper entitled 'Metabolic Studies in a Child with Ménière's Disease' published in your June 1962 issue, Clayton, Birch and Hughes make a reference to our work (1958) which is not altogether in harmony with our views, and we should be glad if you would permit us to restate them.

The general conclusion which we drew from the data presented in the Proceedings of the Royal Society of Medicine (1958), was that a chronic total cationic deficiency could be postulated in Ménière's disease and since the body mechanisms of homeostasis would thus be involved there would also be a part played by the adrenocortical hormones. In a companion paper (1958) we assembled data concerning amongst other matters the sodium/potassium ratio in the endolymph from which it seemed reasonable to deduce that aldosterone has a local effect within the membranous labyrinth. These postulates do not uphold the view (attributed to us) that disturbances of salt and water balance acting as they must through the homeostatic mechanisms have no bearing on the ætiology of Ménière's disease.

The results obtained by Barbara Clayton *et al.*, could also point to adrenocortical over-activity, in terms of cortisone-like secretion, in the days preceding an attack, but in view of the serum electrolyte findings of Talbott and Brown (1940) this over-activity would not necessarily include aldosterone secretion which has a separate control system. Indeed, the results presented by Talbott and Brown, indicate a reduction in aldosterone secretion during an attack. Our own more recent findings (Naftalin and Harrison, 1961 a and b) lead us to suggest that in fact, in the period immediately preceding an attack aldosterone secretion has fallen off, and ADH is active.

The complex of relations of total body content of the various cations, of their serum concentrations, and of the hormonal status influencing their transport across various membranes (renal, intestinal and endolymphatic) seems to us to be at least part of the background of Ménière's disease and since dietary intake of sodium and potassium influences the homeostatic mechanisms it seems likely that water and salt disturbances do affect the course of Ménière's disease.

Yours sincerely,

L. Naftalin
M. Spencer Harrison

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To the Editor

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